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Original article

The Effect of Vitamin D3 Status Correction Through Supplementation on Glycemic Control and Hyperlipidemia in Type 2 Diabetes

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Abstract

Background and Objectives: Vitamin D deficiency is common in patients with type 2 diabetes mellitus (T2DM) and may exacerbate poor glycemic control and dyslipidemia. This study aimed to evaluate the efficacy of high-dose vitamin D3 supplementation in improving glycemic control and lipid parameters in vitamin D-deficient adults with T2DM.
Methods: A double-blind, randomized, placebo-controlled clinical trial was conducted at the Diabetic and Endocrine Center in Sulaimaniyah. A total of 180 adults (aged 40–70 years) with T2DM (HbA1c 7–10%) and vitamin D deficiency (<20 ng/mL) were randomized to receive either vitamin D3 5000 IU/day (n=91) or placebo (n=89) for three months, followed by one month of follow-up. Primary outcome was change in HbA1c; secondary outcomes were fasting blood glucose (FBG), lipid profile, and serum 25(OH)D.
Results: After intervention, the active D3 group showed significant reductions in HbA1c (7.95 ± 0.67% to 7.68 ± 0.69%, p=0.001), FBG (165.64 ± 18.7 to 161.41 ± 19.6 mg/dL, p=0.001), triglycerides (184.82 ± 28.1 to 178.65 ± 27.6 mg/dL, p=0.001), cholesterol (184.15 ± 22.2 to 179.54 ± 20.5 mg/dL, p=0.001), and LDL (113.69 ± 14.7 to 109.59 ± 13.9 mg/dL, p=0.001). HDL increased significantly (35.27 ± 3.2 to 40.07 ± 2.9 mg/dL, p=0.001). Serum 25(OH)D rose from 12.9 ± 2.9 to 34.1 ± 5.3 ng/mL (p=0.001). In contrast, placebo participants demonstrated deterioration in glycemic and lipid markers (all p≤0.001).
Conclusion: High-dose vitamin D3 (5000 IU/day) significantly improved glycemic control, lipid parameters, and serum vitamin D3 status in deficient adults with T2DM, supporting its role as an effective adjunctive therapy.

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1. Introduction:

Diabetes mellitus (DM) is a chronic, multifactorial metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both (1). Among its subtypes, type 2 diabetes mellitus (T2DM) comprises approximately 90% of all global cases, driven largely by insulin resistance (IR) and progressive pancreatic β-cell dysfunction (2). The global prevalence of T2DM has risen dramatically, reaching 462 million cases in 2017, and is projected to increase to 11.3% of the world population by 2030 (3, 4). In Iraq, the age-adjusted prevalence is particularly high at 19.7%, highlighting the urgent public health burden (5).

The fundamental pathogenesis of T2DM

involves a combination of β-cell failure and IR across major metabolic tissues. β-cell inadequacy arises from glucolipotoxicity, oxidative stress, and mitochondrial dysfunction, which impair insulin production and trigger dedifferentiation mechanisms (6, 7). Concurrently, IR in skeletal muscle, liver, and adipose tissue results from disrupted insulin receptor signaling, chronic inflammation, and lipid accumulation, thereby intensifying systemic hyperglycemia (8). Obesity-related adipose dysfunction exacerbates IR through adipokine dysregulation and systemic low-grade inflammation (9). This dual pathology underpins the complex metabolic derangements in T2DM, explaining its strong association with comorbidities including diabetic kidney disease, retinopathy, neuropathy, cardiovascular disease, and steatotic liver disease (10, 11).

Vitamin D3, classically known for regulating calcium and bone homeostasis, has recently emerged as a candidate with broader metabolic effects. Its active metabolite, calcitriol, acts through vitamin D receptors (VDR) on pancreatic β-cells to enhance insulin synthesis and secretion while also improving insulin sensitivity in peripheral tissues by modulating inflammatory and adipokine pathways (12). Observational studies consistently link vitamin D deficiency (25(OH)D <30 ng/mL) with increased risk of T2DM and cardiovascular disease (13). Notably, global estimates suggest deficiency rates exceeding 75% in some populations, making it a critical modifiable risk factor (13). Findings from clinical studies remain heterogeneous. For instance, Banzal and Desai (2020) demonstrated significant HbA1c and fasting glucose reductions after high-dose vitamin D3 supplementation in vitamin D-deficient T2DM patients (14). Conversely, Jamka et al. (2015) concluded that supplementation in overweight individuals, despite elevating serum 25(OH)D, did not improve glucose or insulin metabolism (15). Such discrepancies likely reflect differences in baseline vitamin D status, intervention dose, study duration, and patient characteristics.

This study is novel in its simultaneous evaluation of vitamin D3 supplementation on both glycemic control and lipid profiles in vitamin D-deficient adults with type 2 diabetes. The necessity arises from persistently high diabetes prevalence and the limited efficacy of current therapies in addressing both hyperglycemia and dyslipidemia. Prior trials have produced inconsistent results due to heterogeneous populations, variable dosing regimens, and failure to control confounders. Therefore, the main aim was to rigorously determine whether high-dose daily vitamin D3 (5000 IU) can serve as an effective adjunct therapy in improving metabolic outcomes in this population.

2. Methods

2.1 Study design and setting

A double-blind, randomized, placebo-controlled clinical trial was conducted to evaluate the effect of vitamin D3 supplementation on glycemic control and lipid profiles in patients with T2DM. The study was carried out at the Diabetic and Endocrine Center in Sulaimanyh, a specialized tertiary healthcare facility for metabolic disorders. The overall duration extended four months: a three-month intervention period followed by an additional one-month follow-up for outcome assessments. The trial was reported in accordance with CONSORT guidelines.

2.2. Participants

Eligible participants were identified through the center's electronic health records during routine clinic visits. Adults aged 40–70 years, previously diagnosed with T2DM for at least one year, and documented vitamin D3 deficiency (serum 25(OH)D levels <20 ng/mL) were considered for inclusion. Recruitment occurred consecutively, and

participants who met eligibility requirements were randomly allocated to intervention or placebo groups using a computer-generated allocation sequence in a 1:1 ratio.

Participants were included based on the following criteria: (1) age 40–70 years; (2) confirmed diagnosis of T2DM for ≥1 year; (3) HbA1c between 7–10% at baseline; (4) serum vitamin D3 <20 ng/mL; (5) no use of vitamin D or lipid-lowering supplements; and (6) ability to provide informed consent.

Exclusion criteria comprised: (1) type 1 diabetes mellitus; (2) significant renal impairment (eGFR <60 mL/min/1.73m²); (3) liver disease or malabsorption syndromes; (4) presence of hypercalcemia; (5) pregnancy or lactation; (6) ongoing use of medications influencing vitamin D metabolism (e.g., glucocorticoids); and (7) unusually high sun exposure or excessive physical activity, which could significantly alter vitamin D status independently of supplementation.

Sample size estimation was performed using G*Power software (version 3.1.9.7). Assuming a medium effect size (Cohen's d = 0.5), α = 0.05, power = 0.80, and a two-tailed test, a minimum of 102 participants (51 per group) was required to detect differences in HbA1c reduction. This was inflated to 122 to account for possible attrition. However, to improve external validity and mirror comparable interventional studies, 180 participants were ultimately enrolled.

2.3 Data Collection

At baseline (Month 0), participants completed a standardized questionnaire capturing demographics, socioeconomic indicators, occupation, residence, and comorbid conditions such as hypertension or thyroid disorders. Fasting venous blood samples were collected following an 8–10 hour fast and analyzed for glycated hemoglobin (HbA1c), fasting blood glucose (FBG), serum lipids [low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), and total cholesterol], and serum 25(OH)D levels. Assays were performed using high-performance liquid chromatography for HbA1c, enzymatic colorimetric methods for lipid parameters, and chemiluminescent immunoassay for vitamin D3. Laboratory personnel remained blinded to treatment assignments.

During the active intervention phase (Months 1–3), the treatment group received vitamin D3 supplementation at a standardized dose, while the control group received a placebo identical in appearance. Adherence was assessed via monthly pill counts alongside structured interviews that inquired about missed doses, tolerance, and side effects (e.g., gastrointestinal disturbances, potential hypercalcemia). At the midpoint (Month 2–3), blood sampling was performed to monitor interim glycemic and lipid indices, concomitantly allowing evaluation of treatment adherence and safety.

Post-intervention data were obtained at Month 4 and mirrored baseline measures to enable within- and between-group comparisons. Special emphasis was placed on categorizing outcome variables into clinically relevant ranges such as LDL ≤100 vs. >100 mg/dL and HbA1c 6.5–8% vs. >8%.

2.4 Ethical Approval

This study obtained ethical consideration from the ethical committee of the College of Medicine, University of Sulaimani (No: 380, dated 17/11/2024)

2.5 Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY). Distribution normality was evaluated using the Shapiro–Wilk test. Paired t-tests compared pre- and post-intervention results within groups, while independent t-tests and one-way ANOVA were used for between-group differences. Multivariate linear regression was applied to account for potential confounding factors, including age, gender, and baseline vitamin D3 levels. A p-value of <0.05 was considered statistically significant.

3. Results

Table 1 shows demographic information for two groups of diabetic patients, including the placebo and active D3 groups. The mean age with standard deviation was 51.87 ± 7.573 years in the placebo group and 52.47 ± 8.266 years in the active D3 group. In the placebo group, there were 50 (56.2%) women and 39 (43.8%) men, while the active D3 group had 42 (46.2%) women and 49 (53.8%) men. Employment status in the placebo group showed 24 (27%) individuals as employers, 33 (37.1%) as housewives, and in the active D3 group, 29 (31.9%) as housewives.

73 (80.2%) in the active D3 group and 64 (71.9%) individuals in the placebo group reported no prior medical history. Hypertension was the most common previous condition, affecting 15 (16.9%) patients in the placebo group and 14 (15.4%) patients in the active D3 group. The duration of diabetes in the placebo group included 24 (27%) individuals with 1-2 years, 31 (34.8%) individuals with 3-4 years, 14 (15.7%) individuals with 5-6 years, and 20 (22.5%) individuals with more than 6 years. In the active D3 group, diabetes duration comprised 18 (19.8%) individuals with 1-2 years, 35 (38.5%) individuals with 3-4 years, 19 (20.9%) individuals with 5-6 years, and 19 (20.9%) individuals with over 6 years.

Table 1. Demographic Characteristics in Two Groups (Placebo and Active D3).

Variable		Group		P-value*
		Placebo (n=89)	Active D3 (n=91)	
Age (year)		51.87 ± 7.573	52.47 ± 8.266	0.608
Sex	Female	50 (56.2%)	42 (46.2%)	0.179
	Male	39 (43.8%)	49 (53.8%)	
Occupation	Employer	24 (27%)	13 (14.3%)	0.128
	House wife	33 (37.1%)	29 (31.9%)	
	Retired	8 (9%)	12 (13.2%)	
	Teacher	10 (11.2%)	14 (15.4%)	
	Worker	14 (15.7%)	23 (25.3%)	
Address	Inside town	45 (50.6%)	44 (48.4%)	0.762
	Peripheries	44 (49.4%)	47 (51.6%)	
Other PMH	None	64 (71.9%)	73 (80.2%)	0.354
	HTN	15 (16.9%)	14 (15.4%)	
	HTN + HF	3 (3.4%)	3 (3.3%)	
	HTN + Hypothyroid	3 (3.4%)	0	
	Hypothyroid	2 (2.2%)	0	
	Migraine	2 (2.2%)	1 (1.1%)	
Duration of diabetes	1-2 year	24 (27%)	18 (19.8%)	0.606
	3-4 year	31 (34.8%)	35 (38.5%)	
	5-6 year	14 (15.7%)	19 (20.9%)	
	> 6 years	20 (22.5%)	19 (20.9%)	
Economic status	Low income	59 (66.3%)	60 (65.9%)	N/S
	Moderate	30 (33.7%)	31 (34.1%)	

*P-value based on chi-square and Fisher's exact test

Annual vitamin D3 checkups in the placebo group were performed once for 46 (51.7%) individuals, while 43 (48.3%) individuals had not undergone testing. In the active D3 group,

47 (51.6%) individuals had annual testing once, while 44 (48.4%) individuals had not been tested. The frequency of annual HbA1c monitoring showed that 49 (55.1%) individuals in the placebo group were tested every 3 months, and 40 (44.9%) individuals were tested between 3-6 months. In the active D3 group, 49 (53.8%) individuals were monitored every 3 months, and 42 (46.2%) individuals were tested between 3-6 months. Annual nephrology checkups in the placebo group occurred more than twice for 49 (55.1%) individuals and 1-2 times for 40 (44.9%) individuals. In the active D3 group, 49 (53.8%) individuals had more than two annual visits, while 42 (46.2%) individuals had 1-2 visits (Table 2).

Table 2. D3, HbA1C and [inter_nephro] check in two groups (Placebo and Active D3).

Variable		Group		P-value*
		Placebo (n=89)	Active D3 (n=91)	
D3 check up/year (frequency)	Once	46 (51.7%)	47 (51.6%)	N/S
	Zero	43 (48.3%)	44 (48.4%)	
HbA1C (frequency)	3 months	49 (55.1%)	49 (53.8%)	0.871
	3-6 months	40 (44.9%)	42 (46.2%)	
Checkup/year [inter_nephro]	> 2	49 (55.1%)	49 (53.8%)	0.871
	1-2 times	40 (44.9%)	42 (46.2%)	

*P-value based on chi-square and Fisher's exact test

Table 3 shows blood test values before and after intervention, divided into placebo and active D3 groups. In the placebo group, HbA1c, fasting blood glucose, serum triglycerides, serum cholesterol, and serum LDL levels all increased significantly after intervention compared to baseline, with all increases reaching statistical significance ($P \leq 0.001$). Conversely, vitamin D3 and serum HDL levels significantly decreased after intervention compared to baseline, with these reductions also reaching statistical significance ($P \leq 0.001$).

In the active D3 group, HbA1c, fasting blood glucose, serum triglycerides, serum cholesterol, and serum LDL levels showed significant decreases after the intervention compared to baseline measurements, with all reductions reaching statistical significance ($P \leq 0.001$). Conversely, vitamin D3 and serum HDL levels demonstrated significant increases after the intervention compared to baseline, with these improvements also achieving statistical significance ($P \leq 0.001$).

Table 3. Blood test results pre and post-intervention in the placebo and Active D3 groups.

Blood test	Placebo group		P-value*
	Pre-intervention	Post intervention	
HBA1c (%)	7.911 ± 0.710	8.200 ± 0.9907	0.001
FBG (mg/dL)	171.33 ± 18.746	176.88 ± 18.609	0.001
S.TG (mg/dL)	182.88 ± 25.401	187.74 ± 25.260	0.001
S. cholesterol (mg/dL)	176.69 ± 14.839	181.45 ± 14.191	0.001
Vit D3 (ng/mL)	14.215 ± 3.001	13.215 ± 3.145	0.001
S.HDL (mg/dL)	37.55 ± 2.474	34.60 ± 2.349	0.001
S.LDL (mg/dL)	112.83 ± 13.188	116.00 ± 109.59	0.001
	Active D3 group		
	Pre-intervention	Post intervention	
HBA1c (%)	7.951 ± 0.673	7.681 ± 0.691	0.001

FBG (mg/dL)	165.64 ± 18.746	161.41 ± 19.646	0.001
S.TG (mg/dL)	184.82 ± 28.085	178.65 ± 27.558	0.001
S. cholesterol (mg/dL)	184.15 ± 22.233	179.54 ± 20.517	0.001
Vit D3 (ng/mL)	12.893 ± 2.897	34.142 ± 5.273	0.001
S.HDL (mg/dL)	35.27 ± 3.218	40.07 ± 2.913	0.001
S.LDL (mg/dL)	113.69 ± 14.689	109.59 ± 13.979	0.001

*P-value based on Paired t-test

HbA1c levels before intervention showed that 58 (65.2%) individuals in the placebo group had levels between 6.5-8%, while 31 (34.8%) individuals had levels above 8%. After the intervention, 43 (48.3%) individuals fell within the 6.5-8% range, and 46 (51.7%) individuals remained above 8%. In the active D3 group, 54 (59.3%) individuals were between 6.5-8% before intervention, and 37 (40.7%) individuals were above 8%. After the intervention, 59 (64.8%) individuals were within the 6.5-8% range, while 32 (35.2%) individuals remained above 8%.

LDL levels before intervention showed that 19 (21.3%) individuals in the placebo group had levels at or below 100 mg/dL, while 70 (78.7%) individuals had levels above 100 mg/dL. After intervention, 16 (18%) individuals were at or below 100 mg/dL, and 73 (82%) individuals remained above 100 mg/dL. In the active D3 group, 26 (28.6%) individuals were at or below 100 mg/dL before intervention, and 65 (71.4%) individuals had levels above 100 mg/dL. Following the intervention, 30 (33%) individuals were at or below 100 mg/dL, with 61 (67%) individuals remaining above 100 mg/dL.

HDL levels before and after intervention in all women from both placebo and active D3 groups remained at or below 50 mg/dL. In the placebo group, men before intervention, 33 individuals (84.7%), had levels at or below 40 mg/dL, while six (15.3%) individuals exceeded 40 mg/dL. After the intervention, all men remained at or below 40 mg/dL. In the active D3 group, men before intervention, 46 (93.9%) individuals, had levels at or below 40 mg/dL, while three (6.1%) individuals exceeded 40 mg/dL. After the intervention, 31 (63.3%) individuals remained at or below 40 mg/dL, while 18 (36.7%) individuals exceeded 40 mg/dL. Additional information is presented in Table 4.

Table 4. Individual blood test levels before and after intervention in the placebo and Active D3 groups.

Variable		Intervention			
		Pre-intervention		Post intervention	
		Placebo	Active D3	Placebo	Active D3
HBA1c (%)	6.5 - 8	58 (65.2%)	54 (59.3%)	43 (48.3%)	59 (64.8%)
	> 8	31 (34.8%)	37 (40.7%)	46 (51.7%)	32 (35.2%)
FBG (mg/dL)	80-130	0	0	0	1 (1.1%)
	> 130	89 (100%)	91 (100%)	89 (100%)	90 (98.9%)
S.TG (mg/dL)	≤ 150	0	3 (3.3%)	1 (1.1%)	7 (7.7%)
	> 150	89 (100%)	88 (96.7%)	88 (98.9%)	84 (92.3%)
S. cholesterol (mg/dL)	≤ 200	82 (92.1%)	75 (82.4%)	82 (92.1%)	76 (83.5%)
	> 200	7 (7.9%)	16 (17.6%)	7 (7.9%)	15 (16.5%)
S.LDL (mg/dL)	≤ 100	19 (21.3%)	26 (28.6%)	16 (18%)	30 (33%)
	> 100	70 (78.7%)	65 (71.4%)	73 (82%)	61 (67%)
Female					
HDL (mg/dL)	≤ 50	50 (100%)	42 (100%)	50 (100%)	42 (100%)

	> 50	0	0	0	0
Male					
HDL (mg/dL)	≤ 40	33 (84.7%)	46 (93.9%)	39 (100%)	31 (63.3%)
	> 40	6 (15.3%)	3 (6.1%)	0	18 (36.7%)

Before intervention, all 89 individuals (100%) in the placebo group and all 91 (100%) individuals in the active D3 group had vitamin D3 (25(OH)D) levels below 20 ng/mL, indicating deficiency. After intervention, 87 (97.8%) individuals in the placebo group remained below 20 ng/mL or deficient, while two (2.2%) individuals had levels between 20-29 ng/mL, indicating insufficiency. In the active D3 group, vitamin D3 levels improved, with 10 (11%) individuals showing 20-29 ng/mL or insufficient levels, 80 (87.9%) individuals reaching 30-50 ng/mL or sufficient levels, and one (1.1%) individual reaching 50-100 ng/mL or high levels (Table 5).

Table 5. Individual Vitamin D3 (25(OH)D) levels before and after intervention in the placebo and Active D3 groups.

Variable		Intervention			
		Pre-intervention		Post intervention	
		Placebo	Active D3	Placebo	Active D3
Vitamin D3 (25(OH)D) ng/mL	< 20 Deficient	89 (100%)	91 (100%)	87 (97.8%)	0
	20-29 Insufficient	-	-	2 (2.2%)	10 (11%)
	30-50 Sufficient	-	-	-	80 (87.9%)
	50-100 High	-	-	-	1 (1.1%)
	> 100 Potential Toxicity Risk	-	-	-	-

4. Discussion

The present study was conducted to investigate the specific effect of vitamin D3 supplementation on glycemic control and lipid profile in patients with T2DM who were vitamin D-deficient. The findings provided a clear representation of the efficacy of this intervention among the participants. In the Active D3 group who received vitamin D3 supplementation, the mean serum level of this vitamin significantly increased from 12.89±2.897 ng/mL at baseline to 34.142±5.273 ng/mL after the intervention. This change indicated a successful correction of the deficiency to a sufficient status. In sharp contrast, in the placebo group, serum vitamin D levels significantly decreased from 14.215±3.001 ng/mL to 13.215±3.145 ng/mL. Notable improvements in key parameters of glycemic control accompanied correction of vitamin D deficiency. In the Active D3 group, mean glycated hemoglobin (HbA1c) decreased from 7.951±0.673% mg/dL to 7.681±0.691% mg/dL, while fasting blood glucose (FBG) decreased from 165.64±18.746 mg/dL to 161.41±19.646 mg/dL. Both changes were statistically significant. Conversely, in the placebo group, mean HbA1c and FBG increased from 7.911±0.710% mg/dL and 171.33±18.746 mg/dL to 8.200±0.9907% mg/dL and 176.88±18.609 mg/dL, respectively.

The intervention also demonstrated favorable effects on lipid profile parameters. In the Active D3 group, mean serum triglycerides (S.TG) decreased from 184.82±28.085 mg/dL to 178.65±27.558 mg/dL, LDL decreased from 113.69±14.689 mg/dL to 109.59±13.979 mg/dL, and HDL increased from 35.27±3.218 mg/dL to 40.07±2.913 mg/dL. All of these

changes were statistically significant. These results suggest that vitamin D correction not only improves glycemic control but also influences lipid metabolism.

The improvements in HbA1c and FBG observed in this research match findings from earlier studies. Unlike earlier research that often gave mixed results due to methodological limitations or differences in studied populations, the current evidence supports the growing consensus in the literature. The reductions in HbA1c and FBG in the Active D3 group align with results from several meta-analyses. For example, Farahmand et al. assessed the impact of vitamin D supplementation on glycemic control in T2D patients. They showed benefits in lowering FPG, HbA1c, and HOMA-IR, particularly when higher doses were given for a shorter period (16). Similarly, Afraie et al. reported that supplementing with 50,000 IU of vitamin D significantly lowered HbA1c, FBG, LDL, and systolic blood pressure while increasing serum vitamin D levels. These findings also highlight the potential role of vitamin D in reducing cardiovascular disease (CVD) risk in T2D patients (17)

The current findings regarding the notable increase in serum vitamin D after supplementation align with previous research. For example, Max et al. demonstrated that high-dose vitamin D supplementation not only effectively increased serum 25(OH)D levels in T2D patients but also improved parameters such as glycemic control, blood pressure, and parathyroid hormone levels (18). Additionally, in a meta-analysis by Musazadeh et al., vitamin D supplementation was shown to have beneficial effects on several metabolic biomarkers in individuals with T2D (19).

Findings of the present study regarding improvements in the lipid profile (specifically, significant reductions in triglycerides and LDL together with an increase in HDL within the Active D3 group) are consistent with segments of the existing literature. For instance, Alqadi et al. in Saudi Arabia reported that vitamin D supplementation led not only to improved vitamin D status, but also to marked improvements in HbA1c, fasting blood glucose (FBG), all lipid parameters, as well as renal and hepatic function and hemoglobin levels (20). Similar trends were observed in investigations conducted in Iran (21), and the United States (22), where vitamin D supplementation demonstrated favorable effects on lipid metabolism.

The relationship between vitamin D3 status and glycemic control shown in the present study revealed that higher vitamin D levels in the Active D3 group coincided with better glycemic indices. All participants were deficient before intervention, while most of the Active D3 group reached sufficient levels after supplementation, which was linked to decreases in HbA1c. This finding is consistent with the results of Akhter et al. (23) and Alqahtani et al. (24)Both of whom found an inverse relationship between vitamin D levels and HbA1c. The lack of improvement, or even decrease, in HDL among females may be related to sex-specific factors like body fat distribution, as noted in previous research (25).

The study has certain limitations. The lack of strict control over participants' diet and physical activity could have affected the results. Furthermore, because participants were limited to a single geographic region, and given the high prevalence of vitamin D deficiency in the Middle East, the generalizability of the findings may be limited. Additionally, the three-month duration may not have been sufficient to evaluate long-term effects, indicating that future studies should include longer follow-up periods.

Conclusions

The current study showed that high-dose vitamin D3 supplementation notably

improved glycemic control and various lipid parameters in adults with both vitamin D deficiency and type 2 diabetes. These results emphasize the clinical importance of addressing vitamin D deficiency as part of diabetes management. Practically, adding vitamin D screening into diabetes care protocols could help reduce complications, especially in areas with a high rate of deficiency.

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