RESEARCH ARTICLE

Evaluation of the relationship between vitamin D deficiency and microalbuminuria, glycaemic control, lipid profile in patients with type 2 diabetes mellitus

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Abstract

Objective: To evaluate the relationship of vitamin D deficiency with microalbuminuria, glycaemia and lipid profile in type 2 diabetes mellitus patients.

Method: The retrospective, clinical study was conducted at Family Medicine outpatient clinics of Istanbul Bezmialem Vakıf University, Faculty of Medicine Hospital, Turkey, and comprised data between July 1 and December 31, 2019 related to adult type 2 diabetes patients. Data was classified according to vitamin D levels; <20 ng/mL in group 1, and >20 ng/mL in group 2. Microalbuminuria, glycaemia and lipid parameters were compared between the groups. Data was analysed using SPSS 28.

Results: Of the 148 patients with mean age 56.58±9.76 years, 78 (52.7%) were female and 70 (47.3%) were male. The mean vitamin D level was 22.81±17.85 ng/mL, with 76 patients in group 1 and 72 in group 2. The mean level of glycated haemoglobin was 7.57±1.66%. Total cholesterol, low-density lipoprotein cholesterol, lymphocyte and urine microalbumin/creatinine ratio as well as glycated haemoglobin levels were significantly higher in group 1 patients than those in group 2 ($p<0.05$). Vitamin D levels were significantly negatively associated with glycated haemoglobin and total cholesterol ($p<0.05$).

Conclusion: Lower vitamin D levels were associated with poor glycaemic control, lipid dysregulation, and higher microalbuminuria, suggesting potential roles in glucose metabolism, inflammation and kidney function.

Key Words: HbA1c, Microalbuminuria, Primary care, Type 2 diabetes mellitus, Vitamin D.

(JPMA 75: 1235; 2025) **DOI: https://doi.org/10.47391/JPMA.21055**

Introduction

Diabetes mellitus (DM) is a metabolic disease with a high prevalence, and 300 million individuals are affected globally. DM incidence is projected to continue rising, potentially affecting over 500 million people by 2030. In the United States, DM frequency is approximately 1 million new cases annually. The incidence of diabetes in Turkey has been determined to be 13.7% of the population.[1,2]

Deficiency of vitamin D is widely observed in the diabetic population. Previous reports show that vitamin D status is inversely proportional to hyperglycaemia and hyperlipidaemia.[3] Vitamin D supplementation was found to reduce complications associated with insulin resistance (IR) and fasting glucose in type 2 DM (T2DM) patients. Vitamin D is important in homeostasis, particularly in bone metabolism.[4,5] It has been shown that deficiency in vitamin D is linked to several pathophysiological processes, such as inflammation and endothelial dysfunction, that are prevalent in T2DM.[6]

Diabetic nephropathy (DN) is progressive, and has been shown to be associated with glucose intolerance. About 30% of T2DM patients have nephropathy, and it is linked to end-stage renal disease (ESRD). The albumin-creatinine ratio (ACR) has traditionally served as a diagnostic tool for assessing DN extent. Persistent microalbuminuria, defined as levels 30-300 mg/24 hour in urine sample, or macroalbuminuria, defined as levels >300 mg/24 hour in urine sample, are recognised as the gold standard, non-invasive markers, and predictors for the risk of DN progression to ESRD.[7,8]

Albuminuria has been shown to be associated with low vitamin D levels, and studies have indicated that vitamin D exhibits the ability to reduce the expression of renin, an enzyme implicated in blood pressure regulation and fluid homeostasis. Deficiency of vitamin D has been correlated with chronic kidney disease (CKD), suggesting a potential association between vitamin D status and renal function. Therefore, vitamin D dysregulation may be central to DN.[8]

T2DM and CKD patients often exhibit vitamin D

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Submission complete: 24-06-2024 **First Revision received:**16-09-2024
Acceptance: 04-06-2025 **Last Revision received:** 03-06-2025

deficiency, and vitamin D deficiency is linked to microvascular complications in T2DM patients.[9] The current study was planned to investigate the potential association of vitamin D with microalbuminuria, glycaemic control and lipid profile in T2DM patients.

Materials and Methods

The descriptive, retrospective cross-sectional study was conducted at Family Medicine outpatient clinics of Istanbul Bezmialem Vakıf University, Faculty of Medicine Hospital, Turkey, and comprised data between July 1 and December 31, 2019. Data was classified according to vitamin D levels; <20 ng/mL in group 1, and >20 ng/mL in group.[2] The sample was raised using simple random sampling method, and data was retrieved from the electronic medical records system after approval from the institutional ethics review committee.

The study was carried out according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) criteria.[10] The sample size was calculated with power 0.80, and type 1 error or alpha value 0.05 in line with literature.[11-13]

Those included were T2DM patients of either gender aged at least 18 years. Those excluded were patients with type 1 DM, recent infection with leucocytosis, hypo/hyperthyroidism, hypo/hyperparathyroidism, CKD, chronic rheumatological disease, chronic liver disease, congestive heart failure, malignancy, anaemia or thrombocytopenia. T2DM patients undergoing treatment with medications known to influence the measurement of vitamin D levels, thereby affecting the renin-angiotensin-aldosterone system (RAAS), such as corticosteroids, anticonvulsants, vitamin D supplements and calcium products, as well as those likely to impact complete blood count (CBC) results, including antiplatelet drugs, were also excluded. All the patients provided written informed consent at the last routine appointment.

DM diagnosis was based on the standard criteria recommended by the American Diabetes Association (ADA); fasting blood glucose (FBG) >126 mg/dL, or random blood glucose (RBG) >200 mg/dL, or glycated haemoglobin (HbA1c) >6.5%.[14]

Biochemical parameters and CBC were measured from venous blood samples, while microalbumin, creatinine and microalbumin-creatinine ratio (MCR) were assessed from midstream urine samples. The ratio was classified as normoalbuminuria <30 mg/g, microalbuminuria 30-300 mg/g, and macroalbuminuria >300 mg/g). Blood samples were collected and transferred in non-heparinised tubes for centrifugation to the laboratory. CBC parameters were measured using an automated haematology analyser (Sysmex 1800 t, USA). Biochemical parameters were measured using a chemical auto-analyser (Cobas 8000, Roche, Germany), while other chemical parameters were assessed using a chemical auto-analyser (Toshiba, Japan). Vitamin D level was measured using modular analytics (Cobas e601, Roche, Germany).

Data was analysed using SPSS 28. During the sample selection process, each individual in the population was assigned an equal probability of being included, while variance homogeneity assumption was tested with Levene test. Normality assumption was tested with Kolmogorov-Smirnov test. Missing data was not imputed using statistical methods. Instead, the analyses were conducted based on the available data (complete-case analysis). This approach was chosen because the proportion of missing data was minimal, and it was unlikely to introduce significant bias into the results. Normally distributed continuous variables were presented as mean ± standard deviation, and compared using independent student's t-test. Non-normally distributed variables were expressed as median (minimum-maximum), and compared using the Mann-Whitney U test. Spearman correlation analysis was employed to assess correlations. The relationships between categorical variables were evaluated using the Fisher-Freeman-Halton test. To evaluate the results by age, the median age was calculated, and the participants were divided into two groups based on the cut-off age of 56 years. In univariate analyses, variables with p=0.25 were included in the logistic regression model. The model's goodness-of-fit was assessed using the Hosmer-Lemeshow test, and the Nagelkerke R-squared value was calculated. P<0.05 was considered statistically significant.

Results

Of the 148 patients with mean age 56.58±9.76 years, 78 (52.7%) were female and 70 (47.3%) were male. The mean vitamin D level was 22.81±17.85 ng/mL, with 76 patients in group 1 and 72 in group 2. The mean level of HbA1c was 7.57±1.66%, and mean urine MCR was 88.31±331.56 mg/g (Table 1).

There were no statistically significant differences between groups 1 and 2 regarding age, creatinine, FBG, glomerular filtration rate (GFR), high-density lipoprotein (HDL), triglyceride (TG), white blood cell (WBC), red blood cell (RBC), platelet (PLT), haemoglobin (HGB), haematocrit (HCT), mean platelet volume (MPV), mean corpuscular volume (MCV), and neutrophil levels (p>0.05). However, total cholesterol (TC), low-density lipoprotein (LDL), lymphocyte, urine microalbumin and urine MCR as well as

Table-1: Clinical and laboratory parameters of the patients.

Parameters	Mean ± Std. Deviation	Median (Min-Max)
Vitamin D (ng/mL)	22.81 ± 17.85	19.35 (3.0 - 120.2)
Fasting blood glucose (mg/dL)	159.09 ± 68.75	142 (49 - 482)
HbA1c (%)	7.57 ± 1.66	7.2 (5.18 - 13.51)
Age (years)	56.58 ± 9.76	56 (21.0 - 83.0)
Creatinine (mg/dL)	0.92 ± 0.31	0.85 (0.64 - 3.55)
GFR (mL/min/1.73 m2)	83.89 ± 19.4	86 (12.0 - 135.0)
Urea (mg/dL)	35.71 ± 20.15	31 (16.0 - 199.0)
BUN (mg/dL)	16.67 ± 9.4	14.49 (7.48 - 92.99)
LDL Cholesterol (mg/dL)	118.09 ± 33.2	117 (13.0 - 203.0)
Total Cholesterol (mg/dL)	193.76 ± 43.81	192 (115.0 - 338.0)
HDL Cholesterol (mg/dL)	45.71 ± 9.86	44.8 (29.0 - 76.0)
Triglycerides (mg/dL)	163.88 ± 96.14	144 (45.0 - 728.0)
WBC ($x10^3$/uL)	8.19 ± 2.53	7.91 (4.45 - 24.98)
RBC (x106/uL)	4.84 ± 0.58	4.8 (3.46 - 6.31)
PLT ($x10^3$/uL)	258.53 ± 82.5	251 (79.0 - 816.0)
HGB (g/dL)	13.75 ± 1.77	13.55 (10.25 - 18.85)
HCT (%)	41.63 ± 4.81	41.22 (31.47 - 56.79)
MPV (fL)	8.41 ± 1.44	8.17 (5.95 - 13.6)
MCV (fL)	86.14 ± 5.03	86.33 (64.08 - 100.3)
Neutrophil ($x10^3$/uL)	4.6 ± 1.52	4.44 (1.88 - 11.61)
Lymphocytes ($x10^3$/uL)	2.89 ± 2.77	2.47 (1.11 - 30.33)
Urine Microalbumin (mg/L)	60.42 ± 172.29	15.3 (0.5 - 1419.3)
Urine Creatinine (mg/dL)	113.93 ± 73.22	102.81 (0.81 - 491.26)
Urine Microalbumin/Creatinine	88.31 ± 331.56	11.95 (0.8 - 2821.4)

Std: Standard, Min-Max: Minimum-maximum, HBA1c: Glycated haemoglobin, GFR: Glomerular filtration rate, BUN: Blood-urea-nitrogen, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, WBC: White blood cell, RBC: Red blood cell, HGB: Haemoglobin, HCT: Haematocrit, MPV: Mean platelet volume, MCV: Mean corpuscular volume.

Table-2: Intergroup comparison of baseline laboratory characteristics.

	Vitamin D ≤20 ng/mL (n=76)	Vitamin D >20 ng/mL (n=72)	p
Vitamin D, ng/mL	12.78 ± 3.94	33.39 ± 20.53	<0.001
	12.5 [3 - 19.9]	26 [20 - 120.20]	
Fasting blood glucose, mg/dL	168.88 ± 80.55	148.76 ± 52.17	0.188
	149 [49 - 482]	135 [71 - 323]	
HbA1c, %	7.89 ± 1.86	7.22 ± 1.33	0.027
	7.5 [5.18 - 13.51]	6.87 [5.27 - 11.47]	
Age*	55.19 ± 0.17	58.04 ± 9.07	0.076
Creatinine, mg/dL	0.83 ± 0.50	0.77 ± 0.33	0.482
	0.83 [0.64 - 3.55]	0.86 [0.65 - 1.45]	
GFR, mL/min/1.73 m2	84.50 ± 21.28	83.24 ± 17.32	0.288
	86.5 [12 - 127.42]	85.5 [36.2 - 135]	
Urea, mg/dL	35.26 ± 26.32	36.18 ± 10.38	0.001
	28.5 [17 - 199]	35 [16 - 62]	
BUN, mg/dL	16.38 ± 12.33	16.97 ± 4.77	<0.001
	13.08 [7.94 - 92.99]	16.59 [7.48 - 28.04]	
LDL cholesterol, mg/dL *	124.14 ± 33.22	111.69 ± 32.18	0.022
Total cholesterol, mg/dL*	203.78 ± 43.97	183.93 ± 41.69	0.014
HDL cholesterol, mg/dL*	46.27 ± 50.28	45.15 ± 9.46	0.522
Triglyceride, mg/dL	175.64 ± 103.23	151.62 ± 87.18	0.082
	159 [45 - 728]	136 [50 - 600]	
White blood cell, x103/uL	8.45 ± 3.11	7.90 ± 1.67	0.819
	7.68 [4.45 - 24.98]	7.94 [4.54 - 11.56]	
Red blood cell, x106/uL*	4.84 ± 0.56	4.82 ± 0.59	0.845
Platelet, x103/uL	265.80 ± 101.08	250.84 ± 56.41	0.731
	252 [99 - 816]	248.5 [79 - 387]	
Haemoglobin, g/dL*	13.75 ± 1.65	13.73 ± 1.88	0.957
Haematocrit, %*	41.67 ± 4.48	41.57 ± 5.16	0.896
Mean platelet volume, fL	8.52 ± 1.62	8.29 ± 1.21	0.456
	8.21 [5.95 - 13.6]	8.03 [6.12 - 11.96]	
Mean corpuscular volume, fL	85.99 ± 4.79	86.28 ± 5.29	0.701
	86.16 [68.93 - 98.69]	86.78 [64.08 - 100.3]	
Neutrophil count, x103/uL	4.58 ± 1.78	4.62 ± 1.19	0.161
	4.17 [1.88 - 11.61]	4.63 [2.19 - 6.98]	
Lymphocyte count, x103/uL	3.30 ± 3.76	2.45 ± 0.74	0.038
	2.57 [1.23 - 30.33]	2.33 [1.11 - 4.57]	
Urine Microalbumin, mg/L	95.38 ± 238.01	25.77 ± 39.57	0.006
	18.4 [2.7 - 1419.3]	10.5 [0.5 - 190.6]	
Urine Creatinine, mg/dL	113.63 ± 64.43	114.22 ± 81.74	0.826
	99.18 [0.98 - 303.35]	106.69 [0.81 - 491.26]	
Urine Microalbumin/ Creatinine	139.99 ± 452.61	35.15 ± 90.51	0.006
	14.95 [2.8 - 2821.40]	10.85 [0.80 - 541]	

*Variables with normal distribution are presented. For variables with a normal distribution, descriptive statistics are given as mean ± standard deviation. For non-normally distributed variables, median [min-max] values are additionally provided. A p-value < 0.05 was considered statistically significant.

HBA1c: Glycated haemoglobin, GFR: Glomerular filtration rate, BUN: Blood-urea-nitrogen, LDL: Low-density lipoprotein, HDL: High-density lipoprotein.

blood HbA1c levels were significantly higher in group 1 patients than those in group 2 (p<0.05). Also, group 1 patients had significantly lower mean urea and blood-urea-nitrogen (BUN) levels compared to group 2 patients (Table 2).

The gender distribution between groups 1 and 2 was not significantly different (p=0.758). There was no significant difference between groups 1 and 2 regarding age distribution (p=0.253).

There was a significant but weak negative correlation between vitamin D levels and both urine microalbumin (r=-0.225, p=0.006) and urine MCR (r=-0.253, p=0.002). However, no significant association was found between vitamin D levels and urine creatinine (p=0.697). No significant relationships were found between Vitamin D levels and WBC, haemoglobin (HGB), or FBG levels (p=0.330, p=0.595 and p=0.107, respectively). A weak but statistically significant negative correlation was detected between HbA1c and vitamin D levels (r=-0.169, p=0.040).

Overall, 11 (7.7%) patients had urine MCR >300 mg/g, and 25 (17.6%) had it 30-300 mg/g. In group 1, 8 (11.1%) patients had MCR >300 mg/g, and 15 (20.8%) had it 30-300 mg/g. In group 2, 3 (4.3%) patients had MCR >300

Table-3: Urine microalbumin/creatinine distribution and classification of the renal failure stage.

Category	Total (n, %)	Vitamin D <20 ng/mL (n, %)	Vitamin D >20 ng/mL (n, %)	p-value
Urine Microalbumin/Creatinine				0.155
0-30	106 (74.6%)	49 (68.1%)	57 (81.4%)	
30-300	25 (17.6%)	15 (20.8%)	10 (14.3%)	
>300	11 (7.7%)	8 (11.1%)	3 (4.3%)	
Renal failure/GFR (mL/min/1.73 m^2)				0.286
None (GFR: >90)	62 (41.9%)	35 (46.1%)	27 (37.5%)	
Stage 1 (GFR: 60-89)	72 (48.6%)	34 (44.7%)	38 (52.8%)	
Stage 2 (GFR: 30-59)	11 (7.4%)	4 (5.3%)	7 (9.7%)	
Stage 3 (GFR: 15-29)	2 (1.4%)	2 (2.6%)	0 (0%)	
Stage 4 (GFR: <15)	1 (0.7%)	1 (1.3%)	0 (0%)	

GFR: Glomerular filtration rate.

mg/g, and 10 (14.3%) had it 30-300 mg/g (p=0.155).

GFR stage distribution between the groups were not significantly different (p=0.286) (Table 3).

Logistic regression Model 1 revealed a significant negative association between HbA1c and vitamin D levels (p=0.006, odds ratio (OR)=0.680, 95% confidence interval (CI): 0.516-0.896). TC also showed a significant negative association (p=0.041, OR=0.989, 95% CI:0.978-1.000). Other variables, such as age, urea, BUN, neutrophils, lymphocytes, urine microalbumin and urine MCR, were not significantly associated with vitamin D levels (p>0.05).

In Model 2, only variables with significant associations in univariate analysis (p<0.05) were included, and the model confirmed the findings of Model 1. HbA1c remained significantly associated with vitamin D levels (p=0.008, OR=0.698, 95% CI:0.534-0.912), and TC retained its significant negative association (p=0.032, OR=0.989, 95% CI:0.979-0.999).

Discussion

The current study identified the association of vitamin D deficiency with impaired glucose metabolism, dyslipidaemia and DN in T2DM patients. Almost half of the cohort (51.35%) had vitamin D deficiency. Various studies also showed patients with diabetes had lower vitamin D levels.[9] Vitamin D deficiency happens mostly due to low dietary intake and due to less-then-sufficient sunlight exposure. Furthermore, seasons can affect vitamin D levels, especially during winter; an estimated 50-80% of the total population is affected by vitamin D deficiency or insufficiency.[15] Other causes, like malabsorption, liver or kidney disorder, and/or obesity, lead to vitamin D deficiency. Vitamin D production uses a sunlight ultraviolet B (UVB) ray, which are predominant during morning hours and summer days.[16] Despite having sunlight for two-thirds of the year in the native region, limited time spent outdoors may contribute to the widespread vitamin D deficiency observed in the current patients.

In a randomised controlled study, a correlation was observed between vitamin D deficiency and conditions such as T2DM and various metabolic disorders.[17] Similar result was reported by another study.[18] In line with these findings, the regression analysis in the current study revealed a significant negative association of vitamin D levels with HbA1c and TC, supporting the potential role of vitamin D in glycaemic control and lipid metabolism in T2DM patients.

A study comprising 14,679 patients with IR reported that vitamin D deficiency was related to increased microvascular and macrovascular complications in T2DM patients. Also, in patients with DN, there was a notable reduction in circulating vitamin D levels compared to those without DN.[19] A recent study investigated the association between baseline circulating vitamin D levels and the incidence of macrovascular and microvascular diseases. It was found that a 50 nmol/L difference in circulating vitamin D levels was associated with a 23% reduction in the risk of macrovascular complications. Low circulating vitamin D levels were associated with an increased incidence of both macrovascular and microvascular disease events in individuals with T2DM.[20]

One of the microvascular complications of DM is DN. Relation of DN and vitamin D deficiency has been shown in prospective studies, indicating an independent and inverse association between total vitamin D and microalbuminuria.[21,22] Similarly, the current study found that MCR was significantly higher in patients with vitamin D levels <20 ng/mL. Furthermore, one of the key findings was a statistically significant but weak negative correlation between vitamin D levels and both urine microalbumin and urine MCR. These results reinforce the potential inverse relationship between vitamin D deficiency and DN markers.

A meta-analysis showed that vitamin D supplementation could potentially help with T2DM and T2DM-related nephropathy.[17] Experimental models have shown a reno-protectiveness with vitamin D supplementation.[23] In examining the impact of vitamin D supplementation on DN, research indicates that supplements, such as paricalcitol, an analogue of vitamin D, can effectively inhibit the RAAS, consequently reducing proteinuria in patients with T2DM. The RAAS plays a pivotal role in the pathogenesis of DN, and vitamin D has been shown to

interfere with its activity by inhibiting renin release. Consequently, vitamin D supplementation yields numerous benefits by mitigating renal risk factors, including hypertension and hyperlipidaemia, associated with DN. Crucially, studies have not reported any significant adverse effects or fluctuations in blood pressure linked to vitamin D supplementation. This underscores the potential of vitamin D as a safe and effective adjunctive therapy in managing DN and its associated complications. [3,24]

Numerous studies have suggested an inverse association for vitamin D and complications associated with inflammation. Vitamin D affects the insulin levels and sensitivity; therefore, vitamin D receptors and their tissue distribution are important. Reduced vitamin D levels have been linked to IR and poor glucose control. Key pathways may include pancreatic β-cell dysfunction, IR and systemic inflammation.[25] These findings support the current hypothesis that vitamin D is key in glucose metabolism and regulation, and may influence the risk of developing T2DM.

The current study has limitations, including a small sample size, and missing laboratory data as well as information on diabetes duration. Besides, the study data related to a period of reduced sunlight exposure. Larger, controlled studies are needed to clarify vitamin D's link with glucose regulation and DN.

Conclusion

Lower vitamin D levels were associated with poor glycaemic control, lipid dysregulation, and higher microalbuminuria, suggesting potential roles in glucose metabolism, inflammation and kidney function. There is a clear need for the development and implementation of nationwide strategies, especially within primary care settings, to promote awareness and adherence to vitamin D supplementation.

Acknowledgement: We are grateful to Dr Özge Pasin for statistical support.

Disclaimer: None.

Conflict of Interest: None.

Source of Funding: None.

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AUTHOR'S CONTRIBUTION:
ZIYS & AO: Concept, design, data acquisition, analysis, interpretation, drafting, revision, final approval and agreement to be accountable for all aspects of the work.