

Association of Vitamin D deficiency with dyslipidemia, glycemic control, and microalbuminuria in patients with Type 2 diabetes mellitus in Qatar

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ABSTRACT

Background: Emerging data found that Type 2 diabetes mellitus (T2DM) is associated with Vitamin D deficiency at various frequencies. This study aims to estimate the prevalence of Vitamin D deficiency in T2DM patients in Qatar and the correlation between Vitamin D deficiency and other variables such as dyslipidemia, glycemic control, and microalbuminuria. **Methods:** This retrospective cross-sectional analytical study was conducted in the medical outpatient clinic at Hamad General Hospital. The study involved adult patients (≥ 18 years) with T2DM. The study covered patients admitted between January 1, 2018, and July 31, 2018. Ethical approval was obtained from the Medical Research Committee. **Results:** We recruited 400 subjects with T2DM. Their mean age was 58.97 ± 10.32 years, and the majority were women (52.0%) and Arabs (69.5%). The mean duration of Type 2 diabetes diagnosis was 14.94 ± 8.99 years. The prevalence of Vitamin D deficiency was 29.5%. A comparison between Vitamin D deficiency and non-vitamin D deficiency groups showed a statistically significant difference in terms of fasting blood (FB) sugar ($p < 0.001$), random blood (RB) sugar ($p < 0.001$), hemoglobin A1c (HBA1c) ($p < 0.001$), total cholesterol ($P = 0.001$), low-density lipoprotein cholesterol (LDL [C]) ($p = 0.004$), high-density lipoprotein cholesterol (HDL [C]) ($p < 0.001$), triglyceride ($p < 0.001$), and urinary albumin excretion rate (UAER) ($p = 0.007$). Data analysis showed that a significant negative correlation was found between Vitamin D level and FB sugar ($r = -0.208$, $p < 0.001$), RB sugar ($r = -0.20$, $p < 0.001$), HBA1c ($r = -0.260$, $p < 0.001$), total cholesterol ($r = -0.218$, $p < 0.001$), LDL (C) ($r = -0.176$, $p = 0.004$), triglyceride ($r = -0.342$, $p < 0.001$), and UAER ($r = -0.184$, $p = 0.007$). **Conclusion:** Our study showed a significant correlation between Vitamin D deficiency and the poor control of T2DM, dyslipidemia, and microalbuminuria. The results emphasize the importance of monitoring Vitamin D status in high-risk populations.

Key words: Dyslipidemia, Glycemic control, Microalbuminuria, Type 2 diabetes mellitus, Vitamin D

Type 2 diabetes mellitus (T2DM) is a serious and growing global public health problem with a significant economic burden. Approximately 463 million people are living with diabetes in 2019, and the number is expected to reach 700 million by 2045. A recent modeling study estimates that the prevalence of T2DM among Qataris will rise from 17% in 2012 to at least 24% by 2050 in the absence of urgent and sufficient action [1,2]. Chronic complications, such as chronic kidney diseases and cardiovascular diseases, are well-known outcomes of T2DM progression that reduces patients' quality of life and increases the burden on the health-care system and diabetes mortality. As a result, it is essential to identify the main modifiable factors related to these complications to improve the prognosis of T2DM.

Vitamin D is an important hormone for calcium and phosphorus homeostasis and bone. It was found that T2DM is associated with Vitamin D deficiency at various frequencies. Recently, Vitamin D has acquired a large interest as a possible factor in the pathogenesis and prevention of diabetes. In a systematic review, it was concluded that Vitamin D and calcium deficiency can have a deleterious impact on glycemia, while glucose metabolism can be improved by a combination supplementation with both nutrients [3]. Other systematic review articles suggested the possible role of Vitamin D in the protection against some musculoskeletal disorders, infectious diseases, autoimmune diseases, cardiovascular disease, Type 1 and T2DM, several types of cancer, mental illness, infertility, and adverse pregnancy and birth outcomes [4-6]. Recent trial results are consistent with a large body of evidence from observational studies indicating that Vitamin D plays a role in modulating diabetes risk [7]. However, the causal relationship between Vitamin D deficiency and T2DM

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has not been established by randomized clinical trials, and, thus, will remain a matter of debate.

In Qatar, no previous studies examined the association between Vitamin D and T2DM in adult patients. Therefore, we designed this study to estimate the prevalence of Vitamin D deficiency in T2DM patients in Qatar and assess the correlation between Vitamin D deficiency and other variables such as dyslipidemia, glycemic control, and microalbuminuria.

MATERIALS AND METHODS

Study Design, Population, and Setting

This retrospective cross-sectional analytical study was conducted in the medical outpatient clinic at Hamad General Hospital. The study involved adult patients (≥ 18 years) with T2DM, between January 1, 2018, and July 31, 2018. Ethical approval was obtained from the Medical Research Committee (proposal number # MRC-01-18-289).

Inclusion and Exclusion Criteria

We excluded patients with other types of diabetes, namely, Type 1 diabetes, gestational diabetes mellitus, diabetes due to genetic abnormalities of beta-cell function or insulin action, and drug-induced diabetes. We also excluded pregnant and lactating women, patients taking Vitamin D (D2 and D3 supplements) and calcium supplements, patients with malabsorption owed to bowel disease or post-bariatric surgery, patients taking medications such as anticonvulsants, HIV-AIDS therapy, steroids, rifampin, cholestyramine, or orlistat. On the other hand, adult patients (≥ 18 years) with T2DM who were using the daily anti-diabetic treatment and statin therapy for at least 3 months and who did not meet the above criteria were recruited into this study.

Definitions and Diagnostic Criteria

1. Vitamin D status was classified as follows: Serum Vitamin D level < 20 ng/ml was defined as Vitamin D deficiency. A serum level of 20–30 ng/ml is defined as Vitamin D insufficiency. Moreover, a serum level of > 30 –50 ng/mL was considered as Vitamin D sufficiency (Optimum) [8]. Since there was no consensus on the level of Vitamin D that denotes insufficiency and to avoid confusion or misunderstanding on the part of readers, we have used the term Vitamin D deficiency throughout the text to refer to patients with Vitamin D levels < 20 ng/ml.
2. Urinary albumin excretion rate (UAER) was expressed as μg albumin excreted/mg of creatinine in urine and classified into the following categories: Normal (< 30), mild (30–100), moderate (> 100 –200), severe (> 200 –300), and macroalbuminuria (> 300) $\mu\text{g}/\text{mg}$. The term microalbuminuria was used to describe urinary albumin excretion within 30–300 $\mu\text{g}/\text{mg}$ creatinine [8].
3. Hemoglobin A1c (HbA1c) $\leq 8\%$ was considered as controlled levels and $> 8\%$ as uncontrolled levels [9].
4. Estimated glomerular filtration rate values (eGFR)

were calculated according to the chronic kidney disease epidemiology collaboration formula [10].

Sample Size

A sample size of 400 subjects was obtained, which provided a power of 80% at an alpha value of 0.05.

Data Analysis

Descriptive statistics of qualitative and quantitative data were expressed in the form of frequency along with percentage and mean \pm Standard Deviation (SD). An independent t-test was used to compare the difference in the means of blood glucose, HbA1c, lipids, and UAER between diabetic patients with and without a Vitamin D deficiency. Pearson correlation analyzes were performed to examine the linear relationship between Vitamin D deficiency and dyslipidemia, glycemic control, and microalbuminuria. Data analysis was performed with SPSS software (v 23; IBM Corp, Armonk, NY, USA).

RESULTS

Demographic and Clinical Data

We recruited 400 subjects with T2DM. Their mean age was 58.97 ± 10.32 years (range of 28–86 years) and the majority were women (52.0%) and Arabs (69.5%). The mean duration of Type 2 diabetes diagnosis was 14.94 ± 8.99 years (range of 1–50 years) and mean BMI was 31.48 ± 6.21 kg/m^2 (range of 19.8–64.3 kg/m^2). Table 1 describes the demographic and clinical characteristics of the study population. The prevalence of Vitamin D deficiency was 29.5%.

Comparison between Vitamin D Deficiency and non-vitamin D deficiency groups

A comparison between Vitamin D deficiency and non-vitamin D deficiency groups showed a statistically significant difference in terms of fasting blood (FB) sugar ($p < 0.001$), random blood (RB) sugar ($p < 0.001$), HbA1c ($p < 0.001$), total cholesterol ($p = 0.001$), low-density lipoprotein cholesterol (LDL [C]) ($p = 0.004$), high-density lipoprotein cholesterol (HDL [C]) ($p < 0.001$), triglyceride ($p < 0.001$), and UACR ($p = 0.007$). Table 2 summarizes the comparison between Vitamin D deficiency and non-vitamin D deficiency groups in relation to demographic and clinical aspects.

Correlation between Vitamin D Deficiency and Dyslipidemia and Glycemic Control and Microalbuminuria

The correlations between the level of serum Vitamin D and FB sugar, RB sugar, glycated hemoglobin (HbA1c), total cholesterol, LDL (C), HDL (C), triglyceride, and UACR were described in Table 2. Data analysis showed that a significant negative correlation was found between Vitamin D level and FB sugar ($r = -0.208$, $p < 0.001$), RB sugar ($r = -0.20$, $p < 0.001$), HbA1c

Table 1: Demographic and clinical characteristics of the patients involved in this study

Variable	n (%) / Mean ± SD (Range)
Age	58.97 ± 10.32 (28–86)
Sex	
Male	192 (48.0)
Female	208 (52.0)
Nationality	
Arab	278 (69.5)
Asian	105 (26.3)
African	12 (3.0)
Caucasian	5 (1.2)
Clinical characteristics	
Hypertension	301 (75.25)
Dyslipidemia	340 (85)
CKD	89 (22.25)
Liver disease	11 (2.75)
CAD	94 (23.5)
CVA	15 (3.75)
PVD	17 (4.25)
Retinopathy	119 (29.75)
Neuropathy	66 (16.5)
Vitamin D deficiency	118 (29.5)
BMI	31.48 ± 6.21 (19.8–64.3)
Duration of DM	14.94 ± 8.99 (1–50 years)
Fasting Blood Sugar	7.41 ± 1.88 (3.8–21 mmol/l)
Random Blood Sugar	10.64 ± 3.36 (1.8–22 mmol/l)
HBA1c	8.07 ± 1.48 (5–12.7)
Total cholesterol	4.07 ± 1.19 (1.1–11.9 mmol/l)
LDL (c)	2.15 ± 0.99 (0.40–9.40 mmol/l)
HDL (c)	1.16 ± 0.31 (0.4–2.5 mmol/l)
Triglycerides	1.62 ± 0.92 (0.4–8.5 mmol/l)
Creatinine	105.80 ± 123.29 (15–909 µmol/l)
eGFR	77.09 ± 24.53 (3.40–160 mL/min/1.73m ²)
Vitamin D level	29.89 ± 14.46 (4–81 ng/ml)
Calcium	2.39 ± 0.10 (1.87–2.80 mmol/l)
Phosphorous	1.32 ± 0.36 (0.72–2.44 mmol/l)
UACR	21.34 ± 90.16 (0.3–999.9 µg albumin/mg creatinine)

CKD: Chronic kidney disease, CAD: Coronary artery disease, CVA: Cerebrovascular accident, PVD: Peripheral vascular disease, BMI: Body mass index, LDL (C): Low-density lipoprotein cholesterol, HDL (c): High-density lipoprotein cholesterol, eGFR: Estimated glomerular filtration rate, UACR: Urinary Albumin Excretion Rate

($r = -0.260$, $p < 0.001$), total cholesterol ($r = -0.218$, $p < 0.001$), LDL (C) ($r = -0.176$, $p = 0.004$), triglyceride ($r = -0.342$, $p < 0.001$), and UACR ($r = -0.184$, $p = 0.007$). Table 3 describes the correlation between Vitamin D level and other variables.

DISCUSSION

Recently, researchers began to show increased interest in the association between Vitamin D deficiency and diabetes mellitus. To the best of our knowledge, this is the first study designed to

assess the association between Vitamin D and T2DM in adult patients living in Qatar. The effect of Vitamin D on β -cell function and insulin sensitivity has been observed in animal and human studies. Vitamin D improves insulin production and sensitivity. It facilitates the biosynthetic capacity of β cells and, also, accelerates the conversion of proinsulin into insulin [11-13].

Emerging evidence suggests that Vitamin D is involved in the etiology and pathogenesis of diabetes mellitus [14], leading to the hypothesis that Vitamin D insufficiency is positively correlated with insulin resistance and cardiovascular risk in obese adolescents. This also shows that Vitamin D supplementation improves insulin resistance and cardiovascular risk factors in this population [15]. The reported prevalence of Vitamin D deficiency among patients with T2DM ranges from 62% to 91% [16,17]. In our study, the prevalence of Vitamin D deficiency was 29.5%. The low prevalence was not true, as we involved only the patients with Vitamin D levels of <20 ng/ml.

In general, our findings are in line with most of the recent studies. In the present study, the bivariate analysis showed that FB sugar, RB sugar, and HBA1c were affected and uncontrolled in the Vitamin D deficiency group compared with the non-Vitamin D deficiency Group, in line with the results observed by Anyanwu *et al.* [18]. Moreover, a significant inverse correlation between glycemic control and Vitamin D levels has been reported by several authors [17-19]. This provides further evidence that low serum Vitamin D can play a significant role in impaired glucose metabolism. However, the hypothesis that Vitamin D supplementation may improve insulin resistance and cardiovascular risk factors in this population has not yet been proven, and prescribing supplemental Vitamin D during the early phase of diabetes is still experimental [20].

There were other findings in this study, including the association between Vitamin D deficiency and dyslipidemia. We found significantly increased mean total cholesterol, LDL (C), and triglyceride levels in subjects with Vitamin D deficiency compared with those with the non-Vitamin D deficiency Group. This finding has been reported by several authors [17,21-23]. The functions of Vitamin D are linked to lipid values in several ways: First, Vitamin D regulates calcium metabolism and increases intestinal calcium absorption, thereby reducing intestinal fatty acid absorption [24]. Therefore, a reduction in intestinal fat absorption can lower the cholesterol level. In addition, increasing the calcium concentration promotes the conversion of cholesterol into bile acids in the liver, resulting in reduced cholesterol levels [25].

It was also found that there was an increased frequency of microalbumin in the Vitamin D deficient group compared to the non-Vitamin D deficiency Group. In addition, we also found that Vitamin D level was inversely correlated with microalbumin levels, which agrees with the results of Balla *et al.* and Fiscella *et al.* [26,27]. This suggests that studies to further characterize the role of Vitamin D as a possible risk factor in diabetic nephropathy are needed to assess the impact of maintaining adequate Vitamin D levels on the progression of diabetic nephropathy, as some studies have shown that Vitamin D replacement therapy reduces albuminuria in patients with chronic kidney disease [27,28]. On

Table 2: A comparison between Vitamin D deficiency and non-vitamin D deficiency groups crosschecked with demographic and clinical aspects

Variables	Total	Vitamin D Deficiency (%)	non-Vitamin D deficiency (%)	p-value
Gender (M/F)	192/208	57/61	135/147	1.00
Nationality				
Arab	278	78 (28.1)	200 (71.9)	0.925
Asian	105	38 (36.2)	67 (63.8)	
African	12	1 (8.3)	11 (91.7)	
Caucasian	5	1 (20)	4 (80)	
Clinical aspects				
Hypertension	301	80 (26.6)	221 (73.4)	0.019
Dyslipidemia	340	97 (28.5)	243 (71.5)	0.194
CKD	89	29 (32.6)	60 (67.4)	0.275
Liver disease	11	2 (18.2)	9 (81.8)	0.327
CAD	94	28 (29.8)	66 (70.2)	0.520
CVA	15	4 (26.7)	11 (73.3)	0.530
PVD	17	5 (29.4)	12 (70.6)	0.616
Retinopathy	119	34 (28.6)	85 (71.4)	0.445
Neuropathy	66	17 (25.8)	49 (74.2)	0.284
Age	58.97±10.32 (28–86)	56.212±10.1727	60.131±10.1914	0.001
BMI	31.48±6.21 (19.8–64.3)	31.7059±5.92267	31.3879±6.33677	0.641
FBS	7.41±1.88 (3.8–21)	7.919±2.3112	7.207±1.6412	0.001
RB sugar	10.64±3.36 (1.8–22)	11.4297±3.80929	10.3233±3.10591	0.003
HBA1c	8.07±1.48 (5–12.7)	8.6241±1.57192	7.8449±1.38699	<0.001
Total cholesterol	4.07±1.19 (1.1–11.9)	4.5475±1.43512	3.8750±1.02221	<0.001
LDL (c)	2.15±0.99 (0.40–9.40)	2.4875±1.21508	2.0171±0.85777	<0.001
HDL (c)	1.16±0.31 (0.4–2.5)	1.0680±0.25962	1.2079±0.32755	<0.001
Triglyceride	1.62±0.92 (0.4–8.5)	2.1022±1.23925	1.4206±0.65374	<0.001
Creatinine	105.80±123.29 (15–909)	105.110±112.4449	106.094±127.7520	0.942
UACR	21.34±90.16 (0.3–999.9)	48.7239±155.06557	10.4996±38.51122	<0.001

CKD: Chronic kidney disease, CAD: Coronary artery disease, CVA: Cerebrovascular accident, PVD: Peripheral vascular disease, BMI: Body mass index, LDL (C): Low-density lipoprotein cholesterol, HDL (c): High-density lipoprotein cholesterol, eGFR: Estimated glomerular filtration rate, UACR: Urinary Albumin Excretion Rate, FB: Fasting blood, RB: Random blood

Table 3: Results of simple linear regression with Vitamin D levels as the dependent variable

Variables	Correlation Coefficient (r)	p-value
Hypertension	-0.048	0.339
Dyslipidemia	-0.047	0.345
CKD	0.081	0.104
Liver disease	-0.129	0.010
CAD	0.038	0.449
CVA	0.007	0.893
PVD	0.052	0.303
Retinopathy	0.047	0.352
Neuropathy	-0.022	0.656
Duration of DM	0.009	0.852
Age	0.091	0.070
BMI	-0.113*	0.024
FBS	-0.208	<0.001
RB sugar	-0.184	<0.001
HBA1c	-0.255	<0.001
Total cholesterol	-0.216	<0.001

(Contd...)

Table 3: (Continued)

Variables	Correlation Coefficient (r)	p-value
LDL (c)	-0.173	0.001
HDL (c)	0.211	<0.001
Triglyceride	-0.342	<0.001
Creatinine	-0.041	0.411
GF Rate	0.175	<0.001
Calcium	0.040	0.426
UACR	-0.183	0.001

CKD: Chronic kidney disease, CAD: Coronary artery disease, CVA: Cerebrovascular accident, PVD: Peripheral vascular disease, BMI: Body mass index, LDL (c): Low-density lipoprotein cholesterol, HDL (c): High-density lipoprotein cholesterol, eGFR: Estimated glomerular filtration rate, UACR: Urinary Albumin Excretion Rate, FB: Fasting blood, RB: Random blood

the other hand, some studies have provided conflicting results regarding the association between Vitamin D status and diabetic nephropathy [29-31].

There are limitations to this study that should be considered: First, it was a retrospective analysis and, therefore, relied on secondary data. Second, we involved only patients with Vitamin D levels of <20 ng/l because there was no consensus on the level

of Vitamin D that denotes insufficiency, so there is a possibility that we lost patients in the prevalence estimation. Third, because this was a hospital-based study, the results cannot be generalized. Despite these limitations, this study is the first to highlight the relationship between Vitamin D deficiency and glycemic control, dyslipidemia, and microalbuminuria in Qatar.

CONCLUSION

Our study showed a significant relationship between Vitamin D deficiency and glycemic control, dyslipidemia, and microalbuminuria. The results emphasize the need for better awareness among researchers and clinicians about the consequences of Vitamin D deficiency and the importance of monitoring its status in high-risk populations. Increasing population awareness is also essential to overcome Vitamin D deficiency in the population of Qatar. However, these aspects are worth investigating with large prospective studies and with adequate follow-up.

AUTHORS' CONTRIBUTION

Karuppasamy G wrote the proposal, analyzed the data, and wrote the final manuscript. Al Shokri S proposed the idea, reviewed the literature, and aided in the data collection. Sukik A aided in the data collection, research proposal writing, and data entry. Saleh AO aided in the data collection and data entry. Osman ME aided in research proposal writing, the data analysis, and the revision of the final manuscript. All authors read the manuscript and agree to its publication.

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