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Association Between Vitamin D3 Levels and Diabetic Nephropathy in Patients with Type 2 Diabetes Mellitus

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Abstract

Objectives: To determine the serum Vitamin D3 levels and assess the presence of diabetic nephropathy in patients with Type 2 Diabetes Mellitus.

Study Design and Setting: This cross-sectional study was conducted at Medicine Departments of Bolan and **Jhalawan Medical College** Hospital (1-April-2024 to 31-March-2025).

Methodology: The study included 316 type 2 diabetes patients, aged 35–70 with Type 2 Diabetes for five years or more. Blood and urine samples were taken to assess Vitamin D3 levels and kidney function. Data on blood pressure, diabetes duration, and treatment were collected. Analyzed by SPSS V26, statistical tests assessed the link with diabetic nephropathy.

Results: Mean age was 55.8 ± 8.6 years. Males were 178 (56.3%) and females 138 (43.7%). Most lived in urban areas 204 (64.6%). The average diabetes duration was 9.7± 4.2 years. Treatments were oral hypoglycemic agents in 142 (44.9%), insulin in 81 (25.6%), and both in 93 (29.4%). Vitamin D3 deficiency (<20 ng/mL) was found in 229 (72.5%). Diabetic nephropathy (DN) was present in 189 (59.8%). DN patients had lower Vitamin D3 levels ($p < 0.05$). Hypertension was higher in DN (75.7%) than non-DN (44.1%), $p < 0.001$. Insulin use was also higher in DN (36.5%) than non-DN (11.0%), $p < 0.001$. Hyperlipidemia was more common in DN ($p = 0.001$). Family history showed no significant difference ($p = 0.27$).

Conclusion: Low Vitamin D3 levels were linked to diabetic nephropathy in type 2 diabetes patients. Vitamin D3 may play a role in kidney damage in diabetes.

Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic, progressive metabolic disorder characterized by insulin resistance and relative insulin deficiency, leading to persistent hyperglycemia and a wide spectrum of complications.[1] It is now recognized as one of the most significant global health threats of the 21st century. The International Diabetes Federation (IDF) estimates that in 2019, approximately 463

million adults were living with diabetes, and this number is projected to rise to 700 million by 2045.[2] The escalating prevalence is driven by urbanization, sedentary lifestyles, unhealthy dietary habits, and increasing obesity rates.

Among the many complications of T2DM, diabetic nephropathy (DN) is one of the most serious and burdensome.[3] DN is a microvascular complication that develops gradually over years and is characterized by persistent albuminuria, progressive decline in glomerular filtration rate (GFR), and increased cardiovascular risk. It remains the leading cause of end-stage renal disease (ESRD) globally and contributes significantly to morbidity, mortality, and healthcare costs.[4] The natural history of DN often begins with subtle changes in kidney structure and function, progressing from microalbuminuria to macroalbuminuria and eventually to ESRD if left untreated.

The prevalence of DN among individuals with T2DM varies across regions but is generally reported to be between 20% and 40%.[5] Several well-established risk factors contribute to its development, including poor glycemic control, hypertension, dyslipidemia, obesity, smoking, and genetic predisposition. Beyond the renal consequences, DN is strongly associated with cardiovascular morbidity, which amplifies its clinical impact.[6] Patients with DN often require prolonged medical management, frequent hospital visits, and in advanced stages, renal replacement therapy, placing a substantial economic and emotional burden on both patients and healthcare systems.

In Pakistan, the burden of T2DM has reached alarming levels, with recent national survey data reporting a prevalence of 16.98% among adults.[7] Given the country's limited healthcare infrastructure, high out-of-pocket health expenditures, and large rural population, the management of DN presents a formidable challenge. Hospital-based studies have shown a high frequency of microalbuminuria and early renal involvement among diabetic patients, suggesting that DN often develops silently before it is clinically recognized.[8] This highlights the urgent need for early identification of modifiable risk factors that can delay or prevent progression.

One such potentially modifiable factor is Vitamin D3 deficiency. Vitamin D3 (cholecalciferol) is a fat-soluble vitamin essential for maintaining calcium-phosphorus balance and bone health. In recent decades, research has revealed its extra-skeletal roles, including regulation of immune function, modulation of inflammatory responses, and potential influence on glucose metabolism and insulin sensitivity.[9] Globally, Vitamin D deficiency is widespread, affecting over a billion people, with particularly high prevalence in South Asia due to cultural practices limiting sun exposure, darker skin pigmentation reducing cutaneous synthesis, dietary insufficiency, and urban living.

The possible link between Vitamin D3 deficiency and DN is supported by several biological mechanisms. Vitamin D3 is known to suppress the renin–angiotensin–aldosterone system, reduce oxidative stress, modulate podocyte function, and inhibit pro-inflammatory cytokine release. These pathways are relevant because chronic hyperglycemia in T2DM promotes oxidative damage and inflammation in renal tissue, leading to structural injury and functional decline. International studies have found that lower Vitamin D3 levels are associated with higher proteinuria and reduced kidney function in diabetic patients. For instance, Hong et al. reported that Korean T2DM patients with Vitamin D3 deficiency had significantly higher urinary albumin excretion and lower estimated GFR compared to those with adequate levels. Meta-analyses further suggest that Vitamin D supplementation can modestly reduce proteinuria and may slow the progression of DN, although evidence is still emerging.[10]

In Pakistan, Vitamin D3 deficiency is common across all age groups, including apparently healthy adults. In diabetic populations, deficiency rates are even higher, yet local research directly assessing its relationship with DN is scarce. Moreover, there are no standardized national guidelines recommending routine screening for

Vitamin D3 levels in T2DM patients, nor are there targeted supplementation protocols aimed at renal protection. This gap in evidence and practice underscores the importance of locally conducted studies to clarify whether Vitamin D3 deficiency contributes to DN risk in this population.[8-10]

PATIENTS AND METHODS

This analytical cross-sectional study was conducted in the Department of Medicine at Bolan Medical College, Quetta, and Jhalawan Medical College, Khuzdar. Patients were recruited from both the medical wards and outpatient clinics of these institutions. The study duration was one year, extending from 1-**April-2024 to 31-March-2025**. Ethical approval was obtained from the Institutional Review Board (IRB) of Bolan University of Medical and Health Sciences prior to the commencement of the research (No. 1173 BUMHS/IRB/25, dated 15-02-2024). All procedures adhered to the ethical standards of human subject research as per the Declaration of Helsinki. Written informed consent was obtained from each participant prior to their inclusion in the study.

A non-probability consecutive sampling technique was used to select participants.[7] All eligible patients who met the inclusion criteria and provided informed consent were enrolled. The inclusion criteria specified patients aged between 35 and 70 years, of either sex, with a known diagnosis of T2DM for at least five years. Both male and female patients from various ethnic groups were included, with ethnicity determined by self-report. All participants were required to be willing and able to comply with study procedures, and to sign the written consent form.

Exclusion criteria were established to avoid confounding factors that could influence serum Vitamin D3 levels or kidney function. Patients with a known diagnosis of kidney disease not attributed to diabetes were excluded. Those who had taken Vitamin D supplements within the past three months were not eligible, as supplementation could alter serum Vitamin D3 levels and affect study outcomes. Individuals who had experienced an acute illness or required hospitalization in the preceding four weeks were also excluded, to avoid the impact of acute physiological stress on the study parameters. Exclusions were determined based on patient history and review of available medical records. Recruitment continued until the target sample size was achieved.

The sample size was calculated using OpenEpi software, with an expected frequency of insulin resistance of 71.1%, a precision of ±5%, and a 95% confidence level.[10] The design effect was set at 1 for simple random sampling. Based on these parameters, a sample size of 316 was determined to be sufficient for statistical analysis. The calculated sample size was considered adequate to detect meaningful associations between serum Vitamin D3 levels and DN. Both newly registered and follow-up patients attending the clinics were considered for inclusion.

Demographic data collected for each participant included age, sex, ethnicity, weight, height, and body mass index (BMI). The duration of T2DM was recorded in years. Blood pressure was measured using a mercury sphygmomanometer, with the patient in a seated position after at least five minutes of rest. Two readings were taken at an interval of two minutes, and the average was recorded as the final value.

For laboratory analysis, venous blood samples were collected from all participants under aseptic precautions. Approximately 5 mL of blood was obtained for the measurement of serum 25-hydroxy Vitamin D3 levels, using a chemiluminescence immunoassay (Roche Diagnostics, Mannheim, Germany). Serum Vitamin D3 deficiency was defined as a level below 20 ng/mL, while levels of 20 ng/mL or above were considered sufficient. An additional 5 mL of blood was collected for serum creatinine estimation and glycated hemoglobin (HbA1c) measurement. Serum creatinine was analyzed using an automated chemistry analyzer (Beckman Coulter Inc., Brea, CA, USA), while HbA1c was determined through high-performance liquid

chromatography (HPLC), which is recognized for its accuracy in assessing long-term glycemic control.

Urine samples were collected as spot urine specimens for the measurement of the urine albumin-to-creatinine ratio (UACR). The UACR was analyzed using an immunoturbidimetric method. DN was diagnosed if the UACR was greater than 30 mg/g, or if the estimated glomerular filtration rate (eGFR) was less than 60 mL/min/1.73 m^2 for at least three months. The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, which is widely recommended for its accuracy across different levels of kidney function.

All laboratory tests were conducted in the central laboratory of the respective hospitals, following standard operating procedures. Equipment used for biochemical analyses was regularly calibrated according to the manufacturer's guidelines to ensure the reliability and validity of results.

Data were initially recorded in a structured data collection form, specifically designed for this study by the investigators.[11] The form included sections for demographic details, clinical history, examination findings, and laboratory results. Data entry was performed in SPSS version 26.0 for statistical analysis. Continuous variables, such as age, BMI, blood pressure, serum Vitamin D3, HbA1c, serum creatinine, eGFR, and UACR, were summarized as mean ± standard deviation (SD). Categorical variables, such as gender, residential area, treatment type, comorbidities, Vitamin D3 status, and DN presence, were expressed as frequencies and percentages.

For inferential analysis, serum Vitamin D3 levels were categorized into deficient (<20 ng/mL) and sufficient (≥20 ng/mL) groups. The presence or absence of DN was recorded as a binary variable. Associations between categorical variables, such as Vitamin D3 status and DN, were assessed using the Chi-square test. Continuous variables were compared between groups using the independent t-test when normally distributed, or the Mann–Whitney U test when data did not meet normality assumptions. Normality was assessed using the Shapiro–Wilk test. All statistical tests were two-tailed, and a p-value of less than 0.05 was considered statistically significant. Results were presented both in tables and in narrative form for clarity. Tables were used to display detailed descriptive statistics for demographic variables, clinical characteristics, anthropometric data, and laboratory findings. The narrative text highlighted key trends and important associations observed in the data. Data analysis and results verification were performed independently by a second researcher to minimize errors in statistical computation and interpretation.

This systematic approach ensured that the study design was robust, the data collection process was standardized, and the statistical analysis was appropriate for the research question. The methodology allowed for accurate assessment of the relationship between serum Vitamin D3 levels and DN in patients with T2DM, providing a reliable basis for drawing meaningful conclusions from the study findings.

Results

The demographic characteristics of the study participants are presented in Table 1. A total of 316 individuals were enrolled, with ages ranging from 35 to 70 years and a mean age of 55.8 ± 8.6 years. The majority (64.6%) resided in urban areas, while 35.4% lived in rural regions.

Table 1. Demographic Characteristics of the Study Participants (n = 316)

Variable	n (%) or Mean ± SD
Gender	
Male	178 (56.3)
Female	138 (43.7)

Variable	n (%) or Mean ± SD
Residential Area	
Urban	204 (64.6)
Rural	112 (35.4)

The treatment history and comorbidities of the participants are summarized in Table 2. A family history of diabetes was reported by 73.1% of participants, while 26.9% had no such history. At the time of examination, 6.0% reported a current infection or acute illness, whereas 94.0% did not have any acute health condition.

Table 2. Treatment History and Comorbidities

Variable	n (%) or Mean ± SD
Duration of T2DM (years)	9.7 ± 4.2
Treatment for T2DM	
Oral hypoglycemic agents	142 (44.9)
Insulin	81 (25.6)
Both	93 (29.4)
Comorbidities	
Hypertension	198 (62.7)
Ischemic Heart Disease	66 (20.9)
Hyperlipidemia	111 (35.1)
None	62 (19.6)

The body mass index parameters are shown in Table 3. The mean blood pressure of the participants was 138/86 ± 14/9 mmHg. BMI values indicated that the study population was generally overweight, with a mean of 26.4 ± 3.2 kg/m².

Table 3. Body Mass Index Parameters

Parameter	Mean ± SD
Weight (kg)	71.5 ± 10.4
Height (cm)	164.2 ± 8.3
BMI (kg/m²)	26.4 ± 3.2

The laboratory parameters and DN status are presented in Table 4. The mean urine albumin-to-creatinine ratio was 152.4 ± 88.2 mg/g. Microalbuminuria was the most frequent finding, observed in 53.2% of participants, followed by macroalbuminuria in 25.9% and normal albumin excretion in 20.9%. DN was present in 59.8% of the study population, while 40.2% showed no evidence of the condition.

Table 4. Laboratory Parameters and Diabetic Nephropathy Status

Parameter	Mean ± SD or n (%)
Serum Vitamin D3 (ng/mL)	17.3 ± 6.9
Deficient (<20 ng/mL)	229 (72.5)
Sufficient (≥20 ng/mL)	87 (27.5)
HbA1c (%)	8.2 ± 1.1
Serum Creatinine (mg/dL)	1.2 ± 0.3
Estimated GFR (mL/min/1.73 m²)	68.5 ± 15.7

DISCUSSION

This analytical cross-sectional study demonstrated a strong and statistically significant association between low serum Vitamin D3 levels and the presence of DN in patients with T2DM. A large proportion of participants had both Vitamin D3 deficiency and DN, indicating that inadequate Vitamin D3 status may be an important, potentially modifiable factor in the development and progression of DN. [11] This observation is clinically relevant, as early detection and correction of Vitamin D3 deficiency may represent a low-cost, accessible intervention to reduce the burden of kidney failure and related complications in diabetic populations.

In our cohort, the prevalence of Vitamin D3 deficiency was notably high at 72.5%, reflecting both regional and global trends. This finding underscores the magnitude of the problem in South Asian populations, where environmental, cultural, and dietary factors converge to create widespread deficiency. DN was more frequent among patients with a longer duration of diabetes, consistent with the chronic, progressive nature of microvascular complications. Patients with DN were also more likely to have comorbid hypertension and hyperlipidemia, reinforcing the role of cardiovascular risk factors in kidney disease progression.[12]

Interestingly, although insulin use was more common among DN patients, there was no significant difference in HbA1c between DN and non-DN groups. This suggests that glycemic control alone may not be sufficient to fully explain DN risk, and that other metabolic and nutritional factors including Vitamin D3 status may play an important role. The finding that even patients with a shorter duration of diabetes but low Vitamin D3 levels showed signs of kidney injury raises the possibility that Vitamin D3 deficiency could accelerate the onset of DN.[11. 12]

Our results align with a growing body of international evidence linking Vitamin D3 deficiency with DN. Hong et al. (2021) in Korea found that patients with low Vitamin D3 levels had greater urinary protein excretion and reduced eGFR compared to those with adequate levels. Similarly, Varshney et al. (2019) in India reported that lower Vitamin D3 levels were strongly associated with higher urinary albumin and worse kidney function.[13] These consistent findings across diverse populations support the biological plausibility of the association observed in our study.

Vitamin D3 deficiency is well-documented in South Asia, driven by a combination of factors: limited sun exposure due to indoor lifestyles, cultural clothing practices that restrict skin exposure, high skin melanin content reducing cutaneous synthesis, and diets low in Vitamin D-rich foods.[10-13] Chronic diseases such as diabetes further exacerbate the problem due to altered metabolism, decreased outdoor activity, and comorbidities that affect nutrient absorption and utilization. The high prevalence of deficiency in our cohort mirrors these known trends, and its overlap with DN highlights a critical area for targeted intervention.

Our study adds to the relatively limited body of Pakistani research on this topic. Previous local studies have primarily examined Vitamin D3 deficiency prevalence in the general population or in diabetics without specifically addressing DN. The inclusion of both inpatients and outpatients from two tertiary care centers in our research enhances the representativeness of our sample, providing a broader view of the clinical reality.[14]

The relationship between diabetes duration and DN in our study is consistent with the established natural history of microvascular complications, which often manifest after years of hyperglycemia. However, the marked differences in Vitamin D3 levels between DN and non-DN patients—even in those with shorter disease duration—suggest that deficiency may contribute to earlier renal damage.[15] This aligns with experimental evidence indicating that Vitamin D3 has renoprotective properties, including modulation of the renin–angiotensin–aldosterone system (RAAS), reduction of oxidative stress, and attenuation of podocyte injury.

Our findings regarding comorbidities parallel those of the UK Prospective Diabetes Study (UKPDS), which demonstrated that blood pressure control significantly reduces the risk of microvascular complications, including DN. The higher prevalence of hypertension and hyperlipidemia among DN patients in our study reinforces their role in accelerating renal decline. The lack of significant difference in HbA1c between groups echoes the conclusions of several other studies, which suggest that while glycemic control is necessary, it is not solely predictive of DN risk. This supports the need for a multifactorial approach to DN prevention and management.[13-15]

The implications of these findings are both clinical and public health oriented. At the clinical level, incorporating Vitamin D3 screening into routine care for patients with T2DM, particularly those with risk factors for DN, could enable early identification and treatment of deficiency. Such an approach is practical and relatively inexpensive, especially in resource constrained healthcare systems. Given the simplicity of supplementation and its potential benefits, addressing Vitamin D3 deficiency could be a feasible strategy to help slow DN progression.[16]

From a mechanistic perspective, Vitamin D3's role in modulating RAAS activity, reducing inflammation, and improving endothelial function provides a plausible explanation for its potential protective effects on kidney health. Supplementation could complement existing DN prevention strategies, which already emphasize strict glycemic control, blood pressure management, and lipid regulation. Future interventional studies should examine whether correcting Vitamin D3 deficiency leads to measurable improvements in renal outcomes in diabetic patients.[17]

At the public health level, these findings highlight the potential value of integrating nutritional screening into diabetes management programs. In Pakistan, where both diabetes and Vitamin D3 deficiency are highly prevalent, targeted policies that encourage screening, supplementation, and public awareness could have a substantial impact. Public health campaigns promoting safe sun exposure, dietary modification, and fortified foods could address Vitamin D3 deficiency at the population level.[18]

Additionally, these results suggest the need for updated national clinical guidelines to include Vitamin D3 status as part of comprehensive risk assessment for DN. Implementing such guidelines could standardize care, reduce variation in practice, and improve patient outcomes. Incorporating nutritional assessment alongside traditional cardiovascular risk management could form a more holistic approach to diabetic care, particularly in high-prevalence countries.[16-18]

While our study offers valuable insights, certain limitations must be acknowledged. The cross-sectional design prevents us from establishing causality between low Vitamin D3 levels and DN. Reverse causality is possible, as patients with DN may have lower Vitamin D3 due to impaired renal activation of the vitamin. Longitudinal studies or randomized controlled trials would be better suited to determine the direction and strength of this relationship.[19]

Additionally, we did not measure related biochemical parameters such as parathyroid hormone, serum calcium, or phosphorus, which could help clarify the metabolic context of Vitamin D3 deficiency.[20] Inflammatory markers were also not assessed, despite evidence that Vitamin D3 may influence inflammatory pathways relevant to DN. These additional measures could provide more detailed mechanistic insights.[21]

Another limitation is the absence of assessment for seasonal variation, which can affect Vitamin D3 levels due to changes in sunlight exposure.[21] Furthermore, we did not collect detailed dietary histories or information on physical activity levels, factors known to influence both Vitamin D3 status and kidney health. Without this information, potential confounding effects remain unaccounted for.[22]

Our sample size, while adequate for detecting associations, did not include a healthy non-diabetic control group, which limits our ability to compare Vitamin D3 levels between diabetics and the general population.[23] Including such controls in future

studies could help clarify whether deficiency is disproportionately prevalent in T2DM patients.

Finally, although we took measures to minimize bias, such as using standardized laboratory assays, conducting all analyses in a single laboratory, and excluding patients with recent Vitamin D supplementation or known non-diabetic kidney disease, unmeasured confounding cannot be entirely ruled out.[24] Nonetheless, the study's methodological strengths, including double checking data for accuracy and applying appropriate statistical analyses, support the reliability of the results.[25]

CONCLUSION

This study shows a strong link between low Vitamin D3 levels and DN in people with T2DM. Most patients with DN had Vitamin D3 deficiency. These patients also had worse kidney function. High blood pressure, long diabetes duration, and insulin use were more common in DN patients. Low Vitamin D3 may play a role in kidney damage. Early detection and treatment of Vitamin D3 deficiency may help. It could slow the progress of DN. This finding is important for doctors. Future research and health policies should include Vitamin D3 status in diabetes care.

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DISCLAIMER: This study is intended for academic and scientific purposes only, and the views expressed are solely those of the authors. It should not replace professional medical advice, and readers are encouraged to verify data independently. The authors disclaim liability for any errors or outcomes arising from its use.

Conflict of Interest: The author(s) declare no conflict of interest related to this study. No personal, financial, or professional influences have affected the research design, data collection, analysis, or reporting of findings.

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