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A Comparative Study of Serum 25-Hydroxyvitamin D Levels in Type 2 Diabetes Mellitus Patients With and without Nephropathy

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ABSTRACT

Background: Diabetic nephropathy (DN) is a major microvascular complication of type 2 diabetes mellitus (T2DM) and a leading cause of chronic kidney disease. Vitamin D deficiency has been implicated in the progression of DN due to its role in renal function, inflammation, and the renin-angiotensin system. This study aimed to assess 25-hydroxyvitamin D [25(OH)D] levels in T2DM patients with and without nephropathy and to examine correlations with renal function markers.
Methods: A cross-sectional study was conducted at Hassan Institute of Medical Sciences over one year, including 120 T2DM patients aged 40–75 years. Participants were divided into DN (n = 60) and non-DN (n = 60) groups based on urinary albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR). Data collected included demographic details, duration of diabetes, comorbidities, fasting blood sugar, serum creatinine, UACR, eGFR, and serum 25(OH)D levels. Statistical analysis was performed using SPSS version 21, including t-tests, ANOVA, and Pearson correlation; $p < 0.05$ was considered significant.
Results: The mean age of participants was 58.82 ± 6.07 years, with equal gender distribution. The DN group had significantly higher fasting blood glucose (196.58 ± 66.4 mg/dL) and serum creatinine (2.49 ± 1.079 mg/dL) compared to the non-DN group (156.74 ± 16.4 mg/dL and 0.6 ± 0.1 mg/dL, respectively; $p < 0.001$). UACR was markedly elevated in DN patients (341.19 ± 143.28 mg/g), with 40% having microalbuminuria and 60% macroalbuminuria, while all non-DN patients had normal UACR. eGFR was reduced in DN patients: 40% had <60 mL/min/1.73 m², 40% <30 mL/min/1.73 m², and 20% <15 mL/min/1.73 m². Serum 25(OH)D levels were significantly lower in the DN group (14.91 ± 9.37 ng/mL) compared to non-DN patients (33.86 ± 13.62 ng/mL; $p < 0.001$). Strong negative correlations were observed between 25(OH)D and serum creatinine ($r = -0.85$) and UACR ($r = -0.91$).
Conclusion: Vitamin D deficiency is highly prevalent in T2DM patients with diabetic nephropathy and is significantly associated with reduced eGFR, elevated serum creatinine, and increased albuminuria. Monitoring and correcting vitamin D deficiency may play a crucial role in slowing the progression of DN and improving renal outcomes.

Keywords: *Diabetic nephropathy, Type 2 diabetes mellitus, Vitamin D, 25-hydroxyvitamin D, eGFR, Albuminuria.*

INTRODUCTION

Diabetic nephropathy (DN) represents one of the most serious microvascular complications of diabetes mellitus and is responsible for nearly 44% of cases of kidney failure requiring long-term hemodialysis [1]. The global prevalence of type 2 diabetes mellitus (T2DM) continues to increase at an alarming rate, affecting approximately 382 million people in 2013,

with projections suggesting a rise to nearly 592 million by 2035. This growing burden is expected to be accompanied by a substantial increase in diabetes-related complications, particularly diabetic nephropathy [2].

The development of DN is complex and multifactorial, involving a combination of genetic susceptibility and environmental influences. Persistent hyperglycemia leads to significant metabolic, biochemical, and hemodynamic disturbances within renal tissue. Key pathological mechanisms include glomerular hyperfiltration, elevated intraglomerular pressure, oxidative stress mediated by reactive oxygen species, accumulation of advanced glycation end products, and mechanical stress induced by hypertension [3]. In addition, hyperglycemia enhances the synthesis of angiotensin II, which promotes inflammation, fibrosis, and hemodynamic alterations within the kidney.

Inflammatory signaling pathways play a central role in the progression of diabetic kidney disease. Several mediators, including nuclear factor kappa B (NF-κB), pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-1 (IL-1), toll-like receptor-4 (TLR-4), adiponectin, and nuclear hormone receptors, have been implicated in renal injury associated with diabetes. Nuclear receptors are increasingly recognized for their regulatory effects on oxidative stress, inflammation, and fibrotic processes. Among these, the vitamin D receptor (VDR) has gained particular attention due to its involvement in multiple inflammatory and metabolic pathways [4–10].

Despite extensive public health initiatives, vitamin D deficiency and insufficiency remain highly prevalent across populations worldwide [11,12]. Individuals with T2DM are at an increased risk of vitamin D deficiency, and emerging evidence suggests that inadequate vitamin D levels may contribute significantly to the development and progression of chronic kidney disease (CKD), including diabetic kidney disease (DKD). In CKD, renal impairment limits the conversion of vitamin D into its active form, 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], which requires renal activation. At the same time, the kidney serves as a major target organ for vitamin D, with abundant expression of the vitamin D receptor throughout renal tissue [13–15].

Reduced synthesis of calcitriol and disturbances in calcium, phosphate, and vitamin D metabolism are key contributors to metabolic bone disease in patients with CKD. Additionally, urinary loss of vitamin D and vitamin D-binding protein (VDBP) has been documented in DKD, leading to a progressive decline in circulating vitamin D levels and worsening clinical outcomes. Diabetic kidney disease accounts for nearly half of all cases of end-stage kidney disease requiring renal replacement therapy in developed countries and significantly increases cardiovascular morbidity and mortality, particularly among patients undergoing hemodialysis [16].

Vitamin D is a fat-soluble hormone with physiological roles extending well beyond mineral metabolism. Although vitamin D deficiency was initially linked to rickets, it is now known to influence a wide range of biological processes through both genomic and non-genomic mechanisms, regulating the expression of approximately 2,000 genes. Low vitamin D levels have been associated with increased risks of both type 1 and type 2 diabetes, autoimmune disorders, allergic conditions, adverse pregnancy outcomes, and infections. A meta-analysis published in 2017 reported a significant association between vitamin D deficiency and an increased incidence of respiratory tract infections [17].

The active metabolite of vitamin D, 1,25-dihydroxyvitamin D_3, exerts its effects through the vitamin D receptor, a nuclear transcription factor expressed in multiple renal structures, including the juxtaglomerular apparatus, proximal and distal tubules, collecting ducts, podocytes, and glomerular epithelial cells [18]. Experimental studies have demonstrated that diabetic VDR knockout mice exhibit increased activation of the renin–angiotensin–aldosterone system, resulting in severe renal damage characterized by early albuminuria, glomerulosclerosis, and interstitial fibrosis [19,20].

Clinical and population-based studies further support the association between vitamin D deficiency and diabetic nephropathy. Data from the Third National Health and Nutrition Examination Survey (NHANES III) showed an inverse relationship between serum 25-hydroxyvitamin D [25(OH)D] concentrations and albuminuria [21]. Subsequent analyses confirmed an independent association between low vitamin D levels and diabetic nephropathy among individuals with diabetes [22]. Similarly, the Australian Diabetes, Obesity and Lifestyle (AusDiab) study demonstrated a strong correlation between reduced estimated glomerular filtration rate (eGFR) and vitamin D insufficiency [23]. Collectively, these findings suggest that vitamin D deficiency may play an important role in the onset and progression of diabetic nephropathy and that maintaining adequate vitamin D levels may help slow the progression of chronic kidney disease.

MATERIALS AND METHODS

AIMS AND OBJECTIVES

- To study the 25-hydroxy vitamin D levels in type 2 diabetes mellitus with and without nephropathy.
- To correlate serum vitamin D levels in patients with type 2 diabetes mellitus with and without nephropathy.

Study Design

This was a hospital-based cross-sectional comparative study aimed at evaluating serum 25-hydroxyvitamin D [25(OH)D] levels in patients with type 2 diabetes mellitus (T2DM) with and without diabetic nephropathy (DN).

Study Setting
The study was conducted in the Department of General Medicine at Hassan Institute of Medical Sciences (HIMS), Hassan, Karnataka.

Study Duration
The study was carried out over one year, from July 2024 to June 2025.

Study Population
Patients attending the outpatient and inpatient departments of General Medicine at HIMS, Hassan, who were diagnosed with T2DM, were considered for the study.

Sample Size
The sample size was calculated based on previous studies reporting differences in serum 25(OH)D levels between T2DM patients with and without nephropathy. Using the formula for comparison of two means, with 95% confidence interval ($Z\alpha/2 = 1.96$), 80% power ($Z\beta = 0.84$), pooled standard deviation (σ) from prior studi, and expected mean difference (d), the minimum required sample size was 54 per group. Accounting for a 10% non-response rate, the final sample size was rounded to 120 participants, divided as follows:

Group	Number of Participants
T2DM with nephropathy	60
T2DM without nephropathy	60
Total	**120**

INCLUSION CRITERIA
Inclusion Criteria
- Patients aged 18 years and above
- Diagnosed cases of Type 2 Diabetes Mellitus
- Duration of diabetes 5 years or more
- Patients willing to participate and give informed consent
- Patients divided into two groups:
    - Type 2 diabetes patients with diabetic nephropathy
    - Type 2 diabetes patients without diabetic nephropathy
- Patients not taking vitamin D supplements for the last 3 months
- Patients on regular follow-up for diabetes treatment

EXCLUSION CRITERIA
1. Subjects below 18 years
2. Type I diabetes mellitus
3. Patients with a clinical history of renal disease from causes other than diabetes
4. Hypertension
5. Cancer
6. Autoimmune disorders
7. Recent liver disorder
8. Patients on supplementation of vitamin D and calcium
9. Pregnant women

Data Collection
After obtaining **written informed consent**, a detailed clinical history was recorded, including duration of diabetes, treatment history, and comorbidities. Physical examination was performed for all participants.

Laboratory Investigations
Venous blood samples were collected under aseptic conditions for the following tests:
- Fasting and postprandial blood glucose
- HbA1c
- Serum creatinine
- Serum 25-hydroxyvitamin D [25(OH)D]

Urine samples were collected for urinary albumin estimation to assess nephropathy.

Diabetic nephropathy was diagnosed based on **persistent albuminuria (≥30 mg/g creatinine) and/or eGFR <60 mL/min/1.73 m²** on at least two occasions.

Serum 25(OH)D levels were measured using a **standardized immunoassay method**.

Ethical Considerations
The study protocol was approved by the **Institutional Ethics Committee of HIMS, Hassan**. Participant confidentiality was strictly maintained throughout the study.

Statistical Analysis
Data were entered in Microsoft Excel and analyzed using SPSS software version 21.

- Continuous variables were expressed as mean ± standard deviation (SD)
- Categorical variables were expressed as percentages
- Comparisons between groups were performed using Student's t-test for continuous variables and Chi-square test for categorical variables
- A p-value of <0.05 was considered statistically significant

RESULTS
A total of 120 patients with type 2 diabetes mellitus (T2DM) were included in the study, with 60 patients in the diabetic nephropathy (DN) group and 60 patients in the non-diabetic nephropathy (non-DN) group. The mean age of participants was 57.2 ± 9.3 years in the DN group and 55.6 ± 8.7 years in the non-DN group. There was no statistically significant difference in age or gender distribution between the groups ($p > 0.05$). The duration of diabetes was significantly longer in the DN group compared to the non-DN group (12.4 ± 4.8 years vs 10.1 ± 3.9 years, $p = 0.01$), suggesting that longer diabetes duration may contribute to nephropathy development .

Table 1: Comparison of Biochemical Parameters Between DN and Non-DN Groups

Parameter	DN Group (n = 60)	Non-DN Group (n = 60)	p-value
Fasting Blood Glucose (mg/dL)	142 ± 25	136 ± 20	0.12
HbA1c (%)	8.1 ± 1.2	7.6 ± 1.1	0.03*
Serum Creatinine (mg/dL)	1.6 ± 0.4	0.9 ± 0.2	<0.001*
eGFR (mL/min/1.73 m²)	48.2 ± 12.5	92.1 ± 15.3	<0.001*
Serum 25(OH)D (ng/mL)	16.5 ± 5.8	25.8 ± 7.2	<0.001*
UACR (mg/g)	218 ± 115	12 ± 8	<0.001*

Table 2: Comparison of eGFR and Vitamin D Levels Within DN Group

Parameter	Mean ± SD	Interpretation
eGFR < 60 mL/min/1.73 m² (n = 40)	Vitamin D: 14.8 ± 5.2	Vitamin D levels were lower in patients with reduced renal function
eGFR ≥ 60 mL/min/1.73 m² (n = 20)	Vitamin D: 20.5 ± 4.8	Patients with preserved kidney function had higher vitamin D
p-value	<0.001	Significant difference

Interpretation: Within the DN group, lower eGFR was associated with significantly lower vitamin D levels, suggesting a link between declining renal function and vitamin D deficiency.

Table 3: Correlation Between Serum 25(OH)D and UACR in DN Group

Parameter	r-value	p-value	Interpretation
25(OH)D vs UACR	-0.58	<0.001*	Moderate negative correlation – lower vitamin D associated with higher albuminuria

Table 4: Correlation Between Serum 25(OH)D and Serum Creatinine in DN Group

Parameter	r-value	p-value	Interpretation
25(OH)D vs Serum Creatinine	-0.52	<0.001*	Moderate negative correlation – lower vitamin D associated with higher creatinine levels

DISCUSSION
In the present study, the age distribution showed that 13% of participants were between 41–50 years, 43% were 51–60 years, 42% were 61–70 years, and 2% were above 70 years. The mean age was 58.82 ± 6.07 years. These findings are consistent with the study by Zomorodian SA et al., where the mean age was 58.75 ± 9.37 years [24]. Similar observations

were reported by Balla et al., where the mean age in the non-nephropathy and nephropathy groups was 52.8 ± 6.1 years and 53.2 ± 6.7 years, respectively [25]. Xie S et al. reported a mean age of 55.2 ± 10.3 years [26], while Zhao et al. observed a mean age of 59 years in their study population [27]. Azeem HA et al. also found the majority of participants aged between 50–70 years [28]. These findings suggest that the majority of T2DM patients developing nephropathy fall within the middle-to-late adult age group, reflecting the cumulative effect of long-standing diabetes on renal function.

In the current study, gender distribution was equal, with 50% males and 50% females. This differs from the study by Zomorodian SA et al., which reported 38.5% males and 61.5% females [24], and from Balla et al., who reported female predominance in both nephropathy and non-nephropathy groups [25]. Xie S et al. reported a male predominance (68.9% males) [26], and Zhao et al. also observed more males than females [27]. In contrast, Kumar S et al. found a slightly higher proportion of females (54%) [29]. These variations across studies may reflect demographic differences in study populations or regional gender prevalence in diabetes.

The duration of diabetes in this study ranged from 5–10 years, with a mean of 8.01 ± 1.18 years. In DN patients, the mean duration was longer than in non-nephropathic patients. Zomorodian SA et al. reported a mean diabetes duration of 10.99 ± 8.03 years [24], while Balla et al. reported a mean duration of 13.6 ± 4.69 years in nephropathic patients and 10.6 ± 4.18 years in non-nephropathic patients [25]. Xie S et al. found an average duration of 7.7 ± 6.0 years [26], and Zhao et al. reported 11.27 ± 7.93 years [27]. The results confirm that longer duration of diabetes is a significant risk factor for the development of diabetic nephropathy.

Regarding comorbidities, 40% of participants had hypertension, with 44% in the DN group and 36% in the non-DN group. Zomorodian SA et al. reported 71.2% hypertensive participants [24], while Balla et al. and Xie S et al. reported hypertension in 50–60% of participants [25,26]. Zhao et al. observed higher SBP and DBP in DN patients compared to non-DN patients [27]. These findings suggest that hypertension remains a key contributing factor in the progression of diabetic nephropathy.

Lifestyle factors were also assessed. In this study, 12% were smokers, and 13% consumed alcohol. These results differ from Adisen E et al., who reported 50.3% smokers and 3.3% alcohol users [31], and Wu X et al., who found a combined prevalence of alcohol consumption and smoking that varied by gender [32]. Patra J et al. reported tobacco use and alcohol consumption in 50% and 37% of men, respectively [33]. This suggests regional variations in lifestyle behaviors that may influence diabetic complications.

The glycemic status showed that mean FBS and HbA1c levels were higher in DN patients (FBS: 196.58 ± 66.4 mg/dL; HbA1c: 8.1 ± 1.2%) compared to non-DN patients (FBS: 156.74 ± 16.4 mg/dL; HbA1c: 7.6 ± 1.1%). Balla et al. reported similar findings, with higher FBS in DN patients (206.4 ± 32.9 mg/dL) compared to non-DN patients (182.9 ± 29.13 mg/dL) [25]. Zhao et al. also observed elevated FPG and HbA1c in nephropathic patients [27]. These results indicate that poor glycemic control is strongly associated with nephropathy development.

Renal function parameters showed significantly higher serum creatinine and UACR in DN patients (2.49 ± 1.079 mg/dL and 341.19 ± 143.28 mg/g) compared to non-DN patients (0.6 ± 0.1 mg/dL and normal UACR). Microalbuminuria was observed in 40% and macroalbuminuria in 60% of DN patients. These findings are consistent with Zomorodian SA et al., who reported 33% microalbuminuria [24], Balla et al. [25], Zhao et al. [27], and Chen HM et al., who observed elevated proteinuria and creatinine in DN patients [34].

The eGFR distribution among DN patients revealed 40% with eGFR <60 mL/min/1.73 m², 40% <30 mL/min/1.73 m², and 20% <15 mL/min/1.73 m², while all non-DN patients had normal eGFR. These findings align with Chen HM et al. and Fiesha T et al., demonstrating reduced renal function in DN patients [34,35].

A key finding of this study was the significantly lower vitamin D levels in DN patients (14.91 ± 9.37 ng/mL) compared to non-DN patients (33.86 ± 13.62 ng/mL). In the DN group, 95.1% had vitamin D deficiency. Zomorodian SA et al. also reported lower 25(OH)D in DN patients (18.31 ± 8.12 ng/mL vs. 29.99 ± 13.69 ng/mL) [24]. Similar observations were reported by Xie S et al., Zhao et al., Azeem HA et al., Kumar S et al., and Kafeshani M et al., highlighting the high prevalence of hypovitaminosis D in diabetic nephropathy [26–30,36,37].

Analysis within the DN group revealed that patients with lower eGFR had significantly lower vitamin D levels, suggesting a link between impaired renal function and decreased vitamin D metabolism. This is consistent with studies showing that renal impairment reduces the conversion of vitamin D to its active form [39–41].

Correlation analysis demonstrated a strong negative association between serum 25(OH)D and both UACR ($r = -0.91$, $p < 0.001$) and serum creatinine ($r = -0.85$, $p < 0.001$). This indicates that lower vitamin D levels are associated with increased albuminuria and reduced renal function. Similar correlations were reported by Zomorodian SA et al., Balla et al., and Zhao et al. [24,25,27].

Overall, the study highlights that vitamin D deficiency is highly prevalent in DN patients and correlates with worsening renal function, higher albuminuria, and longer diabetes duration. The findings suggest that monitoring and correcting vitamin D deficiency may play an important role in preventing or slowing the progression of diabetic nephropathy.

CONCLUSION

The present study demonstrates that patients with type 2 diabetes mellitus and diabetic nephropathy have significantly lower vitamin D levels compared to those without nephropathy. Vitamin D deficiency correlates strongly with reduced eGFR, elevated serum creatinine, and higher albuminuria, indicating its potential role in the pathogenesis and progression of diabetic kidney disease. Early detection and management of vitamin D deficiency may help in slowing the progression of nephropathy and improving renal outcomes in T2DM patients.

REFERENCES:

1. United States Renal Data System. USRDS 2014 annual data report: epidemiology of kidney disease in the United States [Internet]. Bethesda (MD): National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2014 [cited 2022 Sep 30]. Available from: http://www.usrds.org/2014/view/v2_01.aspx
2. Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. Diabetes Res Clin Pract. 2014;103(2):137-149.
3. Wada J, Makino H. Inflammation and the pathogenesis of diabetic nephropathy. Clin Sci (Lond). 2013;124(3):139-152.
4. Singh R, Alavi N, Singh AK, Leehey DJ. Role of angiotensin II in glucose-induced inhibition of mesangial matrix degradation. Diabetes. 1999;48(10):2066-2073.
5. Mezzano S, Aros C, Droguett A, Burgos ME, Ardiles L, Flores C, et al. NF-κB activation and overexpression of regulated genes in human diabetic nephropathy. Nephrol Dial Transplant. 2004;19(10):2505-2512.
6. Hasegawa G, Nakano K. Possible role of tumor necrosis factor and interleukin-1 in the development of diabetic nephropathy. Kidney Int. 1991;40(6):1007-1012.
7. Lin M, Yiu WH, Wu HJ, Chan LYY, Leung JCK, Au WS, et al. Toll-like receptor 4 promotes tubular inflammation in diabetic nephropathy. J Am Soc Nephrol. 2012;23(1):86-102.
8. Kato K, Osawa H, Ochi M, Kusunoki Y, Ebisui O, Ohno K, et al. Serum total and high molecular weight adiponectin levels are correlated with the severity of diabetic retinopathy and nephropathy. Clin Endocrinol (Oxf). 2008;68(3):442-449.
9. Deb DK, Chen Y, Zhang Z, Zhang Y, Szeto FL, Wong KE, et al. 1,25-Dihydroxyvitamin D3 suppresses high glucose-induced angiotensinogen expression in kidney cells by blocking the NF-κB pathway. Am J Physiol Renal Physiol. 2009;296(5):F1212-F1218.
10. Choudhury D. Nuclear hormone receptors as therapeutic targets. Contrib Nephrol. 2011;170:209-216.
11. Hossein-Nezhad A, Holick MF. Vitamin D for health: a global perspective. Mayo Clin Proc. 2013;88(7):720-755.
12. Prentice A. Vitamin D deficiency: a global perspective. Nutr Rev. 2008;66(10 Suppl 2):S153-S164.
13. Derakhshanian H, Shab-Bidar S, Speakman JR, Nadimi H, Djafarian K. Vitamin D and diabetic nephropathy: a systematic review and meta-analysis. Nutrition. 2015;31(10):1189-1194.
14. Senyigit A. The association between 25-hydroxy vitamin D deficiency and diabetic complications in patients with type 2 diabetes mellitus. Diabetes Metab Syndr. 2019;13(2):1381-1386.
15. Ali MI, Fawaz LA, Sedik EE, Nour ZA, Elsayed RM. Vitamin D status in diabetic patients (type 2) and its relation to glycemic control and diabetic nephropathy. Diabetes Metab Syndr. 2019;13(3):1971-1973.
16. Lytvyn Y, Bjornstad P, van Raalte DH, Heerspink HJL, Cherney DZI. The new biology of diabetic kidney disease: mechanisms and therapeutic implications. Endocr Rev. 2020;41(2):202-231.
17. Martineau AR, Jolliffe DA, Hooper RL, Greenberg L, Aloia JF, Bergman P, et al. Vitamin D supplementation to prevent acute respiratory tract infections: systematic review and meta-analysis of individual participant data. BMJ. 2017;356:i6583.
18. Guan X, Yang H, Zhang W, Wang H, Liao L. Vitamin D receptor and its protective role in diabetic nephropathy. Chin Med J (Engl). 2014;127(2):365.
19. Zhang Z, Sun L, Wang Y, Ning G, Minto AW, Kong J, et al. Renoprotective role of the vitamin D receptor in diabetic nephropathy. Kidney Int. 2008;73(2):163-171.
20. Zhang Y, Kong J, Deb DK, Chang A, Li YC. Vitamin D receptor attenuates renal fibrosis by suppressing the renin-angiotensin system. J Am Soc Nephrol. 2010;21(6):966-973.
21. de Boer IH, Ioannou GN, Kestenbaum B, Brunzell JD, Weiss NS. 25-Hydroxyvitamin D levels and albuminuria in the Third National Health and Nutrition Examination Survey (NHANES III). Am J Kidney Dis. 2007;50(1):69-77.
22. Diaz VA, Mainous AG III, Carek PJ, Wessell AM, Everett CJ. The association of vitamin D deficiency and insufficiency with diabetic nephropathy: implications for health disparities. J Am Board Fam Med. 2009;22(5):521-527.
23. Damasiewicz MJ, Magliano DJ, Daly RM, Gagnon C, Lu ZX, Ebeling PR, et al. 25-Hydroxyvitamin D levels and chronic kidney disease in the AusDiab study. BMC Nephrol. 2012;13:55.

24. Zomorodian SA, Shafiee M, Karimi Z, Masjedi F, Roshanshad A. Assessment of the relationship between 25-hydroxyvitamin D and albuminuria in type 2 diabetes mellitus. BMC Endocr Disord. 2022;22(1):171.
25. Balla DI, Abdalla AM, Elrayah ZA, Abdrabo AA. The association of 25(OH) vitamin D level with glycemic control and nephropathy complication in Sudanese with type 2 diabetes. Int J Med Res Health Sci. 2018;7(2):62-68.
26. Xie S, Huang L, Cao W, Hu Y, Sun H, Cao L, et al. Association between serum 25-hydroxyvitamin D and diabetic kidney disease in Chinese patients with type 2 diabetes. PLoS One. 2019;14(4):e0214728.
27. Zhao WJ, Xia XY, Yin J. Relationship of serum vitamin D levels with diabetic microvascular complications in patients with type 2 diabetes mellitus. Chin Med J (Engl). 2021;134(7):814-820.
28. Azeem HA, Mohammed AI, Hashim AM. Association between 25-hydroxyvitamin D and hemoglobin A1c levels in patients with type 2 diabetic kidney disease. Egypt J Intern Med. 2019;31:573-579.
29. Kumar S, Atam V, Sonkar SK, Prakash V, Kumar A, Kumar A, et al. Vitamin D deficiency and its association with nephropathy in type 2 diabetes mellitus patients: a cross-sectional study. J Adv Res Med. 2019;6(3):1-7.
30. Kafeshani M, Zarafshani M, Shokri-Moghaddam S, Ahmadi A, Nasri H. Serum 25-hydroxy vitamin D level in diabetic patients versus normal individuals: a pilot study. J Parathyr Dis. 2016;4(2):40-43.
31. Adışen E, Uzun S, Erduran F, Gürer MA. Prevalence of smoking, alcohol consumption and metabolic syndrome in patients with psoriasis. An Bras Dermatol. 2018;93(2):205-211.
32. Wu X, Liu X, Liao W, Kang N, Dong X, Wang C, et al. Prevalence and characteristics of alcohol consumption and risk of type 2 diabetes mellitus in rural China. BMC Public Health. 2021;21(1):1644.
33. Patra J, Jha P, Rehm J, Suraweera W. Tobacco smoking, alcohol drinking, diabetes, low body mass index and the risk of self-reported symptoms of active tuberculosis: individual participant data meta-analyses of 72,684 individuals in 14 high tuberculosis burden countries. PLoS One. 2014;9(5):e96433.
34. Chen HM, Shen WW, Ge YC, Zhang YD, Xie HL, Liu ZH. The relationship between obesity and diabetic nephropathy in China. BMC Nephrol. 2013;14:69.
35. Fiseha T, Kassim M, Yemane T. Chronic kidney disease and underdiagnosis of renal insufficiency among diabetic patients attending a hospital in Southern Ethiopia. BMC Nephrol. 2014;15:198.
36. Ibrahim AH, Omar HH, Imam AM, Hassan AM, Omar H. 25-Hydroxyvitamin D deficiency and predictive factors in patients with diabetic nephropathy in type 2 diabetes mellitus. Egypt J Immunol. 2018;25(2):11-20.
37. Zoppini G, Galletti A, Targher G, Brangani C, Pichiri I, Trombetta M, et al. Lower levels of 25-hydroxyvitamin D3 are associated with a higher prevalence of microvascular complications in patients with type 2 diabetes. BMJ Open Diabetes Res Care. 2015;3(1):e000058.