Correlation of Urinary Protein & Serum Uric Acid with Serum 25-Hydroxyvitamin D Levels in Pre-Dialysis Chronic Kidney Disease Patients.

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ABSTRACT:

Objective: To evaluate the correlation between urinary protein and serum uric acid levels with serum 25-hydroxyvitamin D concentrations in patients with chronic kidney disease who are not yet on dialysis.

Methodology: This is a cross-sectional study performed in the Department of Nephrology at Shaikh Zayed Hospital, Lahore from 10th December 2025 to 10th June 2026. Using non-probability consecutive sampling, 87 pre-dialysis patients with chronic kidney disease were selected. Enrolled were patients between the ages of 18 and 60, with an estimated glomerular filtration rate (eGFR) of 15 and 60 mL/min/1.73 m². Structured proforma used for recording demographic, clinical and laboratory data. Blood and urine samples were subjected to usual hospital procedures for evaluation.

Results: The average age was 30.59 ±8.71 years. Most patients were females and at Stage IV of CKD. The median for the daily urinary protein concentration was 162.50 mg. The average serum uric acid level was 5.30 ± 1.11 mg/dL. The mean serum 25-hydroxyvitamin D concentration was 16.58 ±5.98 ng/mL. Vitamin D exhibited a substantial negative correlation with urinary protein ($r = -0.691$, $p < 0.001$). Additionally, serum uric acid demonstrated a substantial negative correlation ($r = -0.675$, $p < 0.001$).

Conclusion: In pre-dialysis CKD patients, S-25-HD levels were decreased when urinary protein and serum uric acid were elevated. Early risk assessment may be facilitated by these markers.

Keywords: Chronic kidney disease; Pre-dialysis; Proteinuria; Uric acid; Vitamin D.

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Introduction:

Chronic kidney disease (CKD) is a gradual loss of kidney function over a long period. It poses a serious public health challenge. Having an impact on over 10% of the world's population. This implies that CKD may affect more than 800 million individuals.[1] The disease of the kidney is also becoming a more prevalent cause of mortality. According to recent global research, it is currently the ninth most significant risk factor for fatality on a global scale.[2]

Recent research has improved our understanding of CKD and biochemical changes that happen during the disease. There has been an explosion of research on the use of vitamin D in the past couple of years, and vitamin D deficiencies have been associated with undesirable cardiac and renal outcomes.[3] Results from trials/evaluations have been mixed. Another common feature of CKD is hyperuricemia, which might worsen the course of the disease. A recent meta-analysis, however, indicates that some controversy exists with regards to the efficacy of uric acid lowering and the evidence is not uniform.[4]

CKD is also a significant health issue in Pakistan. According to a recent study conducted in Lahore, approximately one-fourth of the high-risk urban population had CKD.[5] Diabetes and hypertension were found to be significantly associated with moderate to severe CKD in another study conducted in Peshawar. In addition, it demonstrated disparities between urban and rural patients.[6] These results show that chronic kidney disease (CKD) is increasing in Pakistan and a lack of local data exists.

The aim of this investigation is to determine the association of S-25-HD with urinary protein and serum uric acid in pre-dialysis CKD patients[7] because these markers could be used to early assess the risk and better monitor the severity of the disease. The importance of this investigation is that it is an under-researched topic in the local context especially in Pakistan. The findings could help doctors to identify high-risk patients at an earlier stage and to improve future management.

Methodology:

The Department of Nephrology at Shaikh Zayed Hospital, Lahore, performed this cross-sectional research study. The study was conducted over a period of six months from 10 December 2025 to 10 June 2026, following ethical approval

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The sample size estimation was guided by the study of Fayed et al.[8], in which urine albumin and serum uric acid were identified as crucial determinants of serum 25-hydroxyvitamin D (S-25-HD) levels in pre-dialysis chronic kidney disease (CKD) patients. Using these variables as the basis for effect size, the calculation was performed at a power of 80% and a 5% level of significance (α = 0.05). The minimum required sample size was determined to be approximately 85 patients. To ensure adequacy and account for potential data loss, the final study population included 87 patients. Patients of either gender aged 18-60 years diagnosed as chronic kidney disease (CKD) having estimated glomerular filtration rate (eGFR) between 15-60 mL/1.73m2 but yet not on dialysis (pre-dialysis CKD) were recruited from the Department of Nephrology, Shaikh Zayed Hospital, Lahore. Patients on dialysis, diagnosed of acute-on-chronic renal failure, history of renal transplantation, having concurrent liver disease, active malignancy or osteoporosis and those on vitamin D supplementation were excluded from the study. For this study purpose CKD was defined as kidney injury or reduced function persisting for more than 3 months, pre-dialysis referred to patients with CKD who had not yet started dialysis. Vitamin D deficiency was determined according to the hospital laboratory's reference range, while Hyperuricemia was defined as serum uric acid levels above the upper limit of normal. Data Collection Demographic and clinical information was recorded using a structured proforma. Confidentiality of patient information was strictly maintained. Urine and blood samples were collected and analysed in the hospital laboratory following standard protocols. The amount of protein in urine was used to measure kidney injury (urinary protein), while blood uric acid concentration (serum uric acid) was used as the measure of uric acid concentration. S-25-HD was the indicator of status of vitamin D. Medications were administered under consultant supervision in accordance with departmental guidelines.

Data were entered and analysed using SPSS version 25. Depending on the distribution, quantitative variables were expressed as mean ± standard deviation or as median with interquartile range, while categorical variables were summarized as frequency and percentage. Normality of quantitative data was assessed using the Shapiro-Wilk test. The Pearson correlation coefficient was applied to evaluate the relationship between serum 25 hydroxy vitamin D (S 25 OH D) levels and urinary protein excretion as well as serum uric acid concentration. To identify independent predictors of S 25 OH D levels, a multivariable linear regression model was employed. A p value <0.05 was considered statistically significant.

Results:

The total of patients included in the study were 87 cases of pre-dialysis CKD. The average age was 30.59 ±8.71 years. There were more females than males. Most patients were in the chronic kidney disease (CKD) stage 4. There were only a few people in Stage III. The mean serum creatinine level was 3.01 ±0.57 mg/dl. The average eGFR was 23.39 ±6.68 mL/min/1.73 m². Median Urinary protein concentration per day was 162.50 mg. The mean serum uric acid level was 5.30 ±1.11 mg/dL. The average 25(OH)D level was 16.58 ±5.98 ng/mL. Most common co-morbidities were diabetes and hypertension. Most patients were treated with Angiotensin-Converting Enzyme Inhibitors / Angiotensin II Receptor Blockers (ACEI/ARB).

Table No 1: Demographic and clinical and biochemical baseline features of Pre-Dialysis CKD patients (n = 87).

Variable	Value
Age, years	30.59 ±8.71
Male sex, n (%)	34 (39.1)
Female sex, n (%)	53 (60.9)
Weight, kg	61.29 ±8.48
Height, cm	162.52 ±8.39
BMI, kg/m²	23.18 ± 2.62
Systolic blood pressure, mmHg	119.66 ±11.38
Diastolic blood pressure, mmHg	74.22 ±7.39
Heart rate, bpm	76.93 ±6.49
CKD Stage III, n (%)	15 (17.2)
CKD Stage IV, n (%)	72 (82.8)
Serum creatinine, mg/dL	3.01 ±0.57
eGFR, mL/min/1.73 m²	23.39 ±6.68
Urine output, mL/day	1374.25 ±274.70
Urinary proteins, mg/day	162.50 (79.90-357.95)
Serum uric acid, mg/dL	5.30 ±1.11
S-25-HD, ng/mL	16.58 ±5.98
Diabetes mellitus, n (%)	29 (33.3)
Hypertension, n (%)	32 (36.8)
Cardiovascular disease, n (%)	7 (8.0)
Hyperuricemia, n (%)	19 (21.8)
Immunocompromised, n (%)	2 (2.3)
Autoimmune disease, n (%)	4 (4.6)
Hepatitis C, n (%)	5 (5.7)
Chronic lung disease, n (%)	2 (2.3)
Peripheral arterial disease, n (%)	1 (1.1)
ACEI/ARB use, n (%)	84 (96.6)
Diuretic use, n (%)	38 (43.7)
Statin use, n (%)	15 (17.2)
Antihypertensive use, n (%)	32 (36.8)
Insulin/oral hypoglycaemic use, n (%)	29 (33.3)

Table 2 illustrates the comparison of S-25-HD, serum uric acid, and urinary protein levels between patients with CKD Stage III and Stage IV. In Stage IV patients, urinary protein levels were marginally higher than those in Stage III patients. Additionally, Stage IV patients exhibited slightly elevated serum uric acid levels. S-25-HD levels were marginally elevated in Stage IV patients. Nevertheless, these discrepancies were not statistically significant. This implies that the variables were comparable between the two CKD stages in this dataset.

Table No 2: Urine protein, serum uric acid and S-25-HD levels according to the stages of chronic kidney disease.

Variable	Stage III (n = 15)	Stage IV (n = 72)	p-value
Urinary proteins, mg/day	127.60 (88.45-308.55)	164.65 (75.78-363.18)	0.933
Serum uric acid, mg/dL	5.10 ± 0.97	5.34 ± 1.14	0.420
S-25-HD, ng/mL	15.74 ± 6.79	16.75 ± 5.83	0.596

In Table 3, it can be seen that both urinary protein and serum uric acid were linked to lower amounts of 25-hydroxyvitamin D. In other words, vitamin D levels went down when urinary protein levels went up. And the same thing was seen with serum uric acid. There was a lot of evidence for both relationships. Urinary protein and blood uric acid were still good predictors of S-25-HD levels in multivariable regression analysis. These results show that both factors were linked to lower vitamin D levels in CKD patients before dialysis.

Table No 3: Correlation and multivariable regression analysis of association of urinary protein, serum uric acid and 25 hydroxy vitamin D in pre dialysis chronic kidney disease patients (n = 87).

Variable	Correlation with serum 25(OH) D (r)	p-value	Regression Coefficient (Multivariable) (B)	95% CI	p-value
Urinary proteins, mg/day	-0.691	<0.001	-0.016	-0.023 to -0.009	<0.001
Serum uric acid, mg/dL	-0.675	<0.001	-2.118	-3.128 to -1.109	<0.001

Figure 1A: Protein in the urine and vitamin D Vitamin D and protein in the urine are shown in the first figure. The dots on the line decrease from left to right. This indicates that the friendship is not good. It indicates that if the body contains low vitamin D levels, there will be high levels of protein in the urine. Urea that has a lot of protein in it means that your kidneys are hurt. As vitamin D levels increase so does the concentration of protein decrease in the urine. Vitamin D and Serum Uric Acid (Figure 1B) Vitamin D and uric acid levels in the blood are shown in the second figure. The slope of this line is also down. This is yet another bad situation. This means that people who don't get enough vitamin D have a lot more uric acid in their bodies. Having too much uric acid can be bad for your health. Just like in the first picture, uric acid levels drop to safe levels as vitamin D levels rise.

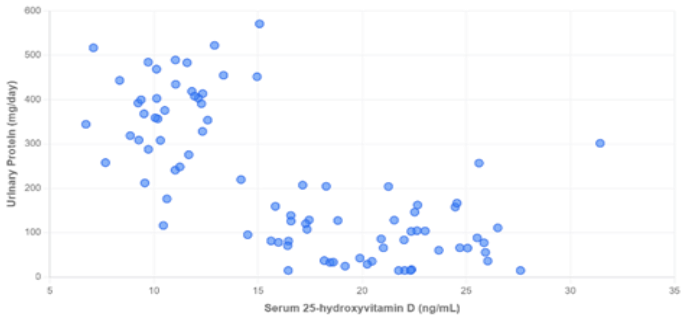


Figure 1A: Relationship of S-25-HD levels with urinary protein in pre-dialysis chronic kidney disease patients.

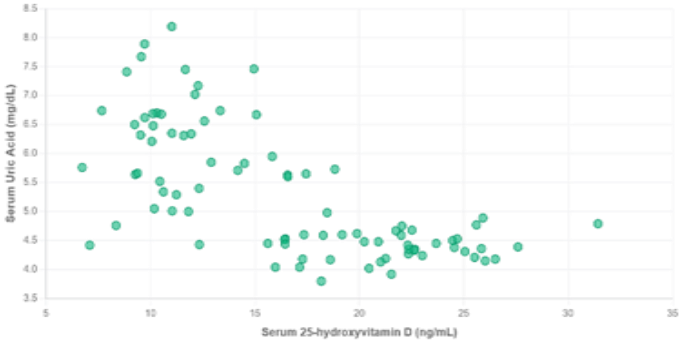


Figure 1B: In patients with pre-dialysis chronic kidney disease there was a positive correlation between serum uric acid and S-25-HD.

Discussion:

This study's major finding was that, in pre-dialysis CKD patients, there was an inverse relationship between serum 25(OH)D and urinary protein and serum uric acid (SUA). Biologically, this discovery is plausible. Chronic kidney disease (CKD) causes vitamin D levels to fall as kidney disease gets worse. Normal vitamin D metabolism and processing are also impaired by kidney disease.[9] Proteinuria may further reduce vitamin D levels by causing the loss of vitamin D-binding protein and related metabolites in urine.[10] The activation of detrimental renal pathways, oxidative stress, inflammation, and endothelial dysfunction may exacerbate kidney injury in the presence of high uric acid. This analysis gives some new data from Lahore on local level as elevated serum uric acid has been found to be associated with low vitamin D levels.[11,12] We have also supported our findings by the evidence of the investigations carried out in Pakistan at the national level at Karachi. Vitamin D deficiency is common in patients with CKD before dialysis and most of the patients improved after vitamin D treatment as Memon et al. showing. The study does not focus its research on urinary protein and uric acid as important correlates but it does support the significant vitamin D deficiency seen in patients in our clinic. In addition, Razzaque et al. found that vitamin D deficiency increased in severity with the progression of the disease from CKD stage 3 to 5. This matches the results that we found in the direction, we saw poorer kidney disease with lower vitamin D.[13,14]

At the regional level, Singh et al.[15] from India studied CKD 3b-5 non-dialysis patients. They found that serum 25-OH vitamin D and urine protein were important predictors of cardiac complications. Their outcome was different from ours, but the study still supports the clinical importance of both vitamin D and proteinuria in non-dialysis CKD. Another regional study by Huang et al. showed that lower 25-hydroxyvitamin D predicted albuminuria progression and decline in kidney function in diabetic kidney disease. This is close to our result because both studies link lower vitamin D with heavier urinary protein loss and worse renal status.[16]

Internationally, Duan et al. showed that those with more severe vascular lesions were more proteinuric, had lower 25(OH)D levels and poorer renal outcomes. However, there was not consistent outcome measurement effects, as seen in our finding that lower vitamin D was associated with more urinary protein.[17] Hsu and colleagues[18] in the CRIC study reported only weak links between 25(OH)D and select cardiovascular markers and no clear link with incident cardiovascular disease. This indicates that vitamin D could be a useful proxy for disease severity, but that vitamin D is not necessarily tightly coupled to hard clinical outcomes.

Interventional work has also given mixed results. Krummel et al.[19] reported a small but significant reduction of albuminuria following high dose native vitamin D. However, Karalliedde et al.[20] reported that calcitriol did not reduce arterial stiffness in type 2 diabetic patients with stage 3 CKD. Galassi et al also found that low active vitamin D was associated with poor renal function in CKD. Basile and coworkers also reported that vitamin D correction may be beneficial for some biochemical or renal markers but not for all of the outcomes in CKD. This also suggests that vitamin D is not just a marker of bone disease, but also may be a marker of renal disease.[21] Obaid et al. also pointed to vitamin D receptor signaling in diabetic kidney disease and progressive proteinuria.[22]

The uric acid finding of our study is also important. Luo et al.[23] showed in a recent meta-analysis that uric acid-lowering therapy may slow CKD progression in patients with asymptomatic hyperuricemia. Yang et al.[24] found that febuxostat was linked with slower decline in eGFR and lower albuminuria. Sharbaf et al.[25] also reported that allopurinol slowed eGFR decline in CKD patients with hyperuricemia. These studies do not prove that uric acid directly lowers vitamin D, but they do support the idea that high uric acid is part of a harmful renal environment. New biomarker work also shows that disturbed mineral metabolism and proteinuria move together during CKD progression, which supports the pattern seen in our study.[26]

Study Limitations:

This study had several limitations. First, it was conducted at a single center, which may restrict the generalizability of the findings. Second, the sample size was relatively small, limiting statistical power. Third, the cross-sectional design precluded the establishment of a cause-and-effect relationship, as all variables were measured at a single time point. Potential confounding factors may also have influenced the results. Serum vitamin D levels, for instance, can be affected by dietary intake, seasonal variation, sunlight exposure, and disease duration, none of which were fully accounted for in this study.

Future Directions:

Further research should include larger, multicenter cohorts to enhance external validity. Prospective studies are needed to determine whether elevated urinary protein and serum uric acid levels truly contribute to declining vitamin D status over time. In addition, interventional trials should assess whether correcting vitamin D deficiency or reducing uric acid levels improves renal outcomes. Future work should also incorporate repeated measures and broader markers of CKD-mineral bone disorder.

Conclusion:

In patients with pre-dialysis chronic kidney disease, serum 25-hydroxy vitamin D [25(OH)D] levels demonstrated an inverse association with both urinary protein excretion and serum uric acid concentration. These parameters may serve as early indicators for assessing the risk of vitamin D deficiency and related complications in this population.

References:

1. Kovesdy CP. Epidemiology of chronic kidney disease: an update 2022. Kidney Int Suppl (2011). 2022 Apr;12 (1):7-11. doi: 10.1016/j.kisu.2021.11.003. Epub 2022 Mar 18. PMID: 35529086; PMCID: PMC9073222.
2. Francis A, Harhay MN, Ong ACM, Tummalapalli SL, Ortiz A, et al. Chronic kidney disease and the global public health agenda: an international consensus. Nat Rev Nephrol. 2024 Jul;20(7):473-485. doi: 10.1038/s41581-024-00820-6. Epub 2024 Apr 3. PMID: 38570631.
3. Vervloet MG, Hsu S, de Boer IH. Vitamin D supplementation in people with chronic kidney disease. Kidney Int. 2023 Oct;104(4):698-706. doi: 10.1016/j.kint.2023.07.010. Epub 2023 Aug 2. PMID: 37541585.
4. Yeung WG, Toussaint ND, Badve SV. Vitamin D therapy in chronic kidney disease: a critical appraisal of clinical trial evidence. Clin Kidney J. 2024 Jul (8):sfae227. doi: 10.1093/ckj/sfae227. PMID: 39119524; PMCID: PMC11306979.
5. Casanova AG, Morales AI, Vicente-Vicente L, López-Hernández FJ. Effect of uric acid reduction on chronic kidney disease. Systematic review and meta-analysis. Front Pharmacol. 2024 Mar 26;15:1373258. doi: 10.3389/fphar.2024.1373258. PMID: 38601468; PMCID: PMC11005459.
6. Khan A, Cheema MF, Fatima R, Cheema SS, Butt Z, et al. Prevalence of Chronic Kidney Disease in a High-Risk Population in Urban Lahore, Pakistan: A Cross-sectional Study. Cureus. 2024 Jun 27;16(6):e63296. doi: 10.7759/cureus.63296. PMID: 39077231; PMCID: PMC11284504.
7. Ali J, Shah S, Nadeem M, Mahmood A, Ahmad U. A Comparative Study of the Epidemiology and Risk Factors of Chronic Kidney Disease Among Rural and Urban Residents in Peshawar, Pakistan. Cureus. 2024 Jul 10;16(7):e64215. doi: 10.7759/cureus.64215. PMID: 39131032; PMCID: PMC11310797.
8. Fayed A, El Nokeety MM, Heikal AA, Sadek KM, Hammad H, et al. Urine albumin and serum uric acid are important determinants of serum 25 hydroxyvitamin D level in pre-dialysis chronic kidney disease patients. Ren Fail. 2019 Nov;41(1):540-546. doi: 10.1080/0886022X.2018.1563552. PMID: 31234687; PMCID: PMC6598477.
9. Alfieri C, Molinari P, Vettoretti S, Fusaro M, Bover J, et al. Native vitamin D in CKD and renal transplantation:

meaning and rationale for its supplementation. J Nephrol. 2024 Jul;37(6):1477-1485. doi: 10.1007/s40620-024-02055-x. Epub 2024 Sep 2. PMID: 39223353.
10. Banerjee S, Sengupta J, Basu S. The clinical relevance of native vitamin D in pediatric kidney disease. Pediatr Nephrol. 2023 Apr;38(4):945-955. doi: 10.1007/s00467-022-05698-9. Epub 2022 Aug 5. PMID: 35930049.
11. Vareldzis R, Perez A, Reisin E. Hyperuricemia: An Intriguing Connection to Metabolic Syndrome, Diabetes, Kidney Disease, and Hypertension. Curr Hypertens Rep. 2024 Jun;26(6):237-245. doi: 10.1007/s11906-024-01295-3. Epub 2024 Jan 25. PMID: 38270791.
12. Du L, Zong Y, Li H, Wang Q, Xie L, et al. Hyperuricemia and its related diseases: mechanisms and advances in therapy. Signal Transduct Target Ther. 2024 Aug 28;9(1):212. doi: 10.1038/s41392-024-01916-y. PMID: 39191722; PMCID: PMC11350024.
13. Memon S, Alam A, Imtiaz S. Nutritional vitamin D is efficacious for reducing intact parathyroid hormone in chronic kidney disease, a prospective interventional study. J Pak Med Assoc. 2022 Jun;72(6):1068-1074. doi: 10.47391/JPMA.2285. PMID: 35751311.
14. Abdul Razzaque MR, Tebha SS, Tukruna A, Arif A, Kogut LM, et al. 25-Hydroxyvitamin-D deficiency in chronic kidney disease stages III, IV, and V in South Asian population: a retrospective cohort. SAGE Open Med. 2023 Jan 19;11:20503121221148613. doi: 10.1177/20503121221148613. PMID: 36741931; PMCID: PMC9893052.
15. Singh S, Aggarwal V, Pandey UK, Sreenidhi HC. Study of left ventricular systolic dysfunction, left ventricular diastolic dysfunction and pulmonary hypertension in CKD 3b-5ND patients-A single centre cross-sectional study. Nefrologia (Engl Ed). 2023 Sep-Oct;43(5):596-605. doi: 10.1016/j.nefroe.2022.06.005. Epub 2022 Dec 21. PMID: 36564233.
16. Huang B, Wen W, Ye S. Correlation Between Serum 25-Hydroxyvitamin D Levels in Albuminuria Progression of Diabetic Kidney Disease and Underlying Mechanisms By Bioinformatics Analysis. Front Endocrinol (Lausanne). 2022 May 12;13:880930. doi: 10.3389/fendo.2022.880930. PMID: 35634488; PMCID: PMC9133500.
17. Duan S, Lu F, Sun R, Chen C, Chen S, et al. 25(OH)D level and vascular lesion scores in kidney histopathology as risk-stratification tool for predicting renal progression in people with type 2 diabetes. Diabetes Metab Syndr. 2024 May;18(5):103037. doi: 10.1016/j.dsx.2024.103037. Epub 2024 May 8. PMID: 38744090.
18. Hsu S, Zelnick LR, Bansal N, Brown J, Denburg M, et al. Vitamin D Metabolites and Risk of Cardiovascular Disease in Chronic Kidney Disease: The CRIC Study. J Am Heart Assoc. 2023 Jul 18;12(14):e028561. doi: 10.1161/JAHA.122.028561. Epub 2023 Jul 8. PMID: 37421259; PMCID: PMC10382125.
19. Krummel T, Ingwiller M, Keller N, Prinz E, Charlin E, et al. Effects of high- vs low-dose native vitamin D on albuminuria and the renin-angiotensin-aldosterone system: a randomized pilot study. Int Urol Nephrol. 2022 Apr;54(4):895-905. doi: 10.1007/s11255-021-02950-3. Epub 2021 Jul 20. PMID: 34286472.
20. Karalliedde J, Fountoulakis N, Corcillo A, Maltese G, Flaquer M, et al. Effect of calcitriol treatment on arterial stiffness in people with type 2 diabetes and stage 3 chronic kidney disease. Br J Clin Pharmacol. 2023 Jan;89(1):279-289. doi: 10.1111/bcp.15484. Epub 2022 Aug 25. PMID: 35942813; PMCID: PMC10087300.
21. Galassi A, Fasulo EM, Ciceri P, Casazza R, Bonelli F, et al. 1,25-dihydroxyvitamin D as Predictor of Renal Worsening Function in Chronic Kidney Disease. Results From the PASCaL-1,25D Study. Front Med (Lausanne). 2022 Mar 2;9:840801. doi: 10.3389/fmed.2022.840801. PMID: 35308556; PMCID: PMC8924653.
22. Obaid AA, Farrash WF, Mujalli A, Singh SK. A Quest for Potential Role of Vitamin D in Type II Diabetes Mellitus Induced Diabetic Kidney Disease. Curr Pharm Des. 2024;30(32):2505-2512. doi: 10.2174/0113816128296168240614071821. PMID: 38963115.
23. Luo Y, Song Q, Li J, Fu S, Yu W, et al. Effects of uric acid-lowering therapy (ULT) on renal outcomes in CKD patients with asymptomatic hyperuricemia: a systematic review and meta-analysis. BMC Nephrol. 2024 Feb 23;25(1):63. doi: 10.1186/s12882-024-03491-4. PMID: 38395818; PMCID: PMC10893702.
24. Yang XH, Zhang BL, Cheng Y, Fu SK, Jin HM. Febuxostat provides renoprotection in patients with hyperuricemia or gout: a systematic review and meta-analysis of randomized controlled trials. Ann Med. 2024 Dec;56(1):2332956. doi: 10.1080/07853890.2024.2332956. Epub 2024 May 13. PMID: 38738384; PMCID: PMC11095284.
25. Sharbaf FG, Bakhtiari E, Faghihi T, Assadi F. Efficacy and Safety of Allopurinol on Chronic Kidney Disease Progression: A Systematic Review and Meta-Analysis. J Pediatr Pharmacol Ther. 2024 Aug;29(4):359-367. doi: 10.5863/1551-6776-29.4.359. Epub 2024 Aug 13. PMID: 39144382; PMCID: PMC11321800.
26. Castillo RF. Pathophysiologic Implications and Therapeutic Approach of Klotho in Chronic Kidney Disease: A Systematic Review. Lab Invest. 2023 Jul;103(7):100178. doi: 10.1016/j.labinv.2023.100178. Epub 2023 May 18. PMID: 37207706.

Authors' contribution	
Sameer Sattar	Conceptualization, study design, methodology, data analysis, and manuscript writing.
Mateen Akram	Supervision, methodology, interpretation of results, and critical revision of the manuscript.
Muhammad Usman Masood Butt	Literature review, data collection, and manuscript writing.
Shazia Abbas	Patient recruitment, data collection, and manuscript review.
Hina Francis	Data interpretation, literature review, and manuscript editing.
Saim Shuja Cheema	Data collection, data organization, and final manuscript review