The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 6, 2025

PREVALENCE OF HYPERURICEMIA IN CKD PATIENTS

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DOI: https://doi.org/10.5281/zenodo.17935008

Keywords
hyperuricemia, prevalence, CKD, Renal impairment

Article History
Received: 01 May 2025
Accepted: 19 June 2025
Published: 29 June 2025

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Abstract

Objective: *The purpose of the research is to find out the prevalence of hyperuricemia in CKD patients*

Study Design: *Cross Sectional study*

Study Duration: *06 months from November 2024 to April 2025*

Study Place: *Department of Medicine, CMH Lahore*

Methods: *The study was a cross-sectional study of 180 CKD patients over a six months period to determine the prevalence of hyperuricemia and its distribution between stages of CKD. Demographic and clinical data, laboratory data were taken care of, serum uric acid levels were taken and eGFR was taken as a staging parameter. Analysis was performed using SPSS version 26 and $p<0.05$ was taken as significant.*

Results: *Total 180 patients were included and the average age of the participants was 57.40 years. There were 102 males and 78 females. The most frequently seen comorbidity was hypertension with (78.3%); diabetes mellitus (52.2%), ischemic heart disease (19.4%), and obesity (17.2%). Hyperuricemia affected 58.9% of 180 CKD patients and increased progressively with advancing CKD stages. It was more common in males and strongly associated with lower eGFR and higher creatinine.*

Conclusion: *Hyperuricemia is highly prevalent in CKD, increasing with detoriating renal function. Early screening is recommended to prevent complications like gout, urate nephropathy, and faster renal function decline.*

INTRODUCTION

Chronic kidney disease (CKD) is a significant health issue of the world and it shows an estimated 10-15 percent prevalence among adult population of the world. It is progressive, and is associated with a variety of complications which pose a significant health burden in regard to morbidity, mortality, and health care cost. Hyperuricemia has been of specific clinical interest among such complications because it is common in patients with CKD and can lead to the development of the disease. The process of hyperuricemia, which is the presence of uric acid in serum exceeding the physiological level, occurs mainly due to the decrease in the uric acid excretion in the kidney, where CKD patients are naturally

susceptible to hyperuricemia. With the deterioration of the kidney functions, the ability to remove uric acid decreases, and, therefore, it gradually accumulates in the bloodstream.[1]

There is also emerging evidence that hyperuricemia is not simply the effect of CKD but can also be a cause of the condition and its development. This two-way interaction has led to the conclusion that there is a need to understand more about the prevalence and clinical consequences of hyperuricemia in CKD cohorts, especially in those areas where the two conditions are commonly observed.[2]

The hyperuricemia prevalence among CKD patients differs widely in studies as much as 30 to more than 60 percent with prevalence, geographical factors, diets, genetic influence as well as methodological variations. In South Asian population, where high-purine diets and metabolic disorders i.e. hypertension, diabetes mellitus, and obesity are prevalent, the pressure of hyperuricemia may be even higher. To achieve early risk stratification and intervention, the magnitude of this problem among CKD cohorts should be identified.[3] The control of hyperuricemia in CKD is fraught with further complications, as drugs to control it should be chosen without excessive care of nephrotoxicity and with a high probability of lowering the level of uric acid. Newer recommendations state more personalized treatment options and the necessity of stronger evidence on the advantages of taking urate-lowering therapy to delay the development of CKD.

Although there is growing awareness of hyperuricemia as an important issue of metabolic concerns in CKD, very little data is available on the same in many developing nations. To be able to improve the clinical management of it, it is crucial to know not only its prevalence but also to direct preventive measures and allocation of resources. The proposed study will answer the question of the prevalence of hyperuricemia in CKD patients, which will provide valuable regional data and will inform further studies on the clinical implications of hyperuricemia.

Methodology:

A cross-sectional observational study was conducted in the Department of Medicine of a tertiary care teaching hospital Lahore. The study was conducted for a period of six months, from November 2024 to April 2025 during which adult patients diagnosed with chronic kidney disease (CKD) were consecutively enrolled from outpatient nephrology clinics and inpatient wards. The objective was to determine the prevalence of hyperuricemia among CKD patients and to assess its distribution across different CKD stages. All individuals presenting with a known diagnosis of CKD, as defined by the Kidney Disease Improving Global Outcomes (KDIGO) guidelines[4], were considered eligible for participation. CKD was classified based on estimated glomerular filtration rate (eGFR) using the CKD-EPI formula and categorized into stages 1–5. A sample size of 180 was determined using an expected prevalence of hyperuricemia of approximately 40–60% among CKD patients, a 95% confidence interval, and a 5% margin of error. Sample size was calculated as per WHO sample size calculator. Patients were recruited using a non-probability consecutive sampling technique. After obtaining informed written consent, demographic and clinical data—including age, gender, duration of CKD, comorbidities (hypertension, diabetes mellitus), and current medications—were collected through a structured questionnaire and medical record review. Physical examination findings were also documented. Blood samples were collected after an overnight fast. Serum uric acid levels were measured using an enzymatic colorimetric method in the hospital laboratory. Hyperuricemia was defined as:

- >7.0 mg/dL in males
- >6.0 mg/dL in females

Serum creatinine, eGFR, electrolytes, fasting glucose, and lipid profile were also measured. CKD staging was assessed based on eGFR values.

Data were entered and analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The prevalence of hyperuricemia was calculated as the proportion of CKD patients with elevated serum uric acid levels. Stratification by CKD stage, gender, and comorbidities was performed using chi-square and independent t-tests. A p-value <0.05 was considered statistically significant.

Inclusion Criteria

Participants were included if they met the following criteria:

1. Adults aged 18 years and above.
2. Diagnosed cases of chronic kidney disease (CKD stages 1–5), as per KDIGO guidelines.
3. Patients willing to provide informed written consent.
4. Patients with available serum uric acid and kidney function test results.

Exclusion Criteria

The following individuals were excluded to avoid confounding factors:

1. Patients with acute kidney injury or rapidly deteriorating renal function.
2. Current use of urate-lowering therapy such as allopurinol, febuxostat, or uricosuric agents.
3. Patients with malignancies, hematological disorders, or autoimmune diseases known to affect uric acid metabolism.
4. Pregnant or lactating women.
5. Patients with chronic alcohol abuse, which can influence uric acid production.
6. Individuals with incomplete laboratory data or inadequate serum samples.

Results:

This study enrolled 180 patients who had been diagnosed with chronic kidney disease (CKD). The average age of the participants is 57.40 years of age with a standard deviation of 13.2 years with a range of 22 years to 85 years. There were 102 males and 78 females out of the total population resulting in a ratio of 1.3:1. The most frequently seen comorbidity was hypertension with 141 of the patients (78.3%); diabetes mellitus was next with 94 of the patients (52.2%). A small number of other comorbidities seen were ischemic heart disease (19.4%), and obesity (17.2%).

Hyperuricemia was found to be prevalent (58.9%) in all CKD patients (n=106). Of these, 67/102 males (65.6%) were hyperuricemic as compared to 39/78 females (50%), which showed that it was more common in males. The average serum uric acid of the hyperuricemic group was 8.4 +/- 1.3mg/dL which is highly higher than the 5.6 +/- 0.9mg/dL in the normouricemic group ($p < 0.001$).

The prevalence of hyperuricemia was found to be continuously growing with progressive stages of CKD strongly and progressively. During the early stages of CKD, 35.7% of the patients reported high levels of uric acid. The level peaked at 48.2% in stage 3, 72.4% in stage 4 and 81.3% in stage 5. This increased tendency was statistically significant ($p < 0.001$), which proves that there is a strong correlation between deteriorating renal function and the rising level of uric acid.

The average eGFR of hyperuricemic group was significantly less (23.4 ± 11.0 mL/min/1.73m^2) as it was in normouricemic patients (34.7 ± 12.5 mL/min/1.73m 2, $p = 0.001$). The creatinine level of serum was also much more elevated in the hyperuricemia group (3.4 ± 1.2 vs. 2.5 ± 1.0, $p = 0.002$

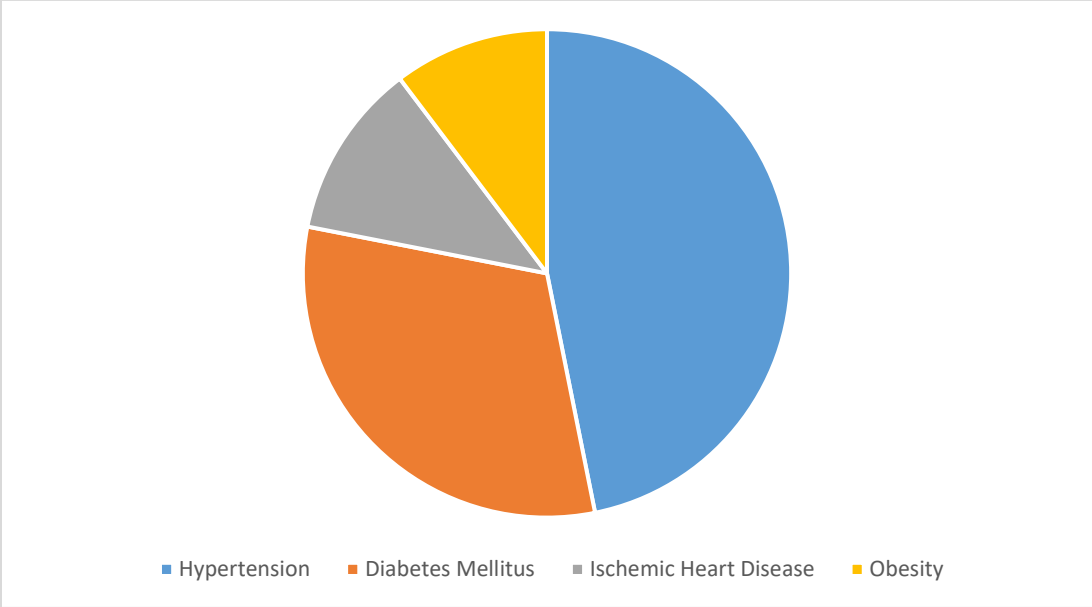
Even though hyperuricemia was also more common in hypertensive (61.7%) and diabetic patients (60.6), these were not statistically significant. A higher percentage of hyperuricemia (68.4) was observed to be more in patients under diuretics especially loop diuretics but not significantly ($p = 0.07$). There was no significant level of BMI categories and uric acid.

The normouricemic and hyperuricemic groups varied in regard to a number of laboratory parameters. Hyperuricemic patients were characterized by an increase in the level of serum potassium, phosphorus, and parathyroid hormone (PTH), but only PTH was statistically significant ($p = 0.03$). The cases of hyperuricemic patients had a slightly lower level of hemoglobin, which was correlated with more advanced CKD, but the difference was not statistically significant.

No significant differences in fasting blood glucose, lipid profile and electrolyte values were found between the two groups and so hyperuricemia was more closely associated with renal dysfunction than metabolism derangements.

Variable	Mean ± SD / n (%)
Age (years)	57.4 ± 13.2
Male	102 (56.7%)
Female	78 (43.3%)

Table 1.1: Demographic data of study subjects



Pie chart 1.2: Comorbid leading to CKD

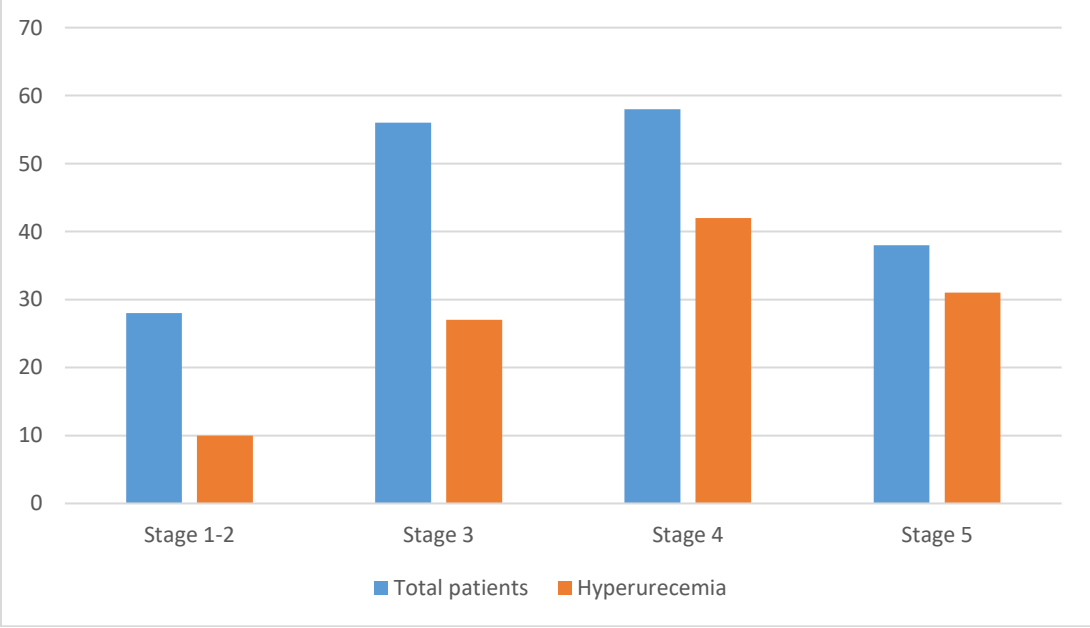


Chart 1.3: Prevalence of Hyperuricemia Across CKD Stages

Parameter	Hyperuricemia (n=106) Mean ± SD	Normouricemia (n=74) Mean ± SD
Serum uric acid (mg/dL)	8.4 ± 1.3	5.6 ± 0.9
eGFR (mL/min/1.73m²)	23.4 ± 11.0	34.7 ± 12.5
Serum creatinine (mg/dL)	3.4 ± 1.2	2.5 ± 1.0
Potassium (mmol/L)	4.9 ± 0.6	4.7 ± 0.5
Phosphorus (mg/dL)	4.9 ± 1.2	4.6 ± 1.1
PTH (pg/mL)	321 ± 140	278 ± 126
Hemoglobin (g/dL)	10.1 ± 1.6	10.5 ± 1.5

Table 1.4: Comparison of Biochemical Parameters Between Groups

Discussion:

The results of this study revealed a high prevalence rate of hyperuricemia in patients with chronic kidney disease (CKD) which is in line with a substantial amount of international and regional literature. An aggregate study of the literature showed the prevalence of approximately in the mid-40 percent range across CKD populations, with high between-study heterogeneity due to variation in case-mix, eGFR distribution, definitions of hyperuricemia and local risk factors.

A number of cohort and cross-sectional studies in various settings have also reported extensive ranges of prevalence, typically reported of 30-70 percent, and proceed to compare steadily rising levels of uric acid with eGFR deterioration.[4]

Findings in the South Asian region and Pakistan reflect the findings globally and also indicate local variations. Pakistan and other countries have reported in single- center and multicenter studies with prevalences ranging over two-thirds to low teens, and based on the population sampled (outpatient CKD, non-dialysis CKD, or haemodialysis patients) and the uric acid cutoffs used.[5]

These variations highlight the effect caused by sampling frame (such a study done only on end-stage renal disease will have more proportions) and population factors including age distribution, prevalence of diabetics and hypertensives, diet and availability of nephrology services.

The recorded progressive correlation between the worsening stage of CKD and increasing prevalence of hyperuricemia in our cohort is biologically feasible and consistent with the previous studies. With a weakening of the renal function, renal urate secretions drop and non-renal compensatory mechanisms fail in most cases to ensure normal serum uric acid levels thus creating accumulation. Further, CKD is coupled with chronic inflammation, oxidative stress and changes in tubular management of urate which further facilitates hyperuricemia.[6]

This pathophysiology facilitates the understanding of why prevalence is usually minimum in levels 1-2, maximum in level 3 and maximum in levels 4-5 and dialysis groups.

On top of descriptive epidemiology, a clinical question of interest is whether hyperuricemia is a simple indicator of less renal clearance or a risk factor by its own that hyperuricemia contributes to the rapid progression of CKD. Causal pathways suggested by observational cohorts and mechanistic studies, such as urate-mediated endothelial dysfunction, activation of renin-angiotensin signaling, intrarenal inflammation and crystal-mediated tubular injury have the capacity to plausibly deteriorate kidney performance.[7]

Coherent evidence of meta-analytic studies also indicates that increased baseline serum uric acid is linked with increased risk of incident CKD and accelerated eGFR loss, and some studies have estimated a substantially higher risk with every 1 mg/dL increase in uric acid. Nonetheless, observational relationships may be confounded by comorbid factors (hypertension, metabolic syndrome, diuretic use) which are both causes of hyperuricemia and causal agents of CKD, therefore causality is a field under active development.[8]

Urate-lowering therapy (ULT) as prevention in the management of CKD progression has delivered inconclusive results. Information available on some smaller trials and post-hoc studies showed a modest

decrease in eGFR with allopurinol or febuxostat although other randomized controlled trials have failed to demonstrate such clear benefit on hard renal endpoints.[9]

Considering such heterogeneity and the possible drug-specific adverse effect (especially in end-stage CKD and in combination with other agents nephrotoxicity), it is nowadays advised in guidelines that the decision-making process should be individualized as opposed to universal ULT in all CKD patients with hyperuricemia. In CKD populations, urate-lowering agents should be carefully selected, dosed and monitored.[10]

In clinical and public-health perspective, a number of implications exist in the high percentage of hyperuricemia among CKD. To begin with, regular monitoring of serum uric acid in CKD clinics can be used to detect at-risk patients prone to gout exacerbation, urate nephropathy and potentially accelerated CKD. Second, the management approach should focus on reversible contributors (e.g., choice of diuretic, optimization of blood pressure and glycemic control, alcohol and diet purine intake) and pharmacotherapy put in the second place as a symptomatic gout treatment option, extremely elevated uric acid levels or isolated instances where ULT may be used with a renal advantage after a multidisciplinary consultation. Third, local studies are required - large, prospective cohorts and properly designed randomized trials in South Asian and Pakistani populations would help define the patterns of prevalence, the interaction of risk factors and the efficacy and safety of ULT in these contexts. Some of the recent studies in the region have given hints of particularly high prevalence in some subgroups of clinical settings (e.g., dialysis patients), which again underline the need of locally relevant evidence.[11,12]

Continuing on the clinical implication of hyperuricemia in chronic kidney disease, it is worth noting that there is a progressive development in the perception of uric acid as a biomarker and mediator of kidney dysfunction. The mechanisms through which serum uric acid levels are associated with CKD progression are the focus of current studies, despite documentation of the associations between the two. In recent work, it has been proposed that the monosodium urate crystals can directly activate the NLRP3 inflammasome to stimulate inflammatory and fibrotic processes in kidney to accelerate the progression of CKD without the need of conventional metabolic drivers (e.g., hypertension and insulin resistance).[13,14]

Regional-based population studies have also been used to support the high burden of hyperuricemia in patients with low eGFR, in which socioeconomic disparities and inaccessibility to preventive nephrology services could have led to late diagnosis and treatment. These results are in line with our study that shows a significant percentage of patients at early CKD stages already had high levels of uric acid which shows that hyperuricemia can show up earlier in disease development than was traditionally thought.[15]

Pharmacological treatment is still a field of controversy. Although xanthine oxidase inhibitors like allopurinol and febuxostat have become extensively popular, there are still concerns that the cardiovascular safety and therapeutic dose of the drugs in elderly CKD is not clear. The neutral effects of uric-acid-lowering therapy on long-term kidney outcome have been reported in large-scale randomized controlled studies such as the CKD-FIX and PERL trials which contradicts the previous assumptions of therapeutic impact. Although these are found, observational studies on East Asian cohorts indicate the possibility of aggressive urate lowering still being beneficial to specific patient groups, which points to the possibility of ethnic and genetic differences in patient response to treatment.[16]

Finally, in CKD, the prevalence of hyperuricemia is associated with a rise in renal damage. Although there has been growing evidence of a relationship between high levels of uric acid and poor renal prognosis, a conclusive evidence on how reduced uric acid can inhibit CKD progression is still pending- especially in different world populations. Until further and more decisive information can be gathered, treatment of hyperuricemia in the context of overall CKD treatment needs to be screened and addressed based on an individual risk and symptomology of a particular patient.

Limitation of Study:
It is a cross-sectional analysis in a singular center, the results might not be representative of the general population of CKD. To get better results, a multi-center study and with more number of participants is required.

Conclusion:
This paper shows that hyperuricemia is very common in patients with chronic kidney disease and there is a very clear tendency of the gradual rise of serum uric acid levels in line with decreasing renal functions. The results corroborate the close relationship between the worsening of kidney function and the decrease in uric acid excretion, especially at advanced stages of CKD. Screening against it regularly should be part of CKD management. Earlier detection would aid in diagnosing those patients who are at more risk of complication like gout, urate nephropathy and perhaps accelerated renal failure. Our research highlights the significance of thorough metabolic assessment in CKD populations.

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