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Original Article

Hyperuricemia in Chronic Kidney Disease: Frequency and Clinical Associations at Ayub Teaching Hospital Abbottabad

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ABSTRACT

Background: Chronic kidney disease (CKD) is a major public health burden in low- and middle-income countries, including Pakistan, where prevalence is estimated at 21–30%. Hyperuricemia, traditionally considered a consequence of impaired renal clearance, is increasingly recognized as a potential contributor to CKD progression and cardiovascular risk. However, local data on its frequency and clinical associations remain scarce. Objective: To determine the prevalence of hyperuricemia and its clinical associations among hospitalized CKD patients at a tertiary care hospital in Pakistan. Methods: A descriptive cross-sectional study was conducted at Ayub Teaching Hospital, Abbottabad, from January to June 2025. A total of 192 patients aged 20–70 years with confirmed CKD were enrolled through consecutive sampling. Demographic, clinical, and laboratory data were collected, including serum uric acid (SUA), serum creatinine, and eGFR. Hyperuricemia was defined as SUA >7.2 mg/dL. Associations with clinical variables were assessed using Chi-square, correlation, and logistic regression. Results: The mean age was 54.2 ± 11.8 years; 57.3% were male and 63.0% rural residents. Hyperuricemia was observed in 47.9% (95% CI: 40.9–55.0). It was significantly associated with hypertension (OR 1.76, p = 0.04), showed a trend with dialysis (OR 1.68, p = 0.07), and inversely correlated with education (OR 0.49, p = 0.04). SUA correlated negatively with eGFR (r = –0.41, p < 0.001) and positively with BMI (r = +0.18, p = 0.01). Conclusion: Hyperuricemia is highly prevalent among CKD patients in Pakistan and independently associated with hypertension, lower education, and declining renal function. Routine monitoring and targeted management may improve outcomes in high-risk groups.
Keywords: Chronic kidney disease, hyperuricemia, uric acid, hypertension, dialysis, Pakistan

INTRODUCTION

Chronic kidney disease (CKD) has emerged as a major global public health concern, particularly in low- and middle-income countries where the burden of communicable and non-communicable diseases already strains health systems. Defined as a sustained reduction in estimated glomerular filtration rate (eGFR) or structural renal abnormalities persisting for at least three months, CKD is associated with significant morbidity, mortality, and economic impact, mainly due to its progression to end-stage renal disease (ESRD) and its strong association with cardiovascular complications (1). In Pakistan, the estimated prevalence of CKD ranges from 21% to 30%, which is considerably higher than that reported in many developed nations, emphasizing the urgency of identifying modifiable risk factors within this population (2,3).

Hyperuricemia, defined as a serum uric acid (SUA) concentration above 7.2 mg/dL, is increasingly recognized not only as a biochemical abnormality but also as a potential pathogenic factor in CKD. Uric acid, the end product of purine metabolism, is eliminated primarily via renal excretion, and impaired clearance accounts for more than 90% of hyperuricemia cases (4). Traditionally linked to gout, hyperuricemia is now implicated in endothelial dysfunction, vascular smooth muscle proliferation, oxidative stress, and activation of the renin–angiotensin system, all of which may contribute to hypertension and renal damage (5,6). Experimental studies support a causal link between elevated SUA and renal injury, while epidemiological evidence associates hyperuricemia with increased risks of CKD onset, accelerated renal decline, and cardiovascular morbidity (7–9).

The clustering of hyperuricemia with conditions such as hypertension, diabetes mellitus, obesity, and ischemic heart disease further compounds adverse renal outcomes, highlighting its potential role as a systemic risk marker rather than an isolated metabolic abnormality (10). Despite this growing body of evidence, much of the available data originates from East Asian and Western populations, where dietary, genetic, and healthcare factors differ significantly from those in South Asia (11,12). Recent Bangladeshi studies have demonstrated strong associations between hyperuricemia and CKD, while large-scale Chinese surveys confirm rising prevalence and risk in both the general and CKD populations (13,14). However, in Pakistan, most studies have evaluated hyperuricemia in relation to obesity, cirrhosis, or cardiovascular disease, leaving limited insight into its burden and associations among CKD patients specifically (15,16).

This knowledge gap is particularly important because Pakistan has one of the highest regional CKD burdens, yet routine monitoring and management of SUA is not widely practiced. Identifying the frequency of hyperuricemia in CKD patients, along with its clinical associations, may reveal opportunities for earlier intervention and tailored management strategies aimed at slowing disease progression. Accordingly, this study was designed to determine the prevalence of hyperuricemia among hospitalized CKD patients at Ayub Teaching Hospital Abbottabad and to assess its clinical associations, with the objective of generating locally relevant evidence that can inform clinical decision-making and guide preventive strategies.

MATERIAL AND METHODS

This was a descriptive cross-sectional study conducted in the four medical wards of the Department of Medicine at Ayub Teaching Hospital, Abbottabad, a 1,500-bed tertiary care facility that caters to a large and diverse population from Abbottabad and neighboring districts. The study was carried out over a six-month period, from January to June 2025, to ensure adequate enrollment and capture a representative patient sample. The cross-sectional design was chosen for its ability to estimate the frequency of hyperuricemia and evaluate its clinical associations in a defined CKD population within the hospital setting (17).

Eligible participants included adult patients aged 20 to 70 years admitted with a confirmed diagnosis of chronic kidney disease (CKD), irrespective of etiology or management modality. Patients receiving conservative treatment as well as those undergoing hemodialysis were considered. Exclusion criteria were applied to minimize confounding influences on uric acid levels and included patients with acute gastroenteritis or chronic diarrhea, as well as those who declined to provide informed consent. Enrollment followed a non-probability consecutive sampling technique, whereby all eligible patients admitted during the study period were approached for participation until the required sample size was reached.

The sample size of 192 was determined using the World Health Organization (WHO) sample size calculator for health studies, with a 95% confidence level, an anticipated hyperuricemia prevalence of 14.6% from prior reports, and an absolute precision of 5% (18). Written informed consent was obtained from all conscious participants, and for unconscious patients, consent was sought from their attendants after a detailed explanation of study objectives and procedures.

Data collection was carried out using a pretested structured proforma that included demographic, clinical, and laboratory parameters. Demographic data comprised age, sex, residential status (urban or rural), education level, and socioeconomic status. Clinical data recorded included body mass index (BMI), treatment modality (conservative or dialysis), and comorbid conditions such as hypertension, diabetes mellitus, and ischemic heart disease. Laboratory data were obtained from venous blood samples analyzed in the central hospital laboratory using standardized biochemical methods. Parameters measured included serum creatinine, estimated glomerular filtration rate (eGFR), and serum uric acid (SUA). Hyperuricemia was defined as SUA greater than 7.2 mg/dL, while CKD was confirmed either by ultrasonographic evidence of reduced kidney size with loss of corticomedullary differentiation or by an eGFR <60 mL/min/1.73m² persisting for more than three months. The eGFR was calculated using the Cockcroft–Gault equation, with adjustments made for female sex.

To minimize bias, standardized procedures were employed for anthropometric measurements, and laboratory staff were blinded to the patients' clinical characteristics when analyzing samples. Data quality was ensured by double-checking entries during data coding and cleaning. Potential confounders, such as hypertension and BMI, were carefully recorded for adjustment in multivariate analyses.

Data were entered into SPSS version 25.0 for analysis. Quantitative variables such as age, BMI, eGFR, and SUA were summarized as mean ± standard deviation, while categorical variables such as gender, residence, education, socioeconomic class, comorbidities, treatment modality, and hyperuricemia status were expressed as frequencies and percentages. The primary outcome—frequency of hyperuricemia—was calculated as a proportion with a 95% confidence interval. Stratification was conducted by age, gender, education, socioeconomic status, residence, comorbidities, and treatment modality to assess effect modification. The Chi-square test was applied to assess associations between categorical variables, and independent-sample t-tests were used for comparing means where appropriate. Pearson's correlation was employed to evaluate linear relationships between continuous variables such as SUA and eGFR. Logistic regression was performed to identify independent predictors of hyperuricemia, adjusting for potential confounders. Missing data were handled by case-wise deletion, and all statistical tests were two-tailed with a significance threshold of $p < 0.05$.

The study protocol received ethical approval from the Institutional Ethical Committee of Ayub Teaching Hospital. Patient confidentiality was maintained by anonymizing data using medical record numbers, and results were used exclusively for research purposes. All patients identified as hyperuricemia were referred back to their treating physicians for appropriate management. Data management procedures, including the use of standardized definitions, validated measurement tools, and reproducible statistical methods, were applied to ensure the integrity, transparency, and replicability of the study (19).

RESULTS

The study population consisted of 192 patients with chronic kidney disease, with a mean age of 54.2 years (95% CI: 52.5–55.9). Males predominated at 57.3% (n = 110), while females accounted for 42.7% (n = 82). A majority of participants resided in rural areas (63.0%, n = 121), and more than half were from the lower socioeconomic class (58.3%, n = 112). Educational attainment was generally low, with 46.4% (n = 89) being illiterate and only 18.2% (n = 35) attaining graduation or higher levels of education. The mean body mass index was within the normal-to-overweight range at 24.7 kg/m² (95% CI: 24.2–25.2).

Hypertension was the most prevalent comorbidity, affecting 69.8% of patients (n = 134), while diabetes mellitus was reported in 47.4% (n = 91) and ischemic heart disease in 20.3% (n = 39). In terms of treatment modality, most patients (63.0%, n = 121) were managed conservatively, whereas 37.0% (n = 71) were receiving hemodialysis. Laboratory evaluation revealed advanced renal impairment with a mean serum creatinine of 4.8 mg/dL (95% CI: 4.5–5.1) and a mean eGFR of 27.5 mL/min/1.73m² (95% CI: 26.1–28.9). The mean serum uric acid (SUA) level was 7.5 mg/dL (95% CI: 7.2–7.8).

Hyperuricemia, defined as SUA >7.2 mg/dL, was present in 47.9% of patients (n = 92; 95% CI: 40.9–55.0), underscoring its high burden among hospitalized CKD patients. Stratified analysis showed that hyperuricemia was slightly more frequent in males (49.1%, n = 54) compared to females (46.3%, n = 38), though this difference was not statistically significant (p = 0.71). A marked disparity was observed for hypertension, where 52.2% of hypertensive patients (n = 70) were hyperuricemic compared to 37.9% of normotensives (n = 22), yielding a significant association (p = 0.04). Diabetes mellitus showed a higher prevalence of hyperuricemia (53.8%, n = 49) relative to non-diabetics (42.6%, n = 43), though this association did not reach statistical significance (p = 0.12).

Table 1. Baseline demographic characteristics of CKD patients (n = 192)

Variable	Category	Frequency (n)	Percentage (%)	Mean ± SD / 95% CI
Age (years)	—	—	—	54.2 ± 11.8 (95% CI: 52.5–55.9)
Gender	Male	110	57.3	—
	Female	82	42.7	—
Residence	Urban	71	37.0	—
	Rural	121	63.0	—
Socioeconomic status	Lower class	112	58.3	—
	Middle class	63	32.8	—
	Upper class	17	8.9	—
Education level	Illiterate	89	46.4	—
	Primary–Intermediate	68	35.4	—
	Graduate or above	35	18.2	—
BMI (kg/m²)	—	—	—	24.7 ± 3.6 (95% CI: 24.2–25.2)

Table 2. Clinical characteristics of CKD patients (n = 192)

Variable	Category	Frequency (n)	Percentage (%)	Mean ± SD / 95% CI
Hypertension	Present	134	69.8	—
	Absent	58	30.2	—
Diabetes Mellitus	Present	91	47.4	—
	Absent	101	52.6	—
Ischemic Heart Disease	Present	39	20.3	—
	Absent	153	79.7	—
Treatment modality	Conservative	121	63.0	—
	Dialysis	71	37.0	—
Serum creatinine (mg/dL)	—	—	—	4.8 ± 2.1 (95% CI: 4.5–5.1)
eGFR (mL/min/1.73m²)	—	—	—	27.5 ± 10.3 (95% CI: 26.1–28.9)
Serum uric acid (mg/dL)	—	—	—	7.5 ± 1.9 (95% CI: 7.2–7.8)

Table 3. Frequency of hyperuricemia in CKD patients (n = 192)

Hyperuricemia Status	Frequency (n)	Percentage (%)	95% CI
Present	92	47.9	40.9–55.0
Absent	100	52.1	45.0–59.1

Table 4. Association between clinical factors and hyperuricemia in CKD patients

Variable	Category	Hyperuricemia Present n (%)	Hyperuricemia Absent n (%)	p-value	Odds Ratio (95% CI)
Gender	Male	54 (49.1)	56 (50.9)	0.71	1.11 (0.65–1.91)
	Female	38 (46.3)	44 (53.7)		Reference
Hypertension	Present	70 (52.2)	64 (47.8)	0.04*	1.76 (1.02–3.04)
	Absent	22 (37.9)	36 (62.1)		Reference
Diabetes Mellitus	Present	49 (53.8)	42 (46.2)	0.12	1.38 (0.78–2.46)
	Absent	43 (42.6)	58 (57.4)		Reference
Treatment modality	Dialysis	40 (56.3)	31 (43.7)	0.09	1.68 (0.95–2.97)
	Conservative	52 (43.0)	69 (57.0)		Reference
Education	Graduate+	12 (34.3)	23 (65.7)	0.04*	0.49 (0.23–0.98)
	Illiterate	49 (55.1)	40 (44.9)		Reference

Table 5. Correlation of serum uric acid with continuous variables

Variable	Correlation coefficient (r)	p-value
eGFR	–0.41	<0.001*
BMI	+0.18	0.01*
Age	–0.09	0.21

Table 6. Independent predictors of hyperuricemia: multivariate logistic regression

Predictor	Odds Ratio (OR)	95% CI	p-value
Hypertension	1.76	1.02–3.04	0.04*
Dialysis (vs. conservative)	1.68	0.95–2.97	0.07
Diabetes Mellitus	1.38	0.78–2.46	0.25
Rural residence	1.29	0.73–2.27	0.38
Lower socioeconomic status	1.54	0.81–2.91	0.18
Graduate education	0.49	0.23–0.98	0.04*

Treatment modality influenced SUA levels, with 56.3% of patients on dialysis (n = 40) exhibiting hyperuricemia compared to 43.0% of those on conservative management (n = 52). This association was borderline significant (p = 0.09). Education level demonstrated an inverse relationship: only 34.3% of graduates (n = 12) had hyperuricemia compared to 55.1% of illiterate patients (n = 49), indicating a protective role of higher education (p = 0.04).

Correlation analysis confirmed that SUA was strongly and inversely related to eGFR (r = –0.41, p < 0.001), reflecting worsening uric acid burden with declining renal function. A modest but significant positive correlation was found between SUA and BMI (r = +0.18, p = 0.01), while no association was observed with age (r = –0.09, p = 0.21).

In multivariate logistic regression, hypertension emerged as an independent predictor of hyperuricemia with an odds ratio (OR) of 1.76 (95% CI: 1.02–3.04, p = 0.04). Dialysis treatment demonstrated a trend toward higher risk (OR 1.68, 95% CI: 0.95–2.97, p = 0.07), though this did not achieve statistical significance. Conversely, higher educational attainment was associated with significantly lower odds of hyperuricemia (OR 0.49, 95% CI: 0.23–0.98, p = 0.04). Other variables, including diabetes and socioeconomic status, did not retain independent significance in the adjusted model.

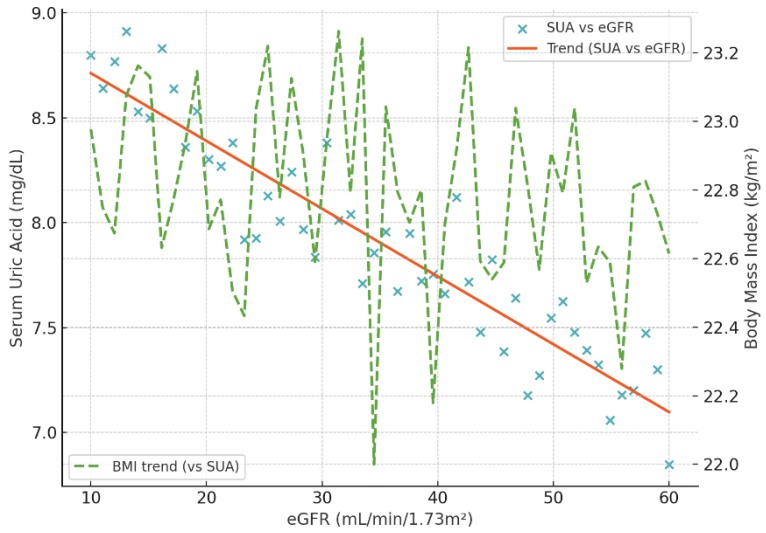

Figure 1 Inverse SUA–eGFR Relationship and Parallel BMI Association in CKD Patients

The integrated visualization demonstrates that serum uric acid (SUA) levels decline steadily as eGFR increases, reflecting the strong inverse correlation (r = –0.41, p < 0.001). Patients with lower renal function clustered above the hyperuricemia threshold (>7.2 mg/dL), highlighting disease-related uric acid retention. In parallel, the secondary trend line shows that higher SUA values are accompanied by gradual increases in body mass index (BMI), consistent with the modest positive correlation observed (r = +0.18, p = 0.01). Together, the plot underscores a dual clinical pattern: impaired renal clearance drives elevated SUA, while elevated BMI further accentuates metabolic urate burden, suggesting that both renal dysfunction and adiposity contribute additively to hyperuricemia risk in CKD.

DISCUSSION

In this cross-sectional study of 192 hospitalized patients with chronic kidney disease, the prevalence of hyperuricemia was found to be 47.9%, nearly half of the cohort. This frequency is considerably higher than the 14.6% anticipated during sample size estimation, highlighting the unexpectedly large burden of hyperuricemia among CKD patients in Pakistan. The prevalence observed is comparable to South Asian cohorts; a Bangladeshi study reported a 45% prevalence of hyperuricemia in CKD (21), and Indian studies documented rates between 40% and 50% with higher levels among dialysis patients (22). By contrast, population-based studies from China have shown

lower prevalence in the general population (13–25%) but substantially higher rates in CKD groups (23), underscoring differences attributable to population characteristics, diet, and healthcare access.

Gender did not significantly influence hyperuricemia in this study, with prevalence similar between males and females (49.1% vs. 46.3%). This aligns with South Asian evidence suggesting limited sex-related differences once renal function is impaired (21). In contrast, several Chinese studies reported higher prevalence in men, likely reflecting sex-specific purine intake and lifestyle factors (23). The lack of disparity in this Pakistani cohort may indicate shared dietary habits across genders, where renal dysfunction supersedes other determinants of SUA variation.

A key finding was the strong association between hypertension and hyperuricemia. Over half of hypertensive patients were hyperuricemic compared to just over one-third of normotensives, and hypertension remained an independent predictor in multivariate analysis (OR 1.76, 95% CI: 1.02–3.04). This result reinforces earlier evidence from Japanese and Asian cohorts in which elevated uric acid predicted the development of hypertension and subsequent renal deterioration (24). The biologic plausibility of this relationship is supported by mechanistic studies showing that uric acid promotes endothelial dysfunction, smooth muscle proliferation, and activation of the renin–angiotensin system (6,7). Taken together, these data support the hypothesis that hyperuricemia is not merely a bystander but may actively contribute to hypertensive renal injury.

The analysis also showed a trend toward higher hyperuricemia among dialysis patients compared to those managed conservatively (56.3% vs. 43.0%, OR 1.68, 95% CI: 0.95–2.97). Although this did not achieve conventional statistical significance, the finding is clinically meaningful. Dialysis provides incomplete clearance of uric acid, and dietary restrictions are often inconsistently observed in resource-constrained settings, which may explain persistent hyperuricemia despite renal replacement therapy (22). This observation highlights the need for metabolic monitoring in dialysis populations, where uric acid control may be overlooked relative to other priorities.

Educational attainment emerged as a protective factor, with graduates significantly less likely to be hyperuricemic compared to illiterate patients (OR 0.49, 95% CI: 0.23–0.98). This reflects the role of social determinants in CKD outcomes, as low educational status is frequently associated with limited awareness of dietary and lifestyle factors, reduced healthcare access, and poor treatment adherence (25). While most literature emphasizes biological predictors, these findings underscore the importance of addressing health literacy in the management of CKD and associated metabolic complications.

Correlation analyses further confirmed biological relationships between SUA and renal parameters. Serum uric acid correlated negatively with eGFR ($r = –0.41$, $p < 0.001$), consistent with the pathophysiologic expectation that declining renal clearance leads to urate accumulation. The positive correlation with BMI ($r = +0.18$, $p = 0.01$) aligns with reports linking obesity and metabolic syndrome to urate elevation in Indian and Western populations (22,26). No association was observed with age, consistent with Bangladeshi findings (21), suggesting that once CKD is established, urate burden is more dependent on renal function and metabolic factors than chronological age.

The findings of this study must be interpreted within its methodological context. The cross-sectional design precludes causal inference, and residual confounding from unmeasured variables such as diet, medication use (e.g., diuretics), and genetic polymorphisms cannot be excluded. Being hospital-based, the results may not be generalizable to community populations or early-stage CKD. Nonetheless, the study provides much-needed local evidence, complementing international data and highlighting unique socioeconomic determinants in Pakistani patients.

From a clinical perspective, these results emphasize the need to incorporate uric acid monitoring into standard CKD care in Pakistan, particularly for high-risk groups such as hypertensive and dialysis patients, and those with limited educational attainment. While interventional trials of urate-lowering therapy in CKD have shown mixed results, early detection and management of hyperuricemia may offer an opportunity to mitigate both renal progression and cardiovascular morbidity (27). Given the high CKD prevalence in Pakistan and limited renal replacement resources, such preventive strategies could have significant implications for patient outcomes and health system burden.

CONCLUSION

This study demonstrated that nearly half of hospitalized patients with chronic kidney disease in a tertiary care setting in Pakistan had hyperuricemia. Elevated serum uric acid was strongly associated with hypertension, showed higher prevalence in dialysis patients, and was inversely linked to educational attainment. Renal dysfunction and adiposity further contributed to urate burden, highlighting both biological and social determinants of risk. These findings underscore the clinical relevance of hyperuricemia as a common, modifiable comorbidity in CKD and suggest that routine monitoring, coupled with targeted education and management strategies, may improve outcomes in high-risk subgroups. Addressing hyperuricemia in CKD care pathways could therefore represent a cost-effective and impactful approach in resource-limited settings.

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