Original Research Article

Correlation of Serum Uric Acid with Severity and Progression of Chronic Kidney Disease

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Abstract

Background: Chronic kidney disease (CKD) is a progressive disorder associated with increasing morbidity, mortality, and risk of end-stage kidney disease. Serum uric acid (SUA) has emerged as a potential biomarker in CKD due to its involvement in oxidative stress, endothelial dysfunction, inflammation, and renal microvascular injury. This study was conducted to evaluate the correlation between serum uric acid levels and severity and progression of chronic kidney disease.

Methods: A hospital-based observational study was conducted among 100 adult patients diagnosed with chronic kidney disease. All participants underwent clinical evaluation and laboratory investigations, including serum uric acid, serum creatinine, blood urea nitrogen, eGFR, and urine protein assessment. CKD severity was categorized according to eGFR-based staging. The association between serum uric acid levels and renal function parameters was analyzed using appropriate statistical methods.

Results: The majority of patients belonged to the 41–60 years age group (46.0%), with male predominance (64.0%). Hypertension was the most common comorbidity (72.0%), followed by diabetes mellitus (48.0%). Hyperuricemia was observed in 62.0% of patients, with a mean serum uric acid level of 7.82 ± 1.86 mg/dL. Serum uric acid showed a significant positive correlation with serum creatinine ($r=0.624$, $p<0.001$) and blood urea nitrogen ($r=0.572$, $p<0.001$) and a significant negative correlation with eGFR ($r=-0.681$, $p<0.001$). Mean uric acid levels increased progressively with advancing CKD stages ($p<0.001$). Logistic regression demonstrated that serum uric acid was an independent predictor of advanced CKD (Stage ≥4) (AOR 1.82, 95% CI 1.32–2.51, $p<0.001$).

Conclusion: Elevated serum uric acid levels are significantly associated with worsening renal function and advanced CKD severity. Serum uric acid may serve as a simple and useful prognostic marker for identifying patients at higher risk of CKD progression.

Keywords: Chronic kidney disease; Serum uric acid; Hyperuricemia; eGFR; CKD progression; Renal function; Prognostic marker.

Introduction

Chronic kidney disease (CKD) is a progressive disorder characterized by persistent abnormalities in kidney structure and function lasting for more than three months, resulting in gradual and irreversible decline in renal capacity.[1] It has emerged as a major global public health concern, affecting approximately 10–13% of adults worldwide and contributing significantly to morbidity, mortality, cardiovascular complications, and healthcare burden. The increasing prevalence of CKD is largely

driven by the rising incidence of diabetes mellitus, hypertension, obesity, metabolic disorders, and population aging.[2,3] Despite advances in renal replacement therapies and supportive care, progression to end-stage kidney disease (ESKD) remains a major clinical challenge, emphasizing the importance of identifying early predictors of disease progression.Traditionally, CKD severity and progression are assessed through estimated glomerular filtration rate (eGFR), albuminuria, and staging systems. However, these parameters may not completely reflect the complex pathophysiological mechanisms involved in progressive renal injury.[4-6] Several non-traditional risk factors, including inflammation, oxidative stress, endothelial dysfunction, metabolic abnormalities, and vascular injury, have been implicated in accelerating renal deterioration. Among these emerging biomarkers, serum uric acid (SUA) has gained considerable attention due to its potential role as both a marker and mediator of CKD progression.[7]Uric acid is the final product of purine metabolism in humans and is primarily eliminated through renal excretion.[8] In patients with CKD, impaired renal clearance leads to accumulation of uric acid and development of hyperuricemia. Although traditionally considered a consequence of reduced kidney function, increasing evidence suggests that elevated serum uric acid may actively contribute to renal damage.[9] Experimental studies have demonstrated that hyperuricemia can induce endothelial dysfunction, oxidative stress, activation of the renin–angiotensin–aldosterone system, vascular smooth muscle proliferation, and inflammatory pathway activation. These mechanisms promote renal microvascular injury, glomerular hypertension, tubulointerstitial fibrosis, and progressive decline in renal function.[10]

Clinical and epidemiological studies have reported a significant association between increased serum uric acid levels and worsening CKD outcomes. Higher SUA concentrations have been linked with reduced eGFR, increased proteinuria, advanced CKD stages, accelerated renal function decline, and increased risk of progression to ESKD.[11] Several observational studies and meta-analyses have supported hyperuricemia as an independent predictor of CKD progression; however, whether uric acid acts as a causal factor or merely reflects impaired renal function remains an area of ongoing investigation. Furthermore, the effectiveness of urate-lowering therapy in preventing CKD progression remains uncertain, necessitating further evaluation of this relationship.[12]

The assessment of serum uric acid as a prognostic marker is particularly relevant in developing countries such as India, where CKD burden is increasing and early identification of high-risk patients is essential for preventing disease progression. Since serum uric acid measurement is inexpensive, routinely available, and easily reproducible, understanding its association with CKD severity may improve risk stratification and individualized patient management.

Therefore, the present study was conducted to evaluate the correlation between serum uric acid levels and the severity and progression of chronic kidney disease among adult patients attending a tertiary care centre, with the aim of determining its potential role as a prognostic marker for renal dysfunction.

Methodology:

The present study was conducted as a hospital-based observational study in the Department of Medicine at a tertiary care centre. The study was designed to evaluate the correlation between serum uric acid levels and the severity and progression of chronic kidney disease among adult patients.The study was conducted over a predefined study period after obtaining approval from the Institutional Ethics Committee. All eligible patients fulfilling the inclusion criteria were enrolled during the study period.

Study Population

The study included 100 adult patients diagnosed with chronic kidney disease who attended the outpatient department or were admitted to the hospital during the study period. Patients were evaluated clinically and biochemically to assess the relationship between serum uric acid levels and CKD severity.A total of 100 patients with chronic kidney disease were included in the study.

Inclusion Criteria

Patients fulfilling the following criteria were included in the study:

- Adult patients aged ≥18 years.
- Patients diagnosed with chronic kidney disease according to established clinical criteria.
- Patients with CKD duration of more than three months.
- Patients willing to participate in the study and provide informed consent.

Exclusion Criteria

Patients were excluded if they had:

- Acute kidney injury or acute worsening of renal function.
- History of recent gout attack.
- Patients receiving urate-lowering therapy.
- Active malignancy or severe systemic illness affecting renal function.
- Pregnancy.

- Patients unwilling to provide informed consent.

Methodology

After obtaining written informed consent, all enrolled patients were evaluated through detailed history taking, clinical examination, and relevant laboratory investigations. Demographic details, including age and sex, were recorded. Clinical history regarding duration of CKD, comorbidities such as diabetes mellitus, hypertension, cardiovascular disease, and medication history was documented.

Clinical Assessment

A detailed clinical examination was performed for all participants. Blood pressure, body weight, and other relevant clinical parameters were recorded. The presence of CKD-related complications and associated comorbidities was assessed.

Laboratory Investigations

Venous blood samples were collected from all patients under aseptic conditions. The following investigations were performed:

- Serum uric acid level.
- Serum creatinine.
- Blood urea nitrogen.
- Estimated glomerular filtration rate (eGFR).
- Serum electrolytes.
- Blood glucose parameters.
- Urine examination including assessment of proteinuria/albuminuria.

Serum uric acid levels were measured using standard biochemical methods. Renal function was assessed using serum creatinine and eGFR values.

Assessment of Chronic Kidney Disease Severity

CKD severity was categorized according to eGFR-based staging criteria. Patients were classified into different CKD stages based on their estimated glomerular filtration rate. The relationship between serum uric acid levels and CKD stage was evaluated.

Assessment of CKD Progression

Progression of CKD was assessed based on decline in renal function parameters, particularly reduction in eGFR and worsening CKD stage during the study evaluation. Serum uric acid levels were compared with renal function indicators to determine their association with disease progression.

Statistical Analysis

The collected data were entered into a structured database and analyzed using SPSS 21statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on data distribution. Categorical variables were presented as frequencies and percentages.The association between serum uric acid levels and CKD severity was assessed using appropriate statistical tests. Correlation between serum uric acid and renal function parameters such as serum creatinine and eGFR was evaluated using Pearson's or Spearman's correlation coefficient depending on data distribution. A p-value of <0.05 was considered statistically significant.

Results:

The present study included 100 patients with chronic kidney disease to evaluate the correlation between serum uric acid levels and severity and progression of CKD.The demographic characteristics of the study participants are presented in Table 1. The majority of patients belonged to the age group of 41–60 years (46.0%), followed by patients aged >60 years (42.0%) and 18–40 years (12.0%). Male predominance was observed, with 64.0% males and 36.0% females. Regarding duration of CKD, most patients had disease duration of 1–5 years (51.0%), followed by <1 year (28.0%) and >5 years (21.0%). A higher proportion of patients were from urban areas (58.0%) compared with rural areas (42.0%) (Table 1).The distribution of associated comorbidities among study participants is shown in Table 2 and Figure 1. Hypertension was the most common comorbidity, present in 72.0% of patients, followed by diabetes mellitus (48.0%). Combined diabetes mellitus and hypertension was observed in 39.0% of patients. Cardiovascular disease was present in 22.0%, dyslipidemia in 31.0%, and a history of gout was reported in 9.0% of participants. The overall distribution of comorbid conditions is illustrated in Figure 1.The severity of CKD based on estimated glomerular filtration rate (eGFR) staging is summarized in Table 3. Among the study participants, the majority belonged to CKD Stage 4 (25.0%), followed by Stage 3b (24.0%), Stage 3a (18.0%), and Stage 5 (15.0%). Early CKD stages were less frequent, with Stage 2 accounting for 14.0% and Stage 1 accounting for 4.0% of patients (Table 3).The distribution of serum uric acid levels among CKD patients is presented in Table 4 and Figure 2. Normal serum uric acid levels were observed in 38.0% of participants, whereas hyperuricemia was present in 62.0% of patients. Mild hyperuricemia (7–8.9 mg/dL) was observed in 34.0%, moderate hyperuricemia (9–10.9 mg/dL) in 20.0%, and severe hyperuricemia (≥11 mg/dL) in 8.0% of patients. The mean serum uric acid level among study participants was 7.82 ± 1.86 mg/dL (Table 4; Figure 2).

The correlation between serum uric acid levels and renal function parameters is shown in Table 5. Serum uric acid demonstrated a significant positive correlation with serum creatinine (r=+0.624,

p<0.001) and blood urea nitrogen (r=+0.572, p<0.001). A significant negative correlation was observed between serum uric acid and eGFR (r=−0.681, p<0.001), indicating that increasing uric acid levels were associated with worsening renal function. Serum uric acid also showed a significant positive correlation with urine protein/albuminuria (r=+0.418, p<0.001) (Table 5).

The association between serum uric acid levels and CKD severity is presented in Table 6. Mean serum uric acid levels increased progressively with advancing CKD stage. Patients with Stage 1–2 CKD had a mean serum uric acid level of 5.92 ± 1.12 mg/dL, which increased to 7.31 ± 1.43 mg/dL in Stage 3, 8.54 ± 1.52 mg/dL in Stage 4, and 9.46 ± 1.67 mg/dL in Stage 5 CKD. This difference across CKD stages was statistically significant (p<0.001) (Table 6), suggesting a strong association between elevated serum uric acid levels and advanced CKD severity.

Multiple logistic regression analysis was performed to identify independent predictors of advanced CKD (Stage ≥4), as shown in Table 7 and Figure 3. Serum uric acid was found to be an independent predictor of advanced CKD, with each 1 mg/dL increase in serum uric acid associated with an 82% higher risk of advanced CKD (Adjusted OR 1.82; 95% CI 1.32–2.51; p<0.001). Other significant predictors included age >60 years (AOR 2.14; 95% CI 1.01–4.54; p=0.047), hypertension (AOR 2.36; 95% CI 1.04–5.33; p=0.039), and serum creatinine (AOR 3.12; 95% CI 1.86–5.23; p<0.001). Diabetes mellitus showed an increased odds ratio but did not reach statistical significance (p=0.145) (Table 7; Figure 3).

Overall, the findings demonstrated that elevated serum uric acid levels were significantly associated with reduced eGFR, higher CKD stages, and increased likelihood of advanced renal disease, supporting its potential role as a prognostic marker for CKD progression.

Table 1: Demographic and Baseline Characteristics of Study Participants (N=100)

Parameter	Category	Number (n)	Percentage (%)
Age group (years)	18–40	12	12.0
	41–60	46	46.0
	>60	42	42.0
Sex	Male	64	64.0
	Female	36	36.0
Duration of CKD	<1 year	28	28.0
	1–5 years	51	51.0
	>5 years	21	21.0
Residence	Urban	58	58.0
	Rural	42	42.0

Table 2: Distribution of Comorbidities Among Study Participants (N=100)

Comorbidity	Present n (%)	Absent n (%)
Diabetes mellitus	48 (48.0)	52 (52.0)
Hypertension	72 (72.0)	28 (28.0)
Diabetes + Hypertension	39 (39.0)	61 (61.0)
Cardiovascular disease	22 (22.0)	78 (78.0)
Dyslipidemia	31 (31.0)	69 (69.0)
History of gout	9 (9.0)	91 (91.0)

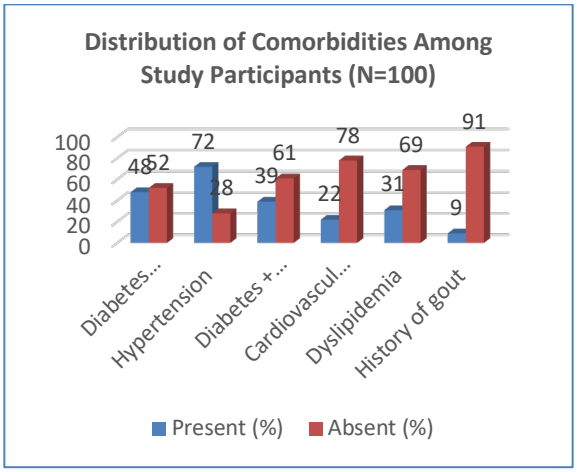

Figure 1 Distribution of Comorbidities Among Study Participants (N=100)

Table 3: Distribution of Patients According to CKD Stage Based on eGFR (N=100)

CKD Stage	eGFR Classification	Number (n)	Percentage (%)

	(mL/min/1.73m²)		
Stage 1	≥90	4	4.0
Stage 2	60–89	14	14.0
Stage 3a	45–59	18	18.0
Stage 3b	30–44	24	24.0
Stage 4	15–29	25	25.0
Stage 5	<15	15	15.0
Total		**100**	**100.0**

Table 4: Distribution of Serum Uric Acid Levels Among Study Participants (N=100)

Serum Uric Acid Level	Number (n)	Percentage (%)
Normal range (<7 mg/dL males, <6 mg/dL females)	38	38.0
Mild hyperuricemia (7–8.9 mg/dL)	34	34.0
Moderate hyperuricemia (9–10.9 mg/dL)	20	20.0
Severe hyperuricemia (≥11 mg/dL)	8	8.0
Mean Serum Uric Acid (mg/dL)	**7.82 ± 1.86**	

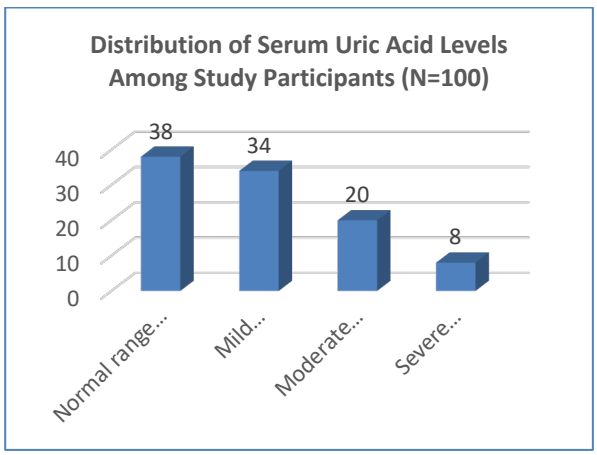

Figure 2 Distribution of Serum Uric Acid Levels Among Study Participants (N=100)

Table 5: Correlation of Serum Uric Acid with Renal Function Parameters (N=100)

Parameter	Correlation coefficient (r)	p-value
Serum creatinine	+0.624	<0.001
eGFR	−0.681	<0.001
Blood urea nitrogen	+0.572	<0.001
Urine protein/albuminuria	+0.418	<0.001

Table 6: Association Between Serum Uric Acid Levels and CKD Severity (CKD Stage) (N=100)

CKD Stage	Mean Serum Uric Acid (mg/dL)	SD	p-value
Stage 1–2	5.92	1.12	
Stage 3	7.31	1.43	
Stage 4	8.54	1.52	
Stage 5	9.46	1.67	
Overall	**7.82**	**1.86**	**<0.001**

Table 7: Logistic Regression Analysis for Predictors of Advanced CKD (Stage ≥4)

Variable	Adjusted OR	95% CI	p-value
Serum uric acid (per 1 mg/dL increase)	1.82	1.32–2.51	<0.001
Age (>60 years)	2.14	1.01–4.54	0.047
Diabetes mellitus	1.76	0.82–3.78	0.145
Hypertension	2.36	1.04–5.33	0.039
Serum creatinine	3.12	1.86–5.23	<0.001

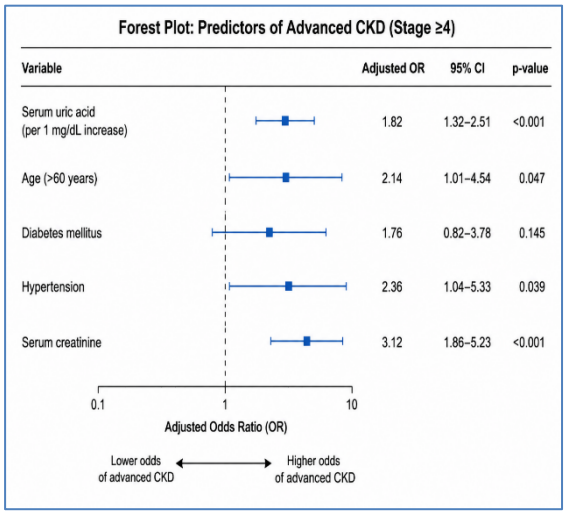

Figure 3 Logistic Regression Analysis for Predictors of Advanced CKD (Stage ≥4)

Discussion

Chronic kidney disease (CKD) is a progressive disorder characterized by gradual decline in renal function and increased cardiovascular morbidity and mortality. Serum uric acid (SUA) has gained attention as a potential biomarker in CKD due to its involvement in oxidative stress, endothelial dysfunction, inflammation, and activation of the renin–angiotensin–aldosterone system. The present study evaluated the correlation between SUA levels and severity and progression of CKD among 100 patients.In the present study, most patients were aged 41–60 years (46.0%), followed by >60 years (42.0%), with male predominance (64.0%) (Table 1). Similar demographic trends have been observed in previous CKD studies, where advanced age and male sex were associated with increased risk of renal dysfunction. Age-related nephron loss, reduced renal blood flow, and declining eGFR contribute to progressive renal impairment.

Hypertension was the most common comorbidity (72.0%), followed by diabetes mellitus (48.0%) and combined diabetes with hypertension (39.0%) (Table 2; Figure 1). These findings support previous evidence identifying diabetes and hypertension as major contributors to CKD progression. Wu et al.[13] reported that these metabolic conditions remain important risk factors, particularly among patients with moderate-to-severe CKD. Hyperuricemia frequently coexists with these risk factors and may further accelerate renal injury.

Advanced CKD stages were predominant in the present study, with Stage 4 CKD observed in 25.0% of patients, followed by Stage 3b (24.0%), Stage 3a (18.0%), and Stage 5 (15.0%) (Table 3). This distribution indicates that many patients presented with significant renal impairment. Reduced renal urate excretion in advanced CKD contributes to elevated SUA levels, which may further promote disease progression.

Hyperuricemia was observed in 62.0% of patients, including mild (34.0%), moderate (20.0%), and severe hyperuricemia (8.0%), with a mean SUA level of 7.82 ± 1.86 mg/dL (Table 4; Figure 2). Previous studies have also demonstrated a high prevalence of hyperuricemia among CKD patients and reported its association with increased risk of CKD progression.

A significant correlation was observed between SUA and renal function parameters. SUA showed a positive correlation with serum creatinine ($r=+0.624$, $p<0.001$) and blood urea nitrogen ($r=+0.572$, $p<0.001$), while a significant negative correlation was observed with eGFR ($r=-0.681$, $p<0.001$) (Table 5). These findings indicate that increasing SUA levels are associated with worsening renal function. Tsai et al.[14] similarly reported that each 1 mg/dL increase in baseline SUA increased the risk of kidney failure progression (adjusted HR 1.07, 95% CI 1.00–1.14, p=0.039).

Serum uric acid levels increased progressively with advancing CKD stages, from 5.92 ± 1.12 mg/dL in Stage 1–2 CKD to 9.46 ± 1.67 mg/dL in Stage 5 CKD ($p<0.001$) (Table 6). This progressive rise suggests that SUA reflects increasing severity of renal dysfunction. Previous systematic reviews and meta-analyses have also demonstrated that higher SUA levels are associated with increased risk of CKD incidence and progression.

Multivariate logistic regression analysis identified SUA as an independent predictor of advanced CKD (Stage ≥4). Each 1 mg/dL increase in SUA increased the odds of advanced CKD by 82% (Adjusted OR 1.82; 95% CI 1.32–2.51; $p<0.001$) (Table 7; Figure 3). Hypertension (AOR 2.36; 95% CI 1.04–5.33; p=0.039) and serum creatinine (AOR 3.12; 95% CI 1.86–5.23; $p<0.001$) were also significant predictors.

Although observational studies consistently demonstrate an association between hyperuricemia and CKD progression, whether SUA acts as a causal factor or merely reflects reduced renal clearance remains uncertain. Clinical trials evaluating urate-lowering therapy have shown inconsistent effects on renal outcomes. Therefore, further prospective studies are required.

Overall, the present study demonstrates that elevated SUA levels are significantly associated with reduced eGFR, advanced CKD stages, and increased risk of severe renal impairment. Serum uric acid may serve as a simple and clinically useful biomarker for early

risk stratification and monitoring of CKD progression.

Conclusion

The present study demonstrated a significant association between elevated serum uric acid levels and severity of chronic kidney disease. Higher serum uric acid concentrations were correlated with increased serum creatinine, reduced eGFR, and advanced CKD stages. Serum uric acid was identified as an independent predictor of advanced CKD, suggesting its potential role as a prognostic marker for renal deterioration. Measurement of serum uric acid may help in early risk stratification and monitoring of patients at increased risk of CKD progression.

Limitations

The study was conducted with a relatively small sample size of 100 patients from a single tertiary care centre, which may limit the generalizability of the findings. The observational cross-sectional design restricted the ability to establish a causal relationship between serum uric acid levels and CKD progression. Long-term follow-up was not performed to assess the impact of changing uric acid levels on renal outcomes. Further multicentric prospective studies with larger sample sizes are required to validate these findings.

Ethical approval

Ethical approval for this study was obtained from the Institutional Ethics Committee prior to the commencement of the study. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and applicable institutional guidelines.

Informed Consent

Written informed consent was obtained from all participants prior to their enrolment in the study.

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Conflict of Interest Declaration

The authors declare no conflict of interest.Data Availability Statement. All data generated or analysed during this study are available from the corresponding author upon reasonable request.

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