



Original Article

Association Between Serum Uric Acid Levels and Progression of Chronic Kidney Disease

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ABSTRACT

Background: Chronic kidney disease (CKD) is a major global health burden associated with progressive loss of renal function and increased cardiovascular morbidity and mortality. Serum uric acid has emerged as a potential risk factor for renal function decline. Several epidemiological studies have demonstrated an association between hyperuricemia and worsening kidney function, although the causal relationship remains under investigation. The present study aimed to evaluate the association between serum uric acid levels and progression of chronic kidney disease in adult patients attending a tertiary care hospital.

Methods: A hospital-based prospective observational study was conducted among 206 adult patients diagnosed with CKD attending the Department of General Medicine from March 2024 to September 2025. Sociodemographic, clinical, and laboratory parameters including serum uric acid, serum creatinine, estimated glomerular filtration rate (eGFR), urine protein status, and CKD stage were recorded. Statistical analysis was performed using SPSS software. Correlation and regression analyses were used to evaluate the relationship between serum uric acid levels and indicators of CKD progression.

Results: The mean age of participants was 56.8 ± 11.2 years, with a male predominance (57.3%). The mean duration of chronic kidney disease was 4.9 ± 2.8 years, and mean BMI was 26.8 ± 3.4 kg/m². Baseline renal assessment showed mean serum creatinine 2.78 ± 1.12 mg/dL, blood urea 68.4 ± 22.7 mg/dL, and eGFR 48.3 ± 18.6 mL/min/1.73m². Mean serum uric acid level was elevated at 7.6 ± 1.8 mg/dL. During follow-up, CKD progression was observed in 82 (39.8%) participants. Baseline serum uric acid demonstrated a significant negative correlation with eGFR ($r = -0.52$, $p < 0.001$) and significant positive correlations with serum creatinine ($r = 0.47$, $p < 0.001$) and blood urea ($r = 0.41$, $p < 0.001$). Participants who experienced CKD progression had significantly higher baseline serum uric acid levels than those without progression (8.4 ± 1.6 mg/dL vs 7.1 ± 1.4 mg/dL, $p < 0.001$). Serum uric acid also showed a significant association with annual decline in eGFR ($r = -0.49$, $p < 0.001$).

Conclusion: Elevated serum uric acid levels are significantly associated with progression of chronic kidney disease and deterioration of renal function in adult CKD patients. Serum uric acid may serve as a useful adjunct biomarker for risk stratification and identification of patients at increased risk of progressive renal impairment. Routine assessment of serum uric acid could facilitate early intervention and closer monitoring, potentially helping to delay CKD progression and its associated complications.

INTRODUCTION

Chronic kidney disease (CKD) is characterized by progressive and irreversible loss of kidney function and represents a major public health challenge worldwide. The global prevalence of CKD is estimated to be approximately 10–13%, and its burden continues to rise due to increasing rates of diabetes mellitus, hypertension, obesity, and aging populations [1].

Hyperuricemia is frequently observed among patients with CKD owing to reduced renal excretion of uric acid. Traditionally considered a consequence of declining renal function, elevated serum uric acid levels are increasingly being recognized as a potential contributor to renal injury [2]. Experimental studies suggest that uric acid may induce endothelial dysfunction, oxidative stress, activation of the renin–angiotensin system, inflammation, and arteriopathy, all of which may accelerate progression of kidney disease [2,3].

Several observational studies have reported that elevated serum uric acid is independently associated with incident CKD, faster decline in estimated glomerular filtration rate (eGFR), increased proteinuria, and greater risk of end-stage renal disease [3–6]. Meta-analyses have further demonstrated a significant association between hyperuricemia and CKD progression across diverse populations [7,8]. Despite these observations, controversy persists regarding whether uric acid is a true causal factor or merely a marker of impaired renal function [2,9].

Hyperuricemia has also been associated with hypertension, metabolic syndrome, cardiovascular disease, and increased all-cause mortality among CKD patients [10]. Recent cohort studies have suggested that elevated serum uric acid levels may predict rapid decline in renal function and worsening CKD stage independent of traditional risk factors [6,11]. Furthermore, several investigators have explored the role of urate-lowering therapy in slowing progression of CKD, although the results remain inconsistent [9,12].

Understanding the relationship between serum uric acid and CKD progression in the Indian population may contribute to improved risk stratification and early identification of high-risk patients. Therefore, the present study was undertaken to evaluate the association between serum uric acid levels and progression of chronic kidney disease in adult patients attending a tertiary care center.

MATERIALS AND METHODS

This hospital-based study was conducted in the Department of General Medicine at Varun Arjun Medical College and Rohilkhand Hospital, Shahjahanpur, Uttar Pradesh, India. The study was designed as a prospective observational hospital-based investigation. Consenting adult patients diagnosed with chronic kidney disease (CKD) attending the outpatient and inpatient services of the Department of General Medicine from March 2024 to September 2025 were enrolled in the study. All participants were followed prospectively during the study period to assess changes in renal function and progression of CKD. The study was carried out over 18 months following approval from the Institutional Ethics Committee.

Sample Size Calculation

The sample size was calculated using the formula:

$$n = Z^2pq/d^2$$

Where:

n = required sample size

Z = 1.96 at 95% confidence interval

p = prevalence of hyperuricemia among patients with chronic kidney disease (35%)

$$q = 1 - p$$

d = absolute precision of 6.5%

$$n = (1.96)^2 \times 0.35 \times 0.65 / (0.065)^2$$

$$n = 20$$

Therefore, the minimum required sample size was calculated to be 206 participants.

Outcome Measures

The primary objective of the study was to assess the association between serum uric acid levels and progression of chronic kidney disease.

The following parameters were evaluated:

- Serum uric acid levels
- Estimated glomerular filtration rate (eGFR)
- Serum creatinine
- Blood urea

- Urine protein status
- CKD stage

Data Entry and Statistical Analysis

Data were entered into Microsoft Excel 2007 and stored securely. Statistical analysis was performed using SPSS software version 26.0.

Continuous variables were expressed as mean \pm standard deviation (SD), whereas categorical variables were expressed as frequency and percentage.

Pearson's correlation coefficient was used to determine the relationship between serum uric acid levels and renal function parameters including eGFR and serum creatinine.

Independent samples t-test and one-way analysis of variance (ANOVA) were used for comparison of continuous variables between groups. Chi-square test was applied for comparison of categorical variables.

Multivariate linear regression analysis was performed to identify independent predictors of renal function decline. A p-value <0.05 was considered statistically significant.

Inclusion Criteria

- Adult patients aged ≥ 18 years.
- Diagnosed cases of chronic kidney disease according to Kidney Disease: Improving Global Outcomes (KDIGO) guidelines [13].
- Patients with CKD stages 2–5 not requiring dialysis.
- Patients willing to provide written informed consent.

Exclusion Criteria

- Acute kidney injury.
- Patients receiving urate-lowering therapy such as allopurinol or febuxostat.
- Patients undergoing maintenance haemodialysis or peritoneal dialysis.
- Chronic liver disease.
- Active malignancy.
- Pregnancy and lactation.
- Autoimmune diseases and chronic inflammatory disorders.
- Active infection at the time of enrolment.
- History of gout or inherited disorders of purine metabolism.

Data Collection Procedure

Data were collected using a structured pre-designed proforma. The procedure included:

1. Obtaining written, informed consent from each participant.
2. Detailed history taking, including personal, family, and treatment history.
3. Thorough clinical examination for evidence of blood pressure, body weight, height, body mass index (BMI), and systemic examination.
4. Laboratory investigations including:
 - Serum uric acid
 - Serum creatinine
 - Blood urea
 - Estimated glomerular filtration rate (eGFR)
 - Complete blood count (CBC)
 - Serum electrolytes
 - Urine routine and microscopic examination
 - Urine protein estimation
 - Fasting blood glucose
 - Lipid profile

Assessment of CKD Progression

Baseline serum uric acid levels were measured at enrolment. Renal function was assessed using serum creatinine and estimated glomerular filtration rate (eGFR) calculated using the CKD-EPI equation [14].

Patients underwent periodic follow-up evaluations at regular intervals during the study period. CKD progression was defined as:

- Decline in eGFR of $\geq 25\%$ from baseline, or

- Advancement to a higher CKD stage according to KDIGO classification, or
- Annualized eGFR decline greater than 5 mL/min/1.73m²/year [13].
- The primary outcome was progression of CKD during follow-up.
- The secondary outcomes included:
 - Rate of decline in eGFR
 - Progression to advanced CKD stages
 - Association between baseline serum uric acid and renal function decline

RESULTS

A total of 206 participants were enrolled in the study. The mean age of the cohort was 56.8 ± 11.2 years, with the majority belonging to the 51–60-year age group (35.0%), followed by >60 years (30.1%), 41–50 years (23.3%), and ≤40 years (11.7%) (Table 1, Figure 1). The study population comprised 118 males (57.3%) and 88 females (42.7%). Farmers (25.2%) and housewives (23.3%) represented the major occupational groups, followed by shopkeepers (15.0%), labourers (13.6%), teachers (9.7%), drivers (7.3%), and retired individuals (5.8%). Most participants were residents of Shahjahanpur (54.4%), while the remaining were from Tilhar (15.5%), Hardoi (11.7%), Powayan (10.7%), and Nigohi (7.8%) (Table 1, Figure 1). The mean duration of chronic kidney disease was 4.9 ± 2.8 years. Anthropometric assessment revealed a mean BMI of 26.8 ± 3.4 kg/m². The mean systolic and diastolic blood pressures were 142 ± 18 mmHg and 86 ± 11 mmHg, respectively. Renal function assessment demonstrated a mean serum creatinine level of 2.78 ± 1.12 mg/dL, blood urea level of 68.4 ± 22.7 mg/dL, and estimated glomerular filtration rate (eGFR) of 48.3 ± 18.6 mL/min/1.73m². Diabetes mellitus was present in 112 participants (54.4%), while hypertension was observed in 154 participants (74.8%). Fifty-two participants (25.2%) were non-hypertensive (Table 2, Figure 2).

The mean serum uric acid level among the study participants was 7.6 ± 1.8 mg/dL. The mean serum creatinine concentration was 2.78 ± 1.12 mg/dL, blood urea level was 68.4 ± 22.7 mg/dL, and estimated glomerular filtration rate (eGFR) was 48.3 ± 18.6 mL/min/1.73m² (Table 3). These findings describe the overall biochemical and renal function profile of the study cohort.

Follow-up assessment demonstrated deterioration in renal function over the study period. Mean serum creatinine increased from 2.78 ± 1.12 mg/dL at baseline to 3.21 ± 1.34 mg/dL at follow-up. Similarly, mean blood urea increased from 68.4 ± 22.7 mg/dL to 78.6 ± 25.4 mg/dL. The mean eGFR declined from 48.3 ± 18.6 mL/min/1.73m² at baseline to 41.2 ± 17.1 mL/min/1.73m² at follow-up (Table 4).

During follow-up, chronic kidney disease progression was observed in 82 participants (39.8%), whereas 124 participants (60.2%) remained clinically stable (Table 5).

Participants who experienced CKD progression had significantly higher baseline serum uric acid levels than those without progression (8.4 ± 1.6 mg/dL vs 7.1 ± 1.4 mg/dL, *p* < 0.001). They also demonstrated higher serum creatinine levels and lower baseline eGFR values compared with participants who did not experience disease progression (Table 6).

Correlation analysis demonstrated a moderate negative correlation between baseline serum uric acid levels and eGFR (*r* = -0.52, *p* < 0.001). Significant positive correlations were observed between serum uric acid and serum creatinine (*r* = 0.47, *p* < 0.001), as well as blood urea levels (*r* = 0.41, *p* < 0.001). Serum uric acid also showed a significant negative correlation with annual decline in eGFR (*r* = -0.49, *p* < 0.001), indicating that higher baseline uric acid levels were associated with greater deterioration of renal function during follow-up (Table 7).

These findings suggest that elevated serum uric acid levels are associated with worsening renal function and increased risk of chronic kidney disease progression among adult CKD patients.

Table 1. Sociodemographic Profile of Study Participants (n = 206)

Variable	Category	n	%
Age (years)	≤40	24	11.7
	41–50	48	23.3
	51–60	72	35.0
	>60	62	30.1
Mean ± SD	—	56.8 ± 11.2	—
Sex	Female	88	42.7
	Male	118	57.3
Occupation	Farmer	52	25.2
	Housewife	48	23.3
	Shopkeeper	31	15.0
	Labourer	28	13.6

	Teacher	20	9.7
	Driver	15	7.3
	Retired	12	5.8
Address	Shahjahanpur	112	54.4
	Tilhar	32	15.5
	Hardoi	24	11.7
	Powayan	22	10.7
	Nighoi	16	7.8

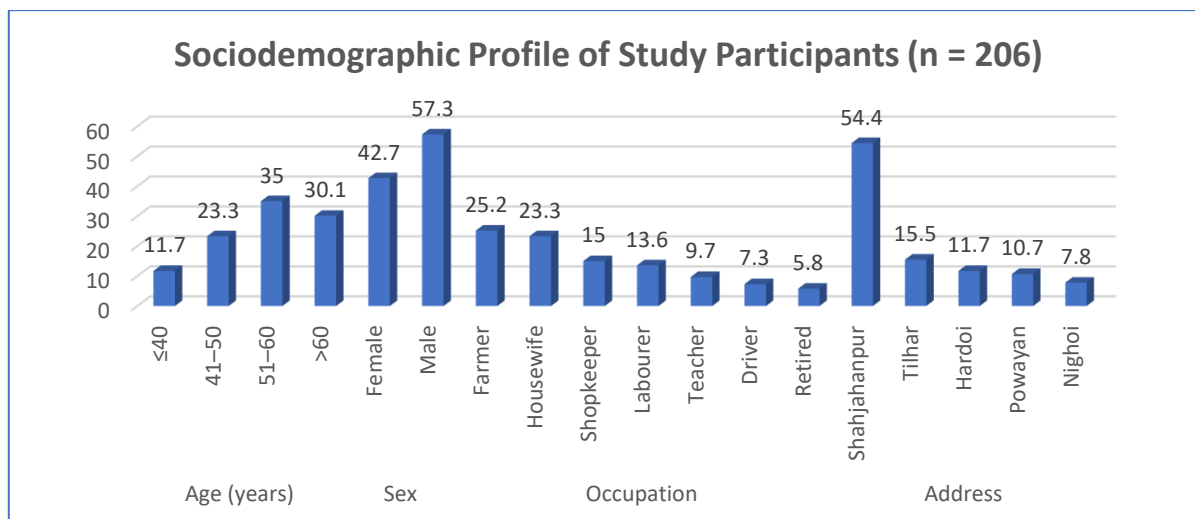


Figure 1 Sociodemographic Profile of Study Participants (n = 206)

Table 2. Clinical and Renal Profile of Study Participants (n = 206)

Parameter	Mean ± SD
Duration of CKD (years)	4.9 ± 2.8
BMI (kg/m ²)	26.8 ± 3.4
Systolic Blood Pressure (mmHg)	142 ± 18
Diastolic Blood Pressure (mmHg)	86 ± 11
Serum Creatinine (mg/dL)	2.78 ± 1.12
Blood Urea (mg/dL)	68.4 ± 22.7
eGFR (mL/min/1.73m ²)	48.3 ± 18.6

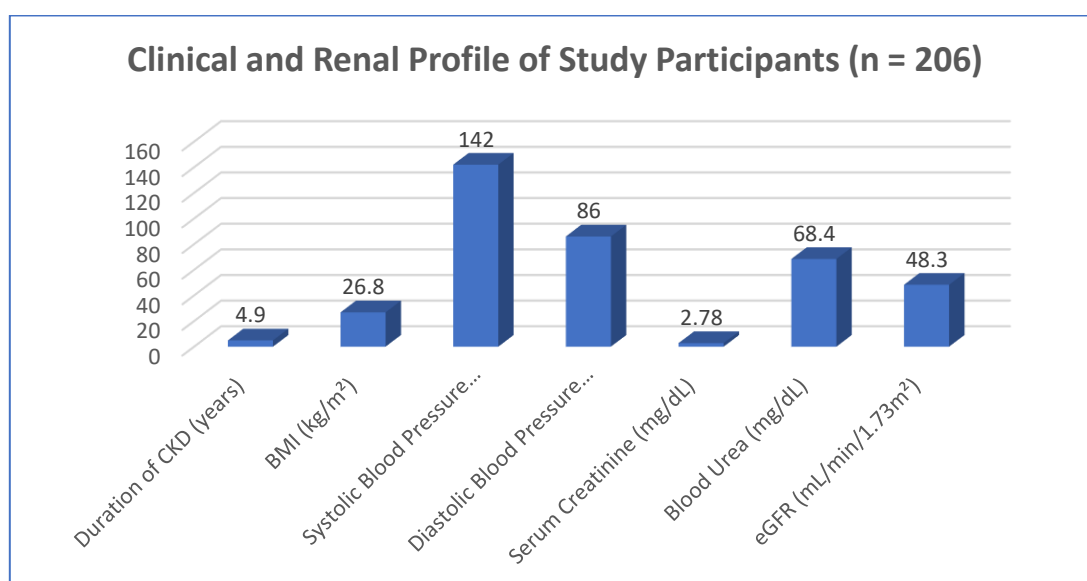


Figure 2 Clinical and Renal Profile of Study Participants (n = 206)

Table 3. Uric Acid and Renal Biochemistry Profile of Study Participants (n = 206)

Parameter	Mean ± SD
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Serum Uric Acid (mg/dL)	7.6 ± 1.8
Serum Creatinine (mg/dL)	2.78 ± 1.12
Blood Urea (mg/dL)	68.4 ± 22.7
Estimated Glomerular Filtration Rate (eGFR) (mL/min/1.73m ²)	48.3 ± 18.6

Table 4. Follow-up Renal Function Parameters of Study Participants (n = 206)

Parameter	Baseline Mean ± SD	Follow-up Mean ± SD
Serum Creatinine (mg/dL)	2.78 ± 1.12	3.21 ± 1.34
Blood Urea (mg/dL)	68.4 ± 22.7	78.6 ± 25.4
eGFR (mL/min/1.73m ²)	48.3 ± 18.6	41.2 ± 17.1

Table 5. Progression of Chronic Kidney Disease During Follow-up (n = 206)

Outcome	n	%
CKD Progression	82	39.8
No Progression	124	60.2

Table 6. Association Between Baseline Serum Uric Acid Levels and CKD Progression (n = 206)

Variable	CKD Progression (n=82)	No Progression (n=124)	p-value
Serum Uric Acid (mg/dL)	8.4 ± 1.6	7.1 ± 1.4	<0.001
Serum Creatinine (mg/dL)	3.18 ± 1.09	2.51 ± 0.97	<0.001
eGFR (mL/min/1.73m ²)	39.6 ± 14.8	54.1 ± 17.3	<0.001

Table 7. Correlation Between Baseline Serum Uric Acid Levels and Renal Function Parameters (n = 206)

Variables Correlated	Pearson's r	p-value
Serum Uric Acid vs eGFR	-0.52	<0.001
Serum Uric Acid vs Serum Creatinine	0.47	<0.001
Serum Uric Acid vs Blood Urea	0.41	<0.001

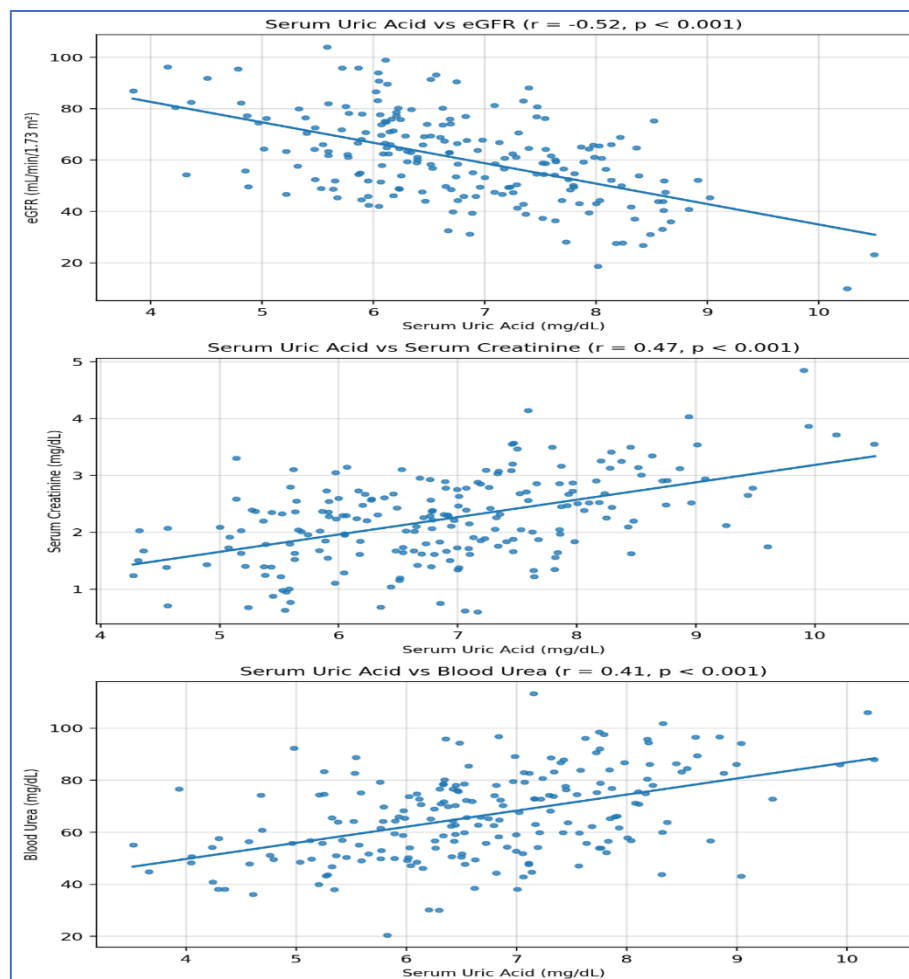


Figure 3 Correlation Between Baseline Serum Uric Acid Levels and Renal Function Parameters (n = 206)

DISCUSSION

The present prospective observational study evaluated the association between serum uric acid levels and progression of chronic kidney disease among adult CKD patients attending a tertiary care centre. The findings demonstrated that elevated serum uric acid levels were significantly associated with deterioration of renal function and increased risk of CKD progression during follow-up. The study population had a mean age of 56.8 ± 11.2 years, with the majority of participants belonging to the fifth and sixth decades of life. A slight male predominance was observed, which is consistent with previous studies reporting a higher prevalence of CKD and hyperuricemia among men [3,4]. Hypertension and diabetes mellitus were common comorbidities in the present cohort, reflecting their established role as major contributors to CKD development and progression [13]. The mean serum uric acid level among study participants was 7.6 ± 1.8 mg/dL, indicating a high prevalence of hyperuricemia in the CKD population. Similar findings have been reported by Chonchol et al. [4], Obermayr et al. [3], and Sonoda et al. [6], who observed that elevated serum uric acid levels were frequently associated with impaired renal function and increased risk of CKD progression. A significant decline in renal function was observed during follow-up, as evidenced by increasing serum creatinine and blood urea levels together with declining eGFR values. Approximately 39.8% of participants experienced CKD progression during the study period. These findings are comparable to those reported by Tsai et al. [9], who demonstrated that elevated serum uric acid levels were associated with accelerated renal function decline and increased risk of progression to advanced stages of CKD. Participants who experienced disease progression had significantly higher baseline serum uric acid levels compared with those who remained clinically stable. This observation supports previous evidence suggesting that serum uric acid may serve as an independent predictor of renal deterioration [3,4,11]. Elevated uric acid levels have been implicated in the development of glomerular hypertension, endothelial dysfunction, oxidative stress, inflammation, and activation of the renin-angiotensin-aldosterone system, all of which contribute to progressive nephron loss and worsening kidney function [1,2]. Correlation analysis revealed a significant negative association between serum uric acid levels and eGFR ($r = -0.52$, $p < 0.001$), indicating that higher uric acid concentrations were associated with lower renal function. Significant positive correlations were also observed between serum uric acid and serum creatinine as well as blood urea levels. These findings are consistent with those reported by Bellomo et al. [5], Mok et al. [10], and Wu et al. [11], who demonstrated that hyperuricemia is associated with declining kidney function across diverse populations. The observed association between serum uric acid and annual decline in eGFR further strengthens the hypothesis that uric acid may play an active role in CKD progression rather than merely representing a consequence of reduced renal clearance. Meta-analyses conducted by Zhu et al. [7] and Gonçalves et al. [8] have similarly reported that elevated serum uric acid levels significantly increase the risk of incident CKD and progression of established renal disease. The clinical implications of the present findings are noteworthy. Serum uric acid estimation is inexpensive, widely available, and routinely performed in clinical practice. Identification of elevated uric acid levels may help clinicians recognize patients at higher risk of progressive renal dysfunction and facilitate closer monitoring and timely intervention. Although the benefits of urate-lowering therapy remain a subject of ongoing investigation, growing evidence suggests that serum uric acid may be a useful biomarker for risk stratification in CKD patients [2,12].

CONCLUSION

The present study demonstrates a significant association between elevated serum uric acid levels and progression of chronic kidney disease in adult CKD patients. Higher baseline serum uric acid levels were associated with greater decline in estimated glomerular filtration rate (eGFR), increased serum creatinine and blood urea levels, and a higher likelihood of disease progression during follow-up. Serum uric acid showed a significant negative correlation with eGFR and a positive correlation with markers of renal dysfunction, suggesting its potential role in the deterioration of kidney function. These findings support the potential utility of serum uric acid as an adjunct biomarker for identifying CKD patients at increased risk of progressive renal impairment. Routine assessment of serum uric acid may facilitate early risk stratification, closer monitoring, and timely therapeutic interventions aimed at slowing disease progression and reducing the burden of advanced chronic kidney disease.

Limitations

This study was a single-centre prospective observational analysis with a relatively modest sample size, which may limit the generalisability of the findings to the broader CKD population. Although participants were followed over time, the observational nature of the study does not permit definitive establishment of a causal relationship between elevated serum uric acid levels and progression of chronic kidney disease. Potential confounding factors such as dietary habits, purine intake, medication use, socioeconomic status, and lifestyle factors were not comprehensively evaluated. Additionally, biomarkers of inflammation, oxidative stress, and endothelial dysfunction, which may influence both serum uric acid levels and renal disease progression, were not assessed. The study was conducted in a single tertiary care centre and excluded patients receiving dialysis or urate-lowering therapy, which may limit the applicability of the findings to all CKD populations. Further large-scale multicentre studies with longer follow-up periods are required to better clarify the role of serum uric acid in CKD progression and its potential as a therapeutic target.

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