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RELATIONSHIP BETWEEN SERUM URIC ACID, SERUM URIC ACID-TO-CREATININE RATIO AND ESTIMATED GLOMERULAR FILTRATION RATE IN PATIENTS WITH STAGE 3–5 CHRONIC KIDNEY DISEASES

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ABSTRACT

Chronic kidney disease (CKD) is a global health problem with high disease progression. Hyperuricemia is frequently observed in CKD; however, its diagnostic value is limited in advanced stages due to decreased renal excretion. Therefore, the serum uric acid–to–creatinine ratio (SUA/SCr) has the potential to serve as a more representative parameter, as it reflects uric acid metabolism adjusted for kidney function. This study aimed to evaluate the correlation between serum uric acid levels, the SUA/SCr ratio, and the estimated glomerular filtration rate (eGFR) in patients with stage 3, 4, and 5 chronic kidney disease (CKD). This was an analytic study with a cross-sectional design with consecutive sampling involving 50 patients with stage 3, 4, and 5 chronic kidney disease (CKD) at Adam Malik Hospital. Serum uric acid and creatinine levels were measured using enzymatic methods, while the estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation. Correlation analysis was performed using Pearson or Spearman tests, and comparisons among CKD stages were conducted using one-way ANOVA or the Kruskal–Wallis test. A p-value of <0.05 was considered statistically significant. Serum uric acid levels showed no significant correlation with CKD stage (r=0.025; p=0.861) or eGFR (r=0.093; p=0.523). In contrast, the SUA/SCr ratio demonstrated a strong negative correlation with CKD stage (r=–0.784; p<0.001) and a strong positive correlation with eGFR (r=0.784; p<0.001). The SUA/SCr ratio also decreased significantly with increasing CKD stage. The serum uric acid-to-creatinine (SUA/SCr) ratio demonstrates a stronger correlation with renal function compared to serum uric acid levels in patients with non-hemodialysis chronic kidney disease (CKD) stages 3, 4, and 5. This parameter may serve as a potentially more sensitive alternative biomarker for assessing declining renal function.

Keywords: chronic kidney disease; estimated glomerular filtration rate; hyperuricemia; renal function decline; serum uric acid; serum uric acid-to-creatinine ratio

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INTRODUCTION

Chronic kidney disease (CKD) represents a major global health problem with an increasing prevalence worldwide (Francis et al., 2024; Stevens et al., 2024). The growing burden of CKD has been observed particularly in patients with advanced stages of the disease, reflecting the cumulative impact of aging populations and the rising prevalence of non-communicable diseases (Francis et al., 2024; Hustrini et al., 2024). CKD is characterized by irreversible structural and functional abnormalities of the kidney, leading to progressive loss of nephron mass and eventual progression to end-stage kidney disease (Chen et al., 2019; Stevens et al., 2024). In addition to renal-related

complications, CKD is closely associated with cardiovascular disease and metabolic disturbances, contributing substantially to morbidity and mortality (Francis et al., 2024; Arzhan et al., 2021).
Uric acid is the final product of purine metabolism and has long been considered a biologically inert molecule (Saito et al., 2021). However, accumulating evidence suggests that uric acid may play an active role in renal injury through mechanisms involving oxidative stress, endothelial dysfunction, inflammation and activation of the renin-angiotensin-aldosterone system (Gherghina et al., 2022; Johnson et al., 2023). Elevated serum uric acid levels have been associated with hypertension, metabolic syndrome and progression of CKD (Copur et al., 2022; Waheed et al., 2021).

Nevertheless, the interpretation of serum uric acid levels in patients with advanced CKD remains challenging, as declining glomerular filtration markedly reduces uric acid excretion, leading to secondary hyperuricemia (Johnson et al., 2018; Johnson et al., 2023). Consequently, serum uric acid levels may not accurately reflect endogenous uric acid production or its pathogenic role in renal dysfunction (Johnson et al., 2023). To address this limitation, the serum uric acid-to-creatinine ratio (SUA/SCr) has been proposed as a parameter that adjusts uric acid levels to renal function (Chen et al., 2022; Silva et al., 2021). Several studies have suggested that this ratio may better reflect uric acid metabolism independent of renal clearance (Chen et al., 2022; Sengsuk et al., 2018). However, data regarding the relationship between SUA/SCr ratio and renal function in patients with advanced CKD remain limited (Silva et al., 2021; Chen et al., 2022). Therefore, the present study aimed to investigate the association between serum uric acid, SUA/SCr ratio and eGFR in patients with CKD stage 3–5

METHOD

Study design and population. The present analytical cross-sectional study was conducted at H. Adam Malik General Hospital, Medan, Indonesia, between May and November 2025. Adult patients (≥18 years of age) diagnosed with stage 3‑5 CKD were consecutively enrolled. Patients with acute kidney injury, active infection, malignancy or incomplete laboratory data were excluded.
Laboratory measurements. Venous blood samples were collected under standardized conditions. Serum uric acid and creatinine levels were measured using enzymatic methods on an automated clinical chemistry analyzer. The SUA/SCr ratio was calculated by dividing serum uric acid (mg/dL) by serum creatinine (mg/dL). Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation. Statistical analysis. Data normality was assessed using the Shapiro-Wilk test. Correlations were analyzed using Pearson or Spearman correlation tests, as appropriate. Comparisons among CKD stages were conducted using one-way ANOVA or Kruskal-Wallis tests. A p-value <0.05 was considered to indicate a statistically significant difference.

All laboratory analyses were performed following standardized pre-analytical, analytical and post-analytical procedures to ensure data reliability. Blood samples were collected once at patient admission, allowed to clot at room temperature, and centrifuged to obtain serum. Samples with evidence of hemolysis, lipemia or icterus were excluded from analysis. Serum uric acid and creatinine measurements were conducted using colorimetric and enzymatic or Jaffe-based methods, respectively, on a Cobas c 503 analyzer (Roche Diagnostics). Daily internal quality control and calibration were performed in accordance with the manufacturer's instructions using commercial control materials. All laboratory results were automatically recorded through the laboratory information system and linked to each participant's medical record number.

RESULT

A total of 50 patients with stage 3–5 chronic kidney disease (CKD) were included in this analytical cross-sectional study using a consecutive sampling method. All participants met the predefined inclusion and exclusion criteria. Data were collected between May and November 2025 at H. Adam Malik General Hospital, Medan, Indonesia. Laboratory assessments included serum uric acid, serum creatinine, and estimated glomerular filtration rate (eGFR), which were analyzed

to evaluate the relationship between serum uric acid, the serum uric acid-to-creatinine ratio (SUA/SCr), and renal function in patients with CKD. As presented in Table 1, the demographic characteristics and baseline laboratory findings of the study population are summarized, including CKD stage distribution, serum uric acid levels, serum creatinine levels, and SUA/SCr ratio categories.

Table 1.
Demographic characteristics and laboratory findings of patients with chronic kidney disease

Characteristic	**f** (%)
Sex	
Male	37 (74)
Female	13 (26)
Age group	
Adults (18–59 years)	24 (48)
Elderly (≥60 years)	26 (52)
Education level	
Elementary school	8 (16)
Junior high school	14 (28)
Senior high school	11 (22)
Diploma (D3)	2 (4)
Bachelor degree	15 (30)
CKD stage	
Stage 3A	10 (20)
Stage 3B	13 (26)
Stage 4	16 (32)
Stage 5	11 (22)
Serum uric acid	
Normal	10 (20)
Elevated	40 (80)
Serum creatinine	
Normal	1 (2)
Elevated	49 (98)
SUA/SCr ratio	
Low	20 (40)
Moderate	9 (18)
High	21 (42)

The demographic and laboratory characteristics of the study population are summarized in Table 1. The majority of patients were male (74%), and more than half were elderly (52%). Most patients were classified as CKD stage 4 (32%), followed by stage 3B (26%), stage 5 (22%), and stage 3A (20%). Laboratory findings demonstrated that 80% of patients had elevated serum uric acid levels with a mean value of 9.62 mg/dL, while 98% exhibited elevated serum creatinine levels with a mean of 3.15 mg/dL, reflecting impaired renal function. Regarding the SUA/SCr ratio, the highest proportion of patients fell into the high category (42%), followed by the low (40%) and moderate (18%) categories. As presented in Table 2, correlation analysis was subsequently performed to evaluate the association between serum uric acid levels and CKD stage and eGFR.

Table 2.
Correlation between serum uric acid and CKD stage and eGFR

Parameter	n	r	p-value
Serum uric acid vs CKD stage[a]	50	0.025	0.861
Serum uric acid vs eGFR[b]	50	0.093	0.523

[a]Spearman correlation

[b]Pearson correlation

Correlation analysis showed no significant association between serum uric acid levels and CKD stage ($r = 0.025$; $p = 0.861$) or eGFR ($r = 0.093$; $p = 0.523$), as presented in Table 2. Although the direction of correlation was positive, the strength was very weak and not statistically significant. These findings indicate that serum uric acid levels alone do not reliably reflect disease severity or renal function decline in patients with advanced CKD. In contrast, as shown in Table 3, the

SUA/SCr ratio demonstrated a strong and statistically significant correlation with both CKD stage and eGFR.

Table 3.
Correlation between SUA/SCr ratio and CKD stage and eGFR

Parameter	n	r	p-value
SUA/SCr vs CKD stage[a]	50	−0.784	<0.001
SUA/SCr vs eGFR[b]	50	0.784	<0.001

[a]Spearman correlation
[b]Pearson correlation

In contrast, the SUA/SCr ratio demonstrated a strong and statistically significant association with renal function. Spearman correlation analysis revealed a strong negative correlation between the SUA/SCr ratio and CKD stage ($r = -0.784$; $p < 0.001$), indicating that the ratio decreased progressively with advancing CKD stage. Pearson correlation analysis further showed a strong positive correlation between the SUA/SCr ratio and eGFR ($r = 0.784$; $p < 0.001$), suggesting that lower eGFR values were associated with lower SUA/SCr ratios. These results highlight the close relationship between the SUA/SCr ratio and CKD progression. As presented in Table 4, further comparative analysis using one-way ANOVA was performed to evaluate differences in serum uric acid levels and eGFR across CKD stages.

Table 4.
Comparison of serum uric acid and eGFR across CKD stages (one-way ANOVA)

Variable	Mean ± SD	p-value
Serum uric acid (mg/dL)		0.502
Stage 3A	7.76 ± 1.69	
Stage 3B	9.49 ± 3.11	
Stage 4	8.97 ± 2.79	
Stage 5	8.65 ± 2.16	
eGFR (mL/min/1.73 m²)		<0.001
Stage 3A	50.97 ± 5.62	
Stage 3B	36.05 ± 2.94	
Stage 4	22.55 ± 4.44	
Stage 5	10.12 ± 3.49	

One-way ANOVA demonstrated no significant differences in serum uric acid levels among CKD stages ($p = 0.502$), indicating that increasing disease severity was not consistently accompanied by changes in serum uric acid concentration. In contrast, eGFR values differed significantly across CKD stages ($p < 0.001$), with a marked decline observed as CKD stage advanced. This finding confirms the progressive deterioration of renal filtration capacity with worsening CKD. As presented in Table 5, further analysis using the Kruskal–Wallis test revealed significant differences in the SUA/SCr ratio across CKD stages.

Table 5.
Comparison of SUA/SCr ratio across CKD stages (Kruskal–Wallis test)

CKD stage	Median (min–max)	p-value
Stage 3A	5.71 (3.08–7.25)	<0.001
Stage 3B	5.10 (2.13–8.72)	
Stage 4	2.89 (1.54–5.32)	
Stage 5	1.33 (0.77–2.47)	

The Kruskal–Wallis test revealed a significant difference in SUA/SCr ratios across CKD stages ($p < 0.001$), with median values progressively decreasing from stage 3A to stage 5, as presented in Table 5. In contrast to the relatively stable serum uric acid levels observed across stages, this decline in the SUA/SCr ratio became more evident with increasing disease severity. This trend was further illustrated in Figure 1, which demonstrated relatively stable serum uric acid levels across CKD stages alongside a pronounced decline in the SUA/SCr ratio as CKD progressed. These findings suggest that the SUA/SCr ratio may serve as a more sensitive marker of renal function deterioration compared with serum uric acid alone.

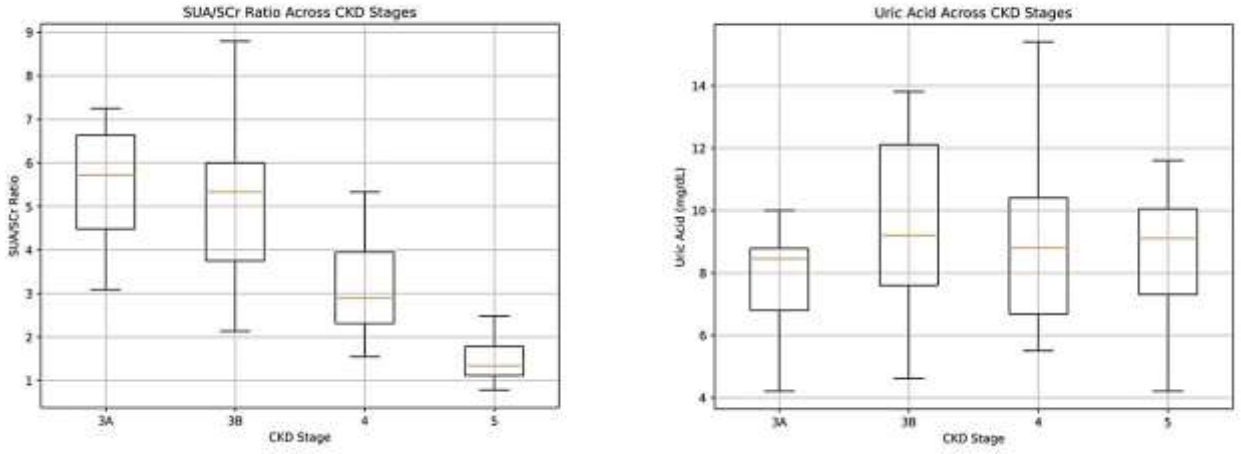

Figure 1. Box plot of serum uric acid and SUA/SCr ratio across CKD stages

DISCUSSION

This study evaluated the relationship between serum uric acid, the serum uric acid-to-creatinine ratio (SUA/SCr), and estimated glomerular filtration rate (eGFR) in patients with stage 3–5 chronic kidney disease (CKD) treated at H. Adam Malik General Hospital. A total of 50 patients were included, with distribution across CKD stages 3A, 3B, 4, and 5. The findings demonstrated that serum uric acid levels were not significantly associated with CKD stage or eGFR, whereas the SUA/SCr ratio showed a strong negative correlation with CKD stage and a strong positive correlation with eGFR. These results highlight the potential role of the SUA/SCr ratio as a more sensitive indicator of renal function decline in advanced CKD.

The demographic characteristics of the study population showed a predominance of male patients (74%), consistent with previous studies reporting a higher prevalence of CKD among men. Yunita et al. (2024) reported that CKD occurred more frequently in men than women among hemodialysis patients (59% vs. 41%), while Salsabila et al. (2023) observed a similar pattern with 68.9% male predominance. This sex-related difference may reflect biological and hormonal factors influencing CKD progression, as men tend to experience a more rapid decline in renal function compared with women.

Sex differences in CKD progression have been attributed to the protective effects of estrogen in women. Kattah and Garovic (2020) reported that estrogen reduces glomerulosclerosis and protects the kidney against ischemic injury, whereas the absence of this hormonal protection in men contributes to faster eGFR decline and a higher likelihood of progression to end-stage kidney disease. These mechanisms may partly explain the higher proportion of male patients observed in the present study.

Age distribution in this study showed that more than half of the patients were aged ≥60 years, reinforcing the well-established association between aging and CKD. Wu et al. (2023) reported that CKD prevalence increases markedly with age, rising from 2.7% in individuals aged 60–64 years to 71.9% in those older than 80 years. Similarly, Tang et al. (2024) demonstrated a significantly higher prevalence of CKD among elderly populations, while Flaherty et al. (2024) observed a sharp increase in CKD prevalence beyond the age of 65 years. Age-related structural and functional changes in the kidney, including nephron loss and reduced renal blood flow, render elderly individuals more susceptible to chronic renal injury.

Educational level also appeared to be an important sociodemographic factor, with 66% of patients having an education level of senior high school or below. Previous studies have shown that lower

educational attainment is associated with a higher risk of CKD due to reduced health literacy, limited access to healthcare information, and poorer disease management. The German CKD Cohort reported that lower education significantly increased the risk of CKD progression and adverse outcome (Winitzki et al., 2022). Similarly, national data from Riskesdas 2018 demonstrated that low educational level significantly increased CKD risk (OR = 1.307; $p < 0.001$) (Hustrini et al., 2022).

Regarding laboratory findings, most patients in this study exhibited elevated serum uric acid levels, with a mean value of 9.62 mg/dL. Hyperuricemia is commonly observed in CKD as both a consequence of impaired renal excretion and a potential contributor to renal damage. Elevated uric acid has been implicated in oxidative stress, activation of reactive oxygen species, tubulointerstitial inflammation, and renal vascular injury, all of which may accelerate CKD progression (Lee et al., 2021). Elevated serum creatinine levels were also observed in nearly all patients, reflecting reduced glomerular filtration and accumulation of nitrogenous waste products in advanced CKD (Avila et al., 2025).

Despite the high prevalence of hyperuricemia, correlation analysis revealed no significant association between serum uric acid levels and CKD stage or eGFR. This finding is consistent with previous studies demonstrating that serum uric acid levels do not differ significantly across advanced CKD stages. Khadka et al. (2018) reported no significant differences in serum uric acid levels among CKD stages 3, 4, and 5, likely due to uniformly impaired uric acid excretion at these stages. Similarly, Sah et al. (2023) found no significant variation in serum uric acid levels across CKD stages despite a high prevalence of hyperuricemia. These findings suggest that serum uric acid alone may not reliably reflect disease severity in advanced CKD.

In contrast, the SUA/SCr ratio demonstrated a strong and statistically significant association with both CKD stage and eGFR. The strong negative correlation between SUA/SCr and CKD stage and the positive correlation with eGFR indicate that the ratio declines progressively as renal function deteriorates. These results are consistent with those of Chen et al. (2022) who reported that SUA/SCr was positively correlated with eGFR and independently associated with reduced renal function. The physiological basis for this observation lies in the differential excretion mechanisms of uric acid and creatinine, with creatinine rising more rapidly than uric acid as glomerular filtration declines.

Similar findings have been reported by Baral et al.(2023) who demonstrated a significant correlation between SUA/SCr and eGFR, with lower ratios observed in patients with more advanced renal impairment. As creatinine is almost entirely eliminated via glomerular filtration, its serum concentration increases sharply with declining eGFR. In contrast, uric acid is eliminated through both renal and extra-renal pathways, resulting in a relatively slower increase. Consequently, the SUA/SCr ratio decreases as kidney function worsens, making it a sensitive indicator of renal function decline.

Taken together, these findings support the clinical utility of the SUA/SCr ratio as a supplementary marker for assessing CKD severity and progression. Compared with serum uric acid alone, the ratio better reflects renal functional decline in patients with advanced CKD. Nevertheless, this study has several limitations, including its single-center design, the absence of longitudinal follow-up, and the lack of adjustment for potential confounders such as diet, hydration status, and medication use. Despite these limitations, the present findings provide evidence that the SUA/SCr ratio may serve as a valuable and accessible biomarker for monitoring renal function deterioration in patients with CKD.

CONCLUSION

In conclusion, this study demonstrates that the serum uric acid-to-creatinine ratio (SUA/SCr) is strongly associated with renal function in patients with stage 3–5 chronic kidney disease. The SUA/SCr ratio showed a strong negative correlation with CKD stage and a strong positive correlation with estimated glomerular filtration rate (eGFR), indicating that lower SUA/SCr values are closely related to more advanced disease and greater renal function impairment. In contrast, serum uric acid levels alone were not significantly associated with either CKD stage or eGFR, despite the high prevalence of hyperuricemia among the study population.

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