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Original Article

Association of Serum Uric Acid with Renal Function Indicators in Patients with Chronic Kidney Disease: A Cross-Sectional Study

Zahoor Ilyas[1], Mazhar Abbas[1]

1. Jinnah Hospital, Lahore, Pakistan

*** Correspondence: zahoorilyas11@gmail.com**

ABSTRACT

Background: Chronic kidney disease is a progressive disorder characterized by declining renal function and metabolic disturbances. Hyperuricemia frequently occurs in patients with impaired kidney function and may reflect or contribute to renal dysfunction. Understanding the relationship between serum uric acid and traditional renal biomarkers may provide additional insights into metabolic changes associated with chronic kidney disease. Objective: To evaluate the association between serum uric acid and serum creatinine levels among patients with chronic kidney disease. Methods: A cross-sectional survey was conducted among 90 patients diagnosed with chronic kidney disease. Serum uric acid and serum creatinine levels were obtained from routine biochemical laboratory measurements. Descriptive statistics were calculated for biochemical parameters, and Pearson's correlation analysis was used to assess the association between serum uric acid and creatinine levels. Statistical analysis was performed using SPSS with a significance threshold of $p < 0.05$. Results: The mean serum uric acid level was 6.35 ± 1.42 mg/dL, while the mean serum creatinine concentration was 2.58 ± 1.36 mg/dL. Male patients constituted 57.8% of the study population. Stage 3 chronic kidney disease was the most prevalent stage (32.2%). Pearson correlation analysis demonstrated a statistically significant positive correlation between serum uric acid and serum creatinine levels ($r = 0.214$, $p = 0.043$). Conclusion: Serum uric acid levels showed a significant positive association with serum creatinine among patients with chronic kidney disease. These findings suggest that elevated uric acid may reflect metabolic alterations associated with declining renal function and may serve as a complementary biomarker in the clinical evaluation of CKD patients. Keywords: Chronic kidney disease, serum uric acid, creatinine, hyperuricemia, renal dysfunction, biomarkers, cross-sectional study.

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INTRODUCTION

Chronic kidney disease (CKD) represents a major global health concern characterized by progressive and irreversible deterioration of renal structure and function. It is clinically defined as abnormalities in kidney structure or function that persist for at least three months and are associated with declining glomerular filtration rate (GFR), metabolic disturbances, and increased risk of cardiovascular morbidity and mortality (1). The prevalence of CKD has increased steadily over recent decades due to the growing burden of hypertension, diabetes mellitus, and population aging, affecting approximately 10–13% of adults worldwide (2). The progressive nature of CKD often results in delayed clinical recognition until significant nephron loss has occurred, emphasizing the importance of identifying biochemical indicators that reflect renal impairment and disease progression.

Serum creatinine remains one of the most widely used laboratory markers for evaluating kidney function. Creatinine is a metabolic byproduct generated from muscle metabolism and is primarily cleared from the bloodstream through glomerular filtration (3). As renal function declines, creatinine excretion decreases, leading to accumulation in the blood and consequently serving as a surrogate indicator for reduced glomerular filtration rate (4). Measurement of serum creatinine therefore forms

the basis for estimating GFR and classifying the severity of CKD, which is critical for guiding clinical management and monitoring disease progression (5). However, creatinine alone may not fully reflect the complex metabolic alterations that occur during renal dysfunction, prompting investigation of additional biochemical markers that may complement renal function assessment.

Serum uric acid, the final product of purine metabolism, has emerged as an important metabolic marker associated with renal and cardiovascular disorders. Under normal physiological conditions, uric acid is filtered through the glomerulus, partially reabsorbed and secreted within renal tubules, and ultimately excreted through urine (6). In patients with impaired renal function, decreased uric acid clearance frequently results in elevated circulating levels, leading to hyperuricemia (7). Increasing evidence suggests that hyperuricemia may contribute not only as a consequence of renal dysfunction but also as a potential factor influencing kidney disease progression through mechanisms involving endothelial dysfunction, oxidative stress, inflammatory activation, and vascular smooth muscle proliferation (8). Experimental and clinical studies have indicated that elevated uric acid levels may stimulate inflammatory pathways and reduce nitric oxide availability, thereby contributing to renal microvascular injury and progressive nephron damage (9).

Several epidemiological investigations have explored the relationship between serum uric acid levels and renal function parameters. Observational studies have reported that higher uric acid concentrations are associated with reduced glomerular filtration rate and increased creatinine levels among individuals with varying stages of kidney disease (10). Longitudinal cohort studies have further demonstrated that hyperuricemia may independently predict the development and progression of CKD as well as increased risk of cardiovascular complications in affected patients (11). Despite these findings, inconsistencies remain regarding the strength of the association between uric acid and creatinine across different populations and clinical contexts, particularly in cross-sectional analyses where demographic and clinical factors may influence biochemical relationships.

Understanding the correlation between serum uric acid and creatinine may therefore provide additional insights into the metabolic disturbances accompanying renal dysfunction and may help clarify whether uric acid levels parallel the severity of kidney impairment. Establishing such relationships could assist clinicians in interpreting laboratory findings more comprehensively and potentially contribute to improved monitoring of CKD patients. However, evidence from certain regional populations remains limited, highlighting the need for further evaluation of biochemical correlations within specific clinical settings.

Therefore, the present study aimed to examine the correlation between serum uric acid and serum creatinine levels among patients diagnosed with chronic kidney disease using a cross-sectional survey design. The study sought to determine whether serum uric acid levels are significantly associated with creatinine concentrations and to evaluate the potential clinical relevance of this relationship in patients across different stages of CKD.

MATERIALS AND METHODS

A cross-sectional survey was conducted to evaluate the association between serum uric acid and serum creatinine levels among patients diagnosed with chronic kidney disease. The cross-sectional design was selected because it allows simultaneous assessment of biochemical variables and their statistical relationships within a defined patient population at a specific point in time, which is appropriate for evaluating correlations between metabolic markers in clinical research (12). The study was carried out in a hospital-based clinical setting where patients with suspected or confirmed kidney disease routinely undergo biochemical evaluation as part of diagnostic and follow-up care.

The study population consisted of adult patients diagnosed with chronic kidney disease according to established clinical criteria indicating persistent impairment of renal structure or function for a duration

of at least three months. Eligible participants included individuals who had documented CKD and available laboratory measurements of both serum uric acid and serum creatinine obtained during routine clinical investigations. Patients were included irrespective of CKD stage provided that complete biochemical data were available. Individuals with missing laboratory records for either of the two primary variables were not included in the final analysis to maintain data integrity and ensure reliable statistical evaluation.

Participants were recruited using a non-probability consecutive sampling approach whereby all eligible CKD patients presenting during the study period were invited to participate until the target sample size was achieved. A total of ninety patients met the inclusion criteria and were included in the final dataset. Data were obtained through review of patient clinical records and laboratory reports generated during routine diagnostic evaluation. This approach ensured that biochemical measurements reflected real-world clinical conditions while maintaining consistency with observational survey methodologies used in clinical epidemiological research (13).

Serum uric acid and serum creatinine levels constituted the primary variables of interest in the study. Serum uric acid concentration was measured in milligrams per deciliter (mg/dL) using standardized enzymatic colorimetric assays based on uricase-mediated reactions. Serum creatinine levels were measured in mg/dL using validated automated biochemical analyzers commonly employed in clinical laboratory practice. Both parameters were recorded as continuous variables for statistical analysis. In addition to biochemical measurements, demographic characteristics including patient sex and clinical classification of CKD stage were recorded to describe the study population and facilitate interpretation of findings in relation to disease severity.

CKD stages were categorized based on internationally accepted clinical classifications derived from estimated glomerular filtration rate values. These categories allowed assessment of the distribution of disease severity among participants and enabled interpretation of biochemical parameters across varying levels of renal impairment (14). Operational definitions of all variables were applied consistently during data extraction to ensure standardization and minimize measurement variability.

Several methodological procedures were implemented to reduce potential sources of bias and confounding. Laboratory measurements were performed using standardized automated systems calibrated according to institutional protocols to ensure accuracy and reproducibility of biochemical results. Data extraction procedures followed uniform criteria to ensure consistent interpretation of laboratory records. Inclusion of consecutive eligible patients minimized selection bias and improved representativeness of the CKD population within the clinical setting. Additionally, analysis focused on objective laboratory measurements rather than subjective clinical assessments, thereby reducing information bias.

The sample size of ninety participants was considered adequate for detecting statistically meaningful correlations between serum uric acid and creatinine levels based on effect sizes reported in previous observational studies investigating metabolic biomarkers in CKD populations (15). This sample size allowed estimation of correlation coefficients with sufficient statistical precision while remaining feasible within the clinical setting in which the study was conducted.

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS). Continuous variables were summarized using descriptive statistics including mean values and standard deviations, while categorical variables were summarized using frequencies and percentages. The primary objective of the analysis was to evaluate the strength and direction of the association between serum uric acid and serum creatinine levels. Pearson's correlation coefficient was calculated to determine the linear relationship between these two continuous biochemical parameters. Statistical significance was evaluated using a two-tailed test with a predefined significance threshold of $p < 0.05$.

To ensure data quality and reproducibility, laboratory results were verified against original reports before entry into the analytical dataset. Data cleaning procedures were conducted prior to statistical analysis to identify inconsistencies or entry errors. Missing values were minimized through inclusion criteria requiring complete biochemical measurements, thereby eliminating the need for imputation procedures and ensuring accuracy of correlation estimates.

Ethical considerations were observed throughout the conduct of the study. Patient confidentiality was maintained by anonymizing all extracted data and removing identifying information from the dataset prior to analysis. The study adhered to recognized ethical principles governing biomedical research involving human participants and followed institutional standards for responsible data handling and reporting (16). All research procedures were conducted in accordance with established ethical guidelines to ensure transparency, scientific integrity, and reproducibility of findings.

RESULTS

A total of 90 patients diagnosed with chronic kidney disease (CKD) were included in the analysis. Descriptive statistics of the primary biochemical variables demonstrated moderate variability across participants. The mean serum uric acid level was 6.35 ± 1.42 mg/dL, with observed values ranging from 3.20 mg/dL to 9.10 mg/dL. The mean serum creatinine concentration was 2.58 ± 1.36 mg/dL, with values ranging from 0.90 mg/dL to 7.40 mg/dL. These findings indicate a wide distribution of renal impairment among the study population.

Table 1. Descriptive Statistics of Serum Biochemical Parameters in CKD Patients (n = 90)

Parameter	Mean ± SD	Minimum	Maximum	95% CI of Mean
Serum Uric Acid (mg/dL)	6.35 ± 1.42	3.20	9.10	6.05 – 6.65
Serum Creatinine (mg/dL)	2.58 ± 1.36	0.90	7.40	2.29 – 2.87

The study population consisted of 52 male patients (57.8%) and 38 female patients (42.2%), demonstrating a slightly higher prevalence of CKD among males in the sample. The gender distribution did not significantly deviate from equal proportions based on chi-square analysis ($\chi^2 = 2.18$, $p = 0.139$), indicating that the observed difference was not statistically significant.

Table 2. Gender Distribution Among CKD Patients (n = 90)

Gender	Frequency	Percentage (%)	χ^2	p-value
Female	38	42.2		
Male	52	57.8	2.18	0.139
Total	90	100		

The distribution of CKD stages among participants demonstrated that Stage 3 CKD was the most common stage, accounting for 29 patients (32.2%), followed by Stage 2 with 20 patients (22.2%). Advanced disease stages were also represented, with Stage 4 observed in 16 patients (17.8%) and Stage 5 in 15 patients (16.7%). Early-stage disease was less frequent, with Stage 1 observed in 10 patients (11.1%). A chi-square goodness-of-fit test demonstrated significant variation in stage distribution ($\chi^2 = 13.27$, $p = 0.010$), indicating that CKD stages were not evenly distributed across the population.

Table 3. Distribution of CKD Stages Among Patients (n = 90)

CKD Stage	Frequency	Percentage (%)	Cumulative %	χ^2	p-value
Stage 1	10	11.1	11.1		
Stage 2	20	22.2	33.3		
Stage 3	29	32.2	65.5		
Stage 4	16	17.8	83.3		
Stage 5	15	16.7	100	13.27	0.010
Total	90	100	—		

Pearson correlation analysis was performed to assess the association between serum uric acid and serum creatinine levels. The analysis revealed a weak but statistically significant positive correlation between the two variables ($r = 0.214$, $p = 0.043$). The 95% confidence interval for the correlation coefficient ranged from 0.02 to 0.39, indicating that higher serum uric acid levels were modestly associated with increased creatinine concentrations among CKD patients.

Table 4. Pearson Correlation Between Serum Uric Acid and Serum Creatinine (n = 90)

Variables	Serum Uric Acid (mg/dL)	Serum Creatinine (mg/dL)
Serum Uric Acid (mg/dL)	1.000	0.214
Serum Creatinine (mg/dL)	0.214	1.000
Significance (2-tailed)	—	0.043
95% CI for r	—	0.02 – 0.39
N	90	90

Overall, the findings demonstrate that serum uric acid levels were moderately elevated among CKD patients and showed a statistically significant positive relationship with serum creatinine levels. Although the correlation coefficient indicates a relatively modest association, the statistically significant p-value suggests that increased uric acid concentrations tend to occur alongside worsening renal function as reflected by higher creatinine levels. The distribution of CKD stages further illustrates that a substantial proportion of patients were in moderate to advanced stages of renal disease, which may contribute to the observed metabolic disturbances.

DISCUSSION

Chronic kidney disease represents a progressive metabolic and systemic disorder in which disturbances of renal filtration, inflammatory signaling, and metabolic homeostasis interact to accelerate renal decline and increase cardiovascular risk. The present study investigated the association between serum uric acid and serum creatinine levels among patients with chronic kidney disease in order to evaluate whether uric acid levels parallel biochemical indicators of renal dysfunction. The findings demonstrated a statistically significant but modest positive correlation between serum uric acid and serum creatinine levels ($r = 0.214$, $p = 0.043$), suggesting that increasing uric acid concentrations tend to accompany worsening renal impairment. This observation supports the concept that hyperuricemia may reflect metabolic consequences of declining kidney function while potentially contributing to pathophysiological processes involved in CKD progression.

The observed mean serum uric acid concentration in this study population was moderately elevated relative to normal physiological ranges, which is consistent with previous clinical investigations reporting higher uric acid levels among patients with reduced renal function. As renal filtration declines, impaired excretion of uric acid leads to accumulation in the circulation, which may promote inflammatory and oxidative mechanisms that further exacerbate renal injury (17). Experimental studies have demonstrated that uric acid can stimulate endothelial dysfunction, activate pro-inflammatory pathways, and impair nitric oxide availability within renal microvasculature, thereby contributing to progressive nephron damage (18). These biological mechanisms provide a plausible explanation for the positive association observed between uric acid and creatinine levels in CKD populations.

Several epidemiological studies have similarly reported significant associations between uric acid levels and markers of renal function. Khadka et al. observed an inverse relationship between uric acid levels and glomerular filtration rate among CKD patients, suggesting that hyperuricemia may accompany progressive renal impairment (19). Likewise, Toyama et al. demonstrated that elevated uric acid concentrations were associated with declining kidney function in a large population cohort, even among individuals with initially normal or mildly reduced renal function (20). Longitudinal research has further suggested that hyperuricemia may independently predict CKD progression and increased cardiovascular risk, highlighting its potential role as a clinically relevant metabolic biomarker (21). The modest correlation observed in the present study aligns with these findings and indicates that uric acid levels may partially reflect the severity of renal dysfunction in CKD patients.

The distribution of CKD stages within the study population further supports the metabolic relationship observed between biochemical parameters. The majority of participants were classified within intermediate to advanced stages of CKD, particularly Stage 3 disease, which is commonly associated with progressive metabolic complications including impaired uric acid clearance. As renal function deteriorates, the kidney's ability to regulate purine metabolism and excrete uric acid becomes increasingly compromised, thereby leading to systemic accumulation of urate and associated metabolic

disturbances (22). Consequently, the observed correlation between serum uric acid and creatinine may represent a reflection of shared underlying renal pathophysiology rather than a purely independent biochemical relationship.

Despite the statistically significant association identified in this study, the magnitude of the correlation coefficient indicates that the relationship between uric acid and creatinine is relatively modest. This suggests that uric acid levels are influenced by multiple physiological and clinical factors beyond renal filtration alone, including dietary purine intake, metabolic syndrome, hypertension, and medication use. Previous studies have similarly reported variability in the strength of association between uric acid and renal function markers across different populations and clinical settings (23). Therefore, while uric acid may provide complementary information regarding metabolic disturbances in CKD, it should not be interpreted as an isolated indicator of renal impairment without consideration of additional clinical variables.

The findings of this study have several potential clinical implications. Monitoring serum uric acid levels alongside traditional renal function markers may provide additional insight into metabolic disturbances accompanying CKD progression. Elevated uric acid levels have been associated with increased cardiovascular risk among CKD patients, and early identification of hyperuricemia may support more comprehensive risk assessment and management strategies (24). Furthermore, the modest but significant association observed in this study reinforces the importance of integrating multiple biochemical indicators when evaluating renal function and disease progression.

Certain limitations should be considered when interpreting the findings of this study. The cross-sectional design limits the ability to establish causal relationships between uric acid levels and renal function decline. Longitudinal studies would be required to determine whether elevated uric acid contributes directly to CKD progression or primarily reflects impaired renal excretion. In addition, the sample size, although adequate for correlation analysis, may limit the generalizability of results to broader populations. Future research involving larger multicenter cohorts and prospective study designs may provide deeper insights into the mechanistic and prognostic role of uric acid in chronic kidney disease.

CONCLUSION

The present study demonstrated a statistically significant positive correlation between serum uric acid and serum creatinine levels among patients with chronic kidney disease, indicating that elevated uric acid concentrations tend to occur alongside worsening renal function. Although the observed association was modest, the findings suggest that serum uric acid may serve as a complementary biochemical marker reflecting metabolic alterations accompanying renal impairment. Monitoring uric acid levels in conjunction with traditional renal function indicators may contribute to improved understanding of metabolic disturbances in CKD and support more comprehensive clinical evaluation of patients with renal disease.

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