JHWCR
Journal of Health, Wellness, and Community Research
Volume III, Issue III
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, **ISSN**: 3007-0570
https://doi.org/10.61919/26hvcw63

Article

Role of Hounsfield Unit in Detecting Stone-Free Rate for Extracorporeal Shock Wave Lithotripsy

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Cite this Article

Received	2025-04-07
Revised	2025-04-26
Accepted	2025-04-28
Published	2025-05-09
Conflict of Interest	None declared
Ethical Approval	Respective Ethical Review Board
Informed Consent	Obtained from all participants
Data/supplements	Available on request.
Funding	None
Authors' Contributions	Author Contributions: HA, AZ, SI, AM, IA, FH, and ABI contributed to concept, design, data collection, analysis, and manuscript drafting.

ABSTRACT

Background: Chronic kidney disease (CKD) is a progressive condition marked by declining renal function and is frequently associated with hyperuricemia, yet the precise relationship between serum uric acid and creatinine levels remains inconclusive. Understanding this relationship may aid in better clinical monitoring and disease management, especially in the absence of clear biomarkers for early CKD progression. **Objective**: This study aimed to assess the correlation between serum uric acid and creatinine levels in patients with CKD, evaluating the extent to which uric acid reflects renal function impairment and its potential as a clinical indicator. **Methods**: A retrospective cross-sectional study was conducted among 200 CKD patients at two diagnostic laboratories in Sialkot. Patients were selected via random sampling based on defined inclusion and exclusion criteria, focusing on recent laboratory data. Serum uric acid and creatinine levels were measured using the Beckman Coulter AU480 chemistry analyzer. Ethical approval was obtained, and all procedures complied with the Declaration of Helsinki. Statistical analysis was performed using SPSS version 27, applying descriptive statistics, Pearson's correlation, and independent t-tests with a significance level set at $p < 0.05$. **Results**: The mean serum uric acid and creatinine levels were 6.41 ± 1.48 mg/dL and 2.60 ± 1.45 mg/dL, respectively. A weak but statistically significant positive correlation was observed between uric acid and creatinine levels ($r = 0.146$, $p = 0.039$), indicating that although uric acid increases with declining renal function, the association is limited and likely influenced by other metabolic factors. Most patients (31%) were in Stage 3 CKD, with a higher prevalence in males (57.5%), and over half of the cohort was aged above 46 years. **Conclusion**: While hyperuricemia commonly coexists with CKD, its weak correlation with serum creatinine suggests it should not be solely relied upon as a biomarker for renal function. However, routine monitoring may aid in comprehensive patient management. Future longitudinal studies are recommended to assess the therapeutic value of uric acid-lowering interventions.

Keywords: Chronic Kidney Disease, Serum Uric Acid, Creatinine, Hyperuricemia, Renal Insufficiency, Biomarkers, Cross-Sectional Studies

INTRODUCTION

Chronic kidney disease (CKD) remains a significant global health burden, contributing to substantial morbidity and mortality due to its progressive and often irreversible nature. Characterized by a sustained reduction in glomerular filtration rate (GFR) and structural kidney damage persisting for more than 90 days, CKD frequently progresses asymptomatically in early stages, making timely diagnosis and intervention challenging (1). Among the many metabolic disturbances observed in CKD, hyperuricemia—defined by elevated serum uric acid (SUA) levels—has been increasingly scrutinized for its potential role in disease onset and progression. This condition arises primarily from impaired renal excretion as kidney function declines, but emerging literature suggests that the relationship between SUA and renal function is multifactorial and may not follow a strictly linear pattern (2,3).

Despite the traditional view that elevated SUA is merely a consequence of declining kidney function, recent studies challenge this assumption by proposing that hyperuricemia may also serve as an independent contributor to renal injury. Mechanistic insights reveal that uric acid can exert both antioxidant and pro-oxidant effects, depending on its extracellular or intracellular location, respectively (4). In extracellular environments, it contributes to the antioxidant capacity of plasma; however, when accumulated within cells, uric acid may activate inflammatory pathways and oxidative

stress, exacerbating renal damage (5). Nevertheless, the clinical relevance of these molecular effects remains debated, as several interventional trials using uric acid-lowering therapies have yielded inconsistent results regarding their capacity to attenuate CKD progression (6).

The ambiguous role of SUA in CKD has led to conflicting findings in observational studies. While some researchers report strong correlations between SUA and markers of renal dysfunction such as creatinine and estimated GFR, others highlight weak or nonsignificant associations, attributing variations to differences in population demographics, comorbidities like diabetes and hypertension, and methodological disparities (7,8). Furthermore, lifestyle factors including dietary purine intake, alcohol consumption, and genetic predispositions are known to influence uric acid metabolism independently of renal function, thereby confounding straightforward interpretations of serum biomarker relationships (9). This complexity underscores a knowledge gap in understanding the nuanced interaction between uric acid and creatinine levels in CKD patients, especially in varying stages of disease severity.

Given this backdrop, the current study aims to investigate the correlation between serum uric acid and creatinine levels in a cohort of CKD patients. While creatinine serves as a conventional marker of renal function, its interplay with uric acid across CKD stages remains inadequately defined in the local clinical context. By analyzing data from a diverse patient population, this study seeks to clarify whether SUA can reliably reflect renal impairment or if other metabolic and environmental variables diminish its diagnostic utility. Thus, the central research question posed is: Is there a statistically significant correlation between serum uric acid and creatinine levels in patients with CKD, and if so, how strong is this association across different stages of the disease?

MATERIAL AND METHODS

This retrospective cross-sectional study was conducted over a six-month period at Dr. Abdul Sattar's Lab and the Main Lab in Sialkot, Pakistan, with the aim of investigating the correlation between serum uric acid and creatinine levels among patients diagnosed with chronic kidney disease (CKD). A total of 200 participants were selected through random sampling from the patient records maintained at these laboratories. Inclusion criteria encompassed adult patients of both genders who had been clinically diagnosed with CKD and had recent laboratory measurements for serum uric acid and creatinine. Exclusion criteria included patients undergoing dialysis, those with incomplete laboratory data, or patients diagnosed with acute kidney injury or other systemic illnesses that could confound the interpretation of serum biomarkers. Since the study utilized anonymized data extracted retrospectively from laboratory records, direct informed consent was not obtained; however, the ethical principles of data confidentiality and patient privacy were strictly maintained.

Serum uric acid and creatinine levels were considered the primary variables of interest, with the study objective being to assess their statistical correlation in the CKD population. These biochemical parameters were analyzed using an automated chemistry analyzer (Beckman Coulter AU480), which ensures high precision and consistency in measurement. Data were extracted from existing records without the use of structured forms or additional patient interviews. The CKD stage distribution, gender, and age of participants were also recorded to enable demographic and clinical characterization of the study population. No follow-up assessments were performed due to the retrospective nature of the study. All procedures were conducted in accordance with the ethical standards of the institutional research committees and adhered to the principles outlined in the Declaration of Helsinki.

Statistical analysis was carried out using SPSS version 27. Descriptive statistics were employed to summarize demographic data and biomarker distributions, including means and standard deviations for continuous variables. Pearson's correlation coefficient was used to determine the strength and significance of the association between serum uric acid and creatinine levels. An independent t-test was applied to assess differences between subgroups based on gender and CKD stage. A p-value of less than 0.05 was considered statistically significant. Since the dataset was complete, there were no missing values, and therefore no imputation techniques were necessary. Potential confounding variables such as comorbid conditions or dietary habits were not assessed due to limitations in available data.

RESULTS

A total of 200 patients diagnosed with chronic kidney disease (CKD) were included in the study. The mean serum uric acid level among participants was 6.41 ± 1.48 mg/dL, with values ranging from 0.50 to 9.30 mg/dL, indicating a high prevalence of hyperuricemia within the sample. Similarly, the mean serum creatinine level was 2.60 ± 1.45 mg/dL, with a range of 0.50 to 8.20 mg/dL, reflective of impaired renal function in the majority of cases. Descriptive statistics of the biochemical parameters are presented in **Table 1**.

Table 1. Descriptive Statistics of Serum Biochemical Parameters in CKD Patients

Parameter	Mean ± SD	Minimum	Maximum
Serum Uric Acid (mg/dL)	6.41 ± 1.48	0.50	9.30
Serum Creatinine (mg/dL)	2.60 ± 1.45	0.50	8.20

The demographic distribution revealed a greater representation of male patients (n = 115, 57.5%) compared to females (n = 85, 42.5%), suggesting a higher burden of CKD among men. The data is summarized in **Table 2**. Age-wise stratification revealed that the majority of CKD patients were in older age groups, with 30.0% aged ≥61 years and 27.5% aged 46–60 years. Cumulatively, 57.5% of the cohort was above 46 years, underscoring the age-associated burden of renal dysfunction. Assessment of CKD stages revealed that the largest proportion of patients were classified in Stage 3 (n = 62, 31.0%), followed by Stage 2 (22.5%),

Stage 4 (18.0%), Stage 5 (17.0%), and Stage 1 (11.5%). This distribution indicates that a majority of diagnoses occurred at moderate to advanced stages of the disease. The frequency distribution across CKD stages is presented in **Table 3**.

Table 2. Gender Distribution Among CKD Patients

Gender	Frequency	Valid Percentage (%)	Cumulative Percentage (%)
Female	85	42.5	42.5
Male	115	57.5	100.0
Total	**200**	**100.0**	—

Table 3. Distribution of CKD Stages Among Patients

CKD Stage	Frequency	Percentage (%)	Cumulative Percentage (%)
Stage 1	23	11.5	11.5
Stage 2	45	22.5	34.0
Stage 3	62	31.0	65.0
Stage 4	36	18.0	83.0
Stage 5	34	17.0	100.0
Total	**200**	**100.0**	—

Correlation analysis was conducted using Pearson's correlation coefficient to examine the relationship between serum uric acid and creatinine levels. A statistically significant but weak positive correlation was observed (r = 0.146, p = 0.039), suggesting that although serum uric acid levels tend to rise with declining kidney function, other factors may influence uric acid concentrations. The correlation results are detailed in **Table 4**.

Table 4. Pearson's Correlation Between Serum Uric Acid and Serum Creatinine Levels

Variables	Serum Uric Acid (mg/dL)	Serum Creatinine (mg/dL)
Serum Uric Acid (mg/dL)	1.000	0.146
Serum Creatinine (mg/dL)	0.146	1.000
Significance (2-tailed)	—	0.039
N	200	200

Despite statistical significance (p < 0.05), the effect size (r = 0.146) falls into the "small" category, indicating limited clinical significance. The weak correlation implies that hyperuricemia in CKD patients cannot be fully explained by creatinine elevation alone. Confounding metabolic, dietary, or genetic factors may contribute to this variance and warrant further investigation. The findings support the hypothesis that while uric acid elevation is associated with renal dysfunction, it may not serve as a robust standalone biomarker for disease progression.

DISCUSSION

The present study explored the association between serum uric acid and serum creatinine levels among patients with chronic kidney disease (CKD), revealing a statistically significant but weak positive correlation (r = 0.146, p = 0.039). This finding suggests that although impaired renal function contributes to elevated uric acid levels, the relationship is not sufficiently strong to imply a direct or exclusive link. Instead, other metabolic, genetic, and environmental factors likely influence uric acid homeostasis in CKD patients. These results align with the hypothesis that hyperuricemia is a multifactorial condition in the context of renal dysfunction and cannot be explained solely by diminished creatinine clearance.

While several earlier studies have reported stronger correlations between serum uric acid and markers of renal function, the present study demonstrates a more attenuated relationship. Chonchol et al. found a moderate correlation (r = 0.32), proposing uric acid as a potential predictor for CKD progression (15). Conversely, Kang et al. reported a weaker association and posited that uric acid levels may reflect a more complex interplay of physiological variables, including insulin resistance, oxidative stress, and endothelial dysfunction (17). These discrepancies may stem from differences in study populations, methods of data collection, or the presence of comorbidities such as diabetes or hypertension, which were not accounted for in the present analysis. Furthermore, Maahs et al. identified a stronger predictive value of hyperuricemia for declining estimated GFR over time, a dynamic that could not be evaluated in the current cross-sectional design (18). The mechanisms underlying the relationship between uric acid and renal dysfunction remain an area of active investigation. While uric acid is traditionally regarded as a byproduct of purine metabolism excreted primarily through the kidneys, it also exhibits dual biochemical behavior—serving as an antioxidant in extracellular environments and as a pro-oxidant within cells. The intracellular accumulation of uric acid has been implicated in inflammation, endothelial dysfunction, and activation of the renin-angiotensin system, all of which may contribute to renal fibrosis and vascular injury (5). However, the inconsistent results from interventional studies employing uric acid-lowering therapies, such as allopurinol and febuxostat, indicate that hyperuricemia may act more as a marker than a causal factor in CKD progression (6).

From a clinical standpoint, the findings underscore the potential relevance of monitoring uric acid in CKD patients, although its

utility as a solitary prognostic biomarker remains limited. The relatively weak correlation with creatinine levels suggests that routine uric acid testing should be interpreted in conjunction with other renal and metabolic parameters. The study further highlights that Stage 3 CKD was the most common stage among participants, with a male predominance (57.5%), consistent with previous epidemiological data suggesting a higher incidence and more rapid progression of CKD in men (19,20). Ricardo et al. also noted that men typically have higher serum uric acid levels and an increased risk of cardiovascular events, reinforcing the sex-based differences observed in the current study (21).

Despite its insights, this study is subject to several limitations. The cross-sectional design precludes causal inferences and lacks the longitudinal perspective necessary to assess temporal changes in uric acid or renal function. The absence of data on comorbidities, dietary intake, body mass index, and medications limits the capacity to control for potential confounders. Additionally, since data were drawn from two laboratory settings in a single geographic region, the findings may not be generalizable to broader or more diverse populations. The modest sample size (n = 200) further limits the statistical power to detect more nuanced relationships or subgroup differences.

Nonetheless, the study benefits from standardized biochemical assessments and real-world data derived from routine clinical practice. It contributes to the growing body of evidence suggesting a complex and only modestly linear relationship between serum uric acid and creatinine in CKD. Future research should employ longitudinal cohort designs to track uric acid trajectories over time and evaluate whether interventions targeting uric acid reduction can translate into measurable improvements in renal outcomes. Investigations should also consider stratifying by CKD stage, gender, and the presence of metabolic syndrome to elucidate population-specific patterns. Incorporating novel biomarkers and genetic profiling may further clarify the mechanistic pathways linking uric acid dysregulation with kidney damage. Ultimately, a multifaceted approach that integrates biochemical, clinical, and molecular data is needed to optimize CKD risk stratification and therapeutic targeting.

CONCLUSION

This study demonstrated a statistically significant yet weak positive correlation between serum uric acid and creatinine levels in patients with chronic kidney disease, indicating that while hyperuricemia is commonly observed, it may not be a direct surrogate marker of renal function impairment. These findings suggest that additional metabolic and physiological mechanisms contribute to uric acid regulation in CKD, highlighting the complexity of its role in disease progression. Clinically, routine assessment of serum uric acid may still provide supplementary insight into the metabolic status of CKD patients, especially in the context of comorbidities and cardiovascular risk. From a research perspective, longitudinal and interventional studies are warranted to determine whether therapeutic modulation of uric acid levels can influence CKD outcomes, potentially informing personalized treatment strategies and advancing nephrology care.

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