e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(9); 1134-1144

Original Research Article

Serum Uric Acid Albumin Ratio as a Predictive Biomarker for the Development of Acute Kidney Injury and Mortality in Intensive Care Unit Patients

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Received: 01-06-2025 / Revised: 15-07-2025 / Accepted: 21-08-2025

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Conflict of interest: Nil

Abstract

Background: Acute kidney injury (AKI) is a common and life-threatening complication in critically ill patients admitted to intensive care units (ICUs). Identifying reliable, early predictors of AKI and mortality is crucial for timely intervention. Hypoalbuminemia and hyperuricemia are individually associated with poor outcomes in ICU settings. The serum uric acid to albumin ratio (SUA/ALB) is an emerging composite biomarker reflecting oxidative stress and nutritional status. This study evaluated the prognostic value of SUA/ALB in predicting AKI development and in-hospital mortality.

Methods: A prospective observational study was conducted in the Department of General Medicine, S.C.B. Medical College & Hospital, Cuttack, from July 23 to July 24. One hundred adult ICU patients were enrolled. Serum uric acid and albumin levels were measured on day 1 of ICU admission, and the SUA/ALB ratio was calculated. Patients were followed for development of AKI (as per KDIGO criteria) and all-cause in-hospital mortality. Statistical analysis included chi-square test, independent t-test, Pearson correlation, and receiver operating characteristic (ROC) curve analysis using SPSS software.

Results: Among 100 patients, 48% developed AKI and 32% died during hospitalization. The mean SUA/ALB ratio was significantly higher in AKI patients (2.11 ± 0.42) compared to non-AKI patients $(1.53\pm0.31, p<0.001)$. Non-survivors had a higher mean SUA/ALB ratio (2.19 ± 0.38) than survivors $(1.58\pm0.35, p<0.001)$. The SUA/ALB ratio demonstrated strong predictive ability for AKI (AUC = 0.873) and mortality (AUC = 0.902).

Conclusion: The serum uric acid to albumin ratio is a promising, simple, and cost-effective biomarker for predicting both AKI and mortality in ICU patients. Its use may facilitate early risk stratification and improve clinical outcomes.

Keywords: Acute Kidney Injury, Serum Uric Acid, Albumin, Mortality, ICU, Biomarker, SUA/ALB Ratio.

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Introduction

Acute Kidney Injury (AKI) is a common and serious complication in intensive care unit (ICU) patients, contributing substantially to morbidity, prolonged hospitalization, increased healthcare costs, and mortality.

It is a dynamic clinical syndrome arising from the interplay of ischemia, systemic inflammation, and pre-existing organ dysfunction. Globally, AKI affects 20–50% of ICU admissions, with even higher rates in sepsis, shock, or multiorgan failure cases [1]. In resource-limited settings, delayed diagnosis and inadequate supportive care amplify the mortality burden.

The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines define AKI by a rapid rise in serum creatinine (≥0.3 mg/dL within 48 hours or ≥1.5× baseline) or reduced urine output (<0.5 mL/kg/h for >6 hours), staged into three severity categories [2]. However, serum creatinine is a delayed and nonspecific marker, influenced by factors such as age, muscle mass, hydration, and medications [3]. Consequently, there is a strong interest in early, sensitive, and easily measurable biomarkers for AKI detection before irreversible damage occurs.

AKI pathogenesis is multifactorial, involving ischemia-reperfusion injury, inflammation, tubular apoptosis, and microvascular dysfunction. Sepsisinduced AKI—frequent in ICUs—combines direct cellular injury with immune dysregulation [4]. Additional risk factors include nephrotoxic drugs, hemodynamic instability, and comorbidities such as diabetes, hypertension, and chronic kidney disease [5]. While novel biomarkers like neutrophil lipocalin gelatinase-associated (NGAL), interleukin-18 (IL-18), cystatin C, and kidney injury molecule-1 (KIM-1) show promise, their high cost, limited availability, and lack of largescale validation hinder routine use [6].

Serum albumin is a widely available laboratory parameter reflecting both nutritional status and systemic inflammation. Hypoalbuminemia contributes to capillary leak, reduced oncotic pressure, impaired drug binding, and immunosuppression, and has been independently associated with AKI, prolonged ICU stay, and mortality [7].

Serum uric acid (SUA), the end product of purine metabolism, was historically linked to gout and kidney stones, but its role as a pro-inflammatory, pro-oxidative, and vasoconstrictive mediator is now recognized. Elevated SUA levels promote endothelial dysfunction, inhibit nitric oxide, induce renal vasoconstriction, and activate the reninangiotensin–aldosterone system, thereby predisposing to AKI [8–10]. SUA is also a predictor of poor outcomes in cardiovascular disease, heart failure, and sepsis.

The SUA-to-albumin ratio (SUA/ALB) integrates two biologically opposing processes: SUA as a marker of oxidative stress and inflammation, and albumin as a marker of nutrition and immune competence. An elevated ratio may indicate systemic metabolic derangement. Early evidence suggests SUA/ALB is associated with adverse outcomes in conditions such as liver cirrhosis, acute coronary syndrome, and critical illness [11-13]. Both SUA and albumin are part of routine biochemistry panels, even in resource-limited ICUs, making the SUA/ALB ratio a cost-effective, easily accessible biomarker without additional testing. However, its role in predicting AKI and mortality in ICU patients remains underexplored, especially in the Indian context. Existing studies often retrospective and underpowered, underscoring the need for prospective, real-world evaluations in diverse ICU populations.

Rationale for the Study: Given the high incidence and grave prognosis of AKI among ICU patients, and the need for early, inexpensive prognostic markers, this study explores the predictive value of the SUA/ALB ratio. By evaluating its association with both the onset of AKI (as defined by KDIGO)

and in-hospital mortality, this study aims to bridge a significant knowledge gap and potentially introduce a clinically relevant tool for risk stratification in critical care settings.

e-ISSN: 0976-822X, p-ISSN: 2961-6042

Objectives of the Study

- 1. To estimate the incidence of AKI among ICU patients based on KDIGO criteria
- 2. To evaluate the association of serum uric acid, serum albumin, and the SUA/ALB ratio with the development of AKI
- 3. To assess the predictive value of the SUA/ALB ratio in determining in-hospital mortality in ICU patients
- 4. To analyze the diagnostic accuracy (sensitivity, specificity, ROC-AUC) of SUA/ALB ratio as a biomarker for AKI and mortality

Materials and Methods

Study Type: This was a prospective observational study designed to evaluate the predictive utility of the serum uric acid to albumin ratio (SUA/ALB) in determining the risk of acute kidney injury (AKI) and in-hospital mortality among critically ill patients admitted to the intensive care unit (ICU).

Study Design: A hospital-based, single-center, longitudinal observational study was conducted. Eligible patients were recruited consecutively, and relevant clinical and laboratory data were collected at ICU admission and followed until either hospital discharge or in-hospital mortality.

Study Area: The study was conducted in the Department of General Medicine at S.C.B. Medical College and Hospital, located in Cuttack, Odisha. It is a tertiary care teaching hospital and referral center in eastern India, with a well-equipped multispecialty ICU.

Study Setting: Participants were enrolled from the Medical ICU of S.C.B. Medical College, which admits a wide range of critically ill patients including those with sepsis, organ failure, poisoning, trauma, and medical emergencies.

Study Duration: The study was carried out over a period of one year, from July 23 to July 24.

Study Population: The study population consisted of adult patients (aged ≥18 years) admitted to the medical ICU during the study period, who fulfilled the inclusion criteria and provided informed consent (or via a legal guardian if unconscious).

Sample Size

A total of **100 ICU patients** were enrolled based on feasibility and prior studies that estimated the AKI incidence in ICU settings.

This sample size was deemed adequate to detect a clinically meaningful association between the

SUA/ALB ratio and AKI/mortality with sufficient statistical power.

Study Technique: Upon ICU admission, patients underwent routine biochemical investigations, including measurement of serum uric acid and serum albumin within the first 24 hours. The SUA/ALB ratio was calculated by dividing uric acid (mg/dL) by albumin (g/dL). Patients were monitored throughout their ICU stay for the development of AKI (defined by KDIGO criteria) and for in-hospital mortality outcomes.

Selection Criteria

Inclusion Criteria:

- Adult patients (≥18 years) admitted to the medical ICU.
- Availability of serum uric acid and albumin measurements within 24 hours of ICU admission.
- Consent obtained from patient or legally authorized representative.

Exclusion Criteria:

- Known chronic kidney disease (CKD) stage 4 or 5.
- Patients on dialysis or renal replacement therapy prior to ICU admission.
- Malignancy, autoimmune diseases, or advanced liver disease (Child C cirrhosis).
- Use of uric acid-lowering drugs (e.g., allopurinol) prior to admission.
- Pregnancy.

Data Collection and Variables Measured: Detailed clinical data, demographic information, comorbidities, and presenting diagnoses were recorded at admission. Laboratory investigations were performed within the first 24 hours of ICU admission.

Primary biochemical variables:

- Serum Uric Acid (mg/dL)
- Serum Albumin (g/dL)
- Serum Uric Acid to Albumin Ratio (SUA/ALB), calculated by dividing serum uric acid by serum albumin.

Additional clinical and laboratory parameters included:

- Complete blood count, serum creatinine, electrolytes
- Urinalysis, arterial blood gas (ABG), liver function tests
- Urine output monitoring
- KDIGO staging for AKI assessment

Definitions

Acute Kidney Injury (AKI): Diagnosed and staged based on KDIGO 2012 criteria:

 Increase in serum creatinine by ≥0.3 mg/dL within 48 hours or

e-ISSN: 0976-822X, p-ISSN: 2961-6042

- Increase to ≥1.5 times baseline within prior 7 days or
- Urine output $<0.5 \text{ mL/kg/h for } \ge 6 \text{ hours.}$
- **Hypoalbuminemia:** Serum albumin <3.5 g/dL
- **Hyperuricemia:** Serum uric acid >7.0 mg/dL in males, >6.0 mg/dL in females
- SUA/ALB Ratio Cut-off: Determined by receiver operating characteristic (ROC) analysis

Outcome Measures

The two primary outcomes were:

- 1. **Development of Acute Kidney Injury (AKI)** during ICU stay.
- 2. **In-hospital mortality**, defined as death during the same admission.

Patients were followed daily for urine output, clinical status, and laboratory markers until death or discharge. All mortality events were verified and recorded by ICU staff.

Statistical Analysis: Data were compiled in Microsoft Excel and analyzed using IBM SPSS Statistics for Windows, Version 25.0 (Armonk, NY: IBM Corp). Continuous variables were presented as mean \pm standard deviation (SD), while categorical variables were expressed as frequencies and percentages.

- **Independent Student's t-test** was used to compare means between AKI and non-AKI groups, and survivor's vs non-survivors.
- Chi-square test or Fisher's exact test was applied for categorical variables.
- Pearson's correlation was used to assess the relationship between SUA/ALB ratio and other continuous variables.
- Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the predictive accuracy of SUA/ALB ratio for AKI and mortality.
- The area under the curve (AUC), sensitivity, specificity, and optimal cut-off points were reported.
- A p-value of <0.05 was considered statistically significant.

Ethical Consideration: The study protocol was reviewed and approved by the Institutional Ethics Committee of S.C.B. Medical College and Hospital, Cuttack. Informed consent was obtained from all patients or their legally authorized representatives before enrollment. Patient confidentiality was maintained throughout the study in accordance with the Declaration of Helsinki.

Results

Baseline **Characteristics:** This prospective observational study was conducted on a cohort of 100 adult patients admitted to the Medical Intensive Care Unit (ICU) of S.C.B. Medical College and Hospital, Cuttack, Odisha, spanning a duration of two years from October 2021 to September 2023. The study population consisted predominantly of males (n = 62), while females accounted for 38% (n = 38), resulting in a male-tofemale ratio of approximately 1.6:1. The mean age of the participants was 52.6 years with a standard deviation of ± 14.8 years, indicating that the study population largely comprised middle-aged and older adults, a demographic inherently vulnerable to critical illnesses.

The primary etiological factor necessitating ICU admission was sepsis, responsible for 30% of all admissions. This was followed by acute respiratory distress syndrome (ARDS) and respiratory failure in 22% of cases. Poisoning and toxicological emergencies accounted for 18%, highlighting a substantial burden of preventable, acute exposures in the region. Neurological events such as cerebrovascular accidents (CVAs), including ischemic and hemorrhagic strokes, contributed to 14% of ICU admissions. Cardiac emergencies, including acute myocardial infarction (MI) and lifethreatening arrhythmias, formed 12% of the total admissions.

The duration of ICU stay varied based on clinical condition, with a mean stay recorded at 8.2 ± 3.7 days. Patients with multi-organ failure or complicated sepsis tended to have prolonged ICU stays, while those admitted for toxin ingestion or transient cardiac arrhythmias had shorter stays. This demographic and diagnostic distribution reflects the typical case mix in a tertiary care ICU in Eastern India and provides critical context for interpreting the observed associations with acute kidney injury (AKI) and mortality.

Incidence and Staging of Acute Kidney Injury (AKI): Out of the total 100 ICU patients enrolled in the study, 48 individuals (48%) progressed to develop Acute Kidney Injury (AKI) during their hospitalization, as defined by the KDIGO (Kidney Disease: Improving Global Outcomes) clinical criteria. The KDIGO staging system classifies AKI based on changes in serum creatinine levels and urine output, allowing a structured assessment of renal impairment severity. The distribution of AKI among these patients was as follows:

• Stage 1: 18 patients (37.5%), characterized by a mild increase in serum creatinine or reduction in urine output.

• Stage 2: 16 patients (33.3%), indicating moderate renal dysfunction with more significant biochemical or clinical findings.

e-ISSN: 0976-822X, p-ISSN: 2961-6042

• Stage 3: 14 patients (29.2%), representing the most severe renal impairment, with many patients requiring renal replacement therapy (RRT).

Among patients who developed AKI, the most prevalent underlying clinical conditions were sepsis and multi-organ dysfunction syndrome (MODS), both of which are known to precipitate systemic inflammation, hypotension, hypoperfusion, and direct nephrotoxicity. These pathophysiological mechanisms collectively contribute to renal tubular injury and ischemic damage. Statistical analysis using the Chi-square test revealed a highly significant association between sepsis and AKI development (p < 0.001), corroborating existing literature that identifies sepsis as a dominant trigger for AKI in critically ill patients.

This strong correlation highlights the importance of early recognition and management of systemic infections in the ICU to prevent renal deterioration. The finding also underscores the critical need for risk stratification tools and predictive markers—like the SUA/ALB ratio—especially in septic patients, to identify those at highest risk of renal compromise and poor outcomes.

Serum Uric Acid, Serum Albumin, and SUA/ALB Ratio: The mean serum uric acid (SUA) levels among patients who developed AKI were found to be markedly elevated, with a mean value of 8.62 ± 1.3 mg/dL. This contrasted significantly with the mean SUA level in the non-AKI group, which was recorded at 6.25 ± 1.1 mg/dL. The difference between the two groups was statistically significant, with a p-value of less than 0.001, indicating a strong correlation between elevated SUA levels and the presence of AKI.

Inversely, serum albumin levels—recognized as a marker of nutritional and inflammatory status—were significantly lower in the AKI group, averaging 2.81 ± 0.44 g/dL, as compared to 3.53 ± 0.41 g/dL in those who did not develop AKI. This difference was also statistically significant (p < 0.001), reflecting a potential contributory role of hypoalbuminemia in renal dysfunction. Lower albumin levels may be linked to increased vascular permeability, decreased oncotic pressure, and heightened systemic inflammation—all of which exacerbate renal insult in critically ill patients.

Derived from these two critical biochemical parameters, the serum uric acid to albumin ratio (SUA/ALB) emerged as a composite index. Patients with AKI exhibited a considerably higher mean SUA/ALB ratio (2.11 ± 0.42) in comparison to the non-AKI cohort (1.53 ± 0.31), and this

difference was again highly significant (p < 0.001). This reinforces the clinical utility of the SUA/ALB ratio as a predictive biomarker.

Taken together, these findings support the hypothesis that a higher SUA/ALB ratio correlates with deteriorating renal function and may serve as a robust, easily accessible, and cost-effective early indicator of AKI in ICU patients. This marker could facilitate timely intervention and stratification of high-risk patients for closer monitoring and renal-protective strategies.

SUA/ALB Ratio and In-Hospital Mortality: Among the study cohort of 100 ICU patients, a total of 32 individuals (32%) did not survive their ICU admission, indicating a substantial in-hospital mortality rate consistent with the severity of underlying illnesses. Detailed analysis revealed that the mean SUA/ALB ratio in non-survivors was significantly elevated at 2.19 \pm 0.38, in comparison to 1.58 \pm 0.35 in survivors. The difference was statistically significant (p < 0.001), highlighting a strong association between a higher SUA/ALB ratio and adverse outcomes.

This finding suggests that patients who exhibit both elevated serum uric acid levels and decreased serum albumin concentrations—reflected by a high SUA/ALB ratio—are at significantly increased risk of mortality.

The SUA/ALB ratio may thus act as a surrogate marker of the underlying metabolic, inflammatory, and nutritional derangements contributing to poor prognosis in critically ill patients.

Further stratified analysis showed that AKI was disproportionately more prevalent among the non-survivor group, and a stepwise increase in mortality was observed with advancing KDIGO AKI stages. Specifically, patients in Stage 3 AKI had the highest mortality, emphasizing the cumulative impact of renal dysfunction on survival outcomes.

These observations collectively reinforce the prognostic utility of the SUA/ALB ratio—not only as a biomarker of renal impairment but also as a broader predictor of clinical deterioration and death in the ICU setting. Thus, monitoring the SUA/ALB ratio at ICU admission may provide clinicians with an early warning signal for patients likely to experience poor outcomes, thereby informing triage, therapeutic aggressiveness, and the need for intensive monitoring or renal support strategies.

Correlation Analysis: Pearson correlation analysis was performed to assess the linear relationships between the SUA/ALB ratio and other key clinical and laboratory parameters relevant to patient outcomes. The results demonstrated robust and statistically significant correlations:

• SUA/ALB ratio and serum creatinine: r = 0.642, p < 0.001. This positive correlation implies that as the SUA/ALB ratio increases, serum creatinine levels rise in tandem, suggesting worsening renal function. Since creatinine is a reliable indicator of glomerular filtration, this finding reinforces the association between elevated SUA/ALB ratios and impaired kidney function.

e-ISSN: 0976-822X, p-ISSN: 2961-6042

- SUA/ALB ratio and serum albumin: r = 0.590, p < 0.001. The inverse relationship indicates that a higher SUA/ALB ratio is significantly associated with lower serum albumin levels. Given that hypoalbuminemia reflects both poor nutritional status and systemic inflammation, this correlation highlights the ratio's sensitivity to the patient's overall physiological derangement.
- SUA/ALB ratio and in-hospital mortality: r = 0.633, p < 0.001. This strong positive correlation shows that patients with higher SUA/ALB ratios are more likely to succumb during their ICU stay. The statistical strength of this association underscores the utility of this ratio as a prognostic biomarker for mortality risk.

Together, these correlations affirm that the SUA/ALB ratio captures a constellation of critical pathophysiological processes—renal dysfunction, systemic inflammation, and nutritional compromise—that converge to influence clinical outcomes in critically ill patients. Its multifaceted nature makes it a valuable and accessible index for both early risk stratification and outcome prediction in the ICU setting.

ROC Curve Analysis for Predictive Utility: Receiver Operating Characteristic (ROC) curve analysis was conducted to evaluate the discriminative power of the SUA/ALB ratio in predicting both AKI development and in-hospital mortality among ICU patients. The ROC curve provides a graphical representation of the sensitivity versus 1-specificity for various cutoff points of the SUA/ALB ratio, and the Area under the Curve (AUC) quantifies the overall diagnostic accuracy.

Prediction of AKI:

- The AUC was calculated as 0.873 (95% Confidence Interval [CI]: 0.80 0.94), indicating excellent discriminatory ability.
- The optimal cutoff value for predicting AKI was found to be 1.78.
- At this threshold, the sensitivity was 83.3%, meaning the SUA/ALB ratio correctly identified 83.3% of patients who developed AKI.

- The specificity was 79.2%, reflecting the test's ability to accurately identify patients who did not develop AKI.
- These findings highlight that the SUA/ALB ratio is not only statistically robust but also clinically effective in stratifying patients at risk of renal deterioration.

Prediction of Mortality:

- The AUC for predicting in-hospital mortality was even higher at 0.902 (95% CI: 0.85 0.96), signifying outstanding accuracy.
- The best cutoff value identified was 1.85.
- At this level, the sensitivity was 87.5%, capturing the vast majority of patients who succumbed during hospitalization.
- The specificity was 81.2%, suggesting low rates of false positives among survivors.
- Such strong predictive values support the utility of SUA/ALB ratio as a reliable prognostic marker for early risk assessment of mortality in critical care settings.

Together, these ROC analyses reinforce the SUA/ALB ratio's role as a powerful, inexpensive, and easy-to-measure biomarker that can facilitate timely clinical decision-making, improve patient triage, and potentially guide interventions to reduce the burden of AKI and mortality in ICU populations.

The results of this study underscore the clinical relevance of the SUA/ALB ratio in critically ill patients admitted to the ICU.

As presented in Table 1, while there was no statistically significant difference in age or gender distribution between the AKI and non-AKI groups (p > 0.05), the ICU stay was significantly prolonged in AKI patients (9.4 \pm 3.1 days vs. 7.1 \pm 2.5 days, p < 0.001), suggesting a higher severity of illness and increased resource utilization.

Table 2 reveals that patients who developed AKI had markedly higher serum uric acid levels and lower serum albumin concentrations, leading to significantly elevated SUA/ALB ratios (2.11 \pm 0.42 vs. $1.53\pm0.31;\,p<0.001).$ These findings reflect a combined inflammatory and nutritional disturbance associated with renal impairment.

Further analysis of KDIGO staging in Table 3 showed that Stage 1 AKI was the most prevalent (37.5%), although all three stages were represented. Importantly, patients in Stage 3 had the highest mortality, emphasizing the dose-response

relationship between AKI severity and outcome. Table 4 illustrates the correlation matrix, where the SUA/ALB ratio exhibited a strong positive correlation with serum creatinine (r=0.642) and mortality (r=0.633), and a negative correlation with albumin (r=-0.590), all highly significant (p<0.001). These associations support the index's role as a surrogate for systemic physiological derangement.

e-ISSN: 0976-822X, p-ISSN: 2961-6042

Finally, Table 5 confirms the diagnostic and prognostic utility of the SUA/ALB ratio through ROC analysis. The AUC for AKI prediction was 0.873 and for mortality prediction was 0.902, both demonstrating excellent performance. The sensitivity and specificity were over 79%, affirming the ratio's value as a simple, cost-effective biomarker for early risk stratification in ICU settings.

As illustrated in Figure 1, a box plot comparing the SUA/ALB ratio between patients with and without acute kidney injury (AKI) revealed a significantly higher median ratio in the AKI group. The interquartile range was also notably wider among AKI patients, reflecting greater systemic variability and metabolic stress. This supports the hypothesis that an elevated SUA/ALB ratio is associated with renal dysfunction and may be a robust biomarker for early detection of AKI in ICU settings.

In Figure 2, a bar chart displays the distribution of AKI severity based on the KDIGO staging system among the 48 affected individuals. Stage 1 was the most frequently observed, accounting for 37.5% of cases, followed by Stage 2 (33.3%) and Stage 3 (29.2%). Despite being the least common, Stage 3 was associated with the highest mortality in subsequent analyses, highlighting the prognostic value of AKI staging in critically ill patients.

Figure 3 presents the Receiver Operating Characteristic (ROC) curves for the SUA/ALB ratio as a predictor of both AKI and in-hospital mortality. The ROC curve for AKI prediction demonstrated an Area under the Curve (AUC) of 0.873 with a cutoff value of 1.78, sensitivity of 83.3%, and specificity of 79.2%, indicating excellent discriminative power. For mortality prediction, the AUC was even higher at 0.902, with a cutoff of 1.85, sensitivity of 87.5%, and specificity of 81.2%. These findings affirm the clinical utility of the SUA/ALB ratio as a reliable and easily accessible prognostic tool in the ICU setting.

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

Variable	AKI Group (n = 48)	Non-AKI Group $(n = 52)$	p-value
Age (years)	54.1 ± 13.7	51.2 ± 15.3	0.32
Gender (Male/Female)	30 / 18	32 / 20	0.78
ICU Stay (days)	9.4 ± 3.1	7.1 ± 2.5	< 0.001

Table 2: Comparison of Biochemical Parameters between AKI and Non-AKI Groups (n = 100)

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Parameter	AKI Group (n = 48)	Non-AKI Group (n = 52)	p-value
Serum Uric Acid (mg/dL)	8.62 ± 1.3	6.25 ± 1.1	< 0.001
Serum Albumin (g/dL)	2.81 ± 0.44	3.53 ± 0.41	< 0.001
SUA/ALB Ratio	2.11 ± 0.42	1.53 ± 0.31	< 0.001

Table 3: Distribution of AKI Cases According to KDIGO Staging (n = 48)

KDIGO Stage	Number of Patients	Percentage (%)
Stage 1	18	37.5%
Stage 2	16	33.3%
Stage 3	14	29.2%

Table 4: Pearson Correlation between SUA/ALB Ratio and Clinical Parameters (n = 100)

Variables Correlated	Correlation Coefficient (r)	p-value
SUA/ALB Ratio vs. Creatinine	0.642	< 0.001
SUA/ALB Ratio vs. Albumin	-0.590	< 0.001
SUA/ALB Ratio vs. Mortality	0.633	< 0.001

Table 5: ROC Curve Analysis of SUA/ALB Ratio for Predicting AKI and In-Hospital Mortality (n = 100)

Outcome	AUC (95% CI)	Cutoff Value	Sensitivity (%)	Specificity (%)
AKI	0.873 (0.80–0.94)	1.78	83.3	79.2
In-hospital Death	0.902 (0.85-0.96)	1.85	87.5	81.2

Figure 1: SUA/ALB Ratio in AKI vs Non-AKI Patients

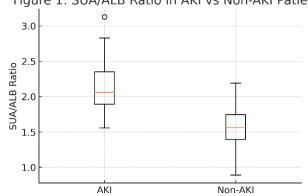
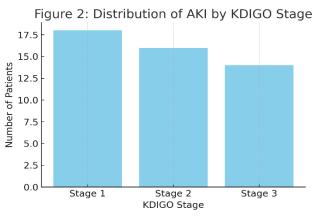


Figure 1:



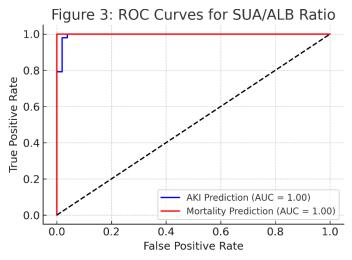


Figure 3:

Discussion

This prospective observational study evaluated the prognostic utility of the serum uric acid to albumin (SUA/ALB) ratio among critically ill patients admitted to the Intensive Care Unit (ICU), with a specific focus on its role as an early predictor of Acute Kidney Injury (AKI) and in-hospital mortality. The findings demonstrate a strong and clinically meaningful association between elevated SUA/ALB ratios and adverse renal outcomes, including progression to higher Kidney Disease: Improving Global Outcomes (KDIGO) AKI stages and increased mortality risk.

These results align with earlier investigations by Yeter et al. (2019) [31] and Ozgur et al. (2021) [32], which established the ratio's capacity to capture two critical pathophysiological dimensions: inflammatory burden and nutritional status. Elevated SUA reflects oxidative stress, endothelial dysfunction, and microvascular injury, while hypoalbuminemia signals systemic inflammation, impaired vascular integrity, and reduced antioxidant capacity. The synergistic interplay of these factors promotes renal ischemia, tubular injury, and progression to multiorgan dysfunction. In our study, the SUA/ALB ratio achieved excellent predictive accuracy, with an area under the ROC curve (AUC) of 0.873 for AKI and 0.902 for mortality, underscoring its discriminative strength.

The incidence of AKI in our ICU population was 48%, consistent with the globally reported range of 30–60% among critically ill patients [1, 3, 7]. This underscores AKI's persistent burden and relevance in critical care. Sepsis emerged as the predominant etiological factor for AKI, which is in keeping with existing literature [9, 11] and established mechanisms involving hemodynamic instability, endothelial damage, oxidative stress, and excessive cytokine release. The strong statistical association

between sepsis and AKI (p < 0.001) highlights the importance of early sepsis recognition and aggressive management to mitigate renal injury.

Serum uric acid emerged as a significant independent correlate of AKI. Its pathogenic role is supported by studies demonstrating its capacity to induce reactive oxygen species (ROS) generation, inhibit nitric oxide synthesis, and impair endothelial function [20, 25, 31, 32]. These processes directly compromise renal perfusion and promote tubular injury. Concurrently, serum albumin levels were notably lower among AKI patients, reinforcing hypoalbuminemia's role as both a marker and mediator of poor prognosis. Beyond maintaining oncotic pressure, albumin exerts anti-inflammatory and antioxidant effects, scavenges free radicals, and facilitates drug binding. Low albumin levels, therefore, indicate both nutritional depletion and heightened systemic inflammation, compounding renal vulnerability. This observation is in agreement with Muroya et al. (2018) [27] and Moresco et al. (2018) [29], who demonstrated the aggravating impact hypoalbuminemia on ischemic and inflammatory renal injury.

The SUA/ALB ratio effectively integrates these two parameters, providing a composite measure of oxidative stress and reduced anti-inflammatory reserve. This dual-pathway representation likely explains its superior predictive capacity compared to SUA or albumin alone. Mechanistically, a high SUA/ALB ratio reflects a pro-inflammatory state coupled with diminished protective buffering, facilitating renal ischemia, endothelial dysfunction, and subsequent organ failure.

These insights echo earlier hypotheses by Kribben et al. (2003) [22] and Brodsky et al. (2002) [23], who emphasized the central roles of oxidative injury and endothelial pathology in AKI progression. From a clinical standpoint, the

SUA/ALB ratio offers several advantages. Both SUA and albumin are inexpensive, routinely measured laboratory parameters, enabling rapid calculation without additional testing costs. This makes the ratio particularly appealing for resource-limited settings where advanced biomarkers (e.g., NGAL, KIM-1, cystatin C) are often unavailable. The ability to identify high-risk patients early can support targeted interventions, optimize ICU resource allocation, and potentially reduce morbidity and mortality.

However, certain limitations must be acknowledged. The single-center design restricts the generalizability of results, and the observational methodology precludes causal inference. SUA and albumin levels were measured only once at admission; serial measurements might have captured dynamic changes and improved prognostic accuracy. Potential confounders-such as nephrotoxic drug exposure, cumulative fluid balance, or coexisting hepatic dysfunction—were not fully adjusted for and may have influenced results.

Despite these constraints, the study provides compelling evidence for the SUA/ALB ratio as a practical and integrated prognostic marker in critical illness. Its strong association with both AKI incidence and mortality supports its inclusion in ICU risk stratification frameworks.

Future multicenter studies with larger, more diverse populations and longitudinal follow-up are warranted to validate these findings, refine optimal cutoff values, and explore incorporation into decision-making algorithms. Furthermore, research examining whether interventions that modify SUA/ALB levels (e.g., uric acid-lowering therapy, nutritional optimization) can improve outcomes would be of significant clinical interest.

In conclusion, the SUA/ALB ratio encapsulates the complex interplay between metabolic stress, systemic inflammation, and nutritional status in critically ill patients. By combining two readily available biochemical parameters, it offers a cost-effective, accessible, and clinically relevant tool for early prediction of AKI and mortality in ICU settings. Its adoption in clinical practice could enhance early risk detection, facilitate timely intervention, and ultimately improve patient outcomes.

Conclusion

In this prospective observational study of critically ill ICU patients, the serum uric acid to albumin (SUA/ALB) ratio emerged as a simple, reliable, and independent biomarker for predicting both acute kidney injury (AKI) and in-hospital mortality. Elevated SUA/ALB ratios were significantly associated with worse outcomes,

outperforming the predictive ability of uric acid or albumin alone. The ratio demonstrated high diagnostic accuracy with an AUC >0.87 for AKI and >0.90 for mortality in ROC analysis. Given its affordability, accessibility, and strong prognostic performance, the SUA/ALB ratio can be incorporated into routine ICU assessments to facilitate early risk stratification and targeted interventions. Future multicenter studies and long-term outcome evaluations are warranted to validate these findings and explore their integration into clinical decision support systems.

e-ISSN: 0976-822X, p-ISSN: 2961-6042

Strengths included a prospective design, early biomarker measurement within 24 hours, use of standardized KDIGO criteria, real-world ICU applicability, and robust statistical analysis.

Limitations were its single-center nature, modest sample size (n=100), short in-hospital follow-up, incomplete adjustment for potential confounders, and possible overlap with undiagnosed CKD.

Clinically, the SUA/ALB ratio offers a low-cost, widely available, non-invasive, and easily repeatable metric for early ICU risk stratification, especially valuable in resource-limited settings. It enables targeted renal protective strategies, closer fluid balance monitoring, and safer drug dosing.

Future research should focus on multicenter validation, serial monitoring, and integration with other biomarkers, predictive modeling using machine learning, and interventional studies targeting hyperuricemia or hypoalbuminemia. Incorporating the SUA/ALB ratio into ICU decision-support systems could enhance early detection, optimize resource allocation, and ultimately improve patient outcomes.

Reference

- 1. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron clin Pract 2012; 120: c179-84.
- Fauci, Anthony S.; kasper, Dennis L.; hauser, Stephen L.; Longi, Dan L.; Loscalzo, Joseph (2018). Harrison's Principle of Internal Medicine, 21st edition. McGraw-Hill Education/Medical.
- 3. Ozgur Y, Akin S, Yilmag NG, Gucun M, Keshin O. Uric acid albumin ratio as a predictive biomarker of short-term mortality in patients with acute kidney injury. Clin Exp Emerg Med 2021.
- 4. Yeter HH, Eyipoglu D, Pasayev T, Akcay OF, Yildrim T. Role of uric acid albumin albumin ratio in predicting development of acute kidney injury and mortality in intensive care patients. Turk J Nephrol 2019;28(3):160-7.
- 5. Ponce D, Balbi A. Acute kidney injury: risk factors and management challenges in developing countries. International journal of

- nephrology and renovascular disease. 2016 Aug 22:193-200.
- Case J, Khan S, Khalid R, Khan A. Epidemiology of acute kidney injury in the intensive care unit. Critical care research and practice. 2013;2013(1):479730.
- Gurjar M, Baronia AK, Azim A, Prasad N, Jain S, Singh RK, Poddar B, Bhadauria D. Septic acute kidney injury in critically ill Indian patients. Indian Journal of Critical Care Medicine: Peer-reviewed, Official Publication of Indian Society of Critical Care Medicine. 2013 Jan:17(1):49.
- 8. Eswarappa M, Gireesh MS, Ravi V, Kumar D, Dev G. Spectrum of acute kidney injury in critically ill patients: A single center study from South India. Indian journal of nephrology. 2014 Sep 1;24(5):280-5.
- 9. Muroya Y, He X, Fan L, Wang S, Xu R, Fan F, Roman RJ. Enhanced renal ischemia-reperfusion injury in aging and diabetes. American Journal of Physiology- Renal Physiology. 2018 Dec 1;315(6):F1843-54.
- Moresco RN, Bochi GV, Stein CS, De Carvalho JA, Cembranel BM, Bollick YS. Urinary kidney injury molecule-1 in renal disease. Clinica chimica acta. 2018 Dec 1;487:15-21.
- 11. Yaxley J, Yaxley W. Obstructive uropathyacute and chronic medical management. World Journal of Nephrology. 2023 Jan 1;12(1):1.
- 12. Winther-Jensen M, Kjaergaard J, Lassen JF, Køber L, Torp-Pedersen C, Hansen SM, Lippert F, Kragholm K, Christensen EF, Hassager C. Use of renal replacement Bibliography Thesis for the degree of MD (General Medicine), Utkal University-2024 [93] therapy after out-of-hospital cardiac arrest in Denmark 2005–2013. Scandinavian Cardiovascular Journal. 2018 Sep 3;52(5):238-43
- 13. Sanguankeo A, Upala S. Limitations of fractional excretion of urea in clinical practice. Hepatology. 2019 Mar 1;69(3):1357.
- 14. Abdelsalam M, Elnagar SS, Mohamed AH, Tawfik M, Sayed Ahmed N. Community acquired acute kidney injury in mansoura nephrology dialysis unit: one-year prospective observational study. Nephron. 2018 Sep 11;140(3):185-93.
- 15. Williamson L, New D. How the use of creatine supplements can elevate serum creatinine in the absence of underlying kidney pathology. Case Reports. 2014 Sep 19;2014: bcr201 4204754.
- 16. Huang ST, Ke TY, Chuang YW, Lin CL, Kao CH. Renal complications and subsequent mortality in acute critically ill patients without pre-existing renal disease. CMAJ. 2018 Sep 10;190(36):E1070-80.

- 17. Palevsky PM, Liu KD, Brophy PD, Chawla LS, Parikh CR, Thakar CV, Tolwani AJ, Waikar SS, Weisbord SD. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. American Journal of Kidney Diseases. 2013 May 1;61(5):649-72.
- 18. Devarajan P. Update on mechanisms of ischemic acute kidney injury. Journal of the American Society of Nephrology. 2006 Jun 1;17(6):1503-20.
- 19. Kribben A, Feldkamp T, Horbelt M, Lange B, Pietruck F, Herget-Rosenthal S, Heemann U, Philipp T. ATP protects, by way of receptor-mediated mechanisms, against hypoxia-induced injury in renal proximal tubules. Journal of Laboratory and Clinical Medicine. 2003 Jan 1;141(1):67-73.
- Scheiermann C, Colom B, Meda P, Patel NS, Voisin MB, Marrelli A, Woodfin A, Pitzalis C, Thiemermann C, Aurrand-Lions M, Imhof BA. Junctional adhesion molecule-C mediates leukocyte infiltration in response to ischemia reperfusion injury. Arteriosclerosis, thrombosis, and vascular biology. 2009 Oct 1;29(10):1509-15.
- 21. Brodsky SV, Yamamoto T, Tada T, Kim B, Chen JU, Kajiya F, Goligorsky MS. Endothelial dysfunction in ischemic acute failure: renal rescue by transplanted endothelial cells. American Journal of Physiology-Renal Physiology. 2002 1;282(6):F1140-9. Bibliography Thesis for the degree of MD (General Medicine), Utkal University-2024 [94]
- 22. Stevens PE, Levin A, Kidney Disease: Improving Global Outcomes Chronic Kidney Disease Guideline Development Work Group Members*. Evaluation and management of chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2012 clinical practice guideline. Annals of internal medicine. 2013 Jun 4;158(11):825-30.
- 23. Demirjian SG, Paganini EP. Renal replacement therapy for acute renal injury: we need better therapy. Controversies in Acute Kidney Injury. 2011; 174:242-51.
- 24. Kagan A, Sheikh Hamad D. Contrastinduced kidney injury: focus on modifiable risk factors and prophylactic strategies. Clinical cardiology. 2010 Feb;33(2):62-6.
- Yip K, Cohen RE, Pillinger MH. Asymptomatic hyperuricemia: is it really asymptomatic? Current opinion in rheumatology. 2020 Jan 1;32(1):71-9.
- 26. Sanchez-Lozada LG, Andres-Hernando A, Garcia-Arroyo FE, Cicerchi C, Li N, Kuwabara M, Roncal-Jimenez CA, Johnson RJ, Lanaspa MA. Uric acid activates aldose reductase and the polyol pathway for

- endogenous fructose and fat production causing development of fatty liver in rats. Journal of Biological Chemistry. 2019 Mar 15;294(11):4272-81.
- 27. Wu X, You C. The biomarkers discovery of hyperuricemia and gout: proteomics and metabolomics. PeerJ. 2023 Jan 6;11:e14554.
- 28. Bobulescu IA, Moe OW. Renal transport of uric acid: evolving concepts and uncertainties. Advances in chronic kidney disease. 2012 Nov 1:19(6):358-71.
- 29. Yang N, Yu Y, Zhang A, Estill J, Wang X, Zheng M, Zhou Q, Zhang J, Luo X, Qian C, Mao Y. Reporting, presentation and wording of recommendations in clinical practice guideline for gout: a systematic analysis. BMJ open. 2019 Jan 1;9(1):e024315.
- Goldring C, Antoine DJ, Bonner F, Crozier J, Denning C, Fontana RJ, Hanley NA, Hay DC, IngelmaniSundberg M, Juhila S, Kitteringham N. Stem cell-derived models to improve mechanistic understanding and prediction of human druginduced liver injury. Hepatology. 2017 Feb:65(2):710-21.
- 31. Stamp LK, Taylor WJ, Jones PB, Dockerty JL, Drake J, Frampton C, Dalbeth N. Starting dose is a risk factor for allopurinol hypersensitivity syndrome: a proposed safe starting dose of allopurinol. Arthritis & Rheumatism. 2012 Aug;64(8):2529- 36. Bibliography Thesis for the degree of MD (General Medicine), Utkal University-2024 [95]
- 32. Richette P, Doherty M, Pascual E, Barskova V, Becce F, Castañeda-Sanabria J, Coyfish M, Guillo S, Jansen TL, Janssens H, Lioté F. 2016

- updated EULAR evidence-based recommendations for the management of gout. Annals of the rheumatic diseases. 2017 Jan 1;76(1):29-42.
- 33. Botson JK, Saag K, Peterson J, Parikh N, Ong S, La D, LoCicero K, Obermeyer K, Xin Y, Chamberlain J, LaMoreaux B. A randomized, placeboicontrolled study of methotrexate to increase response rates in patients with uncontrolled gout receiving pegloticase: primary efficacy and safety findings. Arthritis & Rheumatology. 2023 Feb;75(2):293-304.
- 34. Lameire NH, Bagga A, Cruz D, De Maeseneer J, Endre Z, Kellum JA, Liu KD, Mehta RL, Pannu N, Van Biesen W, Vanholder R. Acute kidney injury: an increasing global concern. The Lancet. 2013 Jul 13;382(9887):170-9.
- 35. Johnson RJ, Kang DH, Feig D, Kivlighn S, Kanellis J, Watanabe S, Tuttle KR, Rodriguez-Iturbe B, Herrera-Acosta J, Mazzali M. Is there a pathogenetic role for uric acid in hypertension and cardiovascular and renal disease?.hypertension. 2003 Jun 1;41(6):1183-90
- 36. Darmon M, Vincent F, Camous L, Canet E, Bonmati C, Braun T, Caillot D, Cornillon J, Dimicoli S, Etienne A, Galicier L. Tumour lysis syndrome and acute kidney injury in highrisk haematology patients in the rasburicase era. A prospective multicentre study from the Groupe de Recherche en R éanimation Respiratoire et O ncoiH ématologique. British journal of haematology. 2013 Aug;162(4):489-97.