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Original Research Article

EARLY PREDICTION OF ACUTE KIDNEY INJURY USING ROUTINE BIOCHEMICAL MARKERS AND CLINICAL RISK FACTORS IN HOSPITALIZED ADULTS: A RETROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Acute kidney injury (AKI) is common among hospitalized adults and is associated with adverse clinical outcomes. Routinely collected clinical and biochemical variables may support early risk assessment. However, candidate predictors must be measured before AKI develops, and prediction models require validation in genuine patient cohorts. **Objective:** To evaluate the associations of routinely available biochemical markers and clinical risk factors with the development of AKI among hospitalized adults. **Materials and Methods:** This retrospective observational study included 100 hospitalized adults aged 18–65 years who were admitted to Central Hospital, Garden Reach, South Eastern Railway, between June 2022 and June 2025. Demographic characteristics, clinical risk factors, nephrotoxic-drug exposure, admission serum creatinine, and serum urea were evaluated. AKI was defined according to the creatinine-based Kidney Disease: Improving Global Outcomes criteria. Multivariable logistic-regression analysis was performed to evaluate factors associated with AKI, and adjusted odds ratios with 95% confidence intervals were reported. **Results:** AKI occurred in 28 of the 100 patients. Patients who developed AKI had higher median age, admission serum creatinine, and serum urea and higher frequencies of sepsis and nephrotoxic-drug exposure. Hypertension was less frequent in the AKI group than in the non-AKI group. In the exploratory multivariable model, age ≥60 years, admission creatinine >1.2 mg/dL, sepsis, and nephrotoxic-drug exposure were associated with AKI. Patients with AKI also had longer hospitalization and more frequent intensive care unit admission. The adjusted estimates had wide confidence intervals and should be interpreted cautiously. **Conclusion:** Routinely available clinical factors and biochemical measurements may help identify hospitalized adults at increased risk of AKI. Older age, sepsis, elevated admission serum creatinine, and nephrotoxic-drug exposure were associated with AKI in the exploratory model. These findings require confirmation in larger multicentre cohorts with complete internal and external validation before clinical implementation.

INTRODUCTION

Acute kidney injury (AKI) is a heterogeneous clinical syndrome characterized by an abrupt decline in kidney excretory function. It is frequently encountered among hospitalized adults, particularly among patients with severe infection, hemodynamic instability, dehydration, major surgery, pre-existing kidney dysfunction, or exposure to nephrotoxic medications. The reported incidence of AKI varies according to the clinical setting, severity of illness, availability of baseline kidney-function measurements, and diagnostic criteria used.[1-4]

AKI is associated with electrolyte and acid–base disturbances, fluid overload, prolonged hospitalization, increased healthcare utilization, kidney-replacement therapy, and mortality. Even when kidney function appears to recover, patients who experience AKI have increased long-term risks of recurrent AKI, chronic kidney disease, kidney failure, cardiovascular complications, and death.[3-5]

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The Kidney Disease: Improving Global Outcomes (KDIGO) criteria define AKI as an increase in serum creatinine of at least 0.3 mg/dL within 48 hours, an increase to at least 1.5 times the baseline value within seven days, or a reduction in urine output.[1] Serum creatinine and urine output therefore remain the most widely used clinical measures for diagnosing and staging AKI.

However, serum creatinine is an imperfect and delayed functional marker. Its concentration may not increase until kidney function has already declined and may be influenced by age, muscle mass, nutritional status, fluid balance, baseline kidney function, and laboratory variation. Accurate determination of baseline creatinine is also important because an inappropriate baseline value can lead to misclassification of AKI.[4,8] Hourly urine-output measurements may be unavailable or unreliable, particularly among patients admitted outside intensive-care settings.

Sepsis is an important clinical context for AKI. Sepsis-associated AKI may occur through interacting hemodynamic, microcirculatory, endothelial, inflammatory, and metabolic mechanisms and may develop even in the absence of overt systemic hypotension.[6] Other potentially relevant clinical factors include older age, diabetes mellitus, dehydration, reduced pre-existing kidney reserve, critical illness, and exposure to nephrotoxic medications.[3,4]

Novel biomarkers, including neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, cystatin C, and the product of tissue inhibitor of metalloproteinases-2 and insulin-like growth factor-binding protein 7, may provide information regarding tubular injury or cellular stress before substantial changes in serum creatinine occur. However, variation in performance, cost, availability, and clinical interpretation currently restricts their routine application in many healthcare settings.[7,12]

Prediction models using routinely available demographic, clinical, and laboratory information have therefore been developed to identify hospitalized patients at increased risk of AKI.[9] Such approaches may be useful only when predictors are measured before AKI onset, the intended prediction time and outcome window are clearly defined, and model performance is assessed using appropriate internal and external validation.[11]

The present study was undertaken to evaluate the associations of routinely available biochemical markers and clinical risk factors with the development of AKI among hospitalized adults. An exploratory multivariable model was also constructed to examine the combined associations of selected clinical and biochemical variables with AKI.

Aim and Objectives

Aim

To evaluate the associations of routinely available biochemical markers and clinical risk factors with the development of AKI among hospitalized adults.

Objectives

The objectives were to determine the occurrence and severity of AKI, compare selected demographic, clinical, and biochemical variables between patients with and without AKI, evaluate factors associated with AKI using an exploratory multivariable logistic-regression model, and compare selected hospital outcomes according to AKI status.

MATERIALS AND METHODS

Study Design and Setting

This retrospective observational study was conducted at Central Hospital, Garden Reach, South Eastern Railway. The study included 100 hospitalized adults aged 18–65 years who were admitted between June 2022 and June 2025. Medical records of eligible patients were reviewed to evaluate routinely available biochemical markers and clinical risk factors associated with the development of AKI during hospitalization.

Study Population

A total of 100 hospitalized adults aged 18–65 years were included in the analysis. Patients were classified into AKI and non-AKI groups according to the Kidney Disease: Improving Global Outcomes criteria. Only patients who met the predefined eligibility criteria and had the required baseline or admission and follow-up serum creatinine measurements were included.

Inclusion Criteria

Patients were included if they met all of the following criteria:

- Hospitalized adults aged 18–65 years.
- Admission between June 2022 and June 2025.
- Availability of a baseline or admission serum creatinine measurement.
- Availability of at least one follow-up serum creatinine measurement during hospitalization.
- Availability of the demographic and clinical information required for the analysis.
- Only the first eligible admission for each patient during the study period.

Exclusion Criteria

Patients were excluded if they met any of the following criteria:

- Age younger than 18 years or older than 65 years.
- Maintenance dialysis before hospital admission.
- Previous kidney transplantation.
- Absence of the serum creatinine measurements required to apply the prespecified AKI definition.
- Repeat admission after the first eligible admission during the study period.
- Incomplete clinical records containing insufficient information for the planned analysis.

Variables and Outcomes

Candidate variables included age, sex, diabetes mellitus, hypertension, sepsis, dehydration,

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nephrotoxic-drug exposure, admission serum creatinine, and serum urea.

Group-specific data for chronic kidney disease, serum sodium, serum potassium, serum albumin, hemoglobin, and total leukocyte count were unavailable and were therefore not included in the comparative analysis.

The primary outcome was the development of creatinine-defined AKI during hospitalization. Secondary outcomes included AKI severity, length of hospital stay, intensive care unit admission, dialysis requirement, and in-hospital mortality. The timing of intensive care unit admission relative to AKI onset could not be reliably determined from the available records.

AKI Definition and Temporal Requirements

AKI was defined as an increase in serum creatinine of ≥0.3 mg/dL within 48 hours or an increase to ≥1.5 times the baseline value within seven days, in accordance with KDIGO criteria.

AKI was identified using serum-creatinine criteria because reliable serial urine-output measurements were not consistently available in the retrospective records.

Clinical and biochemical variables recorded at admission or during the first 24 hours of hospitalization were considered candidate predictors. Predictor measurements obtained after documented AKI onset were not included in the prediction analysis.

Statistical Analysis

Continuous variables were summarized as medians, and categorical variables were presented as frequencies and percentages. Categorical variables were compared between the AKI and non-AKI groups using Pearson's chi-square test when expected cell counts were adequate and Fisher's exact test when expected cell counts were small. Unadjusted odds ratios with 95% confidence intervals were calculated for categorical clinical characteristics and outcomes. Multivariable logistic-regression analysis was used to evaluate factors associated with AKI, and adjusted odds ratios with 95% confidence intervals were reported. All tests were two-sided, and a p-value <0.05 was considered statistically significant. Analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of World College of Medical Sciences and Research and Hospital (approval number: _____). Written informed consent was obtained from all participants. Patient confidentiality was maintained throughout the study, and all procedures were conducted in accordance with institutional ethical requirements and the principles of the Declaration of Helsinki.

RESULTS

Study Population and Demographic Characteristics

A total of 100 patients were included, of whom 28 developed AKI and 72 did not. The median age was higher in the AKI group than in the non-AKI group. The sex distribution did not differ significantly between the groups (p=0.491; Table 1).

Table 1: Demographic Characteristics According to AKI Status

Demographic characteristic	No AKI (n=72)	AKI (n=28)
Median age, years	50	56
Male sex	44 (61.1%)	15 (53.6%)
Female sex	28 (38.9%)	13 (46.4%)

Table Note: Values are presented as median or n (%). The sex distribution did not differ significantly between the groups (Pearson's chi-square p=0.491). AKI, acute kidney injury.

Comorbidities and Clinical Risk Factors

Sepsis and nephrotoxic-drug exposure were significantly more frequent among patients who developed AKI. Sepsis was associated with seven-fold greater unadjusted odds of AKI (OR, 7.00; 95% CI, 2.53–19.38; p<0.001), whereas nephrotoxic-drug exposure was associated with 3.59-fold greater unadjusted odds (95% CI, 1.40–9.23; p=0.006). Diabetes mellitus, hypertension, and dehydration were not significantly associated with AKI. [Table 2]

Table 2: Comorbidities and Clinical Risk Factors According to AKI Status

Clinical characteristic	No AKI (n=72)	AKI (n=28)	Unadjusted OR (95% CI)	p-value
Diabetes mellitus	31 (43.1%)	14 (50.0%)	1.32 (0.55–3.17)	0.531
Hypertension	37 (51.4%)	11 (39.3%)	0.61 (0.25–1.49)	0.277
Sepsis	9 (12.5%)	14 (50.0%)	7.00 (2.53–19.38)	<0.001
Dehydration	18 (25.0%)	10 (35.7%)	1.67 (0.65–4.26)	0.284
Nephrotoxic-drug exposure	14 (19.4%)	13 (46.4%)	3.59 (1.40–9.23)	0.006

Table Note: Values are presented as n (%). Unadjusted odds ratios are presented with 95% confidence intervals. P-values were calculated using Pearson's chi-square test. AKI, acute kidney injury; CI, confidence interval; OR, odds ratio.

Admission Kidney-Function Markers

Patients who developed AKI had higher median admission serum creatinine and serum urea values than patients without AKI. Median serum creatinine was 1.11 mg/dL in the AKI group and 1.03 mg/dL in

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the non-AKI group. The corresponding median serum urea values were 35.8 mg/dL and 27.6 mg/dL, respectively. [Table 3]

Table 3: Admission Kidney-Function Markers According to AKI Status

Admission marker	No AKI (n=72)	AKI (n=28)
Serum creatinine, mg/dL	1.03	1.11
Serum urea, mg/dL	27.6	35.8

Table Note: Values are presented as medians. AKI, acute kidney injury.

Occurrence and Severity of Acute Kidney Injury

AKI occurred in 28 of the 100 patients. Among the 28 patients with AKI, stage 2 was the most frequent category and occurred in 20 patients (71.4%). Stage 3 AKI occurred in five patients (17.9%), whereas stage 1 AKI occurred in three patients (10.7%). [Table 4; Figure 1]

Table 4: Occurrence and Severity of Acute Kidney Injury

AKI category	Number of patients	Percentage of total population (n=100)	Percentage among AKI cases (n=28)
No AKI	72	72.00%	NA
Any AKI	28	28.00%	100.00%
Stage 1 AKI	3	3.00%	10.70%
Stage 2 AKI	20	20.00%	71.40%
Stage 3 AKI	5	5.00%	17.90%

Table Note: Percentages in the total-population column were calculated using n=100. Percentages in the AKI-only column were calculated using n=28. AKI, acute kidney injury; NA, not applicable.

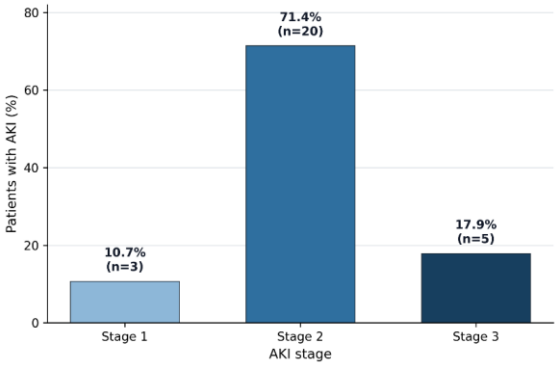

Figure 1: Distribution of AKI Severity Among Patients Who Developed AKI

Figure Note: Bars show the number and percentage of patients in KDIGO stages 1–3. Percentages were calculated using the 28 patients with AKI as the denominator. AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes.

Multivariable Associations with Acute Kidney Injury

In multivariable logistic-regression analysis, age ≥60 years, admission serum creatinine >1.2 mg/dL, sepsis, and nephrotoxic-drug exposure were associated with increased odds of AKI. Sepsis had the largest adjusted association with AKI (adjusted OR, 17.02; 95% CI, 3.97–72.98; p<0.001). Serum urea >40 mg/dL was not associated with AKI after adjustment (adjusted OR, 0.86; 95% CI, 0.15–5.04; p=0.867). The wide confidence intervals indicate statistical imprecision (Table 5; Figure 2).

Table 5: Multivariable Associations with Acute Kidney Injury

Predictor	Adjusted odds ratio	95% confidence interval	p-value
Age ≥60 years	9.55	2.26–40.34	0.002
Admission creatinine >1.2 mg/dL	5.87	1.28–26.95	0.023
Serum urea >40 mg/dL	0.86	0.15–5.04	0.867
Sepsis	17.02	3.97–72.98	<0.001
Nephrotoxic-drug exposure	7.23	1.87–28.03	0.004

Table Note: All five variables were included in one multivariable logistic-regression model. Adjusted odds ratios are presented with 95% confidence intervals. AKI, acute kidney injury; CI, confidence interval.

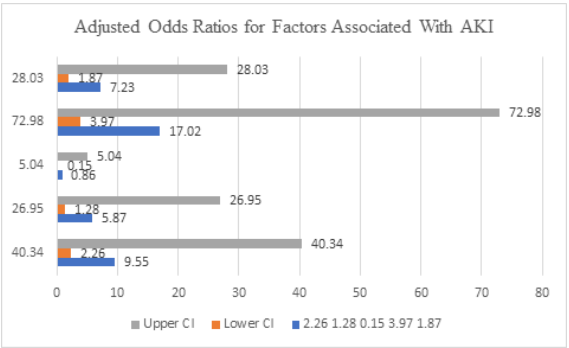

Figure 2: Adjusted odds ratios and 95% confidence intervals for factors associated with acute kidney injury

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Figure Note: Points indicate adjusted odds ratios, and horizontal lines indicate 95% confidence intervals. The vertical reference line at an odds ratio of 1 indicates no adjusted association. The horizontal axis is presented on a logarithmic scale. AKI, acute kidney injury; CI, confidence interval.

Hospital Outcomes

Patients with AKI had a longer median hospital stay than patients without AKI (10.5 versus 5 days). ICU admission was significantly more frequent in the AKI group (53.6% versus 5.6%; OR, 19.62; 95% CI, 5.61–68.63; p<0.001). Dialysis was required in four patients with AKI and no patients without AKI (Fisher's exact p=0.005). In-hospital mortality was numerically higher in the AKI group, but the difference was not statistically significant (10.7% versus 1.4%; Fisher's exact p=0.065). [Table 6]

Table 6: Hospital Outcomes According to AKI Status

Hospital outcome	No AKI (n=72)	AKI (n=28)
Median length of hospital stay, days	5	10.5
ICU admission	4 (5.6%)	15 (53.6%)
Dialysis requirement	0	4 (14.3%)
In-hospital mortality	1 (1.4%)	3 (10.7%)

Table Note: Values are presented as median or n (%). Pearson's chi-square test was used for ICU admission, whereas Fisher's exact test was used for dialysis requirement and in-hospital mortality. The timing of ICU admission relative to AKI onset could not be reliably determined. AKI, acute kidney injury; ICU, intensive care unit.

DISCUSSION

The present study identified associations between routinely available clinical variables and creatinine-defined AKI. Patients who developed AKI had higher median age, admission serum creatinine, and serum urea and higher frequencies of sepsis and nephrotoxic-drug exposure. These findings are directionally consistent with established AKI risk patterns.[1-4]

Sepsis showed the largest adjusted association with AKI. This direction is clinically plausible because sepsis-associated AKI may involve altered renal microcirculation, endothelial dysfunction, inflammation, oxidative stress, metabolic changes, and tubular injury.[6] Because of the retrospective observational design, this association should not be interpreted as evidence of causation.

Nephrotoxic-drug exposure was also associated with AKI. Medication class, dose, duration, cumulative exposure, concurrent treatment, baseline kidney function, and timing relative to AKI may influence this association.[3,4]

Age ≥60 years was associated with AKI in the exploratory model. However, because the study population was restricted to patients aged 18–65 years, this predictor represented only patients aged 60–65 years. The estimate should therefore be interpreted cautiously, and the number of patients in this subgroup should be reported.

Serum urea was higher in the AKI group but was not independently associated with AKI after adjustment. Urea is influenced by kidney filtration, hydration, protein intake, catabolism, gastrointestinal bleeding, and treatment effects. Its role cannot be excluded because the confidence interval was wide and the study included only 28 AKI events.

Routine-variable prediction models may provide a practical approach where specialized biomarkers are unavailable.[7,9,12] However, the small number of AKI events and wide confidence intervals indicate that the present model may be unstable. Complete model evaluation requires internal validation, calibration assessment, transparent handling of missing data, and external validation in an independent cohort.[11]

Prediction alone does not guarantee clinical benefit. A randomized trial showed that an electronic AKI alert by itself did not improve AKI severity or clinical outcomes.[10] Any prediction system should therefore be connected to a defined clinical response and evaluated prospectively.

The findings should be interpreted in view of the small sample, limited number of AKI events, wide confidence intervals, incomplete laboratory information, unclear timing of selected predictors, and absence of calibration and external validation. Larger multicentre studies are required to confirm the observed associations and evaluate the model's clinical usefulness.

Strengths

The study evaluated inexpensive and routinely available biochemical and clinical variables and used standardized creatinine-based KDIGO criteria for AKI classification. It also examined AKI severity and clinically relevant hospital outcomes. The use of a parsimonious exploratory model may support practical risk assessment; however, its performance requires validation in larger independent cohorts.

Limitations

This study has several limitations. Its retrospective, single-centre design may introduce selection bias and limit generalisability. The sample included only 100 patients and 28 AKI events, reducing statistical precision and increasing the risk of model overfitting. Patients older than 65 years were excluded, further restricting applicability. Incomplete documentation of urine output, comorbidities, medication exposure, and illness severity may have caused misclassification. AKI was assessed using serum-creatinine criteria alone. The timing of ICU admission relative to AKI onset could not be reliably determined, limiting its interpretation as a post-AKI

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outcome. Missing laboratory dispersion measures, model calibration, and external validation also limit interpretation. Larger multicentre prospective studies are required to confirm these findings and assess clinical usefulness.

CLINICAL AND RESEARCH IMPLICATIONS

Routine clinical factors and biochemical measurements may support early identification of patients at increased risk of AKI. Future studies should use larger multicentre cohorts with prespecified predictor timing, transparent missing-data handling, adequate sample-size calculations, and complete assessment of discrimination and calibration. Internal validation should be followed by evaluation in an independent cohort. A prospective impact study would then be required to determine whether model-guided assessment improves clinical decision-making or patient outcomes.

CONCLUSION

Routinely available clinical factors and biochemical measurements were associated with AKI among hospitalized adults. Older age, sepsis, elevated admission serum creatinine, and nephrotoxic-drug exposure were associated with increased odds of AKI in the exploratory model. However, the small sample, wide confidence intervals, and absence of calibration and external validation limit clinical interpretation. Larger multicentre studies are required before the model can be recommended for clinical use.

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