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Multi variate logistic regression model for predicting Risk Factors for Acute Kidney Injury in Septic Patients: A Single-Center Study from Peshawar, Pakistan.

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Abstract

Background: Sepsis-associated acute kidney injury (SA-AKI) is a common and serious complication of sepsis, but evidence from resource-limited South Asian settings is limited. ***Objective:*** This study assessed the prevalence, risk factors, and outcomes of AKI among septic patients at the Institute of Kidney Diseases, Peshawar, Pakistan. ***Methods:*** A retrospective cohort study included 200 adult patients with sepsis admitted between August 2025 and March 2026. Sepsis was defined using Sepsis-3 criteria and AKI was diagnosed and staged according to KDIGO criteria. Demographic, clinical, laboratory, treatment, and outcome variables were analyzed using comparative statistics and multivariable logistic regression. ***Results:*** AKI developed in 88 patients (44.0%). Among AKI cases, 31 (35.2%) had stage 1, 28 (31.8%) had stage 2, and 29 (33.0%) had stage 3 AKI. Independent predictors included chronic kidney disease, age >60 years, vasopressor use, MAP <65 mmHg, vancomycin exposure, and higher SOFA score. Patients with AKI had higher mortality, greater dialysis requirement, and longer hospital and ICU stays. ***Conclusion:*** SA-AKI was highly prevalent and clinically important in this cohort. Early risk stratification and renal protective care may improve septic patient outcomes.

Keywords: Acute kidney injury, sepsis, KDIGO, SOFA score, mortality, Pakistan, IEEE citation.

INTRODUCTION

Sepsis is a life-threatening syndrome caused by a dysregulated host response to infection and is a leading contributor to organ failure, prolonged hospitalization, and death worldwide [1]. Acute kidney injury (AKI) is among the most frequent organ complications of sepsis. It causes abrupt deterioration in renal filtration, dysregulation of fluids and electrolytes, accumulation of nitrogenous waste products, and increased need for renal replacement therapy. When AKI develops in patients with sepsis, it is commonly referred to as sepsis-associated acute kidney injury (SA-AKI) [2], [3].

SA-AKI is clinically important because its underlying mechanisms are complex and differ from those of purely ischemic or nephrotoxic AKI. In the past, renal hypoperfusion was considered the main cause of AKI during sepsis. However, more recent evidence suggests that SA-AKI can develop even when overall renal blood flow is maintained.

Several pathophysiologic processes can contribute to this, including systemic inflammation, endothelial dysfunction, changes in renal microvascular circulation, oxidative stress, mitochondrial dysfunction, tubular epithelial cell injury, and exposure to nephrotoxic drugs [4], [5]. The difficulty of the diagnosis is complicated by these pathophysiologic mechanisms and by serum creatinine, which usually elevates when considerable kidney injury is present already.

The burden of SA-AKI is a serious issue for low- and middle-income countries, where results of treatment are often deteriorated due to late hospital admission, lack of intensive care beds, insufficient monitoring, and lack of renal

replacement therapy. Global research indicates that AKI is common among critically ill sepsis patients [6], but the risk factors and results can greatly vary depending on regional infection patterns, antibiotic policy, CKD rates, referral process, and available resources.

Nevertheless, there is very little information on the epidemiology of SA-AKI in Pakistan. Local research is necessary for a complete understanding of the scope of the problem and identification of local risk factors.

For the purpose of filling this information gap, we conducted a study on the prevalence, risk factors, and clinical outcomes of AKI among adult sepsis patients in a tertiary renal and critical care center in Peshawar, Pakistan. We concentrated on the commonly available clinical parameters such as age, comorbidity, MAP, use of vasopressors, SOFA score, baseline renal function, and nephrotoxic drugs exposure.

MATERIALS AND METHODS

This retrospective cohort study was carried out in the Institute of Kidney Diseases (IKD) located in Peshawar, Pakistan, which provides nephrology, intensive care, dialysis, and renal transplant facilities. The patients involved in the study were those admitted into the institution from August 30, 2025, to March 1, 2026. The protocol for this study was approved by the institutional review board (IKD/IRB/2026/112). Informed consent was waived in the current study as it does not have direct contact with the participants. Patients who were admitted to the hospital with sepsis and were at least 18 years old were screened. Patients who met the Sepsis-3 criteria for sepsis, had a hospital stay of at least 24 hours, and had adequate clinical and laboratory documentation were included. An rise in the SOFA score of at least two points was used to operationalize the definition of sepsis, which was characterised as suspected or confirmed infection with acute organ failure [1], [7]. Patients with maintenance dialysis-dependent end-stage renal disease, previous kidney transplantation, pregnancy, hospital stay less than 24 hours, or incomplete critical data were excluded to reduce confounding and improve diagnostic reliability.

AKI was defined and staged using Kidney Disease: Improving Global Outcomes (KDIGO) criteria based on serum creatinine and urine output changes [2]. SA-AKI was defined as new-onset AKI occurring within seven days of sepsis diagnosis. Baseline creatinine was taken from the most recent value within three months before admission when available; otherwise, admission creatinine was used as baseline. CKD was defined as documented CKD or estimated glomerular filtration rate below 60 ml/min/1.73 m2 for at least three months.

Data were collected from patient records using a case report form. The variables recorded were age, sex, admission unit, smoking status, diabetes mellitus, hypertension, chronic kidney disease (CKD), ischemic heart disease, chronic liver disease, heart failure, and malignancy. Clinical data included mean arterial pressure (MAP), hypotension lasting more than 30 minutes, use of vasopressors, mechanical ventilation, lactate level, SOFA score, delay in antibiotic administration, and the source of sepsis. Nephrotoxic drug exposure during the first 72 hours was also recorded. Serum creatinine, blood urea nitrogen, urine output, dialysis requirement, hospital stay, ICU stay, and in-hospital mortality were also noted.

Continuous variables were presented as mean ± standard deviation or median with interquartile range, depending on the distribution of the data. Categorical variables were presented as numbers and percentages. The independent t-test or Mann–Whitney U test was used to compare continuous variables, and the chi-square test or Fisher's exact test was used for categorical variables. Variables with a p-value <0.10 in univariate analysis were entered into the multivariate logistic regression analysis. Adjusted odds ratios (aOR) along with their 95% confidence intervals (CI) were calculated to analyze independent relationships. Additionally, ROC curves were performed to calculate discrimination power of the SOFA score between patients developing AKI and not developing AKI. Statistical significance was accepted at $p < 0.05$ for two-sided tests.

RESULTS

In this study, 200 adult patients suffering from sepsis were studied. Their mean age was 48.9±18.6 years. There were 106 males (53.0%), 116 urban dwellers (58.0%), and 88 ICU admissions (44.0%). Some of the common comorbidities noted among these patients included hypertension (48.0%), DM (45.5%), CKD (39.5%), malignancy (36.0%), chronic liver disease (35.5%), and heart failure (34.5%). Signs of critical illness along with hemodynamic instability were prevalent among the patients. 47.5% of the patients had a mean arterial pressure of less than 65 mmHg, while 46.0% of the patients needed vasopressors.

AKI developed in 88 patients, giving an overall prevalence of 44.0%. Among patients with AKI, 31 (35.2%) had stage 1, 28 (31.8%) had stage 2, and 29 (33.0%) had stage 3 AKI. Patients with AKI were older, had more frequent ICU admission, diabetes, hypertension, CKD, MAP <65 mmHg, prolonged hypotension, vasopressor use, mechanical ventilation, higher

SOFA scores, vancomycin exposure, higher baseline and peak creatinine, higher blood urea nitrogen, and oliguria compared with patients without AKI.

TABLE I. KEY BASELINE CHARACTERISTICS BY AKI STATUS

Variable	AKI (n=88)	No AKI (n=112)	p-value
Age, years	54.2 +/- 16.4	44.7 +/- 19.2	<0.001
Age >60 years	38 (43.2%)	32 (28.6%)	0.032
ICU admission	51 (58.0%)	37 (33.0%)	<0.001
Diabetes mellitus	48 (54.5%)	43 (38.4%)	0.022
Hypertension	52 (59.1%)	44 (39.3%)	0.005
CKD	48 (54.5%)	31 (27.7%)	<0.001
MAP <65 mmHg	52 (59.1%)	43 (38.4%)	0.003
Vasopressor use	53 (60.2%)	39 (34.8%)	<0.001
Mechanical ventilation	51 (58.0%)	38 (33.9%)	0.001
SOFA score	10.1 +/- 3.2	7.2 +/- 3.5	<0.001
Vancomycin use	52 (59.1%)	41 (36.6%)	0.002

In multivariable logistic regression, six variables remained independently associated with SA-AKI. CKD had the strongest association (OR 3.12, 95% CI 1.68-5.79, p<0.001), followed by age >60 years (OR 2.84, 95% CI 1.52-5.31, p=0.001), vasopressor use (OR 2.68, 95% CI 1.44-4.98, p=0.002), MAP <65 mmHg (OR 2.41, 95% CI 1.29-4.50, p=0.006), vancomycin exposure (OR 2.15, 95% CI 1.17-3.95, p=0.014), and SOFA score (OR 1.21 per point, 95% CI 1.11-1.32, p<0.001). The SOFA score showed good predictive performance for AKI, with AUC 0.782 (95% CI 0.718-0.846). A cut-off score of at least 8 had 71.6% sensitivity, 68.8% specificity, 64.1% positive predictive value, and 75.5% negative predictive value.

TABLE II. MULTIVARIABLE PREDICTORS AND CLINICAL OUTCOMES

Measure	Result	95% CI / Comparator	p-value
CKD	OR 3.12	1.68-5.79	<0.001
Age >60 years	OR 2.84	1.52-5.31	0.001
Vasopressor use	OR 2.68	1.44-4.98	0.002
MAP <65 mmHg	OR 2.41	1.29-4.50	0.006
Vancomycin exposure	OR 2.15	1.17-3.95	0.014
SOFA score	OR 1.21 per point	1.11-1.32	<0.001
Mortality	20.5% vs 7.1%	AKI vs no AKI	0.005
Dialysis requirement	28.4% vs 5.4%	AKI vs no AKI	<0.001
Hospital stay	12.3 vs 8.4 days	AKI vs no AKI	<0.001
ICU stay	5.2 vs 2.1 days	AKI vs no AKI	<0.001

Clinical outcomes were significantly worse among patients who developed AKI. In-hospital mortality was 20.5% in the AKI group and 7.1% in the non-AKI group (p=0.005). Dialysis was required in 28.4% of AKI patients compared with 5.4% of those without AKI (p<0.001). Hospital length of stay was longer in the AKI group (12.3 +/- 5.2 vs. 8.4 +/- 4.6 days, p<0.001), and ICU stay was also longer (5.2 +/- 3.8 vs. 2.1 +/- 2.4 days, p<0.001). Outcomes worsened with increasing AKI severity. Mortality increased from 9.7% in stage 1 to 17.9% in stage 2 and 34.5% in stage 3 AKI.

DISCUSSION

This study found that SA-AKI affected nearly one-half of adult septic patients admitted to a tertiary kidney center in Peshawar. The prevalence of 44.0% is consistent with international evidence showing that AKI is common among critically ill patients with sepsis [3], [6]. The distribution of AKI severity was clinically important: approximately one-third of AKI cases were stage 3, and stage 3 patients had the highest dialysis requirement and mortality. These findings show that SA-AKI is common in patients with sepsis and is associated with more severe illness and worse outcomes.

CKD was the strongest independent risk factor for AKI in our study. Patients with CKD have less renal reserve, which may make them more susceptible to kidney injury during sepsis, particularly in the presence of inflammation, changes in blood pressure, and exposure to nephrotoxic drugs [8]. Older age was also independently associated with AKI. This may be related to the higher burden of comorbidities, vascular disease, changes in drug handling, and reduced nephron mass seen with increasing age. Based on these findings, septic patients with pre-existing CKD and older patients should be considered at higher risk of AKI from the time of hospital admission.

Another important route was haemodynamic instability. AKI was independently predicted by MAP <65 mmHg and vasopressor usage, indicating the importance of renal hypoperfusion, shock severity, and microcirculatory dysfunction. Sepsis recommendations prioritise source management, haemodynamic resuscitation, early detection, and timely antimicrobial treatment [5]. The SOFA score performed well in ROC and also predicted AKI in this sample. SOFA may be a useful bedside tool for early SA-AKI risk categorisation in hospitals with limited resources since it is currently used for organ dysfunction evaluation.

Exposure to vancomycin was linked to AKI on its own. Vancomycin is frequently required for presumed resistant Gram-positive infections, although critically sick patients may be more susceptible to nephrotoxicity, especially if they have high trough targets, extended treatment, concomitant nephrotoxins, or unstable renal function [9], [10]. Antimicrobial stewardship, renal dosage modification, analysis of nephrotoxic combinations, therapeutic drug monitoring where possible, and daily reevaluation of continued vancomycin requirement are all supported by the findings.

There were many discrepancies with the results. Patients with AKI had longer stays at the hospital and the ICU, required dialysis, and had almost three times higher mortality rate than patients without AKI. These results agree with previous studies demonstrating a relationship between AKI, longer hospitalization periods, costs, and death rate [11]. It is essential to acknowledge the importance of a bundled renal protection program based on sepsis early identification, timely administration of antibiotics, optimization of MAP, reduction of non-essential nephrotoxins, monitoring of creatinine and urine output, as well as early involvement of nephrologists in case of any complications such as fluid and electrolyte imbalances in case of AKI.

Several limitations of this study should be addressed. The retrospective design of this study makes it dependent on the availability of medical records only. Besides, there cannot be any conclusions regarding causality because of the observational design. Moreover, this study took place in one center with a small sample size that can affect generalizability of the results for other Pakistani hospitals. Some variables that can influence the development and outcomes of AKI were unavailable, such as the long-term renal function recovery, antibiotic serum concentrations, exact amounts of vasopressors used, fluid balance, and inflammatory markers. In cases when the recent creatinine values were not available, baseline creatinine was calculated based on the admission values that can affect AKI diagnosis. Nevertheless, the study provides local data about SA-AKI defined according to the KDIGO criteria of AKI and Sepsis-3 criteria.

CLINICAL IMPLICATIONS AND FUTURE WORK

The findings of this study may help clinicians identify septic patients who are at higher risk of AKI. Closer monitoring of renal function should be considered in patients older than 60 years, those with pre-existing CKD, patients requiring vasopressors, those with a MAP below 65 mmHg, patients receiving vancomycin, and those with a high SOFA score. Simple measures such as hourly urine output monitoring, daily serum creatinine measurement, or more frequent testing when clinically indicated, should be used during the first week. Nephrotoxic medications should be reviewed regularly, and fluid status should be assessed carefully.

Early nephrology review should be considered when serum creatinine starts to rise, oliguria continues, potassium levels increase, metabolic acidosis develops, or fluid overload becomes significant.

The prevention of AKI becomes particularly critical in such settings as hospitals where the facilities of dialysis and ICU are limited. The local protocols for sepsis should incorporate means of preventing the deterioration of the kidneys. These measures may include early antibiotic treatment, source control, maintaining MAP in required range, dosage adjustment based on the level of renal function, avoiding unnecessary use of NSAIDs and aminoglycosides, and determining the trough level of vancomycin whenever possible.

As for the next research steps, the prospective, multicenter study design must be considered. Moreover, the early biomarkers of tubular injury assessment, exact fluid balance recording with vasopressor dosages, and follow-up after discharge are important for further investigations.

CONCLUSION

SA-AKI was common in this study conducted from a single center of Pakistan in sepsis patients. SA-AKI patients have longer duration of hospitalization and ICU stay, increased mortality rate, and the chance of dialysis. Risk factors of developing AKI include chronic kidney disease (CKD), age > 60, MAP < 65 mmHg, vasopressor use, vancomycin use, and increased SOFA score.

Prevention of the clinical effect of SA-AKI requires proper identification of high-risk patients, maintenance of appropriate perfusion pressure, careful use of nephrotoxic drugs and antibiotics, and monitoring of renal function. Consultation with

nephrology at an early stage should always be done when there is a decline in renal function. The best way forward is to conduct multicenter trials in Pakistan.

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