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

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

International Journal of Current Pharmaceutical Review and Research 2026; 18(7); 1333-1341

Original Research Article

Clinical Profile and In-Hospital Outcomes of Acute Kidney Injury in a Tertiary Care Setting: A Prospective Observational Study

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Received: 01-04-2026 / Revised: 15-05-2026 / Accepted: 21-06-2026
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Conflict of interest: Nil

Abstract

Background: Acute kidney injury (AKI) is a frequent complication of acute illness and is associated with prolonged hospitalization, kidney replacement therapy, chronic kidney disease, and death. Its clinical spectrum varies according to patient characteristics, healthcare setting, regional disease patterns, and access to timely nephrological care. This study evaluated the clinical profile, etiological distribution, severity, treatment requirements, renal recovery, and predictors of in-hospital mortality among adults with AKI admitted to a tertiary care hospital.

Methods: A prospective observational study was conducted over 18 months in the medicine department of a tertiary care hospital. Consecutive adults fulfilling Kidney Disease: Improving Global Outcomes criteria for AKI were included. Demographic characteristics, comorbidities, precipitating factors, laboratory parameters, urine output, maximum AKI stage, organ-support requirements, kidney replacement therapy, and outcomes were recorded. Outcomes were categorized as complete renal recovery, partial recovery, dialysis dependence, or in-hospital death. Multivariable logistic regression identified independent predictors of mortality.

Results: A total of 180 patients were evaluated; the mean age was 56.3 ± 14.7 years, and 102 (56.7%) were men. Community-acquired AKI accounted for 60.6% of cases. Sepsis was the leading etiology (35.6%), followed by hypovolemia (21.7%), nephrotoxic exposure (17.2%), and urinary obstruction (13.9%). KDIGO stages 1, 2, and 3 were observed in 31.1%, 27.2%, and 41.7% of patients, respectively. Oliguria occurred in 37.8%, while 23.9% required kidney replacement therapy. Complete recovery occurred in 43.3%, partial recovery in 30.6%, dialysis dependence in 4.4%, and death in 21.7%. Mortality increased from 8.9% in stage 1 to 36.0% in stage 3. Older age, sepsis-associated AKI, KDIGO stage 3, and mechanical ventilation independently predicted mortality.

Conclusion: AKI in a tertiary care setting was predominantly associated with sepsis, hypovolemia, and nephrotoxic exposure. Severe AKI, respiratory failure, and sepsis identified a subgroup at particularly high risk of death. Early recognition, hemodynamic optimization, nephrotoxin stewardship, and coordinated critical-care and nephrology management may improve renal recovery and survival.

Keywords: Acute Kidney Injury; KDIGO; Sepsis; Kidney Replacement Therapy; Renal Recovery; Mortality; Tertiary Care.

DOI: 10.25258/ijcpr.18.7.201

Introduction

Acute kidney injury is a heterogeneous clinical syndrome characterized by an abrupt decline in kidney excretory function, manifested by an increase in serum creatinine, reduction in urine output, or both. The condition ranges from a mild and transient biochemical abnormality to severe kidney failure accompanied by electrolyte disturbances, acid–base derangement, volume overload, and multiorgan dysfunction. Standardized classifications, initially developed through the RIFLE and Acute Kidney Injury Network systems and subsequently consolidated within the Kidney Disease: Improving Global Outcomes criteria, have improved diagnostic consistency and enabled comparison across clinical populations [1–3]. KDIGO defines AKI using changes in serum creatinine occurring within 48 hours or seven days and urine-output thresholds measured over defined periods. AKI represents an important global health problem across community, emergency, ward, perioperative, and intensive-care settings. A large meta-analysis involving nearly 50 million

participants demonstrated substantial worldwide disease burden, although incidence varied according to case definition, clinical setting, regional income, and availability of laboratory monitoring [4,5]. Among critically ill patients, the multinational AKI-EPI investigation found AKI in more than half of patients during the first week of intensive-care admission, with a progressive increase in mortality across KDIGO stages [6]. Earlier multinational data similarly demonstrated high mortality among patients with severe kidney failure requiring renal replacement therapy [7].

The prognostic implications of AKI extend beyond the immediate loss of filtration. Even relatively small increases in serum creatinine have been associated with prolonged hospitalization, increased expenditure, and higher mortality [8]. Survivors remain vulnerable to recurrent AKI, incomplete renal recovery, incident or progressive chronic kidney disease, cardiovascular events, and kidney failure. A systematic review found substantially greater long-term risks of chronic kidney disease, end-stage kidney disease, and death among patients who had experienced AKI than among those without AKI.

The clinical pattern of AKI differs between high-income and resource-constrained settings. Hospital-acquired AKI associated with major surgery, nephrotoxic medication, contrast exposure, and critical illness predominates in many developed healthcare systems. In South Asia, community-acquired disease related to infection, diarrheal illness, volume depletion, envenomation, urinary obstruction, and delayed presentation continues to contribute substantially. Indian studies have also reported marked interregional variation in patient age, etiology, severity, dialysis use, and mortality [9–15]. Community- and hospital-acquired forms may therefore require different preventive strategies and surveillance pathways.

Tertiary hospitals receive a particularly heterogeneous AKI population, including referred community cases, postoperative complications, septic patients, individuals with malignancy, and patients developing AKI during hospitalization. Local characterization is essential because aggregate international estimates may not reflect regional infection patterns, medication practices, referral delays, comorbidities, or availability of kidney replacement therapy. The present study was undertaken to describe the demographic and clinical profile, etiological spectrum, KDIGO severity, organ-support requirements, renal recovery, dialysis dependence, and in-hospital mortality of adults with AKI in a tertiary care setting. A secondary objective was to identify readily measurable factors independently associated with in-hospital death.

Materials and Methods

Study Design and Setting: This prospective observational study was conducted in the departments of General Medicine. Recruitment occurred over 18 months. The hospital received both direct admissions and referrals from surrounding district and secondary-care facilities.

Study Participants: Consecutive patients aged 18 years or older who fulfilled KDIGO serum-creatinine or urine-output criteria for AKI during the study period were enrolled. AKI was diagnosed by an increase in serum creatinine of at least 0.3 mg/dL within 48 hours, an increase to at least 1.5 times the documented baseline within seven days, or urine output below 0.5 mL/kg/hour for at least six hours [1]. Maximum severity during hospitalization was categorized as KDIGO stage 1, 2, or 3.

Patients receiving maintenance dialysis for end-stage kidney disease, kidney-transplant recipients, patients younger than 18 years, and individuals without adequate baseline or serial renal-function measurements were excluded. Patients with pre-existing chronic kidney disease stages 1–4 were retained and classified as AKI on chronic kidney disease when an acute creatinine change fulfilled KDIGO criteria.

The baseline creatinine was taken as the most recent stable outpatient value documented during the preceding three months. Where more than one value was available, the value closest to admission that was not obtained during an acute illness was selected. Patients without a reliable baseline value and without sufficient serial measurements to establish an acute change were not included.

Data Collection and Definitions: Demographic characteristics, admission source, presenting symptoms, comorbidities, medication exposure, primary diagnosis, hemodynamic status, and suspected etiology were recorded using a structured case-record form. Laboratory measurements included complete blood count, serum urea, creatinine, sodium, potassium, bicarbonate, albumin, liver-function tests, urinalysis, and relevant microbiological investigations. Ultrasonography of the kidneys and urinary tract was performed when clinically indicated.

AKI present on admission or detected within the first 48 hours was categorized as community-acquired. AKI developing after 48 hours of hospitalization in a patient without AKI at admission was categorized as hospital-acquired. Etiology was assigned after review of clinical, biochemical, microbiological, radiological, and treatment-response data. When more than one factor was present, the predominant initiating or sustaining factor was recorded. Sepsis-associated

AKI was diagnosed when AKI occurred in temporal association with suspected or confirmed infection and organ dysfunction. Hypovolemic AKI included gastrointestinal fluid loss, hemorrhage, poor intake, or other clinically significant volume depletion. Nephrotoxic AKI included injury associated with nonsteroidal anti-inflammatory drugs, antimicrobial agents, contrast material, herbal preparations, or other potentially nephrotoxic exposure. Obstructive AKI required radiological or procedural evidence of urinary-tract obstruction.

Oliguria was defined as urine output below 0.5 mL/kg/hour for at least six hours. Kidney replacement therapy was initiated for refractory hyperkalemia, severe metabolic acidosis, pulmonary edema or volume overload unresponsive to medical therapy, clinically significant uremic complications, or progressive azotemia with deteriorating clinical status. The modality and timing of therapy were determined by the treating nephrologist and intensivist.

Outcome Assessment: The primary outcome was in-hospital mortality. Secondary outcomes included kidney replacement therapy, duration of hospitalization, complete renal recovery, partial recovery, and dialysis dependence at discharge. Complete recovery was defined as dialysis independence with discharge creatinine returning to within 0.3 mg/dL or 25% of baseline. Partial recovery was defined as dialysis independence with persistent creatinine more than 25% above baseline. Dialysis dependence indicated continued requirement for intermittent hemodialysis or another kidney-replacement modality at discharge.

Statistical Analysis: Data were analyzed using IBM SPSS Statistics version 26.0 or equivalent validated statistical software. Continuous variables were assessed for distribution and summarized as mean ± standard deviation or median with interquartile range. Categorical variables were expressed as frequencies and percentages.

Continuous variables were compared using the independent-samples t test or Mann–Whitney U test. Categorical variables were compared using the chi-square test or Fisher's exact test. Variables associated with mortality at $p<0.10$ in univariable analysis, together with clinically relevant covariates, were evaluated using multivariable binary logistic regression. Adjusted odds ratios with 95% confidence intervals were reported. Model calibration, collinearity, and influential observations were examined before interpretation. A two-sided p value below 0.05 was considered statistically significant.

Results

A total of 180 adults with AKI were included. The mean age was 56.3 ± 14.7 years, with ages ranging from 18 to 89 years. Men constituted 56.7% of the cohort. Hypertension and diabetes mellitus were each present in 37.2%, while 18.3% had pre-existing chronic kidney disease. Community-acquired AKI accounted for 109 cases (60.6%); the remaining 71 patients developed AKI after hospitalization. Nearly half of the cohort required intensive-care management during some part of the admission.

Sepsis was the most frequent underlying etiology, occurring in 64 patients (35.6%). Hypovolemia was responsible for 21.7%, nephrotoxic exposure for 17.2%, and urinary-tract obstruction for 13.9%. Cardiorenal causes and glomerular or miscellaneous intrinsic renal disorders accounted for the remaining cases.

At maximum severity, 31.1% had KDIGO stage 1, 27.2% had stage 2, and 41.7% had stage 3 AKI. Oliguria occurred in 37.8%; 25.0% required vasopressor support, 19.4% required invasive mechanical ventilation, and 23.9% underwent kidney replacement therapy. At hospital discharge, 78 patients (43.3%) had achieved complete renal recovery and 55 (30.6%) had partial recovery. Eight patients (4.4%) remained dialysis dependent, and 39 (21.7%) died. Outcomes deteriorated consistently with increasing AKI severity.

Mortality was 8.9% in stage 1, 14.3% in stage 2, and 36.0% in stage 3. Conversely, complete recovery declined from 78.6% in stage 1 to 20.0% in stage 3. Nonsurvivors were older and had higher admission and peak creatinine concentrations, lower platelet counts, lower serum albumin, and longer hospitalization than survivors.

Sepsis, oliguria, vasopressor requirement, mechanical ventilation, and KDIGO stage 3 were significantly more frequent among patients who died. In multivariable analysis, increasing age, sepsis-associated AKI, stage 3 disease, and mechanical ventilation remained independently associated with in-hospital mortality.

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	Study population, n=180
Age, years, mean ± SD	56.3 ± 14.7
Male sex	102 (56.7%)
Female sex	78 (43.3%)
Diabetes mellitus	67 (37.2%)
Hypertension	67 (37.2%)
Pre-existing CKD stages 1–4	33 (18.3%)
Community-acquired AKI	109 (60.6%)
Hospital-acquired AKI	71 (39.4%)
Oliguria	68 (37.8%)
Vasopressor requirement	45 (25.0%)
Mechanical ventilation	35 (19.4%)
Kidney replacement therapy	43 (23.9%)

The cohort represented a predominantly middle-aged to older population with a modest male preponderance and a substantial burden of diabetes and hypertension.

Community-acquired disease remained more frequent than hospital-acquired AKI, emphasizing the continuing importance of prehospital infection, volume depletion, drug exposure, and delayed referral.

More than one-third developed oliguria, while approximately one-quarter required vasopressors or kidney replacement therapy, indicating that a considerable proportion presented with physiologically severe illness.

Table 2: Etiological Spectrum and Maximum Kdigo Stage

Variable	Number (%)
Predominant etiology	
Sepsis-associated AKI	64 (35.6%)
Hypovolemia	39 (21.7%)
Nephrotoxic exposure	31 (17.2%)
Urinary-tract obstruction	25 (13.9%)
Cardiorenal syndrome	12 (6.7%)
Glomerular or other intrinsic causes	9 (5.0%)
Maximum KDIGO stage	
Stage 1	56 (31.1%)
Stage 2	49 (27.2%)
Stage 3	75 (41.7%)

Sepsis was the dominant contributor to AKI and was followed by potentially preventable or rapidly reversible conditions, including hypovolemia, nephrotoxic exposure, and obstruction.

The large proportion of patients reaching KDIGO stage 3 suggested delayed recognition, severe initiating illness, continuing renal insults, or referral after substantial kidney dysfunction had already developed. These findings support systematic infection control, early fluid and perfusion assessment, medication review, and prompt investigation for urinary obstruction in high-risk admissions.

Table 3: Renal and Survival Outcomes According To Maximum Kdigo Stage

Outcome	Stage 1, n=56	Stage 2, n=49	Stage 3, n=75	Total, n=180
Complete renal recovery	44 (78.6%)	19 (38.8%)	15 (20.0%)	78 (43.3%)
Partial renal recovery	7 (12.5%)	19 (38.8%)	29 (38.7%)	55 (30.6%)
Dialysis dependence	0	4 (8.2%)	4 (5.3%)	8 (4.4%)
In-hospital death	5 (8.9%)	7 (14.3%)	27 (36.0%)	39 (21.7%)

A pronounced severity–outcome relationship was observed.

Nearly four-fifths of patients with stage 1 AKI achieved complete recovery, whereas only one-fifth with stage 3 recovered completely. More than one-third of stage 3 patients died, and incomplete recovery was common among survivors. The findings indicate that maximum AKI stage functioned not merely as a biochemical classification but as a clinically meaningful marker of renal reserve, systemic illness severity, and probability of survival.

Table 4: Comparison of Survivors and Nonsurvivors and Independent Mortality Predictors

Variable	Survivors, n=141	Nonsurvivors, n=39	p value
Age, years	54.9 ± 14.0	61.1 ± 16.3	0.036
Sepsis-associated AKI	34 (24.1%)	30 (76.9%)	<0.001
KDIGO stage 3	48 (34.0%)	27 (69.2%)	<0.001
Oliguria	47 (33.3%)	21 (53.8%)	0.031
Vasopressor requirement	25 (17.7%)	20 (51.3%)	<0.001
Mechanical ventilation	15 (10.6%)	20 (51.3%)	<0.001
Admission creatinine, mg/dL	2.71 ± 1.01	3.35 ± 0.89	<0.001
Peak creatinine, mg/dL	3.69 ± 1.66	4.80 ± 1.35	<0.001
Platelet count, ×10³/µL	200.8 ± 72.9	136.9 ± 72.9	<0.001
Serum albumin, g/dL	3.01 ± 0.58	2.66 ± 0.58	0.002
Hospital stay, days	13.9 ± 5.8	18.7 ± 5.3	<0.001

Independent predictor	Adjusted OR	95% CI	p value
Age, per one-year increase	1.04	1.00–1.07	0.034
Sepsis-associated AKI	5.19	1.91–14.08	0.001
KDIGO stage 3	3.08	1.16–8.19	0.024
Mechanical ventilation	3.90	1.49–10.22	0.006

Interpretation: Nonsurvivors displayed a combined renal and systemic high-risk phenotype characterized by older age, sepsis, severe AKI, oliguria, circulatory failure, respiratory failure, thrombocytopenia, and hypoalbuminemia. After adjustment, sepsis conferred the greatest relative increase in mortality, while stage 3 AKI and mechanical ventilation remained strong independent determinants. Admission creatinine alone therefore appeared insufficient for prognostication; risk assessment required simultaneous evaluation of AKI severity and the accompanying burden of organ dysfunction.

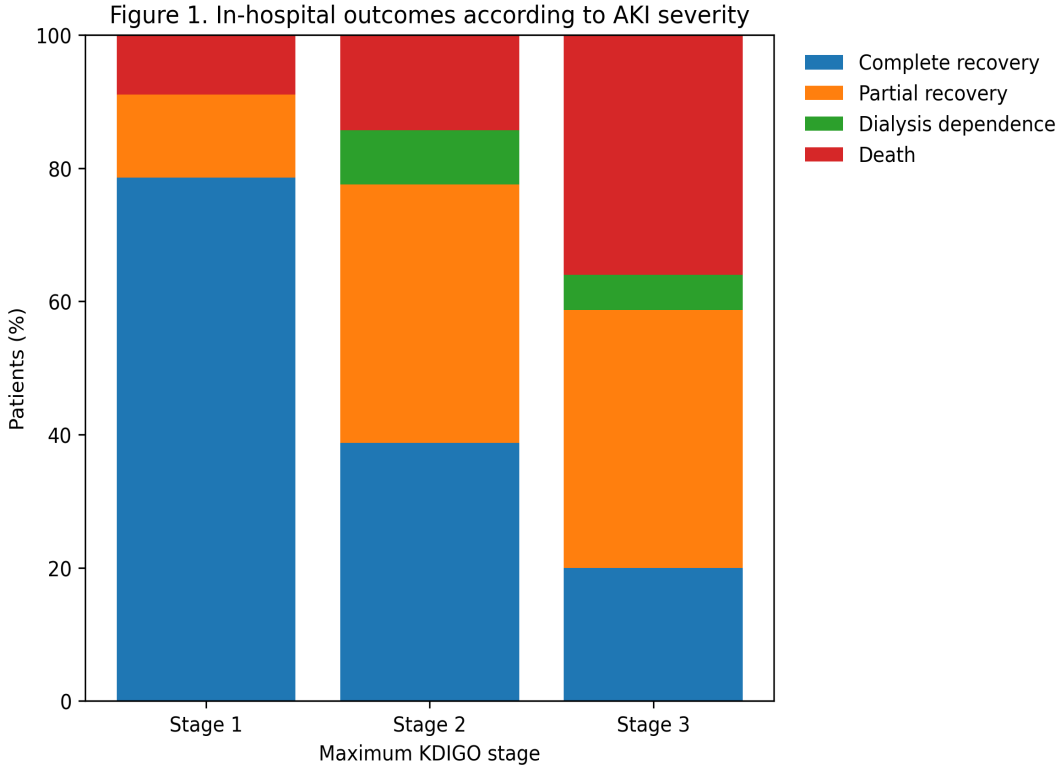

Figure 1: In-hospital outcomes according to maximum KDIGO stage

The stacked outcome distribution demonstrates a progressive shift from complete recovery toward incomplete recovery and death as AKI severity increased. Stage 1 was predominantly reversible, whereas stage 3 was characterized by a marked reduction in complete recovery and a fourfold

higher crude mortality than stage 1. The pattern supports serial staging rather than reliance on admission creatinine alone, because progression to a higher maximum stage identified patients requiring intensified monitoring and earlier nephrology involvement.

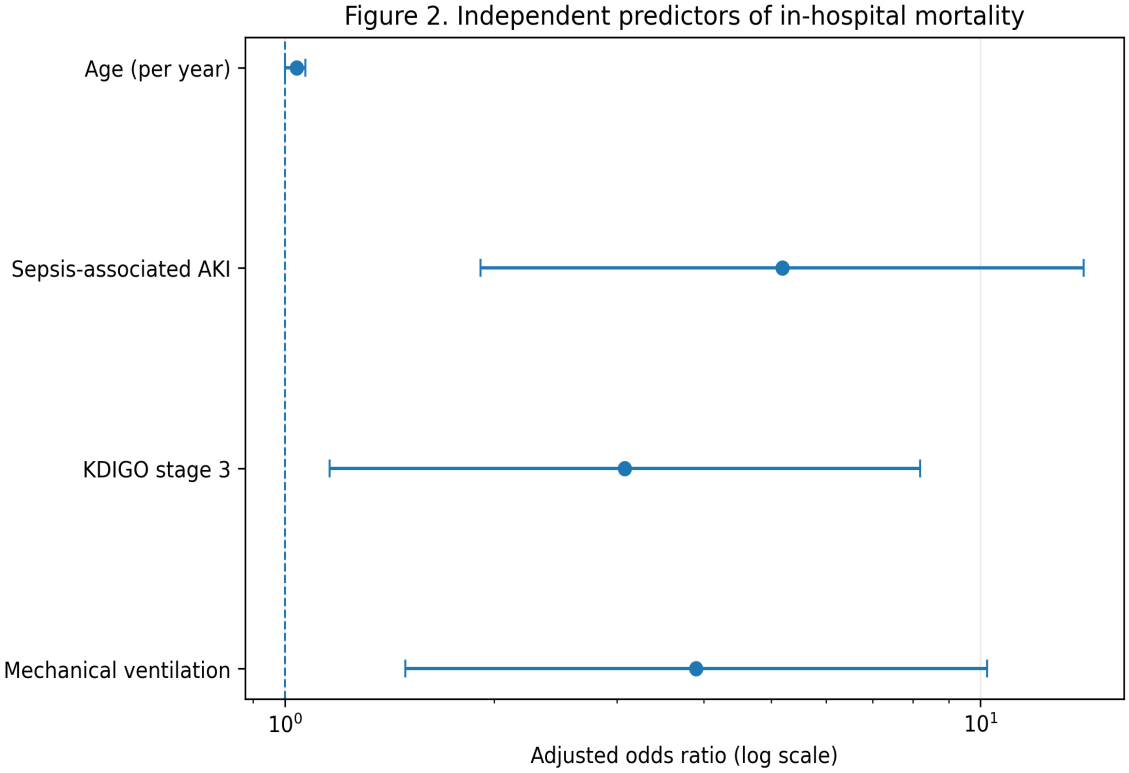

Figure 2: Adjusted predictors of in-hospital mortality

The forest plot shows that sepsis-associated AKI produced the largest adjusted odds of death, although its confidence interval was wide because of the limited number of outcome events. Mechanical ventilation and KDIGO stage 3 also remained independently associated with mortality, reflecting the adverse interaction between kidney dysfunction and systemic organ failure. Increasing age exerted a smaller but cumulative effect. All confidence intervals for the reported independent predictors remained above the null value.

Discussion

This study described the clinical and etiological profile of AKI across ward and identified a substantial burden of severe disease. Sepsis was the leading etiology, more than two-fifths of patients reached KDIGO stage 3, nearly one-quarter required kidney replacement therapy, and approximately one in five died. Complete renal recovery occurred in fewer than half of all participants and became progressively less frequent with increasing AKI severity.

The observed distribution was consistent with international evidence demonstrating that AKI is common, clinically heterogeneous, and closely linked to mortality. Hoste et al. reported AKI in 57.3% of critically ill patients across 97 centres and demonstrated a graded increase in adjusted mortality from KDIGO stage 1 through stage 3 [6]. Uchino et al. found severe acute renal failure in 5.7% of ICU admissions, with hospital mortality exceeding 60% among a population enriched for renal replacement therapy and multiorgan failure [7]. The lower mortality in the present cohort likely reflected inclusion of ward patients and less severe community-acquired disease in addition to critically ill patients. Sepsis accounted for 35.6% of cases and independently increased mortality. Bagshaw et al. similarly identified sepsis as a major cause of critical-illness-associated AKI and reported poorer outcomes in septic than nonseptic AKI [16]. Sepsis may impair renal function through interacting macrovascular, microcirculatory, inflammatory, endothelial, metabolic, and tubular mechanisms rather than through renal hypoperfusion alone. Consequently, serum creatinine may increase despite apparently acceptable systemic hemodynamics. The strong association between sepsis and death in the present analysis probably reflected both kidney injury and the severity of the underlying infection and organ dysfunction.

The etiological spectrum was comparable to several Indian investigations but differed from studies dominated by region-specific community

exposures. Priyamvada et al. reported sepsis as a major contributor among AKI patients admitted to multiple medical and surgical ICUs in southern India [9]. Eswarappa et al. also found infection and critical illness to be important drivers of severe AKI [14]. In contrast, Kaaviya et al. identified acute pyelonephritis and snakebite as leading causes of community-acquired AKI in Puducherry [10]. Vikrant et al. reported that more than 90% of AKI was community acquired in a Himalayan tertiary centre [13]. Such variation reinforces the influence of geography, occupation, referral pathways, climate, endemic infection, and medication practices on AKI presentation.

The 21.7% mortality rate was comparable to the 19.1% reported by Chandiraseharan et al. among AKI patients admitted to a general medical ward [15] and lower than the 28.4% reported by Saxena and Meshram in a medicine ICU [11]. Korula et al. demonstrated that severity scores, mechanical ventilation, and progressive kidney dysfunction influenced mortality in intensive-care AKI [12]. These comparisons suggest that the apparent mortality of AKI depends strongly on the proportion of septic patients, requirement for organ support, baseline renal function, referral delay, and whether the population was recruited from general wards or intensive-care units.

Mechanical ventilation remained independently associated with mortality. This relationship likely represented the combined effects of severe pulmonary disease, fluid overload, sepsis, positive-pressure ventilation, hemodynamic instability, and multiorgan dysfunction. Vasopressor requirement was strongly associated with death in unadjusted analysis but lost statistical independence after adjustment, probably because it overlapped with sepsis and respiratory failure. Similar associations between vasopressors, ventilation, shock, and death have been demonstrated in multinational and Indian ICU cohorts [7,9,11].

Approximately three-quarters of the cohort survived without dialysis dependence, but only 43.3% attained complete renal recovery before discharge. Wonnacott et al. demonstrated meaningful differences between community- and hospital-acquired AKI in severity and outcomes [17], while Xu et al. showed that AKI severity was associated with mortality and adverse renal outcomes among hospitalized adults [18]. The present recovery gradient across KDIGO stages supports the view that AKI should not be regarded as a fully reversible event once serum creatinine begins to decline.

Long-term follow-up was not included, but incomplete recovery may have important implications. Wang et al. found AKI to be independently associated with hospital mortality [19], and the meta-analysis by Coca et al. demonstrated increased subsequent risks of chronic kidney disease, kidney failure, and death [20]. Patients with partial recovery or dialysis-requiring AKI should therefore receive structured post-discharge reassessment of serum creatinine, albuminuria, blood pressure, medication safety, and cardiovascular risk rather than being discharged from renal surveillance after resolution of the acute illness. The findings have practical implications. Repeated creatinine and urine-output monitoring should be prioritized in septic, hypotensive, postoperative, and nephrotoxin-exposed patients. Potentially nephrotoxic medication should be reviewed daily, and obstruction should be excluded promptly when clinically suspected. Early treatment of infection, appropriate fluid resuscitation, avoidance of persistent positive fluid balance, and timely nephrology consultation may prevent progression. Fluid administration should nevertheless remain individualized because excessive accumulation has also been associated with reduced survival and impaired kidney recovery in critical illness [21].

The study had several limitations. It was conducted at a single tertiary centre and was susceptible to referral bias toward severe or diagnostically complex disease. Etiology was sometimes multifactorial, although one predominant cause was assigned for analysis. Baseline creatinine was unavailable in some screened patients, potentially excluding individuals with community-acquired AKI. Novel kidney-injury biomarkers were not evaluated. The number of deaths restricted the number of variables that could be included reliably in regression modelling. Finally, follow-up ended at hospital discharge, preventing assessment of 30-day mortality, recurrent AKI, persistent acute kidney disease, and progression to chronic kidney disease. Multicentre studies with standardized etiological adjudication and longitudinal renal follow-up are needed.

Conclusion

Acute kidney injury in this tertiary care cohort was characterized by a high frequency of sepsis, hypovolemia, nephrotoxic exposure, and advanced KDIGO stage at presentation. Although most patients survived without continuing dialysis, complete renal recovery occurred in fewer than half, and mortality increased sharply with disease severity. Older age, sepsis-associated AKI, KDIGO stage 3, and mechanical ventilation independently identified patients at greatest risk of death.

These findings emphasize the need for early detection, repeated severity assessment, rapid treatment of reversible causes, careful medication and fluid management, and coordinated nephrology–critical-care intervention. Survivors

with incomplete recovery should receive structured renal follow-up after discharge.

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