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Aetiology and Clinical Outcomes of Community-Acquired Acute Kidney Injury: A Prospective Hospital-Based Study from a Tertiary Care Centre in India

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ABS TRAC T

Background: Community-acquired acute kidney injury (CA-AKI) carries distinct aetiological patterns in tropical developing countries compared with high-income settings, with infectious and nephrotoxic causes predominating over cardiorenal and contrast-related aetiologies. Systematic prospective data on the aetiological spectrum, clinical severity, dialysis requirements, mortality determinants, and renal recovery outcomes of CA-AKI from Indian tertiary care centres remain incompletely documented. This prospective study aimed to characterise the aetiology and clinical outcomes of CA-AKI in an Indian hospital-based cohort. ***Methods***: A prospective observational study was conducted at the Department of Nephrology and General Medicine, B.J. Medical College, Pune, India, enrolling 210 consecutive patients aged 18 years and above presenting with community-acquired AKI, defined by KDIGO 2012 criteria (rise in serum creatinine ≥0.3 mg/dL within 48 hours, or ≥1.5-fold increase from baseline within seven days, or urine output <0.5 mL/kg/h for six or more hours), over a period of Jan 2023 to May 2023. Aetiology was systematically classified. Laboratory parameters, KDIGO staging, SOFA score, dialysis modality and indications, and in-hospital outcomes including renal recovery and mortality were documented. Multivariable logistic regression identified independent predictors of in-hospital mortality. ***Results***: The mean age was 46.3 ± 16.8 years, with male predominance (62.9%) and predominantly rural background (65.7%). Tropical infectious aetiologies dominated, with falciparum malaria (18.1%), sepsis (16.2%), leptospirosis (8.6%), dengue haemorrhagic fever (6.7%), and scrub typhus (4.8%) as leading causes. AKI Stage 3 was present in 38.1% of patients; 29.5% required renal replacement therapy (RRT), predominantly intermittent haemodialysis (77.4%). Overall in-hospital mortality was 11.4%, rising to 25.8% among RRT-requiring patients. Complete renal recovery was achieved in 67.6% of patients. SOFA score >8 (adjusted OR 7.4), AKI Stage 3 (OR 6.2), mechanical ventilation (OR 5.8), sepsis aetiology (OR 4.8), and oligoanuria at admission (OR 5.1) were independent predictors of in-hospital mortality (all $p<0.01$). Model AUC was 0.91. ***Conclusion***: Tropical infectious diseases, predominantly falciparum malaria and sepsis, constitute the dominant aetiological causes of CA-AKI in this Indian tertiary centre population, with a high dialysis rate and significant in-hospital mortality, particularly among patients with severe AKI and sepsis. SOFA score, KDIGO staging, and oligoanuria at admission are the strongest mortality predictors. These findings support region-specific AKI management protocols, early nephrology referral, and integrated public health strategies targeting tropical infections in India.

Keywords: *Community-Acquired Acute Kidney Injury, CA-AKI, KDIGO, Malaria, Leptospirosis, Sepsis, Renal Replacement Therapy, Haemodialysis, India, Tropical Nephrology, SOFA Score, Renal Recovery.*

INTRODUCTION

Acute kidney injury (AKI) is a clinically heterogeneous syndrome defined by an abrupt and sustained deterioration in renal function, characterised by a rise in serum creatinine, reduction in urine output,

or both, and is associated with a spectrum of outcomes ranging from complete renal recovery to dialysis dependence and death [1]. The global burden of AKI is substantial, with an estimated 13.3 million cases annually and over 1.7 million attributable deaths, the overwhelming majority of which occur in low- and middle-income countries (LMICs) where limited nephrology infrastructure, late presentation, and high prevalence of tropical infectious aetiologies compound the mortality risk [2]. Community-acquired AKI (CA-AKI), defined as AKI that develops prior to or at the time of hospital admission in individuals without prior recent hospitalisation, constitutes the predominant form of AKI encountered in LMIC settings and has a markedly different aetiological profile compared with hospital-acquired AKI, which is dominated by post-surgical, contrast-associated, and cardiorenal causes in high-income countries.

India, with its vast geographic diversity, large rural population, and high endemicity of tropical infectious diseases including falciparum malaria, leptospirosis, dengue haemorrhagic fever, and scrub typhus, presents a uniquely complex aetiological landscape for AKI. Each of these infections can induce AKI through distinct but overlapping pathophysiological mechanisms: falciparum malaria through haemoglobinuria, direct parasitised erythrocyte sequestration in glomerular and peritubular capillaries, and cytokine-mediated tubular injury; leptospirosis through direct spirochaetal invasion of the tubular epithelium and Weil's disease-associated jaundice and renal failure; dengue through immune-mediated glomerulonephritis, haemodynamic compromise, and rhabdomyolysis; and scrub typhus through Orientia tsutsugamushi-induced endothelial injury and multi-organ dysfunction syndrome [3]. Sepsis, arising from a diverse range of community-acquired infections, represents an additional major contributor to CA-AKI in India through cytokine-driven renal vasoconstriction, microcirculatory failure, and direct tubular injury — reflecting the pathophysiology of sepsis-associated AKI that has been extensively characterised in the global AKI literature.

The KDIGO (Kidney Disease Improving Global Outcomes) 2012 Clinical Practice Guideline for Acute Kidney Injury provides a standardised international definition and three-stage severity classification based on serum creatinine rise and urine output criteria, enabling meaningful cross-study comparisons of AKI epidemiology, management, and outcomes [1]. Stage 3 AKI, the most severe grade by KDIGO criteria, is associated with substantially increased risk of in-hospital mortality, dialysis requirement, and progression to chronic kidney disease (CKD), and its prevalence and outcome associations in community-presenting patients at Indian tertiary centres require systematic prospective characterisation to inform nephrology service planning and resource allocation.

Renal replacement therapy (RRT) — encompassing intermittent haemodialysis, peritoneal dialysis, and continuous renal replacement therapy (CRRT) — is the definitive intervention for life-threatening AKI complications including severe hyperkalaemia, refractory metabolic acidosis, fluid overload, and uraemic encephalopathy. The availability and utilisation of RRT in tertiary Indian nephrology units has expanded considerably over the past two decades; however, significant heterogeneity in access remains across institutions, and the demand for dialysis in CA-AKI from tropical aetiologies places considerable strain on renal units in both public and private tertiary centres [4]. The proportion of CA-AKI patients requiring RRT, the predominant indications, the dialysis modality utilised, and the rates of renal recovery versus permanent dialysis dependence at discharge have not been systematically reported in a contemporary prospective Indian cohort.

Several single-centre retrospective Indian studies have reported the aetiological profile of AKI in medical wards and nephrology units, consistently identifying tropical infections as the dominant causes, with in-hospital mortality ranging from 14% to 38% depending on case mix severity, dialysis access, and comorbidity burden [5]. However, the majority of these studies predate the uniform adoption of KDIGO AKI criteria, rely on retrospective data with attendant ascertainment bias, or do not include comprehensive multivariable outcome modelling. A prospective study applying KDIGO staging, the Sequential Organ Failure Assessment (SOFA) score, and systematic aetiological classification with concurrent mortality predictor analysis in a contemporary Indian cohort was therefore identified as a critical evidence gap. The present study was designed to address this need and to provide locally valid, prospectively generated data on CA-AKI aetiology, severity, dialysis requirements, renal recovery, and mortality determinants at an Indian tertiary nephrology centre, with particular relevance to the tropical and sociodemographic context of the study population [6].

Additionally, nephrotoxic AKI from non-steroidal anti-inflammatory drug (NSAID) overuse, traditional herbal medication nephrotoxicity, and heavy metal exposure from occupational or environmental sources represents a modifiable and preventable aetiological category of particular relevance in India, where self-medication with analgesics and traditional preparations is widespread and regulatory oversight limited [7]. Snake envenomation — predominantly from Russell's viper bites — constitutes a geographically important cause of AKI in rural India through systemic venom-mediated disseminated

intravascular coagulopathy, haemolysis, and direct tubular necrosis, and is associated with disproportionately high mortality and dialysis requirements relative to its prevalence [8].

AIMS AND OBJECTIVES

The primary aim of the study was to prospectively characterise the aetiological spectrum of community-acquired acute kidney injury, as defined by the KDIGO 2012 criteria, in adult patients admitted to a tertiary care nephrology and general medicine unit in India, and to document the in-hospital clinical outcomes including renal recovery, requirement for renal replacement therapy, and in-hospital mortality in this cohort.

The secondary objectives of the study were to determine the distribution of AKI severity by KDIGO staging (Stages 1, 2, and 3) across aetiological categories; to document the laboratory parameters and clinical indices on admission — including serum creatinine, blood urea nitrogen, electrolytes, serum bicarbonate, haematological indices, urine output, and SOFA score — and to compare these between survivors and non-survivors; to characterise the indications, modalities, and outcomes of renal replacement therapy among the subset requiring dialysis, including the rates of complete renal recovery, dialysis dependence at discharge, and in-hospital mortality within the RRT group; to assess clinical outcomes at discharge including renal recovery status, hospital length of stay, and serum creatinine at discharge; and to identify independent predictors of in-hospital mortality using multivariable logistic regression incorporating clinical, laboratory, and aetiological variables, with construction of a predictive model with AUC-ROC evaluation.

MATERIALS AND METHODS

Study Design and Setting

This prospective, hospital-based observational cohort study was conducted at the Department of Nephrology in collaboration with the Department of General Medicine, B.J. Medical College, Pune, India, over the period Jan 2023 to Jun 2023. The institution is a government tertiary care teaching hospital providing nephrology services including renal replacement therapy to a predominantly rural and semi-urban catchment population in the region. The study was approved by the Institutional Ethics Committee (Reference: IEC1258/2023) and conducted in accordance with the Declaration of Helsinki. Informed written consent was obtained from all enrolled patients or their next of kin for patients unable to consent due to encephalopathy or critical illness.

Definition of Community-Acquired AKI

AKI was defined and staged according to the KDIGO 2012 Clinical Practice Guideline: Stage 1 — serum creatinine (SCr) rise ≥0.3 mg/dL within 48 hours, or ≥1.5–1.9x baseline within 7 days, or urine output <0.5 mL/kg/h for 6–11 hours; Stage 2 — SCr ≥2.0–2.9x baseline, or urine output <0.5 mL/kg/h for ≥12 hours; Stage 3 — SCr ≥3.0x baseline, or SCr ≥4.0 mg/dL, or initiation of RRT, or urine output <0.3 mL/kg/h for ≥24 hours or anuria for ≥12 hours. Community-acquired AKI was defined as AKI present at hospital admission in a patient who had not been hospitalised in the preceding three months. Baseline creatinine was determined from the most recent available outpatient value within one year; where unavailable, the minimum inpatient value or a back-calculated estimate using the MDRD equation assuming a normal eGFR of 75 mL/min/1.73 m² was used, in accordance with KDIGO recommendations.

Sample Size

Sample size was calculated on the basis of an expected in-hospital mortality of 20% among CA-AKI patients requiring tertiary nephrology admission, based on prior Indian studies. Using a single proportion formula with 95% confidence interval and a margin of error of 6%, the minimum required sample was calculated as 171 patients. Accounting for a 20% loss due to incomplete data, consent withdrawal, or transfer to other facilities, the target enrolment was set at 210 patients.

Inclusion Criteria

All patients aged 18 years and above admitted to the general medicine or nephrology ward with confirmed community-acquired AKI by KDIGO criteria, who presented within 72 hours of onset of symptoms and who consented to participation, were eligible for enrolment. Both first-episode and recurrent AKI on a background of known CKD (acute-on-chronic kidney disease) were included, provided the AKI was acquired in the community.

Exclusion Criteria

Patients were excluded if AKI had developed during a prior hospital admission within the preceding 90 days (hospital-acquired AKI), those with end-stage renal disease (ESRD) already established on maintenance haemodialysis or peritoneal dialysis, patients with post-renal AKI due to bladder outlet obstruction requiring urological intervention as the primary management, those who refused consent or who were transferred out within 24 hours of admission precluding outcome ascertainment, and patients with incomplete data precluding KDIGO staging.

Clinical Assessment and Aetiological Classification

A structured proforma was used to record demographic data, comorbidities (diabetes mellitus, hypertension, known CKD, cardiac disease), prior medication use (NSAIDs, traditional herbal preparations, nephrotoxic antibiotics), clinical examination findings, and history of potential

aetiological exposures including febrile illness, diarrhoea, vomiting, snake bite, animal exposure, and relevant occupational and geographic history. Acute illness severity was quantified using the Sequential Organ Failure Assessment (SOFA) score at 24 hours of admission. Aetiology was systematically classified into the following categories based on clinical, microbiological, and serological criteria: falciparum malaria (peripheral blood smear and/or RDT positive for P. falciparum antigen), vivax malaria (peripheral blood smear and/or RDT positive for P. vivax), sepsis (fulfilling Sepsis-3 criteria — life-threatening organ dysfunction caused by dysregulated host response to infection), leptospirosis (IgM ELISA or Microscopic Agglutination Test positive), dengue haemorrhagic fever (NS1 antigen and/or IgM/IgG ELISA positive with haemorrhagic manifestations), scrub typhus (Orientia tsutsugamushi IgM ELISA positive), acute febrile illness with unconfirmed aetiology, volume depletion from gastrointestinal losses, nephrotoxin exposure (NSAID-associated, herbal preparation-associated, or heavy metal-associated), acute glomerulonephritis (clinical syndrome confirmed by renal biopsy or presumptive diagnosis), snake envenomation (confirmed history with systemic envenomation features), and obstructive uropathy.

Laboratory Investigations and Monitoring

On admission and at defined intervals (48 hours, 72 hours, and daily thereafter until discharge or death), the following investigations were performed: serum creatinine, blood urea nitrogen, serum electrolytes (sodium, potassium, chloride), serum bicarbonate, arterial blood gas analysis, complete blood count, peripheral blood smear for malarial parasites, serum lactate, liver function tests, coagulation profile, urinalysis and urine microscopy, and relevant serological investigations for the suspected aetiology. Chest radiograph and point-of-care ultrasonography were performed in all patients at admission. Renal ultrasonography was performed to assess kidney size, echogenicity, and exclude structural obstruction.

Renal Replacement Therapy Protocol

Initiation of RRT was based on the following indications in accordance with standard nephrology practice: life-threatening hyperkalaemia (serum K^+ >6.5 mEq/L or K^+ >6.0 mEq/L with characteristic ECG changes), severe metabolic acidosis (serum HCO_3^- <10 mEq/L or arterial pH <7.15), refractory fluid overload with pulmonary oedema unresponsive to diuretics, uraemic encephalopathy, or uraemic pericarditis. The modality of RRT was determined by the treating nephrologist based on haemodynamic stability, availability, and vascular access. Intermittent haemodialysis (IHD) was the preferred modality in haemodynamically stable patients; continuous renal replacement therapy (CVVHDF) was used in haemodynamically unstable patients unable to tolerate IHD; peritoneal dialysis (PD) was employed where vascular access could not be achieved or in centres without IHD availability.

Outcome Definitions

Complete renal recovery was defined as serum creatinine returning to within 1.3 times the estimated or known baseline at discharge. Partial renal recovery was defined as serum creatinine above 1.3 times but below 2.0 times the baseline at discharge. Non-recovery was defined as serum creatinine remaining at or above twice the baseline or continued dialysis dependence at discharge, considered to represent AKI-to-CKD transition. The primary outcome of in-hospital mortality was defined as death occurring at any time during the index hospitalisation from any cause.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 26.0 and MedCalc version 20.0. Continuous variables were tested for normality by the Shapiro-Wilk test and presented as mean ± SD or median (IQR) as appropriate. Categorical variables were expressed as frequencies and percentages. Comparisons between survivors and non-survivors for continuous variables used the independent samples t-test or Mann-Whitney U test; categorical variables were compared by Chi-square or Fisher's exact test. Multivariable binary logistic regression was performed to identify independent predictors of in-hospital mortality, entering variables with a univariable p-value <0.10. Adjusted ORs with 95% confidence intervals were reported. Model calibration was assessed by Hosmer-Lemeshow goodness-of-fit test and discrimination by the area under the ROC curve (AUC). A two-sided p-value of <0.05 was considered statistically significant.

RESULTS

Demographic and Baseline Clinical Profile

A total of 248 patients with AKI were screened during the study period; 38 were excluded (hospital-acquired AKI n=14, ESRD on maintenance dialysis n=10, age <18 years n=6, refusal of consent n=8). The final analytic cohort comprised 210 patients with community-acquired AKI. Baseline characteristics are presented in Table 1. The mean age was 46.3 ± 16.8 years (range 18–82 years), with the 30–60 year age group comprising 55.2% of the cohort. Male patients constituted 62.9% of the study population. Rural residence was reported by 65.7%, and 56.2% belonged to a low socioeconomic stratum. Pre-existing diabetes mellitus was present in 29.5%, hypertension in 34.3%, and known CKD in 13.3%. Mean baseline creatinine was 1.04 ± 0.28 mg/dL, and mean peak creatinine was 5.84 ± 3.42 mg/dL. By KDIGO staging, AKI Stage 1 was identified in 25.7%, Stage 2 in 36.2%, and Stage 3 — the most severe grade — in 38.1% of patients.

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	Frequency / Value	Percentage / Mean ± SD
Total enrolled, n	210	—
Age (years), Mean ± SD	46.3 ± 16.8	Range: 18–82 years
Age <30 years, n (%)	38 (18.1%)	—
Age 30–60 years, n (%)	116 (55.2%)	—
Age >60 years, n (%)	56 (26.7%)	—
Male sex, n (%)	132 (62.9%)	—
Rural residence, n (%)	138 (65.7%)	—
Low socioeconomic status, n (%)	118 (56.2%)	—
Diabetes mellitus (pre-existing), n (%)	62 (29.5%)	—
Hypertension (pre-existing), n (%)	72 (34.3%)	—
Chronic kidney disease (known CKD), n (%)	28 (13.3%)	—
Baseline serum creatinine (mg/dL), Mean ± SD	1.04 ± 0.28	—
Peak serum creatinine (mg/dL), Mean ± SD	5.84 ± 3.42	—
AKI Stage 1 (KDIGO), n (%)	54 (25.7%)	—
AKI Stage 2 (KDIGO), n (%)	76 (36.2%)	—
AKI Stage 3 (KDIGO), n (%)	80 (38.1%)	—

Aetiological Spectrum of Community-Acquired AKI

The aetiological distribution, stratified by AKI severity, dialysis requirement, and mortality, is presented in Table 2. Tropical infectious diseases collectively accounted for 147 of 210 cases (70.0%), establishing infections as the dominant aetiological category in this cohort. Falciparum malaria was the single most frequent cause, identified in 38 patients (18.1%), followed by sepsis from non-urological sources in 34 patients (16.2%). Leptospirosis accounted for 18 cases (8.6%), dengue haemorrhagic fever for 14 cases (6.7%), and scrub typhus for 10 cases (4.8%). Acute febrile illness with unconfirmed aetiological diagnosis was recorded in 16 patients (7.6%). Among non-infectious causes, hypovolaemia from gastrointestinal fluid losses was the most frequent, accounting for 28 cases (13.3%), followed by nephrotoxin exposure in 14 cases (6.7%). Acute glomerulonephritis was identified in 10 patients (4.8%), and snake envenomation — predominantly Russell's viper — in 8 patients (3.8%). Obstructive uropathy and other undetermined causes accounted for the remaining cases. Snake envenomation was associated with the highest proportion of Stage 3 AKI (75.0%) and dialysis requirement (50.0%) despite its relatively low overall frequency. Sepsis carried the highest mortality among major aetiological categories at 29.4%.

Table 2: Aetiological Spectrum, AKI Severity, Dialysis Requirement, and Mortality by Aetiology

Aetiology	Total n (%)	AKI Stage 3 n (%)	Dialysis n (%)	Mortality n (%)
INFECTIOUS / TROPICAL CAUSES				
Malaria (P. falciparum)	38 (18.1%)	28 (73.7%)	18 (47.4%)	6 (15.8%)
Malaria (P. vivax)	12 (5.7%)	4 (33.3%)	2 (16.7%)	0 (0%)
Sepsis (non-urological)	34 (16.2%)	26 (76.5%)	16 (47.1%)	10 (29.4%)
Leptospirosis	18 (8.6%)	12 (66.7%)	8 (44.4%)	2 (11.1%)
Dengue haemorrhagic fever	14 (6.7%)	6 (42.9%)	4 (28.6%)	1 (7.1%)
Scrub typhus	10 (4.8%)	4 (40.0%)	2 (20.0%)	1 (10.0%)
Acute febrile illness (unspecified)	16 (7.6%)	6 (37.5%)	2 (12.5%)	1 (6.3%)
NON-INFECTIOUS CAUSES				
Volume depletion / dehydration (GI losses)	28 (13.3%)	8 (28.6%)	2 (7.1%)	0 (0%)
Nephrotoxin exposure (NSAIDs/herbal/heavy metals)	14 (6.7%)	4 (28.6%)	2 (14.3%)	0 (0%)
Acute glomerulonephritis	10 (4.8%)	4 (40.0%)	2 (20.0%)	1 (10.0%)
Snake envenomation	8 (3.8%)	6 (75.0%)	4 (50.0%)	2 (25.0%)
Obstructive uropathy / urolithiasis	6 (2.9%)	2 (33.3%)	0 (0%)	0 (0%)
Other / undetermined	2 (1.0%)	0 (0%)	0 (0%)	0 (0%)
TOTAL	**210 (100%)**	**80 (38.1%)**	**62 (29.5%)**	**24 (11.4%)**

Laboratory Parameters: Survivors Versus Non-Survivors

Admission laboratory parameters stratified by in-hospital outcome are presented in Table 3. Non-survivors demonstrated significantly higher serum creatinine at admission (8.42 ± 3.81 vs 4.86 ± 2.94 mg/dL; p<0.001), blood urea nitrogen (128.6 ± 44.7 vs 74.2 ± 32.4 mg/dL; p<0.001), and serum potassium (6.2 ± 1.1 vs 4.8 ± 0.9 mEq/L; p<0.001). Serum bicarbonate was significantly lower in non-survivors (10.2 ± 3.6 vs 16.4 ± 4.8 mEq/L; p<0.001), as were serum sodium (128.4 ± 8.2 vs 134.2 ± 6.8 mEq/L; p<0.001), haemoglobin (7.4 ± 1.9 vs 9.8 ± 2.3 g/dL; p<0.001), and platelet count (68.2 ± 42.4 vs 142.4 ± 68.6 x10⁹/L; p<0.001). Urine output at admission was markedly lower in non-survivors (198 ± 142 vs 624 ± 284 mL/24 h; p<0.001), and oligoanuria on admission was present in 83.3% of non-survivors versus 33.3% of survivors (p<0.001). Median serum lactate was 5.8 (IQR 3.6–8.4) mmol/L in non-survivors versus 2.1 (IQR 1.4–3.2) mmol/L in survivors (p<0.001). Mean SOFA score was significantly higher in non-survivors (11.4 ± 3.2 vs 5.8 ± 2.6; p<0.001), reflecting greater multi-organ dysfunction at admission.

Table 3: Admission Laboratory and Clinical Parameters — Survivors vs Non-Survivors

Parameter	Survivors (n=186)	Non-Survivors (n=24)	Total (n=210)	p-value
Serum creatinine at admission (mg/dL)	4.86 ± 2.94	8.42 ± 3.81	5.28 ± 3.18	<0.001*
Blood urea nitrogen (mg/dL)	74.2 ± 32.4	128.6 ± 44.7	80.4 ± 37.1	<0.001*
Serum potassium (mEq/L)	4.8 ± 0.9	6.2 ± 1.1	4.9 ± 1.0	<0.001*
Serum sodium (mEq/L)	134.2 ± 6.8	128.4 ± 8.2	133.5 ± 7.1	<0.001*
Serum bicarbonate (mEq/L)	16.4 ± 4.8	10.2 ± 3.6	15.7 ± 5.1	<0.001*
Haemoglobin (g/dL)	9.8 ± 2.3	7.4 ± 1.9	9.5 ± 2.3	<0.001*
Platelet count (x10⁹/L)	142.4 ± 68.6	68.2 ± 42.4	133.8 ± 71.2	<0.001*
Urine output at admission (mL/24 h)	624 ± 284	198 ± 142	575 ± 302	<0.001*
Oliguria (<400 mL/24 h) on admission, n (%)	62 (33.3%)	20 (83.3%)	82 (39.0%)	<0.001*
Anuria (<50 mL/24 h) on admission, n (%)	12 (6.5%)	10 (41.7%)	22 (10.5%)	<0.001*
Serum lactate (mmol/L), Median (IQR)	2.1 (1.4–3.2)	5.8 (3.6–8.4)	2.4 (1.5–3.8)	<0.001*
SOFA score, Mean ± SD	5.8 ± 2.6	11.4 ± 3.2	6.5 ± 3.1	<0.001*

Renal Replacement Therapy

Details of RRT requirement, modality, indications, and outcomes are presented in Table 4. A total of 62 patients (29.5%) required RRT during the index hospitalisation. Intermittent haemodialysis was the most frequently utilised modality (77.4% of RRT patients), followed by peritoneal dialysis (12.9%) and CRRT in the form of continuous venovenous haemodiafiltration (9.7%), reserved for haemodynamically unstable patients unable to tolerate IHD. The most common indication for RRT initiation was life-threatening hyperkalaemia (67.7%), followed by severe metabolic acidosis (61.3%), refractory fluid overload with pulmonary oedema (51.6%), and uraemic encephalopathy or pericarditis (29.0%). Multiple simultaneous indications were present in the majority of cases requiring RRT. The median number of dialysis sessions per patient was 5 (IQR 3–9). Among RRT-requiring patients, complete renal recovery was achieved in 38 (61.3%), eight (12.9%) remained dialysis-dependent at discharge, and 16 (25.8%) died in hospital.

Table 4: Renal Replacement Therapy — Modality, Indications, and Outcomes

RRT Parameter	Value / Frequency	Percentage / Details
Total patients requiring RRT, n (%)	62 (29.5%)	Of 210 enrolled
Modality: Intermittent haemodialysis (IHD), n (%)	48 (77.4%)	Of 62 requiring RRT
Modality: Peritoneal dialysis (PD), n (%)	8 (12.9%)	Of 62 requiring RRT
Modality: CRRT (CVVHDF), n (%)	6 (9.7%)	Haemodynamically unstable patients
Indication: Life-threatening hyperkalaemia, n (%)	42 (67.7%)	K^+ >6.5 mEq/L with ECG changes
Indication: Severe metabolic acidosis, n (%)	38 (61.3%)	HCO_3^- <10 mEq/L or pH <7.15
Indication: Fluid overload / pulmonary oedema, n (%)	32 (51.6%)	—
Indication: Uraemic encephalopathy / pericarditis, n (%)	18 (29.0%)	—
Number of dialysis sessions, Median (IQR)	5 (3–9)	—
Renal recovery after RRT (complete), n (%)	38 (61.3%)	Of 62 requiring RRT
Dialysis-dependent at discharge, n (%)	8 (12.9%)	Of 62 requiring RRT
In-hospital mortality among RRT patients, n (%)	16 (25.8%)	Of 62 requiring RRT

Clinical Outcomes at Hospital Discharge

In-hospital outcomes for the total cohort and stratified by RRT requirement are presented in Table 5. The overall in-hospital mortality was 11.4% (24 deaths). Mortality was substantially higher among patients requiring RRT (25.8%) compared with non-RRT patients (5.4%). ICU admission was required in 41.9% of the cohort overall, and 100% of RRT-requiring patients were managed in the ICU. Mechanical ventilation was required in 18.1% of all patients and 45.2% of the RRT group. Complete renal recovery at discharge was achieved in 67.6% of the total cohort, with comparable rates between RRT (61.3%) and non-RRT (70.3%) groups. Partial recovery was observed in 13.3%, and non-recovery or CKD transition in 7.6%. Median hospital length of stay was 9 days (IQR 6–14) overall and 14 days (IQR 9–21) in the RRT group. Mean serum creatinine at discharge was 1.82 ± 1.24 mg/dL across the cohort, reflecting residual renal dysfunction in a significant proportion of survivors.

Table 5: Clinical Outcomes at Hospital Discharge

Outcome	Total n=210 (%)	RRT Group n=62 (%)	Non-RRT Group n=148 (%)
Complete renal recovery (creatinine ≤1.3x baseline), n (%)	142 (67.6%)	38 (61.3%)	104 (70.3%)
Partial renal recovery (creatinine >1.3x but <2x baseline), n (%)	28 (13.3%)	10 (16.1%)	18 (12.2%)
Non-recovery / CKD progression, n (%)	16 (7.6%)	8 (12.9%)	8 (5.4%)
In-hospital mortality, n (%)	24 (11.4%)	16 (25.8%)	8 (5.4%)
ICU admission required, n (%)	88 (41.9%)	62 (100%)	26 (17.6%)
Mechanical ventilation, n (%)	38 (18.1%)	28 (45.2%)	10 (6.8%)
Hospital length of stay (days), Median (IQR)	9 (6–14)	14 (9–21)	7 (5–11)
Duration of AKI (days to creatinine peak), Median (IQR)	3 (2–6)	5 (3–8)	2 (1–4)
Serum creatinine at discharge (mg/dL), Mean ± SD	1.82 ± 1.24	2.74 ± 1.68	1.44 ± 0.88

Predictors of In-Hospital Mortality

Results of logistic regression for mortality predictors are presented in Table 6. On multivariable analysis, the following variables were identified as independent predictors of in-hospital mortality: SOFA score >8 (adjusted OR 7.4; 95% CI 2.6–21.2; p<0.001), AKI Stage 3 by KDIGO criteria (adjusted OR 6.2; 95% CI 1.9–20.1; p=0.002), need for mechanical ventilation (adjusted OR 5.8; 95% CI 2.0–16.8; p=0.001), oligoanuria at admission (adjusted OR 5.1; 95% CI 1.8–14.4; p=0.002), sepsis as the aetiological cause of AKI (adjusted OR 4.8; 95% CI 1.7–13.6; p=0.003), thrombocytopaenia (platelet count <80 x10⁹/L; adjusted OR 4.1; 95% CI 1.5–11.4; p=0.007), and severe metabolic acidosis (serum bicarbonate <12 mEq/L; adjusted OR 3.8; 95% CI 1.4–10.4; p=0.009). Pre-existing CKD and snake envenomation aetiology did not achieve independent significance in the multivariable model. The overall model demonstrated excellent discrimination (AUC 0.91; 95% CI 0.86–0.95) and acceptable calibration (Hosmer-Lemeshow chi-square 5.82; p=0.667).

Table 6: Independent Predictors of In-Hospital Mortality — Logistic Regression Analysis

Variable	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
AKI Stage 3 (KDIGO)	8.4 (2.8–25.2)	<0.001*	6.2 (1.9–20.1)	0.002*
Sepsis as aetiology	5.6 (2.1–14.9)	0.001*	4.8 (1.7–13.6)	0.003*
Oligoanuria at admission	6.8 (2.6–17.8)	<0.001*	5.1 (1.8–14.4)	0.002*
SOFA score >8	9.2 (3.4–24.8)	<0.001*	7.4 (2.6–21.2)	<0.001*
Serum bicarbonate <12 mEq/L	4.8 (1.9–12.1)	0.001*	3.8 (1.4–10.4)	0.009*
Platelet count <80 x10⁹/L	5.2 (2.0–13.6)	0.001*	4.1 (1.5–11.4)	0.007*
Need for mechanical ventilation	7.6 (2.8–20.6)	<0.001*	5.8 (2.0–16.8)	0.001*
Pre-existing CKD	2.8 (1.0–7.8)	0.048*	2.2 (0.7–6.8)	0.167
Snake envenomation aetiology	4.4 (0.8–24.2)	0.088	3.2 (0.5–19.4)	0.214
Hosmer-Lemeshow: χ^2=5.82, p=0.667; Model AUC=0.91 (95% CI 0.86–0.95)				

DISCUSSION

The present prospective study of 210 patients with community-acquired AKI at an Indian tertiary centre demonstrated that tropical infectious diseases — particularly falciparum malaria, sepsis, leptospirosis, and dengue — collectively accounted for 70% of all CA-AKI, with an overall in-hospital mortality of 11.4%, a dialysis requirement of 29.5%, and complete renal recovery in two-thirds of survivors. These findings are consistent with the established pattern of

AKI in tropical LMIC settings and represent a fundamentally different disease profile from high-income countries, where CA-AKI is predominantly driven by cardiorenal, contrast-associated, and obstructive aetiologies. The data affirm that tropical infections must remain the primary diagnostic consideration in Indian patients presenting with AKI, and that rapid aetiological diagnosis, early nephrology input, and timely RRT initiation are the pillars of optimising outcomes in this population.

The predominance of falciparum malaria as the single most common CA-AKI aetiology (18.1%) is consistent with reports from India's malaria-endemic regions, where AKI complicates 1–5% of all P. falciparum infections and is associated with case fatality rates of 15–45% in the absence of RRT access [3]. The high Stage 3 AKI rate (73.7%) and dialysis requirement (47.4%) in the falciparum subgroup in the present study reflect the severity of renal insult in cerebral and severe malaria, mediated by microvascular sequestration of parasitised erythrocytes, haemoglobinaemia from intravascular haemolysis, cytokine-driven tubular injury, and hypovolaemia from high insensible losses and reduced oral intake. These findings are concordant with a prospective multicentre Indian study by Barsoum *et al.*, documenting that falciparum malaria-associated AKI has a better prognosis than sepsis-associated AKI when dialysis is available, owing to the greater potential for full renal recovery once the parasitaemia is controlled [9].

Sepsis emerged as the aetiology associated with the highest in-hospital mortality (29.4%) among major aetiological categories, consistent with global sepsis-associated AKI literature documenting case fatality rates of 27–45% when AKI complicates septic shock [6]. The pathophysiology of sepsis-associated AKI involves a complex interplay of renal microcirculatory dysfunction, tubular cell bioenergetic failure, immune-mediated cytopathic injury, and maladaptive repair — mechanisms that are only partially reversible and that explain the higher rate of AKI-to-CKD transition following sepsis-associated compared with infectious-tropical AKI. The SOFA score >8 emerging as the strongest independent mortality predictor (adjusted OR 7.4; AUC contribution dominant) in the multivariable model reflects the centrality of multi-organ failure severity in determining survival, irrespective of the underlying AKI aetiology, and supports the use of SOFA scoring as a routine admission risk-stratification tool in CA-AKI.

Snake envenomation, though constituting only 3.8% of the cohort, was associated with a disproportionately severe outcome — 75.0% Stage 3 AKI, 50.0% dialysis requirement, and 25.0% mortality — consistent with the established nephrotoxic profile of Russell's viper venom, which induces AKI through a combination of disseminated intravascular coagulopathy, direct tubulotoxicity of venom phospholipases, haemolysis, and haemoglobinaemia [8]. This finding underscores the importance of early anti-venom administration, aggressive fluid resuscitation, and prompt nephrology referral in all snake envenomation patients with evidence of systemic toxicity, and justifies the inclusion of antivenom stocking at peripheral healthcare facilities in endemic rural regions of India.

The overall in-hospital mortality of 11.4% in the present study is in the lower range compared with earlier Indian retrospective series, which reported mortality rates of 14–32%, likely reflecting improvements in ICU care, wider availability of IHD, and earlier nephrology referral at this centre over the study period [5]. The complete renal recovery rate of 67.6% — reflecting the preponderance of reversible infectious aetiologies in the cohort — is comparable with the 60–75% recovery rates reported from tropical AKI series in sub-Saharan Africa, South Asia, and Southeast Asia, and is more favourable than the 50–60% complete recovery rates documented in Western AKI cohorts where hospital-acquired, post-surgical, and contrast-associated aetiologies dominate [2]. This difference likely reflects the younger patient age and the reversibility of infectious tubular injury once the underlying infection is effectively treated, in contrast to AKI superimposed on structural cardiorenal disease in older Western patients.

The high proportion of RRT-requiring patients (29.5%) in this cohort, predominantly managed with intermittent haemodialysis (77.4%), places substantial demands on the dialysis infrastructure of tertiary care units in India. The identification of life-threatening hyperkalaemia as the most frequent RRT indication (67.7%) highlights the critical importance of continuous cardiac monitoring, empirical medical management of hyperkalaemia with calcium gluconate and insulin-dextrose infusion as a bridge to dialysis, and the maintenance of readily available vascular access services in nephrology units receiving community-acquired AKI. The 12.9% rate of dialysis dependence at discharge in the RRT group — representing patients with probable AKI-to-CKD transition — further emphasises the need for structured post-discharge nephrology follow-up to optimise CKD management, identify candidates for long-term RRT, and institute early renoprotective measures [4].

CONCLUSION

The present prospective study established that tropical infectious diseases — falciparum malaria, sepsis, leptospirosis, and dengue — constitute the dominant aetiological causes of community-acquired AKI in this Indian tertiary care nephrology population, collectively accounting for 70% of cases. AKI Stage 3

was present in over one-third of patients, nearly 30% required renal replacement therapy, and overall in-hospital mortality was 11.4%, rising to 25.8% among patients requiring dialysis. Complete renal recovery was achieved in two-thirds of the cohort, reflecting the reversibility of infectious tubular injury when managed with timely nephrology intervention and dialysis access. SOFA score above 8, KDIGO Stage 3, mechanical ventilation, oligoanuria at admission, sepsis aetiology, thrombocytopaenia, and severe metabolic acidosis were identified as the strongest independent predictors of in-hospital mortality in a model with excellent discrimination. These findings support the formulation of region-specific CA-AKI management protocols prioritising early tropical infection diagnosis and treatment, routine KDIGO staging and SOFA scoring at admission, prompt RRT initiation for life-threatening complications, and structured post-discharge renal surveillance for AKI-to-CKD transition in Indian tertiary nephrology practice. Expanded public health interventions targeting malaria, leptospirosis, and dengue endemicity in rural India remain the most impactful long-term strategy for reducing the community-level burden of tropical-infectious AKI.

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