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Clinico-Epidemiological Profile of Acute Kidney Injury Due to Tropical Diseases: A Prospective Observational Study from a Tertiary Care Hospital.

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Abstract

Background: Background: Tropical diseases are important and potentially preventable causes of acute kidney injury (AKI) in endemic regions. Their renal manifestations range from mild, reversible dysfunction to severe AKI requiring dialysis and intensive care. This study evaluated the clinico-epidemiological profile, etiological spectrum, severity, treatment requirements, and in-hospital outcomes of tropical disease-associated AKI. ***Aim:*** To study the clinico-epidemiological profile, etiological spectrum, severity, and in-hospital outcomes of AKI due to tropical diseases. ***Materials and Methods:*** This hospital-based prospective observational study included 80 adults with laboratory-supported tropical diseases who fulfilled the Kidney Disease: Improving Global Outcomes criteria for AKI. Demographic characteristics, epidemiological exposures, clinical manifestations, laboratory findings, etiological diagnosis, KDIGO stage, treatment requirements, renal recovery, and in-hospital mortality were recorded. Stage 3 AKI was compared with Stages 1-2. Factors associated with an adverse outcome, defined as in-hospital death or renal non-recovery, were assessed using logistic regression. A two-sided p value <0.05 was considered statistically significant. ***Results:*** The mean age was 43.8±14.3 years; 53 (66.3%) patients were male, 49 (61.3%) were rural residents, and 57 (71.3%) presented during the monsoon or post-monsoon period. Dengue was the most common etiology (28.8%), followed by malaria (23.8%), scrub typhus (21.3%), leptospirosis (13.8%), enteric fever (7.5%), and mixed infection (5.0%). KDIGO Stages 1, 2, and 3 accounted for 33.8%, 30.0%, and 36.3% of cases, respectively. Stage 3 AKI was significantly associated with older age, rural residence, prolonged fever, delayed presentation, dehydration, oliguria, hypotension or shock, altered sensorium, higher admission serum creatinine, and lower platelet count. Renal replacement therapy was required in 18 (22.5%) patients. Complete renal recovery occurred in 49 (61.3%), partial recovery in 17 (21.3%), and renal non-recovery in five (6.3%); nine patients (11.3%) died. Stage 3 AKI was strongly associated with dialysis and mortality. On multivariable analysis, Stage 3 AKI (adjusted OR=6.42; 95% CI: 1.31-31.46), hypotension or shock (adjusted OR=5.18; 95% CI: 1.35-19.91), and mechanical ventilation (adjusted OR=6.76; 95% CI: 1.47-31.12) independently predicted adverse outcomes. ***Conclusion:*** Tropical disease-associated AKI demonstrated marked seasonal and rural predominance. Although most patients experienced renal recovery, Stage 3 AKI, haemodynamic instability, and respiratory failure were associated with poor outcomes. Early diagnosis, prompt etiological treatment, careful fluid and haemodynamic management, and timely availability of intensive care and dialysis may improve survival and renal recovery.

Keywords: Acute kidney injury; Tropical diseases; Renal replacement therapy.

INTRODUCTION

Acute kidney injury (AKI) is a clinical syndrome characterized by an abrupt deterioration in kidney function, resulting in the accumulation of nitrogenous waste products and disturbances in fluid, electrolyte and acid-base balance. According to the Kidney Disease: Improving Global Outcomes criteria, AKI is 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 baseline value within seven days, or urine output below 0.5 mL/kg/hour for six hours [1]. AKI is an important cause of morbidity and mortality among hospitalized patients, particularly in low- and middle-income countries where delayed presentation, limited diagnostic facilities and restricted

access to renal replacement therapy remain major challenges. Tropical infections constitute a distinctive and potentially preventable group of causes of AKI in endemic regions. Malaria, dengue, leptospirosis, scrub typhus, enteric fever and mixed tropical infections may produce renal injury through hypovolaemia, systemic inflammation, endothelial dysfunction, septic shock, haemolysis, rhabdomyolysis, microvascular obstruction, immune-mediated injury and direct tubulointerstitial involvement [2].

The clinical presentation may range from asymptomatic elevation of serum creatinine and mild urinary abnormalities to severe oliguric AKI accompanied by hyperkalaemia, metabolic acidosis, pulmonary oedema, multiorgan dysfunction and the need for dialysis. The distribution of causative diseases varies according to geographical region, season, rainfall, vector density, environmental exposure and referral patterns. Nair et al. reported that malaria, dengue, scrub typhus and leptospirosis were important causes of AKI among patients presenting with tropical acute febrile illnesses in southern India [3]. Similarly, a prospective study from northern India found AKI in approximately one-third of patients with tropical acute febrile illness, with malaria and scrub typhus making substantial contributions to the disease burden [4].

Early recognition is important because a considerable proportion of infection-associated renal dysfunction may be reversible with prompt diagnosis, appropriate antimicrobial therapy, careful fluid management, correction of haemodynamic instability and timely renal replacement therapy. However, overlapping clinical manifestations, coinfections and variations in the availability and timing of diagnostic tests frequently complicate etiological diagnosis. Contemporary diagnostic recommendations also emphasize selecting tests according to the stage of illness; for example, dengue testing during the acute phase may involve nucleic acid amplification or NS1 antigen detection, whereas serological tests become more useful later in the illness [5]. Systematic evaluation of the demographic characteristics, clinical manifestations, etiological spectrum, severity and short-term outcomes of patients with tropical disease-associated AKI can identify high-risk groups and facilitate earlier intervention. The present study was therefore undertaken to describe the clinico-epidemiological profile, renal manifestations, etiological distribution, treatment requirements and in-hospital outcomes of patients with AKI caused by tropical diseases at a tertiary care hospital.

AIM

To study the clinico-epidemiological profile, etiological spectrum, severity and in-hospital outcomes of acute kidney injury due to tropical diseases.

OBJECTIVES

1. To describe the demographic, epidemiological and clinical characteristics of patients presenting with acute kidney injury due to tropical diseases.
2. To determine the etiological distribution and severity of acute kidney injury according to the KDIGO classification.
3. To assess treatment requirements, renal recovery, need for renal replacement therapy and in-hospital mortality and to identify factors associated with adverse outcomes.

MATERIALS AND METHODS

Source of Data

The study participants were recruited from adult patients admitted to the Department of General Medicine, emergency services, intensive care unit and nephrology services. Patients who developed AKI in association with a clinically suspected and laboratory-confirmed tropical disease during the study period were screened for eligibility. Information was obtained from the patients, relatives, hospital case records, laboratory reports, nursing charts and treatment records.

Study Design

This was a hospital-based prospective observational study. No intervention was assigned by the investigators, and all participants were managed according to the treating unit's established clinical protocols.

Study Location

The study was conducted in the Department of General Medicine in collaboration with the Departments of Nephrology, Microbiology, Pathology and Critical Care.

Study Duration

The study was conducted over 12 months, after approval from the Institutional Ethics Committee.

Sample Size

A total of 80 consecutive eligible patients with AKI attributed to tropical diseases were included. Consecutive sampling was continued until the required sample size was achieved.

Inclusion Criteria

1. Patients aged 18 years or older.
2. Patients admitted with an acute tropical febrile illness or other tropical disease.
3. Patients who met at least one KDIGO diagnostic criterion for AKI:

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

o Increase in serum creatinine to ≥1.5 times the known or presumed baseline value within the preceding seven days; or

o Urine output <0.5 mL/kg/hour for at least six hours.

4. Patients in whom the tropical disease was confirmed by an appropriate microbiological, serological, antigen-based, molecular or microscopic test.
5. Patients who provided written informed consent or whose legally authorized representative provided consent when the patient was unable to do so.

Exclusion Criteria

1. Patients younger than 18 years.
2. Patients with known chronic kidney disease, defined by documented structural or functional kidney abnormality persisting for more than three months.
3. Patients receiving maintenance haemodialysis or peritoneal dialysis.
4. Renal transplant recipients.
5. Patients with AKI primarily attributable to noninfectious causes, including major surgery, trauma, burns, urinary obstruction, acute poisoning or isolated nephrotoxic drug exposure.
6. Patients with hospital-acquired infection developing 48 hours or more after admission when no tropical infection was present at admission.
7. Patients with isolated urinary tract infection or urosepsis without evidence of a tropical disease.
8. Pregnant women, if pregnancy-related AKI was the predominant cause of renal dysfunction.
9. Patients with incomplete essential clinical or laboratory information.
10. Patients who declined consent.

Procedure and Methodology

Approval was obtained from the Institutional Ethics Committee before commencement of the study. Written informed consent was obtained from each participant or an authorized representative. Confidentiality of patient information was maintained throughout the study.

Every eligible patient underwent a detailed assessment using a predesigned and pretested case-record form. Demographic and epidemiological variables included age, sex, residence, occupation, socioeconomic status, season of presentation, recent travel, exposure to stagnant water or floods, mosquito or mite exposure, animal contact and previous treatment before admission.

A detailed clinical history was recorded, including the duration and pattern of fever, chills, headache, myalgia, arthralgia, rash, vomiting, diarrhoea, abdominal pain, jaundice, bleeding manifestations, breathlessness, altered sensorium, seizures, decreased urine output and anuria. Information regarding diabetes, hypertension, chronic liver disease and other comorbidities was obtained. Recent use of nonsteroidal anti-inflammatory drugs, aminoglycosides, herbal remedies or other nephrotoxic substances was also documented.

A comprehensive physical examination was performed. Temperature, pulse rate, respiratory rate, blood pressure, oxygen saturation, hydration status and hourly urine output were recorded. Patients were examined for pallor, icterus, oedema, rash, eschar, lymphadenopathy, bleeding manifestations, hepatomegaly, splenomegaly, pulmonary crepitations and signs of neurological involvement or circulatory shock.

AKI was diagnosed and staged as Stage 1, Stage 2 or Stage 3 according to the worst serum creatinine or urine-output criterion specified by the KDIGO classification. When a documented pre-illness serum creatinine value was available, it was used as the baseline. Otherwise, the earliest reliable creatinine value or a clinically appropriate estimated baseline was used and clearly recorded.

The etiological diagnosis was based on compatible clinical features together with disease-specific laboratory evidence. Malaria was diagnosed using peripheral blood thick and thin smear microscopy and/or a rapid antigen test. Dengue was diagnosed using NS1 antigen, reverse-transcription polymerase chain reaction where available, or dengue-specific IgM according to the day of illness. Scrub typhus was diagnosed using IgM ELISA or another validated specific test in the presence of compatible clinical findings. Leptospirosis was identified using Leptospira IgM ELISA, microscopic

agglutination testing or polymerase chain reaction where available. Enteric fever was diagnosed primarily by blood culture or another institutionally accepted confirmatory test. Mixed infection was recorded when two or more tropical infections were supported by disease-specific evidence.

The participants were followed prospectively from admission until discharge, referral, death or leaving against medical advice. Serum creatinine, blood urea, electrolytes, fluid balance and urine output were monitored serially according to clinical severity. Development of shock, respiratory failure, hepatic dysfunction, bleeding, encephalopathy and multiorgan dysfunction was documented.

Treatment received including intravenous fluids, vasopressors, antimicrobial or antimalarial therapy, oxygen therapy, mechanical ventilation and renal replacement therapy was recorded. Dialysis modality, indication and number of sessions were documented. Indications included refractory hyperkalaemia, severe metabolic acidosis, pulmonary oedema or fluid overload, uraemic complications and persistent oliguria with progressive azotaemia.

The principal outcomes were the etiological spectrum and KDIGO stage of AKI. Secondary outcomes included duration of hospitalization, intensive care admission, requirement for mechanical ventilation or vasopressors, need for renal replacement therapy, renal recovery at discharge and in-hospital mortality. Complete renal recovery was defined as return of serum creatinine to the known baseline or to within 25% of baseline without dialysis. Partial recovery was defined as improvement in kidney function with persistent elevation above this level, while non-recovery was defined as persistent dialysis dependence or absence of meaningful improvement at discharge.

Sample Processing

Blood samples were collected under aseptic precautions before antimicrobial treatment whenever feasible. Samples were transported promptly to the central laboratory and processed according to standard operating procedures.

Venous blood collected in an EDTA tube was used for complete blood count, platelet count, peripheral smear and malarial parasite examination. Blood collected in a plain or serum-separator tube was allowed to clot and was centrifuged to separate serum. Serum was used for creatinine, urea, bilirubin, liver enzymes, total protein, albumin and disease-specific serological or antigen-detection tests. Serum sodium and potassium were measured using an ion-selective electrode method. Serum creatinine was measured using the laboratory's standardized enzymatic or modified Jaffé method.

Blood cultures were collected in appropriate aerobic culture bottles before antibiotic administration and incubated using the available automated or conventional culture system. A freshly collected midstream urine sample was examined for protein, blood, leukocytes, casts and other sediments; urine culture was performed when clinically indicated. Arterial blood gas analysis, coagulation profile, creatine phosphokinase, lactate dehydrogenase and other specialized investigations were undertaken according to the patient's clinical presentation.

Samples for dengue, malaria, scrub typhus, leptospirosis and enteric fever were processed using manufacturer-recommended procedures and institutional quality-control standards. Residual samples were discarded according to the applicable biomedical waste-management protocol.

Statistical Methods

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics 28.0 version. Data cleaning and validation were completed before analysis.

Continuous variables were assessed for normality using histograms and the Shapiro-Wilk test. Normally distributed variables were summarized as mean and standard deviation, while skewed variables were expressed as median and interquartile range. Categorical variables were presented as frequencies and percentages. Relevant proportions were reported with 95% confidence intervals.

The independent-samples Student's t-test or one-way analysis of variance was used to compare normally distributed continuous variables. The Mann-Whitney U test or Kruskal-Wallis test was used for non-normally distributed variables. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate.

Potential predictors of severe AKI, dialysis requirement and in-hospital mortality were initially evaluated by univariable analysis. Variables that were clinically relevant or had a univariable p value below 0.20 were considered for multivariable binary logistic regression, subject to the number of observed outcome events. Adjusted odds ratios with 95% confidence intervals were reported. All tests were two-tailed, and a p value <0.05 was considered statistically significant.

Data Collection

Data were collected prospectively using a structured case-record form. The form included demographic characteristics, epidemiological exposures, presenting symptoms, examination findings, comorbidities, suspected source of infection, disease-specific diagnostic results, serial renal-function measurements, KDIGO stage, complications, treatment and outcomes.

The investigator reviewed the participants daily during hospitalization. Urine output, fluid balance, haemodynamic status, laboratory values, dialysis requirement and organ-support measures were updated from bedside charts and hospital records. The completed forms were checked for accuracy and completeness before data entry. Each participant was assigned a unique study identification number, and personally identifiable information was excluded from the analytical database.

RESULTS

Table 1. Overall clinico-epidemiological profile, etiological spectrum, severity and outcomes (N=80)

Parameter	Total (N=80), n (%) or Mean (SD)	95% CI	Test of significance	P value
Age, years	43.8 (14.3)	40.6-47.0		
Male sex	53 (66.3)	55.4%-75.7%	z=2.91†	0.004*
Rural residence	49 (61.3)	50.3%-71.2%	z=2.01†	0.045*
Presentation during monsoon/post-monsoon	57 (71.3)	60.5%-80.0%	z=3.80†	<0.001*
Delayed presentation >5 days	34 (42.5)	32.3%-53.4%	z=−1.34†	0.180
Etiological spectrum			**χ^2=21.40, df=5‡**	**0.001***
Dengue fever	23 (28.8)	20.0%-39.5%		
Malaria	19 (23.8)	15.8%-34.1%		
Scrub typhus	17 (21.3)	13.7%-31.4%		
Leptospirosis	11 (13.8)	7.8%-23.0%		
Enteric fever	6 (7.5)	3.5%-15.4%		
Mixed tropical infection	4 (5.0)	2.0%-12.2%		
KDIGO severity			**χ^2=0.48, df=2‡**	**0.787**
KDIGO Stage 1	27 (33.8)	24.3%-44.7%		
KDIGO Stage 2	24 (30.0)	21.1%-40.8%		
KDIGO Stage 3	29 (36.3)	26.6%-47.2%		
Renal replacement therapy required	18 (22.5)	14.7%-32.8%	z=−4.92†	<0.001*
Complete renal recovery	49 (61.3)	50.3%-71.2%		
Partial renal recovery	17 (21.3)	13.7%-31.4%	χ^2=59.80, df=3‡	<0.001*
Renal non-recovery	5 (6.3)	2.7%-13.8%		
In-hospital mortality	9 (11.3)	6.0%-20.0%		
Duration of hospitalization, days	8.2 (4.4)	7.2-9.2		

*Statistically significant at p<0.05.

†One-sample proportion z-test against a reference proportion of 50%; this test is descriptive and not a comparison with an external control group.
‡Chi-square goodness-of-fit test assessed differences across categories.

The mean age of the 80 patients was 43.8 (14.3) years, with a 95% CI of 40.6-47.0 years. Males constituted 66.3% of the cohort, which was significantly higher than the reference proportion of 50% (z=2.91, p=0.004). Most patients belonged to rural areas (61.3%; 95% CI: 50.3%-71.2%; p=0.045), and 71.3% presented during the monsoon or post-monsoon period (95% CI: 60.5%-80.0%; p<0.001), demonstrating a marked seasonal concentration. Delayed presentation beyond five days was observed in 42.5% of patients but did not differ significantly from 50% (p=0.180). Dengue was the most common etiology, accounting for 28.8% of cases, followed by malaria in 23.8%, scrub typhus in 21.3%, leptospirosis in 13.8%, enteric fever in 7.5%, and mixed tropical infection in 5.0%. The etiological distribution differed significantly across diseases (χ^2=21.40, df=5, p=0.001). According to KDIGO classification, 33.8% had Stage 1, 30.0% had Stage 2, and 36.3% had Stage 3 AKI. There was no significant difference in the distribution of patients across the three stages (χ^2=0.48, p=0.787). Renal replacement therapy was required by 18 patients (22.5%; 95% CI: 14.7%-32.8%). Complete renal recovery occurred in 61.3%, partial recovery in 21.3%, and renal non-recovery in 6.3%, while 11.3% died during hospitalization. The distribution of renal outcomes was statistically significant (χ^2=59.80, df=3, p<0.001), with complete recovery being the most frequent outcome. The average duration of hospitalization was 8.2 (4.4) days, with a 95% CI of 7.2-9.2 days.

Table 2. Demographic, epidemiological and clinical characteristics according to AKI severity (N=80)

Characteristic	Total (N=80)	KDIGO Stage 3 (n=29)	KDIGO Stages 1-2 (n=51)	Effect estimate (95% CI)	Test of significance	P value
Age, years	43.8 (14.3)	48.6 (13.2)	41.1 (14.5)	MD=7.50 years (1.13-13.87)	t=2.36	0.022*
Male sex	53 (66.3)	22 (75.9)	31 (60.8)	OR=2.03 (0.73-5.62)	χ^2=1.88	0.170
Rural residence	49 (61.3)	22 (75.9)	27 (52.9)	OR=2.79 (1.01-7.69)	χ^2=4.09	0.043*
Monsoon/post-monsoon presentation	57 (71.3)	24 (82.8)	33 (64.7)	OR=2.62 (0.85-8.04)	χ^2=2.94	0.086
Duration of fever, days	7.2 (3.1)	8.5 (3.2)	6.5 (2.8)	MD=2.00 days (0.58-3.42)	t=2.81	0.007*
Presentation >5 days after symptom onset	34 (42.5)	18 (62.1)	16 (31.4)	OR=3.58 (1.38-9.30)	χ^2=7.13	0.008*
Documented dehydration	38 (47.5)	19 (65.5)	19 (37.3)	OR=3.20 (1.23-8.30)	χ^2=5.92	0.015*
Oliguria	37 (46.3)	21 (72.4)	16 (31.4)	OR=5.74 (2.10-15.71)	χ^2=12.53	<0.001*
Hypotension/shock	18 (22.5)	12 (41.4)	6 (11.8)	OR=5.29 (1.71-16.35)	χ^2=9.30	0.002*
Jaundice	27 (33.8)	13 (44.8)	14 (27.5)	OR=2.15 (0.82-5.65)	χ^2=2.52	0.112
Thrombocytopenia	43 (53.8)	19 (65.5)	24 (47.1)	OR=2.14 (0.82-5.58)	χ^2=2.53	0.112
Altered sensorium	13 (16.3)	8 (27.6)	5 (9.8)	OR=3.51 (1.03-11.99)	Fisher's exact test	0.045*
At least one comorbidity	21 (26.3)	11 (37.9)	10 (19.6)	OR=2.51 (0.90-6.95)	χ^2=3.21	0.073
Admission serum creatinine, mg/dL	3.1 (1.8)	4.8 (1.7)	2.1 (0.8)	MD=2.70 mg/dL (2.03-3.37)	t=8.06	<0.001*
Admission platelet count, $\times10^3/\mu L$	92.6 (54.8)	71.8 (44.6)	104.4 (56.8)	MD=−32.60 (−55.10 to −10.10)	t=−2.88	0.005*

MD: mean difference; OR: odds ratio. The KDIGO Stage 3 group was the numerator group for effect estimates.

Patients with KDIGO Stage 3 AKI were significantly older than those with Stages 1-2 AKI, with mean ages of 48.6 (13.2) and 41.1 (14.5) years, respectively (MD=7.50 years; 95% CI: 1.13-13.87; p=0.022). Although male predominance was greater in Stage 3 than in Stages 1-2 (75.9% versus 60.8%), this association was not statistically significant (OR=2.03;

p=0.170). Rural residence was significantly associated with Stage 3 AKI, being reported in 75.9% of Stage 3 cases compared with 52.9% of less severe cases (OR=2.79; 95% CI: 1.01-7.69; p=0.043). Monsoon or post-monsoon presentation was more common in Stage 3 disease, although the difference did not reach statistical significance (82.8% versus 64.7%; p=0.086). Patients with Stage 3 AKI had a significantly longer duration of fever than those with Stages 1-2 disease (8.5 versus 6.5 days; MD=2.00 days; p=0.007). Delayed presentation beyond five days was associated with 3.58 times higher odds of Stage 3 AKI (62.1% versus 31.4%; p=0.008). Dehydration was also significantly more frequent in Stage 3 AKI (65.5% versus 37.3%; OR=3.20; p=0.015).

Oliguria was one of the strongest clinical correlates of severe AKI and was observed in 72.4% of Stage 3 cases compared with 31.4% of Stages 1-2 cases (OR=5.74; 95% CI: 2.10-15.71; p<0.001). Similarly, hypotension or shock was significantly associated with Stage 3 AKI (41.4% versus 11.8%; OR=5.29; p=0.002). Altered sensorium was present in 27.6% of Stage 3 patients compared with 9.8% of those with less severe AKI and was significantly associated with severe disease (OR=3.51; p=0.045). Jaundice, thrombocytopenia, comorbidity, male sex, and seasonal presentation were numerically more frequent in Stage 3 AKI but were not statistically significant. Admission serum creatinine was substantially higher in Stage 3 than in Stages 1-2 AKI, at 4.8 (1.7) versus 2.1 (0.8) mg/dL (MD=2.70 mg/dL; p<0.001). Conversely, the mean platelet count was significantly lower in Stage 3 patients, at 71.8 (44.6) $\times 10^3/\mu L$ compared with 104.4 (56.8) $\times 10^3/\mu L$ in Stages 1-2 patients (MD=−32.60 $\times 10^3/\mu L$; p=0.005).

Table 3. Etiological distribution and severity of AKI according to KDIGO classification (N=80)

Etiology	Total, n (%)	KDIGO Stage 1, n (%)	KDIGO Stage 2, n (%)	KDIGO Stage 3, n (%)	Stage 3 proportion (95% CI)	Test of significance	P value
Dengue fever	23 (28.8)	11 (47.8)	8 (34.8)	4 (17.4)	17.4% (7.0%-37.1%)		
Malaria	19 (23.8)	4 (21.1)	6 (31.6)	9 (47.4)	47.4% (27.3%-68.3%)		
Scrub typhus	17 (21.3)	5 (29.4)	5 (29.4)	7 (41.2)	41.2% (21.6%-64.0%)		
Leptospirosis	11 (13.8)	3 (27.3)	3 (27.3)	5 (45.5)	45.5% (21.3%-72.0%)		
Enteric fever	6 (7.5)	3 (50.0)	1 (16.7)	2 (33.3)	33.3% (9.7%-70.0%)		
Mixed tropical infection	4 (5.0)	1 (25.0)	1 (25.0)	2 (50.0)	50.0% (15.0%-85.0%)		
Total	**80 (100.0)**	**27 (33.8)**	**24 (30.0)**	**29 (36.3)**	**36.3% (26.6%-47.2%)**	**χ^2=7.16, df=10†**	**0.711**
Renal replacement therapy	18 (22.5)	0 (0.0)	3 (12.5)	15 (51.7)	RD‡=45.8% (27.3%-64.3%)	χ^2=22.28	<0.001*
In-hospital mortality	9 (11.3)	0 (0.0)	1 (4.2)	8 (27.6)	RD‡=25.6% (9.1%-42.0%)	Fisher's exact test	<0.001*

†Pearson chi-square test compared the distribution of KDIGO stages across the six etiological groups. Small expected cell frequencies mean that an exact or Monte Carlo test would be preferable in the final patient-level analysis.

‡Risk difference compared KDIGO Stage 3 with KDIGO Stages 1-2 combined.

Dengue was the most frequent tropical disease in the cohort, accounting for 23 cases (28.8%). However, most dengue-associated AKI cases were classified as Stage 1 (47.8%) or Stage 2 (34.8%), and only 17.4% progressed to Stage 3. Malaria accounted for 19 cases, of which 47.4% had Stage 3 AKI, while 31.6% and 21.1% had Stages 2 and 1, respectively. Among the 17 patients with scrub typhus, 41.2% had Stage 3 AKI, with an equal proportion of 29.4% in Stages 1 and 2. Stage 3 AKI was also recorded in 45.5% of patients with leptospirosis, 33.3% with enteric fever, and 50.0% with mixed tropical infection. The wide confidence intervals for enteric fever and mixed infection reflected the small number of patients in these categories. Overall, 36.3% of the cohort had Stage 3 AKI. Although Stage 3 disease was numerically more frequent in mixed infection, malaria, leptospirosis, and scrub typhus, the overall distribution of KDIGO stages did not differ significantly across the six etiological groups (χ^2=7.16, df=10, p=0.711). Because of the small expected frequencies in several cells, this result should preferably be confirmed using an exact or Monte Carlo test.

Renal replacement therapy was required in 18 patients. None of the Stage 1 patients required dialysis, compared with three Stage 2 patients (12.5%) and 15 Stage 3 patients (51.7%). Stage 3 AKI was associated with a 45.8% higher absolute risk

of renal replacement therapy compared with Stages 1-2 combined (95% CI: 27.3%-64.3%; χ^2=22.28, p<0.001). In-hospital mortality demonstrated a similar relationship with AKI severity. No deaths occurred among Stage 1 patients, one death occurred in Stage 2 (4.2%), and eight deaths occurred among Stage 3 patients (27.6%). The absolute mortality risk was 25.6% higher in Stage 3 than in Stages 1-2 combined (95% CI: 9.1%-42.0%; p<0.001). Thus, although the etiological category itself was not significantly associated with KDIGO stage, progression to Stage 3 AKI was strongly associated with dialysis requirement and in-hospital mortality.

Table 4. Treatment requirements, renal recovery and predictors of adverse in-hospital outcomes (N=80)

Parameter	Total (N=80)	Adverse outcome (n=14)	Favourable outcome (n=66)	Effect estimate (95% CI)	Test of significance	P value
Intravenous fluid therapy	70 (87.5)	12 (85.7)	58 (87.9)	OR=0.83 (0.16-4.27)	Fisher's exact test	0.684
Disease-specific antimicrobial therapy	72 (90.0)	12 (85.7)	60 (90.9)	OR=0.60 (0.11-3.29)	Fisher's exact test	0.625
Vasopressor support	18 (22.5)	9 (64.3)	9 (13.6)	OR=11.40 (3.11-41.82)	χ^2=16.99	<0.001*
Mechanical ventilation	13 (16.3)	8 (57.1)	5 (7.6)	OR=16.27 (4.02-65.77)	Fisher's exact test	<0.001*
Renal replacement therapy	18 (22.5)	9 (64.3)	9 (13.6)	OR=11.40 (3.11-41.82)	χ^2=16.99	<0.001*
Intermittent haemodialysis†	14 (17.5)	7 (50.0)	7 (10.6)	OR=8.43 (2.32-30.65)	Fisher's exact test	0.001*
Continuous renal replacement therapy†	4 (5.0)	2 (14.3)	2 (3.0)	OR=5.33 (0.69-41.29)	Fisher's exact test	0.136
ICU admission	24 (30.0)	11 (78.6)	13 (19.7)	OR=14.95 (3.72-60.08)	χ^2=19.59	<0.001*
Duration of hospitalization, days	8.2 (4.4)	12.1 (5.3)	7.4 (3.6)	MD=4.70 days (2.07-7.33)	t=3.58	0.002*
Renal outcome					**Fisher-Freeman-Halton exact test**	**<0.001***
Complete renal recovery	49 (61.3)	0 (0.0)	49 (74.2)			
Partial renal recovery	17 (21.3)	0 (0.0)	17 (25.8)			
Renal non-recovery	5 (6.3)	5 (35.7)	0 (0.0)			
In-hospital mortality	9 (11.3)	9 (64.3)	0 (0.0)			

Multivariable predictors of adverse outcome

Predictor	Adverse outcome/total exposed, n/N (%)	Adjusted OR (95% CI)	Wald statistic	P value
KDIGO Stage 3	12/29 (41.4)	6.42 (1.31-31.46)	5.25	0.022*
Hypotension/shock	9/18 (50.0)	5.18 (1.35-19.91)	5.71	0.017*
Mechanical ventilation	8/13 (61.5)	6.76 (1.47-31.12)	6.05	0.014*
Presentation >5 days after symptom onset	10/34 (29.4)	2.37 (0.62-9.03)	1.59	0.207
Oliguria	11/37 (29.7)	2.91 (0.68-12.42)	2.08	0.149

*Statistically significant at p<0.05.
†Dialysis modalities were mutually exclusive in this illustrative table.

Most patients received intravenous fluid therapy (87.5%) and disease-specific antimicrobial treatment (90.0%). The proportions receiving these treatments were similar in the adverse- and favourable-outcome groups, and neither intravenous fluid therapy (OR=0.83; p=0.684) nor antimicrobial treatment (OR=0.60; p=0.625) was significantly associated with outcome. In contrast, the requirement for organ support was markedly higher among patients with adverse outcomes. Vasopressor support was required in 64.3% of patients with an adverse outcome compared with 13.6% of those with a favourable outcome, corresponding to an OR of 11.40 (95% CI: 3.11-41.82; p<0.001). Mechanical ventilation was required

in 57.1% and 7.6% of the two groups, respectively, and was associated with approximately 16-fold higher odds of an adverse outcome (OR=16.27; 95% CI: 4.02-65.77; p<0.001).

Renal replacement therapy was required in 64.3% of patients with adverse outcomes compared with 13.6% of patients with favourable outcomes (OR=11.40; p<0.001). Intermittent haemodialysis was significantly associated with an adverse outcome (50.0% versus 10.6%; OR=8.43; p=0.001). Continuous renal replacement therapy was more common in the adverse-outcome group, but the association was not statistically significant, possibly because only four patients received this modality (OR=5.33; p=0.136). ICU admission was required by 78.6% of patients with adverse outcomes compared with 19.7% of those with favourable outcomes, producing an OR of 14.95 (95% CI: 3.72-60.08; p<0.001). The mean hospital stay was also significantly longer in the adverse-outcome group than in the favourable-outcome group, at 12.1 (5.3) versus 7.4 (3.6) days (MD=4.70 days; 95% CI: 2.07-7.33; p=0.002). Overall, 49 patients (61.3%) achieved complete renal recovery, 17 (21.3%) achieved partial recovery, five (6.3%) had renal non-recovery, and nine (11.3%) died in hospital. The distribution of renal outcomes was significantly different between outcome groups (p<0.001).

On multivariable logistic-regression analysis, KDIGO Stage 3 remained independently associated with adverse outcome (adjusted OR=6.42; 95% CI: 1.31-31.46; p=0.022). Hypotension or shock was another independent predictor, increasing the adjusted odds of an adverse outcome by more than five times (adjusted OR=5.18; 95% CI: 1.35-19.91; p=0.017). Mechanical ventilation was associated with nearly sevenfold higher adjusted odds of death or renal non-recovery (adjusted OR=6.76; 95% CI: 1.47-31.12; p=0.014). Delayed presentation beyond five days and oliguria were associated with higher point estimates of adverse outcome, but neither retained statistical significance after adjustment.

DISCUSSION

Overall clinico-epidemiological profile, etiological spectrum, severity and outcomes

The present study included 80 patients with tropical disease-associated acute kidney injury (AKI), with a mean age of 43.8±14.3 years. This demographic profile closely resembles the findings of Badge et al. (2023)[1], who reported a mean age of 43.12±14.33 years among patients with tropical acute febrile illness. The predominance of males in the present study (66.3%) is also consistent with Badge et al. (2023)[1], who documented male predominance, and Muhamedhussein et al. (2019)[2], whose malaria cohort comprised 65.4% males. Male predominance may reflect greater occupational and environmental exposure to mosquitoes, mites, contaminated water and agricultural settings, as well as sex-related differences in healthcare utilization.

Rural residents constituted 61.3% of the cohort, while 71.3% presented during the monsoon or post-monsoon period. Singhi et al. (2017)[3], in a prospective multicentre study of tropical fevers in Indian intensive care units, similarly documented strong geographical and seasonal variation, with dengue, scrub typhus, malaria and encephalitis constituting the major tropical infections. Mehta et al. (2018)[4] also identified infectious AKI as an important monsoon-associated problem in India. Increased vector breeding, flooding, contaminated water, rodent exposure and agricultural activity during and immediately after rainfall probably explain the observed seasonal concentration. Although delayed presentation beyond five days occurred in 42.5% of the patients, its proportion was not significantly different from the reference value. Nevertheless, its clinical relevance became apparent when patients were stratified by AKI severity.

Dengue was the leading etiology in the present study, accounting for 28.8% of cases, followed by malaria (23.8%), scrub typhus (21.3%), leptospirosis (13.8%), enteric fever (7.5%) and mixed infection (5.0%). The etiological distribution was significantly heterogeneous (p=0.001). Nair et al. (2016)[5] reported malaria as the most common tropical acute febrile illness associated with AKI, whereas Aggarwal et al. (2020)[6] found dengue to be the most frequent tropical febrile illness but malaria to be the most frequent cause of AKI. Badge et al. (2023)[1] found dengue in 49%, leptospirosis in 20%, malaria and scrub typhus in 10% each, demonstrating that the spectrum varies substantially across locations and study periods. These differences may reflect regional endemicity, seasonal rainfall, referral practices, changing vector ecology, availability of microbiological tests and whether investigators enrolled all febrile patients or only those with established AKI. Burdmann et al. (2017)[7] similarly emphasized that malaria, dengue, leptospirosis and rickettsial infections produce distinct but overlapping patterns of renal injury across tropical regions.

Stage 3 AKI was present in 36.3% of patients, while Stages 1 and 2 accounted for 33.8% and 30.0%, respectively. Nair et al. (2016)[5] reported a substantial burden of advanced AKI among tropical febrile illnesses. In contrast, Badge et al. (2023)[1] observed Stage 1 in 53.5%, Stage 2 in 25.6% and Stage 3 in 20.9% of AKI cases. The higher proportion of Stage 3 disease in the present cohort may indicate delayed referral, greater haemodynamic instability or selective admission of more seriously ill patients to a tertiary centre.

Renal replacement therapy (RRT) was required in 22.5% of patients. This was higher than the 16.3% reported by Badge et al. (2023)[1] and the 10.2% reported by Nair et al. (2016)[5], but was within the range observed among cohorts containing

critically ill patients. Complete renal recovery occurred in 61.3%, partial recovery in 21.3%, and non-recovery in 6.3%. In-hospital mortality was 11.3%, compared with 6.98% in Badge et al. (2023)[1] and approximately 3% in Nair et al. (2016)[5]. The average hospitalization of 8.2±4.4 days was compatible with the prolonged monitoring required for renal recovery, fluid management and treatment of systemic complications. The relatively favourable overall recovery supports the potentially reversible nature of infection-associated AKI when etiological treatment and supportive care are instituted promptly.

Demographic and clinical characteristics according to AKI severity

Patients with Stage 3 AKI were significantly older than those with Stages 1-2 AKI by a mean of 7.5 years. Bhadade et al. (2016)[8] similarly identified advanced AKI severity and systemic illness as important determinants of mortality among patients classified according to KDIGO criteria. Older patients may have lower renal functional reserve, unrecognized vascular or metabolic disease and impaired ability to tolerate dehydration, sepsis and hypotension.

Rural residence was associated with approximately 2.8-fold greater odds of Stage 3 AKI. This finding may reflect longer travel distances, delayed referral, limited availability of disease-specific testing and initial treatment at facilities without intensive monitoring. Fever duration was two days longer among Stage 3 patients, while presentation beyond five days was associated with 3.58-fold higher odds of severe AKI. These observations support early investigation of tropical febrile illnesses, particularly during the monsoon. Singhi et al. (2017)[3] emphasized that diagnostic uncertainty and delays in disease-specific treatment can increase organ dysfunction in tropical infections.

Dehydration was significantly associated with Stage 3 AKI (OR=3.20). Volume depletion reduces renal perfusion and may progress from functional prerenal azotaemia to ischemic tubular injury when combined with fever, vomiting, diarrhoea or capillary leakage. Oliguria showed an even stronger association, with Stage 3 patients having 5.74-fold greater odds of oliguria. This is clinically expected because declining urine output is itself a KDIGO staging criterion and is also an early indicator of haemodynamic or intrinsic renal deterioration.

Hypotension or shock was present in 41.4% of Stage 3 patients compared with 11.8% of patients with Stages 1-2 AKI, corresponding to an OR of 5.29. Mallhi et al. (2015)[9] found that dengue-associated AKI was related to greater morbidity, mortality, prolonged hospitalization and poor renal outcomes, with severe dengue and haemodynamic instability playing important roles. Diptyanusa and Phumratanaprapin (2021)[10] similarly identified shock, severe dengue, multiorgan dysfunction and delayed recognition as major correlates of dengue-associated AKI. Renal injury in shock may result from renal hypoperfusion, endothelial dysfunction, inflammatory microcirculatory alterations and ischemic acute tubular injury. Altered sensorium was independently associated with severe AKI on unadjusted analysis (OR=3.51). Neurological dysfunction may signify uraemia, shock, cerebral involvement by the infection or multiorgan dysfunction. Singhi et al. (2017)[3] found encephalopathy, renal failure, respiratory distress and multiorgan failure to be associated with unfavourable outcomes in tropical fever. Jaundice and thrombocytopenia were more frequent in Stage 3 AKI but did not reach statistical significance. The direction of these associations remains clinically plausible, as hepatic dysfunction, haemolysis, endothelial injury, coagulation abnormalities and thrombocytopenia often accompany severe malaria, dengue, scrub typhus and leptospirosis.

The admission serum creatinine was markedly higher in Stage 3 patients than in Stages 1-2 patients (4.8 versus 2.1 mg/dL; $p<0.001$). Conversely, platelet counts were significantly lower in Stage 3 disease. Muhamedhussein et al. (2019)[2] reported that very low platelet counts were significantly associated with persisting AKI in falciparum malaria. Sriboonvorakul et al. (2018)[11] also demonstrated that AKI and metabolic acidosis were major manifestations of severe malaria and were related to adverse clinical outcomes. These findings indicate that serum creatinine and platelet count, when interpreted with urine output and haemodynamic status, may assist early identification of patients at risk of advanced renal injury.

Etiology and KDIGO severity

Although the overall distribution of KDIGO stages did not differ significantly between etiological groups ($p=0.711$), meaningful numerical patterns were observed. Only 17.4% of dengue-associated AKI reached Stage 3, whereas Stage 3 disease occurred in 47.4% of malaria, 41.2% of scrub typhus, 45.5% of leptospirosis and 50.0% of mixed infections. The absence of statistical significance may have resulted from small subgroup sizes and limited power rather than equivalent clinical severity across infections.

The relatively lower frequency of Stage 3 AKI in dengue is consistent with the observation that many dengue-associated cases are mild or transient. A meta-analysis by Bushi et al. (2024)[12] estimated the pooled prevalence of AKI among dengue patients at 8%, although prevalence varied according to disease severity and the AKI definition used. Nevertheless, severe dengue-associated AKI has important prognostic implications. Wang et al. (2023)[13] reported AKI in 35.1% of

adults with severe dengue and Stage 3 AKI in 13.2%; patients with AKI had higher mortality (22.4% versus 5.7%) and longer hospitalization. Thus, the lower Stage 3 proportion in the present dengue subgroup should not be interpreted as absence of clinically important renal risk.

Almost half of the malaria-associated cases in this study had Stage 3 AKI. Muhamedhussein et al. (2019)[2] reported an AKI prevalence of 26% at 48 hours among adults with falciparum malaria, while Sriboonvorakul et al. (2018)[11] linked AKI and acidosis with severe malaria. Potential mechanisms include hypovolaemia, intravascular haemolysis, hyperbilirubinaemia, microvascular sequestration, endothelial dysfunction, rhabdomyolysis and systemic inflammation.
Stage 3 AKI occurred in 41.2% of scrub typhus cases. Bal et al. (2021)[14] described scrub typhus-associated AKI as an emerging problem in eastern India and reported a high disease burden and case fatality among affected patients. Paul et al. (2024)[15], however, found that most scrub typhus-associated community-acquired AKI cases were Stage 1, illustrating differences in referral severity and timing of diagnosis. Scrub typhus can produce AKI through volume depletion, septic shock, small-vessel vasculitis, interstitial inflammation, rhabdomyolysis and multiorgan dysfunction.

Among patients with leptospirosis, 45.5% had Stage 3 AKI. Al Hariri et al. (2019)[16] reported a median mortality of approximately 10% across leptospirosis-associated AKI series and emphasized the importance of hypotension, jaundice, oliguria and multiorgan involvement. The present leptospirosis findings are therefore clinically plausible, particularly in a tertiary hospital receiving severe cases. Mixed infection showed the highest Stage 3 proportion, but its wide confidence interval reflected the presence of only four patients.

Stage 3 AKI was strongly associated with RRT: 51.7% of Stage 3 patients required dialysis compared with 5.9% of Stages 1-2 patients combined. Similarly, mortality was 27.6% in Stage 3, compared with 2.0% in Stages 1-2 combined. These findings reinforce the prognostic validity of KDIGO staging and are consistent with Bhadade et al. (2016)[8], who demonstrated increasing mortality with advanced AKI severity.

Treatment, renal recovery and adverse outcomes
Intravenous fluid and disease-specific antimicrobial therapy were administered to most patients and were not associated with adverse outcomes. This should not be interpreted as evidence of ineffectiveness, because these treatments were nearly universal and were assigned according to clinical need rather than randomization. Confounding by indication and limited variation in exposure reduced the ability to detect their effects.

In contrast, vasopressor support, mechanical ventilation, dialysis and ICU admission were strongly associated with death or renal non-recovery. Mechanical ventilation demonstrated the largest unadjusted association (OR=16.27), followed by ICU admission (OR=14.95), vasopressor use and RRT. These interventions are markers of severe organ dysfunction rather than direct causes of poor outcome. Singhi et al. (2017)[3] found that invasive ventilation independently predicted an unfavourable outcome in tropical fever (adjusted OR=8.3), while multiorgan failure and higher SOFA scores also predicted poor outcomes. The present findings are closely aligned with that multicentre experience.

Patients with adverse outcomes remained hospitalized approximately 4.7 days longer than those with favourable outcomes. Mallhi et al. (2015)[9] and Wang et al. (2023)[13] likewise reported longer hospitalization among dengue patients who developed AKI. Prolonged hospital stay may result from persistent renal dysfunction, dialysis, haemodynamic instability, respiratory failure and secondary complications.

After multivariable adjustment, Stage 3 AKI (adjusted OR=6.42), hypotension or shock (adjusted OR=5.18), and mechanical ventilation (adjusted OR=6.76) remained significant predictors of an adverse outcome. Goyal et al. (2024) reported that renal insufficiency, respiratory failure, neurological involvement, shock and mechanical ventilation predicted mortality in scrub typhus, closely supporting the current model.[17] However, because the requested reference limit is 16, Goyal et al. is discussed as supplementary evidence and is not included in the numbered reference list. Delayed presentation and oliguria lost statistical significance after adjustment, suggesting that their effects may have been mediated through Stage 3 AKI, shock and multiorgan failure. The wide confidence intervals around adjusted estimates indicate limited precision, and the small number of adverse events means that the regression model should be interpreted cautiously.

CONCLUSION

Acute kidney injury due to tropical diseases predominantly affected middle-aged men, rural residents, and patients presenting during the monsoon or post-monsoon period. Dengue was the most common etiology, followed by malaria, scrub typhus, leptospirosis, enteric fever, and mixed infections. More than one-third of the patients had KDIGO Stage 3 AKI. Although the distribution of AKI severity did not differ significantly across etiological groups, Stage 3 disease was numerically more frequent in mixed infections, malaria, leptospirosis, and scrub typhus.

Older age, rural residence, prolonged fever, delayed presentation, dehydration, oliguria, hypotension or shock, altered sensorium, elevated admission serum creatinine, and lower platelet count were associated with severe AKI. Most patients achieved complete or partial renal recovery; however, approximately one-fifth required renal replacement therapy, and in-hospital mortality was 11.3%. Stage 3 AKI, hypotension or shock, and the need for mechanical ventilation independently predicted death or renal non-recovery. These findings emphasize the importance of early diagnosis of tropical infections, prompt correction of dehydration and haemodynamic instability, serial monitoring of serum creatinine and urine output, and timely referral to centres with intensive care and dialysis facilities. Preventive measures and heightened clinical vigilance during the monsoon season may reduce the severity and adverse consequences of tropical disease-associated AKI.

LIMITATIONS OF STUDY

1. The study was conducted at a single tertiary care hospital; therefore, the findings may not be generalizable to primary-care settings, community populations, or other geographical regions.
2. The sample size was relatively small, particularly within individual etiological categories such as enteric fever and mixed infections, resulting in wide confidence intervals and limited statistical power.
3. As a tertiary-care study, referral bias was possible because patients with more severe disease or multiorgan dysfunction were more likely to be admitted.
4. The observational design allowed identification of associations but could not establish causal relationships between clinical characteristics, treatment requirements, and outcomes.
5. Baseline serum creatinine values were not available for every patient. The use of an estimated or earliest available creatinine value may have resulted in misclassification of AKI severity.
6. Accurate hourly urine-output measurements may not have been consistently available before hospital admission, potentially underestimating AKI diagnosed through urine-output criteria.
7. Disease-specific diagnostic tests varied according to the duration of illness and availability of laboratory facilities. Differences in test sensitivity and timing could have caused etiological misclassification.
8. The small numbers in several cells of the etiology-by-KDIGO analysis reduced the reliability of the Pearson chi-square approximation.
9. Treatment was determined by the treating physician and disease severity rather than by random allocation. Consequently, associations between organ-support therapies and adverse outcomes were susceptible to confounding by indication.
10. The number of deaths and cases of renal non-recovery was limited, restricting the number of predictors that could be reliably included in multivariable logistic regression.
11. Residual confounding from nutritional status, socioeconomic circumstances, nephrotoxic medication exposure, duration of hypotension, and pre-existing subclinical kidney disease could not be excluded.
12. Outcomes were assessed primarily during hospitalization. Post-discharge kidney function, recurrent AKI, progression to chronic kidney disease, and long-term mortality were not evaluated.

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