



AN OBSERVATIONAL STUDY ON INCIDENCE OF DIFFERENT ETIOLOGIES OF ACUTE KIDNEY INJURY IN A TERTIARY CARE HOSPITAL

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DOI: <https://doi.org/10.59551/IJHMP/25832069/2026.7.3/4.103>

How to cite this article: Islam SKT, Roy D, Mondal A. An Observational Study on Incidence of Different Etiologies of Acute Kidney Injury in a Tertiary Care Hospital. *Indian J. Health Care, Med. Pharm. Pract.* 2026;7(3-4):27-32.

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Received: 24 Aug, 2026, Decision for Acceptance: 01 Oct, 2026

ABSTRACT

Background: Acute kidney injury (AKI) is common across hospital settings and is associated with significant morbidity and mortality. Its etiological spectrum varies widely by region, and short-term outcome depends on the underlying cause and severity of renal and metabolic derangement.

Objective: To determine the common etiologies of AKI, evaluate outcome according to etiology, and identify parameters predicting short-term outcome in a tertiary care setting.

Methods: This observational, descriptive, single-centre study was conducted in the Department of General Medicine, IPGME&R and SSKM Hospital, from May 2023 to April 2024. Inpatients aged 13 years or above with clinical and/or biochemical evidence of AKI were included; patients with diabetes mellitus or chronic kidney disease were excluded. A sample of 100 patients was selected by weekly randomisation and followed for 7 days for symptom evolution, investigation trends, and outcome. Categorical variables were compared using the chi-square test and continuous variables using the t-test or rank-sum test, as appropriate, with SPSS v24.

Results: Of 100 patients, 52% were male, with a mean age of 40.6 years overall. Oliguria was the most common presenting feature (71%). Acute gastroenteritis (28%), snakebite (16%), and sepsis (15%) were the leading etiologies, together accounting for 59% of cases. Renal/intrinsic AKI was the most common type (62%), followed by prerenal (28%) and postrenal (10%) causes. Hemodialysis was required in 51% of patients. At 1 week, 67% had fully recovered, 19% partially recovered, and 14% died; sepsis accounted for 42.9% of deaths. Higher serum urea, creatinine, and potassium levels, and requirement of renal replacement therapy, were each significantly associated with poorer outcome ($p < 0.05$).

Conclusion: Acute gastroenteritis, snakebite, and sepsis were the leading causes of AKI in this cohort. Serum urea, creatinine, and potassium levels, along with the need for renal replacement therapy, were significant predictors of short-term outcome, supporting their use in early risk stratification of AKI patients.

KEYWORDS: Acute Kidney Injury, Etiology, Outcome, Hemodialysis, Renal Replacement Therapy.

Introduction

Acute kidney injury (AKI) denotes an abrupt decline in glomerular filtration rate sufficient to impair the elimination of nitrogenous waste and other uraemic toxins, and has supplanted the older term acute renal failure following the development of standardised consensus definitions and staging criteria[1,2]. AKI is a global public health concern associated with substantial morbidity, mortality, and healthcare cost, and even apparently complete recovery of renal function after an episode of AKI is now recognised to carry an increased long-term risk of chronic kidney disease[2].

The Kidney Disease: Improving Global Outcomes (KDIGO) staging criteria, based on the magnitude of serum creatinine rise and the degree of oliguria, provide a uniform framework for diagnosing and grading AKI across clinical settings[3]. A meta-analysis of the world incidence of AKI estimated that approximately one in five adults admitted to hospital, and a substantially higher proportion of those admitted to intensive care, develop some degree of AKI, with wide regional variation in reported incidence[4].

The epidemiology and aetiological spectrum of AKI differ markedly between developed and developing countries. In high-income settings, AKI is predominantly hospital-acquired, occurring in elderly patients with multiple comorbidities in the context of sepsis, major surgery, or nephrotoxic drug exposure, whereas in tropical, resource-limited settings AKI more often arises in the community, affecting younger and previously healthy individuals, and is frequently caused by a single, identifiable insult such as severe volume depletion from diarrhoeal illness, infection, obstetric complications, or animal envenomation[5,6]. Large multinational surveys coordinated by the International Society of Nephrology have highlighted this disparity and the disproportionate burden of preventable AKI-related deaths in low-resource settings, prompting global initiatives aimed at earlier recognition and prevention[7,8].

Snakebite envenomation remains an important, largely tropical cause of AKI, with a spectrum of renal injury ranging from mild, self-limiting derangement to cortical necrosis requiring long-term dialysis, depending on the species involved and the timeliness of antivenom administration[9]. Similarly, community-acquired AKI related to infective diarrhoeal illness, sepsis, and other tropical infections continues to constitute a substantial proportion of cases in Indian and other South Asian case series, in contrast to the largely iatrogenic, hospital-acquired pattern described in Western cohorts[5,10].

Given this wide variability in aetiological pattern by region and healthcare setting, the present study was undertaken to determine the incidence of different etiologies of AKI among patients admitted to a tertiary care hospital in eastern

India, to evaluate the consequences of AKI according to the underlying cause, and to identify clinical and biochemical parameters that predict short-term outcome in this population.

Materials and Methods

This observational, descriptive study was conducted in the Department of General Medicine, IPGME&R and SSKM Hospital, a single tertiary care centre, over one year from May 2023 to April 2024, after approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants, and the study was conducted in accordance with the Declaration of Helsinki.

All inpatients aged 13 years or above admitted under the Department of General Medicine with clinical and/or biochemical evidence of AKI, as per KDIGO criteria, were eligible. Patients with diabetes mellitus, chronic kidney disease, or age below 13 years were excluded.

Sample size was calculated using the formula $n = Z^2pq/d^2$, based on a reported South Asian AKI prevalence of 7% [4], a 95% confidence interval ($Z=1.96$), and a maximum allowable error of 0.05, yielding a sample size of approximately 100. Simple random sampling was used for patient selection: one day of the week (Wednesday) was randomly selected by lottery, and on each Wednesday over 50 of the 52 study weeks (excluding the first and last), two AKI patients identified from the ward records were randomly selected using a chit method, giving a total of 100 patients.

Enrolled patients were followed for 7 days for the development of symptoms and requirement of renal replacement therapy (RRT). Demographic, social, and clinical data, including duration of oliguria, were recorded on a structured proforma. Baseline investigations included a complete haemogram, urea, creatinine, electrolytes, liver function tests, fasting blood glucose, HbA1c, arterial blood gas, urine routine and microscopic examination (with culture where applicable), urine albumin-creatinine ratio, and ultrasonography of the kidneys, ureters, and bladder in all patients; autoimmune and vasculitis workup (ANA, C3, C4, anti-dsDNA, ENA, antiphospholipid antibody profile) and renal biopsy were performed in selected patients based on clinical features. Investigations were repeated on day 7, with additional special investigations and imaging incorporated as they became available.

For continuous variables, means with standard deviations or medians were computed as appropriate; normally distributed continuous variables were compared using the t-test, and non-normally distributed variables using the rank-sum test. Categorical variables were compared using the chi-square test, and correlation between ordinal variables was assessed using Spearman's correlation coefficient. A p-value <0.05 was considered statistically significant. Data were analysed using SPSS version 24.

Results

Of 100 patients, 52 (52.0%) were male and 48 (48.0%) were female (male:female ratio 1.08:1). The mean age was 38.59 years for males, 42.77 years for females, and 40.61 years overall (youngest 14 years, oldest 80 years). The largest proportion of patients (31.0%) were aged 41–55 years. Oliguria was the most common presenting feature (71.0%), followed by nausea/vomiting (30.0%), fever (17.0%), oedema (16.0%), and drowsiness (10.0%) (Table 1).

Table 1: Demographic and clinical profile

Parameter	n	%
Male	52	52.0%
Female	48	48.0%
Age 13–25 years	22	22.0%
Age 25–40 years	27	27.0%
Age 41–55 years	31	31.0%
Age 56–65 years	13	13.0%
Age >65 years	7	7.0%
Oliguria	71	71.0%
Nausea/vomiting	30	30.0%
Fever	17	17.0%
Oedema	16	16.0%
Drowsiness	10	10.0%

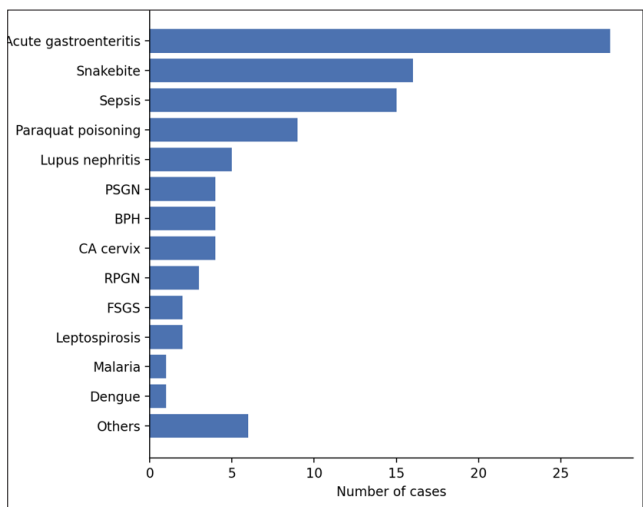


Figure 1. Etiological distribution of acute kidney injury

Acute gastroenteritis (28.0%), snakebite (16.0%), and sepsis (15.0%) were the three most common causes of AKI, together accounting for 59% of cases, followed by paraquat poisoning (9.0%) and lupus nephritis (5.0%) (Figure 1).

Among the 14 patients who died within the first week, sepsis was the leading cause (42.9%), followed by paraquat poisoning (21.4%) and carcinoma cervix (14.3%); acute gastroenteritis, lupus nephritis, and pyrexia of unknown origin each accounted for one death (7.1% each) (Table 2).

Table 2: Causes of mortality within the first week

Etiology	n	%
Sepsis	6	42.9%
Paraquat poisoning	3	21.4%
Carcinoma cervix	2	14.3%
Acute gastroenteritis	1	7.1%
Lupus nephritis	1	7.1%
Pyrexia of unknown origin	1	7.1%

Renal (intrinsic) AKI was the most common type (62.0%), followed by prerenal (28.0%) and postrenal (10.0%) causes. Serum urea was fairly evenly distributed across the three bands examined, with a slightly higher proportion in the 100–150 mg/dl group (35.0%). Half of patients (50.0%) had serum creatinine between 2 and 4 mg/dl. Most patients (87.0%) had a normal serum potassium; hyperkalaemia was present in 9.0% and hypokalaemia in 4.0% (Table 3).

Table 3: Investigation findings

Parameter	n	%
Prerenal AKI	28	28.0%
Renal (intrinsic) AKI	62	62.0%
Postrenal AKI	10	10.0%
Serum urea <100 mg/dl	33	33.0%
Serum urea 100–150 mg/dl	35	35.0%
Serum urea >150 mg/dl	32	32.0%
Serum creatinine <2 mg/dl	22	22.0%
Serum creatinine 2–4 mg/dl	50	50.0%
Serum creatinine >4 mg/dl	28	28.0%
Serum K ⁺ <3.5 mEq/L	4	4.0%
Serum K ⁺ 3.5–5.5 mEq/L	87	87.0%
Serum K ⁺ >5.5 mEq/L	9	9.0%

Just over half of patients (51.0%) required at least one session of hemodialysis, while 49.0% were managed conservatively. At the end of 1 week, 67.0% had fully recovered, 19.0% had partially recovered, and 14.0% had died (Figure 2).

No statistically significant association was found between type of AKI (prerenal, renal, or postrenal) and outcome ($p=0.5$). A statistically significant positive correlation was found between higher serum urea and poorer outcome (Spearman's $\rho=0.997$, $p<0.05$), and between higher serum creatinine and poorer outcome (Spearman's $\rho\approx 1$, $p<0.001$); among patients with serum creatinine >4 mg/dl, only 25.0% fully recovered, compared with 100% of those with creatinine <2 mg/dl. Both hyperkalaemia and hypokalaemia were significantly associated with poorer outcome ($p<0.001$). Outcome also differed significantly by

treatment modality ($p<0.001$): 93.9% of patients managed conservatively fully recovered, compared with 41.2% of those who received hemodialysis, among whom 27.5% died — consistent with hemodialysis being required preferentially in more severely ill patients rather than itself worsening prognosis (Table 4).

To clarify whether the association between serum creatinine and outcome held independently within each treatment group, outcome was further analysed by creatinine level separately among conservatively managed and hemodialysed

patients. Among the 49 patients managed conservatively, full recovery occurred in 100.0% of those with creatinine <2 mg/dl and 88.5% of those with creatinine 2–4 mg/dl (3 of 26 partially recovered), with no deaths in this group; only one conservatively managed patient had creatinine >4 mg/dl, and this patient also fully recovered ($p=0.001$, chi-square). Among the 51 patients who received hemodialysis, none had creatinine <2 mg/dl; outcome worsened markedly with rising creatinine, from 62.5% full recovery and no deaths in the 2–4 mg/dl band to 22.2% full recovery and 51.9% mortality in the >4 mg/dl band ($p=0.0002$) (Table 5).

Table 4: Association between clinical/biochemical parameters and outcome

Parameter	Full recovery	Partial recovery	Death	p-value
Prerenal AKI (n=28)	96.4%	0.0%	3.6%	0.5
Renal AKI (n=62)	56.5%	24.2%	19.3%	
Postrenal AKI (n=10)	40.0%	40.0%	20.0%	
Urea <100 mg/dl (n=33)	81.8%	18.2%	0.0%	<0.05
Urea 100–150 mg/dl (n=35)	68.6%	22.9%	8.6%	
Urea >150 mg/dl (n=32)	50.0%	15.6%	34.4%	
Creatinine <2 mg/dl (n=22)	100.0%	0.0%	0.0%	<0.001
Creatinine 2–4 mg/dl (n=50)	76.0%	24.0%	0.0%	
Creatinine >4 mg/dl (n=28)	25.0%	25.0%	50.0%	
K ⁺ <3.5 mEq/L (n=4)	50.0%	0.0%	50.0%	<0.001
K ⁺ 3.5–5.5 mEq/L (n=87)	75.9%	16.1%	8.0%	
K ⁺ >5.5 mEq/L (n=9)	11.1%	33.3%	55.6%	
Conservative treatment (n=49)	93.9%	6.1%	0.0%	<0.001
Hemodialysis (n=51)	41.2%	31.4%	27.5%	

Table 5: Outcome by serum creatinine level within treatment-modality subgroups

Treatment / creatinine	Full recovery	Partial recovery	Death	p-value
Conservative, creatinine <2 mg/dl	22	0	0	0.001
Conservative, creatinine 2–4 mg/dl	23	3	0	
Conservative, creatinine >4 mg/dl	1	0	0	
Conservative, creatinine >4 mg/dl	0	0	0	0.0002
Hemodialysis, creatinine 2–4 mg/dl	15	9	0	
Hemodialysis, creatinine >4 mg/dl	6	7	14	

Discussions

In this cohort of 100 patients with AKI admitted to a tertiary care hospital in eastern India, acute gastroenteritis, snakebite, and sepsis were the three leading etiologies, together accounting for well over half of cases, and renal (intrinsic) AKI was the predominant type. This community-acquired, single-insult pattern of AKI, dominated by volume depletion, infection, and envenomation, is characteristic of AKI in tropical, resource-limited settings, and contrasts with the largely hospital-acquired, comorbidity-driven pattern described in high-income countries.

The male predominance observed in this cohort (52.0%, male:female ratio 1.08:1) was modest compared with some other series. Kandoth et al., in a study of children with AKI requiring dialysis in India, reported a male:female ratio of 1.8:1, with delay in seeking medical attention more common among female patients, a pattern often attributed to differential health-seeking behaviour rather than a true difference in disease incidence between sexes[11]. Mehta et al., in the PICARD cohort of adults with AKI in intensive care units in the United States, reported a somewhat higher male predominance (59% male, mean age 59.5 years)[12], while Bernieh et al., describing the pattern of AKI in a tertiary hospital in the United Arab Emirates, reported a male:female ratio of 1.3:1 with a mean age of 56.2 years[13]. The mean age in the present cohort (40.6 years) was notably lower than in each of these comparator series, consistent with the predominantly community-acquired, infection- and toxin-related aetiological pattern observed here, which tends to affect younger, previously healthy individuals, in contrast to the older, comorbid populations typically described in ICU-based or high-income cohorts.

The overall mortality in this cohort (14.0%) fell within the wide range reported across the AKI literature. Asmus et al., in a retrospective cohort study from Germany using KDIGO criteria, reported an in-hospital mortality of 26% among AKI patients, considerably higher than in the present study, though their cohort was drawn from a broader, more comorbid hospital population rather than a single-insult, community-acquired pattern[14]. The comparatively lower mortality in the present cohort may reflect both the younger age profile and the predominance of reversible causes such as acute gastroenteritis and snakebite, which, with timely fluid resuscitation or antivenom therapy, often permit fuller renal recovery than AKI secondary to sepsis or multiorgan dysfunction.

Consistent with previous reports, higher serum urea and creatinine, and hyperkalaemia, were each significantly associated with poorer outcome in this cohort, and both hemodialysis requirement and mortality were concentrated among patients with more severe biochemical derangement. This pattern mirrors the general clinical experience that the degree of biochemical derangement at presentation reflects the severity of the underlying insult and the physiological

reserve of the patient, rather than hemodialysis itself conferring risk; in the present study, patients managed conservatively had a substantially higher rate of full recovery than those requiring hemodialysis, most plausibly because dialysis was preferentially required in more severely ill patients. Notably, the creatinine-outcome relationship persisted even within the hemodialysis subgroup itself, where mortality rose from 0% among patients with creatinine 2–4 mg/dl to 51.9% among those with creatinine >4 mg/dl despite all receiving RRT, indicating that creatinine at presentation captures underlying illness severity that dialysis does not fully offset, rather than merely tracking which patients happened to need dialysis. No significant association was found between the anatomical type of AKI (prerenal, renal, or postrenal) and outcome in this cohort, in keeping with the general principle that severity and reversibility of the underlying insult, rather than the anatomical category of injury per se, more directly determine short-term prognosis.

Conclusion

Acute gastroenteritis, snakebite, and sepsis were the leading causes of AKI in this cohort of patients admitted to a tertiary care hospital in eastern India, and renal (intrinsic) AKI was the predominant type. Just over half of patients required hemodialysis, and 14% died within the first week, with sepsis the leading cause of death. Higher serum urea, creatinine, and potassium levels, and the requirement for renal replacement therapy, were each significantly associated with poorer short-term outcome, supporting their use as simple, readily available markers for early risk stratification in patients with AKI.

Source of Funding

Nil.

Conflict of Interest

Nil.

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