Spectrum of Etiopathogenesis of Acute Kidney Disease in Indoor Patients: A Prospective Observational Study from a Tertiary Care Centre in Rajasthan, India

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Abstract

Background: Acute kidney disease (AKD) represents the transitional state of kidney dysfunction between acute kidney injury (AKI) and chronic kidney disease (CKD), and in hospitalised (indoor) patients it arises from a broad, multifactorial spectrum of prerenal, intrinsic and postrenal insults. The present study was planned to study the spectrum of etiopathogenesis of acute kidney disease in indoor patients at a tertiary care centre of Rajasthan, India.

Materials and Methods: This hospital-based prospective observational study was conducted in the Department of General Medicine, Jaipur National University Institute of Medical Sciences and Research Centre, Jaipur, over 15 months from the date of approval of the college research committee and the ethics committee. One hundred patients aged more than 18 years with acute kidney injury attending the outpatient department or admitted to the wards/intensive care units were enrolled after applying the inclusion and exclusion criteria. Information was collected using a pre-designed, semi-structured proforma, and all patients underwent detailed history, clinical examination and routine laboratory investigations, including complete blood count and renal function tests. Data were analysed using SPSS-25; quantitative variables were expressed as mean ± standard deviation and qualitative data as percentages, with $p < 0.05$ considered statistically significant.

Results: Of the 100 patients, 63% were males and 37% females, with a mean age of 50.91 ± 14.87 years; 39% were aged above 60 years. The mean body mass index was 22.32 ± 2.14 kg/m². Diabetes was present in 45%, hypertension in 33% and coronary artery disease in 23% of patients. Decreased urine output (67%) was the most common presenting feature, followed by hypotension (63%), swelling (60%) and fever (57%). Sepsis was present in 46% and oliguria in 67% of patients. The mean blood urea, serum creatinine and eGFR were 93.42 ± 5.21 mg/dl, 2.56 ± 0.67 mg/dl and 43.21 ± 7.63 ml/min/1.73 m², respectively. Sepsis was the most common etiology of AKI (46%), followed by infections (25%), haemorrhage (15%), poisoning (13%) and drug-induced injury (5%). Eighty-two per cent of patients were discharged and 18% expired. Patients who expired had significantly higher blood urea (104.08 ± 8.65 vs 93.72 ± 5.31 mg/dl; $p = 0.001$) and serum creatinine (4.40 ± 0.49 vs 2.58 ± 0.68 mg/dl; $p = 0.001$) and significantly lower eGFR (37.29 ± 4.40 vs 42.86 ± 7.77 ml/min/1.73 m²; $p = 0.004$). Sepsis (77.8% vs 39%; $p = 0.003$) and oliguria (100% vs 59.8%; $p = 0.001$) were significantly associated with mortality.

Conclusion: Acute kidney injury predominantly affected older adults with a male predominance and a high burden of comorbidities. Sepsis and oliguria were common and showed a strong, statistically significant association with mortality, while patients who expired had significantly deranged renal parameters. Early identification of high-risk patients—particularly those with sepsis, oliguria and markedly deranged renal parameters—along with prompt and aggressive management, is essential to improve outcomes and reduce mortality in acute kidney injury.

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Keywords: Acute kidney disease, Acute kidney injury, Etiopathogenesis, Sepsis, Oliguria, Indoor patients, KDIGO, Mortality

Introduction

Acute Kidney Disease (AKD) is a recently recognized and increasingly important clinical entity that represents a transitional state of kidney dysfunction between acute kidney injury (AKI) and chronic kidney disease (CKD).[1] While AKI is characterized by a rapid decline in renal function occurring within 48–72 hours and CKD by structural or functional kidney damage persisting for more than three months, AKD spans the intermediate phase—defined as evidence of kidney damage or decreased function lasting more than seven days but less than 90 days.[2] This continuum is vital to understand because AKD often develops either due to unresolved AKI or may occur de novo in systemic conditions causing subacute renal damage.[3] The recognition of AKD

helps bridge the diagnostic and therapeutic gap between AKI and CKD and underscores a critical window of opportunity to initiate interventions aimed at renal recovery before irreversible damage sets in. Clinically, AKD can present with persistent elevations in serum creatinine, reduced urine output, or structural abnormalities evidenced by imaging or biomarkers. Unlike AKI, which often triggers an immediate response due to its abrupt onset, AKD may evolve subtly and progressively, making it harder to detect, especially in hospitalized patients where overlapping morbidities may mask its presentation. Patients with AKD have a significantly increased risk of progressing to CKD or end-stage renal disease (ESRD), and they also face heightened risks for cardiovascular events and death. In the hospital setting, particularly among indoor patients with severe systemic illnesses, surgical interventions, or exposure to nephrotoxins, the risk of developing AKD is markedly elevated.[4,5]

The 2012 KDIGO AKI Workgroup first proposed criteria for AKD, encompassing any acute renal condition such as AKI, a glomerular filtration rate (GFR) <60 ml/min/1.73 m² for less than 3 months, a reduction in GFR by ≥35% or an increase in serum creatinine (SCr) by >50% within 3 months, or evidence of kidney damage lasting under 3 months.[6] Subsequently, in 2017, the 16th Acute Disease Quality Initiative (ADQI) Workgroup redefined AKD as the persistence of AKI stage 1 or higher, according to KDIGO, for ≥7 days following an initiating insult; although an inciting event is often identifiable, it is not mandatory for establishing an AKD diagnosis.[7]

The spectrum of etiopathogenesis of Acute Kidney Disease, especially in indoor or hospitalized patients, is broad, multifactorial, and clinically nuanced. It reflects a variety of sustained insults to renal hemodynamics, tubular structure, glomerular integrity, and interstitial tissues. In prerenal AKD, the prolonged reduction in renal perfusion without timely correction leads to ischemic damage, primarily affecting the renal tubules. This is often seen in hospitalized patients suffering from sepsis, septic shock, major surgeries (such as cardiac or abdominal operations), trauma, or gastrointestinal losses causing volume depletion.[8] Hemodynamic instability in the setting of vasodilatory shock, massive blood loss, or heart failure can persist and cause ongoing hypoperfusion, leading initially to reversible AKI but eventually transitioning into AKD due to cumulative ischemic injury and failure of repair mechanisms. In intrinsic AKD, the causes are diverse and commonly encountered in hospitalized patients.[9] Acute tubular necrosis (ATN) that fails to resolve within a week is a common pathway, especially in ICU patients who are exposed to repeated episodes of hypotension, nephrotoxic drugs (like aminoglycosides, amphotericin B, cisplatin, or radiocontrast agents), and systemic inflammation.[10] Acute interstitial nephritis, often drug-induced (from antibiotics like beta-lactams, proton pump inhibitors, or NSAIDs), can present insidiously and progress to subacute renal dysfunction if not recognized early.[11] Autoimmune glomerulonephritides such as ANCA-associated vasculitis, lupus nephritis, or anti-GBM disease may not follow an acute course and instead exhibit a smoldering pattern of renal inflammation contributing to AKD.[12] Additionally, multiple myeloma-related light chain cast nephropathy and thrombotic microangiopathies are frequently seen in hospital patients and represent intrinsic causes that lead to persistent renal damage.

Infectious causes are also critical in this spectrum—particularly in tropical countries or immunocompromised indoor patients—where leptospirosis, malaria, dengue, and disseminated tuberculosis can result in prolonged renal insult through immune mechanisms or direct pathogen-induced injury.[13]

Postrenal AKD is another important category, particularly in elderly or cancer patients, where partial or intermittent urinary outflow obstruction from prostatic hypertrophy, pelvic malignancies, retroperitoneal fibrosis, or ureteric stones causes gradually progressive hydronephrosis and pressure-induced renal parenchymal atrophy. This may not produce the acute oligoanuria typical of AKI but rather manifests as a steady decline in renal function over days to weeks. In indoor patients, iatrogenic factors form a substantial part of the etiological spectrum of AKD. This includes not only nephrotoxic drug exposure but also inappropriate or aggressive fluid management, repeated contrast imaging, unrecognized obstructive uropathy during hospitalization, and complications from interventions such as cardiopulmonary bypass, liver transplant, or stem cell transplant. In the recovery phase of AKI, maladaptive repair mechanisms—including incomplete tubular regeneration, persistent endothelial dysfunction, microvascular loss, and interstitial fibrosis—play a key role in driving the transition into AKD.

Moreover, patients with underlying comorbidities like diabetes mellitus, systemic hypertension, chronic liver disease, or cardiovascular disorders are particularly vulnerable to AKD even without a frank AKI episode, due to subclinical insults accumulating in a hospital environment.[14] AKD in indoor patients therefore represents a dynamic and heterogenous spectrum of kidney dysfunction triggered by persistent or repetitive insults rather than a single acute event. Recognizing the etiopathogenic spectrum of AKD in hospitalized patients is crucial for timely diagnosis, targeted therapy, and prevention of irreversible renal damage or progression to CKD. This necessitates vigilant monitoring of renal function beyond the acute phase, judicious use of nephrotoxic agents, correction of hemodynamic abnormalities, early imaging for obstructive uropathy, and a high index of suspicion in patients with systemic illnesses or poor renal reserve.

With this background, the present study was planned to study the spectrum of etiopathogenesis of acute kidney disease in indoor patients in a tertiary care centre of Rajasthan, India. The aim of this study was to identify the high-risk patients, characterizing etiological factors, developing preventive strategies, optimizing clinical management, assessing healthcare outcomes, exploring novel biomarkers, promoting multidisciplinary approaches, and enhancing patient education. The objective was to study the screening, refining treatment protocols, evaluating biomarkers, and fostering collaboration to improve early detection, prevention, and management of acute kidney disease in indoor patients.

Materials and Methods

Study Design and Setting

This was a hospital-based prospective observational study conducted in the Department of General Medicine, Jaipur National University, Institute of Medical Sciences and Research Centre, Jagatpura, Jaipur, Rajasthan. The study was conducted for a period of 15 months from the date of approval of the college research committee and the ethical committee.

Sample Size

Based on the previous studies conducted, the sample size was calculated using the formula $n = Z^2 \times P \times (1 - P) / d^2$, where Z is the Z statistic for the level of confidence (for a 95% level of confidence, which is conventional, the Z value is 1.96), P is the expected prevalence or proportion (in proportion of one; if 8%, P = 0.08) and d is the precision (in proportion of one; if 5%, d = 0.05). Putting the above values in the formula gave a sample size of 96, which was rounded up to 100 patients.

Inclusion Criteria

- Patients with age group > 18 years.
- All acute kidney injury patients enrolled in the Department of General Medicine, Institute of Medical Sciences and Research Centre, Jaipur National University.

Exclusion Criteria

- Patients not willing to give consent for study.
- End stage renal disease (ESRD) with GFR < 15 ml/min/m².
- Patients who were in the intensive care unit (ICU) for less than 24 hours.
- Pregnant females.
- Presumed life expectancy of less than 48 hours after admission.
- Renal vasculitis or post renal etiology for AKI.
- Patients who had just undergone renal transplantation or were prior to kidney transplantation.

Methodology

All patients with acute kidney injury attending the outpatient department or admitted in the ward/ICUs of the Department of General Medicine, Jaipur National University, Institute of Medical Sciences and Research Centre, Jaipur, Rajasthan during the 15-month period from the date of approval of the college research committee and ethics committee were taken for the study, considering the inclusion and exclusion criteria. Information was collected through a pre-designed, semi-structured proforma for each patient. Qualifying patients underwent detailed history, clinical examination, and laboratory investigations. The patient's clinical history and examination findings were recorded prospectively. The patients were informed about their enrolment into the study and the study implications. Routine laboratory investigations performed were complete blood count (CBC) and renal function tests, including serum urea and creatinine.

Statistical Analysis

The data were entered and cleaned using MS-Excel and analysed statistically using SPSS-25. Quantitative variables were expressed as mean value ± standard deviation or median ± interquartile range. Qualitative data were expressed as percentages (%) and proportion. Appropriate statistical tests were used to infer association between two variables, and a p value of < 0.05 was considered statistically significant.

Ethical Considerations

The protocol was presented before the ethical committee for approval. After due approval from the ethical committee, the study was initiated. Before including any patient for participation in the study, a voluntary written consent for participation was obtained from either the patient and/or his/her legally acceptable representative. Information obtained from patients was kept confidential and was only used for scientific reasons, and no personal identity of the participant was disclosed at any point in time. There was no additional risk to patients because of their participation in the study. Participation in the study was purely voluntary in nature.

Funding

No funding was required. The investigations were done as a part of routine investigations.

Results

In our study, a total of 100 patients who were admitted with acute kidney injury fulfilling the inclusion and exclusion criteria were included.

Table 1: Distribution of patients according to gender

Gender	Frequency (n)	Percentage (%)
Males	63	63
Females	37	37
Total	100	100

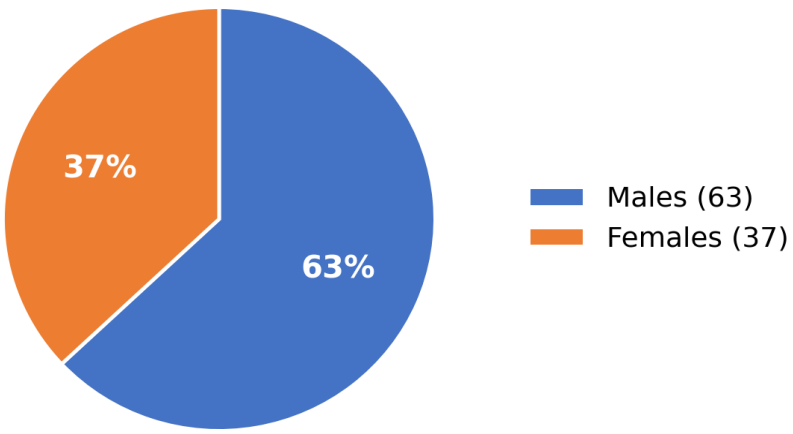

Fig 1: Distribution of patients according to gender

Table 1 and Figure 1 show the distribution of patients according to gender. In our study, 63% of patients were males while 37% of patients were females.

Table 2: Distribution of patients according to age

Age	Frequency (n)	Percentage (%)
18 to 30 years	13	13
31 to 45 years	21	21
46 to 60 years	27	27
> 60 years	39	39
Total	100	100
Mean ± standard deviation	50.91 ± 14.87	—

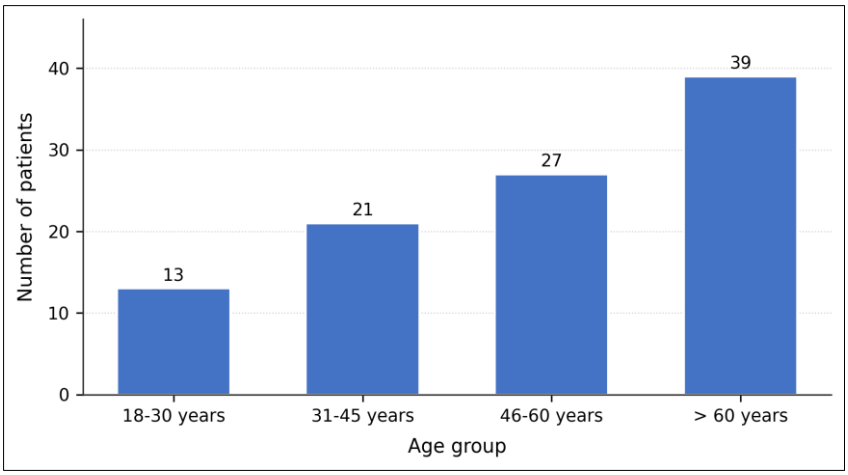

Fig 2: Distribution of patients according to age

Table 2 and Figure 2 show the distribution of patients according to age. Thirty-nine patients were aged above 60 years, followed by 27 patients in the age group of 46 to 60 years. Twenty-one patients were aged between 31 to 45 years and only 13 patients were in the age group of 18 to 30 years. The mean age in our study was 50.91 ± 14.87 years.

Table 3: Distribution of patients according to body mass index

Body mass index (BMI)	Frequency (n)	Percentage (%)
Normal (18.5 to 22.9 kg/m²)	59	59
Overweight (23 to 24.9 kg/m²)	24	24
Obese (≥ 25 kg/m²)	17	17
Total	100	100
Mean ± standard deviation	22.32 ± 2.14	—

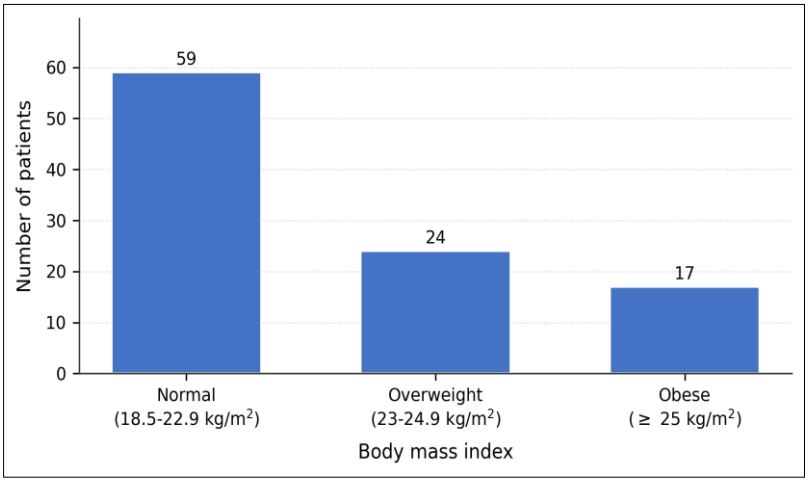

Fig 3: Distribution of patients according to body mass index

Table 3 and Figure 3 show the distribution of patients according to body mass index (BMI). Fifty-nine patients were in the normal BMI category while 24 patients were overweight in our study. Seventeen patients had BMI ≥ 25 kg/m², making them classified as obese. The mean body mass index in our study was 22.32 ± 2.14 kg/m².

Table 4: Distribution of patients according to comorbidities

Comorbidities	Frequency (n)	Percentage (%)
Diabetes	45	45
Hypertension	33	33
Coronary artery disease	23	23

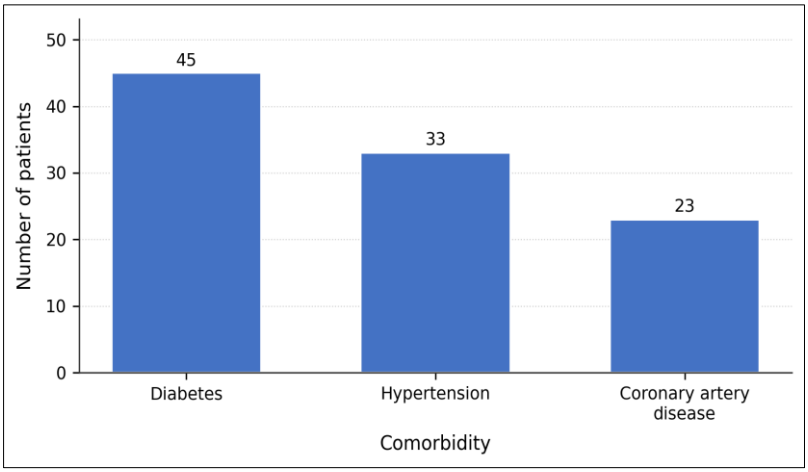

Fig 4: Distribution of patients according to comorbidities

Table 4 and Figure 4 show the distribution of patients according to comorbidities. Diabetes was present in 45 patients while hypertension was observed in 33 patients. Twenty-three patients had coronary artery disease (CAD) as a comorbid condition in the present study.

Table 5: Distribution of patients according to clinical symptoms

Clinical symptoms	Frequency (n)	Percentage (%)
Decrease urine output	67	67
Hypotension	63	63
Swelling	60	60
Fever	57	57
Vomiting	54	54
Yellowish discoloration	40	40
Loose stools	40	40
History of poisoning	13	13

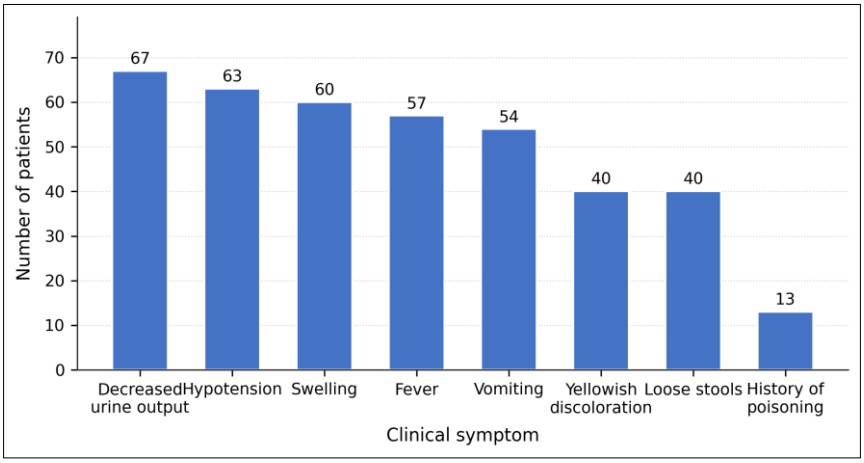

Fig 5: Distribution of patients according to clinical symptoms

Table 5 and Figure 5 summarize the clinical presentation of patients in the study population. Decreased urine output was the most common presenting feature, observed in 67% of patients, highlighting the frequent involvement of renal dysfunction in sepsis. Hypotension was noted in 63% of cases, reflecting the high prevalence of septic shock or circulatory compromise. Fever was present in 57% of patients, indicating that although common, it was not a universal finding. Gastrointestinal symptoms were also frequent, with vomiting reported in 54% and loose stools in 40% of patients. Swelling, suggestive of fluid overload or capillary leak, was observed in 60% of cases. Yellowish discoloration, indicating possible hepatic involvement or hyperbilirubinemia, was seen in 40% of patients. A history of poisoning was reported in 13% of cases, representing a relatively uncommon but notable associated factor.

Table 6: Distribution of patients according to sepsis

Sepsis	Frequency (n)	Percentage (%)
Present	46	46
Absent	54	54
Total	100	100

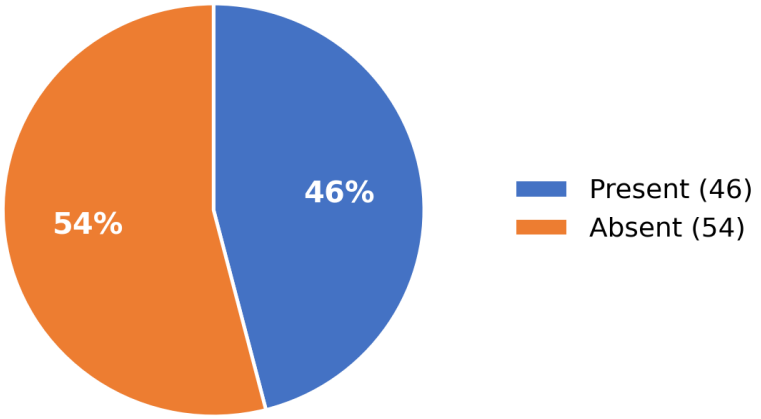

Fig 6: Distribution of patients according to sepsis

Table 6 and Figure 6 show the distribution of patients according to sepsis. It was present in 46% of patients in our study.

Table 7: Distribution of patients according to oliguria

Oliguria	Frequency (n)	Percentage (%)
Present	67	67
Absent	33	33
Total	100	100

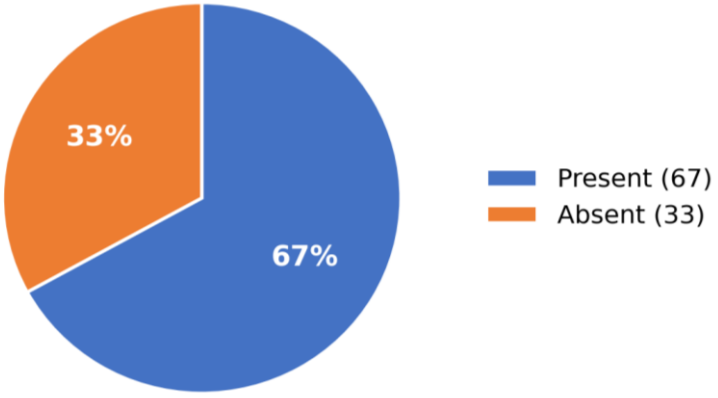

Fig 7: Distribution of patients according to oliguria

Table 7 and Figure 7 show the distribution of patients according to the presence or absence of oliguria in our study. Oliguria was found to be present in 67% of patients.

Table 8: Descriptive statistics for clinical parameters

Variables	Mean	Standard deviation
Pulse rate (beats per minute)	81.75	7.13
Systolic blood pressure (mm Hg)	130.88	7.93
Diastolic blood pressure (mm Hg)	88.38	5.42

Table 8 shows the descriptive statistics for various clinical parameters. The mean pulse rate in our study was 81.75 ± 7.13 beats per minute. The average systolic and diastolic blood pressure in the present study were 130.88 ± 7.93 mm Hg and 88.38 ± 5.42 mm Hg respectively.

Table 9: Descriptive statistics for laboratory parameters

Laboratory parameters	Mean	Standard deviation
Haemoglobin (g/dl)	11.28	1.64
Total leukocyte count (per mm³)	9234.92	1844.15
Platelets (thousands per mm³)	269.23	45.77
Blood urea (mg/dl)	93.42	5.21
S. Creatinine (mg/dl)	2.56	0.67
eGFR (ml/min/1.73 m²)	43.21	7.63

Table 9 shows the descriptive statistics for various laboratory parameters. The mean haemoglobin level in the study population was 11.28 ± 1.64 g/dl. The average total leukocyte count and platelet count were 9234.92 ± 1844.15 per mm³ and 269.23 ± 45.77 thousands per mm³ respectively. The average serum urea and creatinine levels were 93.42 ± 5.21 mg/dl and 2.56 ± 0.67 mg/dl respectively. The average eGFR was 43.21 ± 7.63 ml/min/1.73 m².

Table 10: Distribution of patients according to etiology of AKI

Etiology of AKI	Frequency (n)	Percentage (%)
Sepsis	46	46
Infections	25	25
Haemorrhage	15	15
Poisoning	13	13
Drug induced	05	05

*Etiologies are not mutually exclusive; one patient can have one or more etiology.

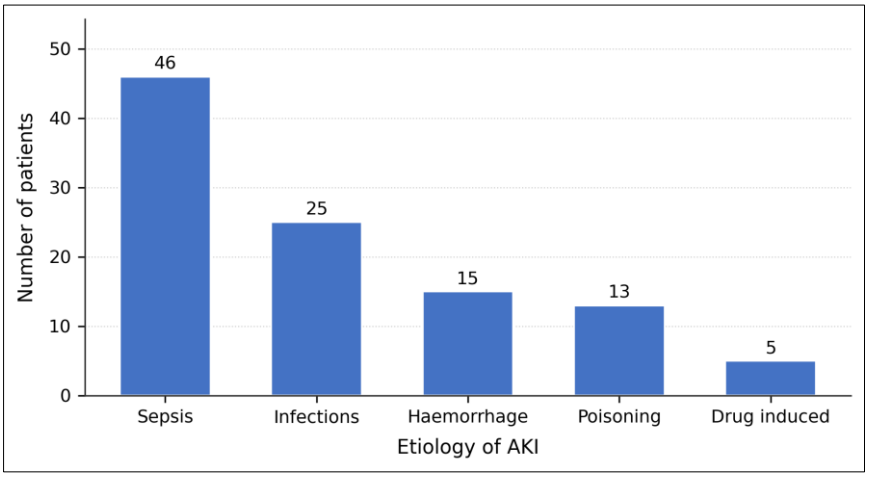

Fig 8: Distribution of patients according to etiology of AKI

Table 10 and Figure 8 show the distribution of patients according to etiology of AKI. Sepsis was the most common cause of AKI, which was present in 46% of patients, followed by infections which were present in 25% of patients. Haemorrhage was observed in 15% of patients while poisoning as the cause of AKI was present in 13% of patients. Drug-induced AKI was observed in 5% of patients.

Table 11: Distribution of patients according to outcome

Outcome	Frequency (n)	Percentage (%)
Discharged	82	82
Expired	18	18
Total	100	100

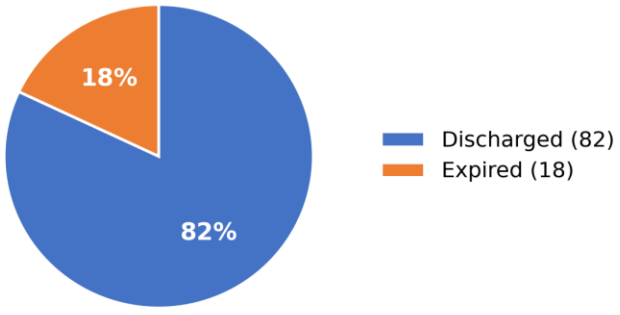

Fig 9: Distribution of patients according to outcome

Table 11 and Figure 9 show the distribution of patients according to outcome. Eighty-two per cent of patients were discharged after recovering while 18% of patients expired during their stay in hospital.

Table 12: Comparison between biochemical indices based on outcome

Biochemical indices	Discharged		Expired		p-value*
	Mean	SD	Mean	SD	
Blood urea (mg/dl)	93.72	5.31	104.08	8.65	0.001
S. Creatinine (mg/dl)	2.58	0.68	4.40	0.49	0.001
eGFR (ml/min/1.73 m²)	42.86	7.77	37.29	4.40	0.004

*p-value < 0.05 statistically significant

Table 12 shows the comparison between biochemical indices based on outcome in the present study. In our study, we found that the mean blood urea level was higher among patients who expired (104.08 ± 8.65 mg/dl) as compared to patients who were discharged (93.72 ± 5.31 mg/dl) and the difference between the two was statistically significant (p = 0.001). The mean serum creatinine level was higher among patients who expired (4.40 ± 0.49 mg/dl) as compared to patients who were discharged (2.58 ± 0.68 mg/dl) and the difference between the two was statistically significant (p = 0.001). The average eGFR was 37.29 ± 4.40 ml/min/1.73 m² among patients who expired, which was significantly lower than in patients who were discharged (42.86 ± 7.77 ml/min/1.73 m²), and the difference between the two was statistically significant (p = 0.004).

Table 13: Comparison between sepsis and outcome

Sepsis	Outcome		p-value*
	Discharged n (%)	Expired n (%)	
Present	32 (39)	14 (77.8)	0.003
Absent	50 (61)	04 (22.2)	
Total	82 (100)	18 (100)	

*p-value < 0.05 statistically significant; Chi-square test applied

Table 13 shows the comparison between sepsis and outcome in the present study. It can be seen that among patients who expired, 77.8% of patients had sepsis as compared to 39% of patients who were discharged. The association between sepsis and outcome was statistically significant (p = 0.003).

Table 14: Comparison between oliguria and outcome

Oliguria	Outcome		p-value*
	Discharged n (%)	Expired n (%)	
Present	49 (59.8)	18 (100)	0.001
Absent	33 (40.2)	00 (00)	
Total	82 (100)	18 (100)	

*p-value < 0.05 statistically significant; Chi-square test applied

Table 14 shows the comparison between oliguria and outcome in the present study. It can be seen that among patients who expired, 100% of patients had oliguria as compared to 59.8% of patients who were discharged. The association between oliguria and outcome was statistically significant (p = 0.001).

Discussion

Acute kidney injury (AKI) constitutes a major global health concern and is one of the most frequently encountered complications among hospitalized patients, particularly in tertiary care centers catering to critically ill and high-risk populations. It is a multifaceted clinical syndrome characterized by an abrupt decline in renal function, leading to disturbances in fluid, electrolyte, and acid–base balance, and often reflecting the combined effects of systemic illness, hemodynamic instability, infection, and underlying chronic diseases. The incidence of AKI has been steadily rising due to increasing life expectancy, higher prevalence of diabetes mellitus, hypertension, cardiovascular disorders, and greater exposure to invasive procedures, nephrotoxic agents, and severe infections.

The clinical spectrum of AKI is highly variable, ranging from mild and transient elevations in serum creatinine to severe renal failure requiring renal replacement therapy. Importantly, AKI is not merely a reversible biochemical abnormality but a powerful predictor of adverse outcomes, including prolonged hospitalization, progression to chronic kidney disease, and increased short- and long-term mortality. The presence of sepsis and oliguria often signifies a more severe disease state, reflecting systemic inflammation, impaired perfusion, and multiorgan involvement, and is consistently associated with poorer prognosis. Despite advances in diagnostic modalities and supportive care, outcomes in AKI remain suboptimal, largely due to delayed recognition, heterogeneity in presentation, and coexistence of multiple comorbid conditions. Against this background, the present study was undertaken to comprehensively evaluate patients admitted with acute kidney injury, analyze their clinical and biochemical profiles, and identify factors influencing outcomes. The following is a discussion of our study findings with those of previous studies to check for generalizability of results.

Gender

In our study, 63% of patients were males while 37% of patients were females. In a study by Rout N *et al*,[15] among the AKI patients, 51% of patients were male and 48.5% were females. In another similar study by Kathyayani M *et al*,[16] there were 99 males and 51 females. In a study by Bhudhrani

D *et al*,[17] 246 participants were analysed, with a predominance of males (65.85%) over females (34.15%), resulting in a male-to-female ratio of 1.56:1. In another study by Eswarappa M *et al*,[18] 63.6% were males. In a study by Rao J *et al*,[19] 71.6% were males. In a study by Kashinkunti M *et al*,[20] 56.6% were males. In another study by Suresh V *et al*,[21] out of 237 patients with AKI, 62% were males.

Age

Thirty-nine patients were aged above 60 years, followed by 27 patients in the age group of 46 to 60 years. Twenty-one patients were aged between 31 to 45 years and only 13 patients were in the age group of 18 to 30 years. The mean age in our study was 50.91 ± 14.87 years. In a study by Rout N *et al*,[15] the mean age of the patients was 34 ± 16 years. In another similar study by Kathyayani M *et al*,[16] the average age of the patients was 43.97 years, and their ages ranged from 18 to 75 years. In another study by Bhudhrani D *et al*,[17] the mean age of participants was 50.6 ± 18.00 years, with ages ranging from 18 to 80 years; the most affected age group was individuals over 60 years (39.42%), followed by the 50–60 years group (14.88%), while the least affected group was 18–20 years (4.2%). In another study by Eswarappa M *et al*,[18] 41.8% of patients were aged more than 60 years. In a study by Rao J *et al*,[19] the mean age of the cohort was 54 years. In a study by Kashinkunti M *et al*,[20] most of the patients (43.3%) were in the 31–50 years age group.

Comorbidities

Diabetes was present in 45 patients while hypertension was observed in 33 patients. Twenty-three patients had coronary artery disease (CAD) as a comorbid condition in the present study. In another similar study by Rout N *et al*,[15] the majority of patients in the AKI cohort were nondiabetic (only 4.8% were diabetic) and had no history of hypertension (only 6.7% were hypertensive). In a study by Eswarappa M *et al*,[18] diabetes mellitus (30.6%) and hypertension (29.2%) were the most prevalent comorbid conditions, highlighting a high burden of metabolic and vascular risk factors; coronary heart disease was present in 11.4% of patients, while chronic liver disease accounted for 9.2%. Other comorbidities included COPD (7.0%), malignancy (6.8%), and cerebrovascular accident (2.8%). In a study by Kashinkunti M *et al*,[20] comorbidity was seen in 41 (34.2%) patients, of which diabetes was the most common (43.9%).

Clinical Features

The clinical presentation of patients showed that decreased urine output was the most common presenting feature, observed in 67% of patients, highlighting the frequent involvement of renal dysfunction in sepsis. Hypotension was noted in 63% of cases, reflecting the high prevalence of septic shock or circulatory compromise. Fever was present in 57% of patients, indicating that although common, it was not a universal finding. Gastrointestinal symptoms were also frequent, with vomiting reported in 54% and loose stools in 40% of patients. Swelling, suggestive of fluid overload or capillary leak, was observed in 60% of cases. Yellowish discoloration, indicating possible hepatic involvement or hyperbilirubinemia, was seen in 40% of patients. A history of poisoning was reported in 13% of cases, representing a relatively uncommon but notable associated factor. In another study by Bhudhrani D *et al*,[17] decreased urine output was the most common manifestation, observed in 150 patients (60%), indicating frequent renal involvement in sepsis. This was followed by swelling in 140 patients (56%) and hypotension in 130 patients (53%), reflecting significant circulatory compromise and capillary leak commonly seen in severe sepsis. Fever was present in 95 (39%) patients, underscoring its role as a common but not universal presenting symptom. Gastrointestinal manifestations were also prominent, with vomiting reported in 85 patients (35%) and loose motions in 70 patients (29%). Yellowish discoloration, suggestive of hepatic dysfunction or hyperbilirubinemia, was noted in 65 patients (27%). A history of poisoning was observed in 25 patients, representing a comparatively smaller proportion of the study population. In another study by Deshpande N *et al*,[22] decreased urine output, swelling over feet and face, and vomiting were the most common presenting symptoms, comprising 44%, 45% and 34% respectively. In a study by Suresh V *et al*,[21] fever was the most common symptom (48%).

Oliguria

Oliguria was found to be present in 67% of patients. In another similar study by Kathyayani M *et al*,[16] 72.6% of patients were oliguric. In another study by Bhudhrani D *et al*,[17] 60% of the patients were having oliguria. In another study by Eswarappa M *et al*,[18] 67% (n = 335) of the patients were oliguric and 33% of the patients non-oliguric.

Biochemical Indices

The mean haemoglobin level in the study population was 11.28 ± 1.64 g/dl. The average total leukocyte count and platelet count were 9234.92 ± 1844.15 per mm³ and 269.23 ± 45.77 thousands per mm³ respectively. The average serum urea and creatinine levels were 93.42 ± 5.21 mg/dl and 2.56 ± 0.67 mg/dl respectively. The average eGFR was 43.21 ± 7.63 ml/min/1.73 m². In a study by Rao J *et al*,[19] the mean haemoglobin was 9.6 g/dl, urea 121.7 mg/dl, creatinine 5.0 mg/dl, sodium 135.7 mmol/L, and potassium 4.2 mmol/L. KDIGO staging revealed 45.9% in G2, 41.9% in G3, and 12.2% in G4, while urine albumin was absent in 71.6%, mild in 20.3%, and moderate in 8.1%. In another study by Suresh V *et al*,[21] common laboratory abnormalities included anaemia (70%), hyperkalaemia (36%) and proteinuria (29%).

Etiology of AKI

Sepsis was the most common cause of AKI, which was present in 46% of patients, followed by infections which were present in 25% of patients. Haemorrhage was observed in 15% of patients while poisoning as the cause of AKI was present in 13% of patients. Drug-induced AKI was observed in 5% of patients. In a study by Rout N *et al*,[15] 70.8% had medical AKI, 7.7% had surgical AKI and 21.3% had obstetric AKI; puerperal sepsis was the leading cause and infection was associated with 58%. In another study by Kathyayani M *et al*,[16] the predominant causes of AKI were sepsis (30.6%), followed by poisoning (21.3%), snake bite (19.3%), diarrheal disease (12.7%), dengue (8%), pancreatitis (3.3%), scrub typhus (1.3%), and post-renal causes (0.6%), with wasp bite accounting for 0.6%. Similar findings were reported by Bagshaw *et al*.,[23] who emphasized sepsis as a key contributor to AKI in critically ill patients. In another study by Bhudhrani D *et al*,[17] pre-renal causes, such as acute gastroenteritis (33%) and hepatic causes (20%), were

prominent, highlighting the significance of fluid loss and circulatory disturbances in precipitating renal impairment; intrinsic renal causes, including snake bites (25%) and nephrotoxic drug exposure (23%), indicated substantial nephrotoxicity as a leading cause of AKI in the region, while post-renal causes, primarily benign prostate hypertrophy (44%), emphasized the importance of urinary tract obstruction as another significant contributor to AKI cases. In another study by Eswarappa M *et al*,[18] sepsis was the most common cause, accounting for 38.6% of cases, followed by acute gastroenteritis (10.4%) and obstetric causes (8.4%); cardiac causes (6.8%), hepatic causes (6.0%), surgical causes (6.0%), and tropical infections such as malaria, dengue, and leptospirosis (6.4%) were other important contributors, while drug-induced AKI constituted 5.6% of cases and less frequent causes included snake bite (2.6%), rapidly progressive glomerulonephritis (1.8%), multiple myeloma (1.6%), H1N1 influenza (1.2%), and poisoning (1.0%). A recent Indian study by Singh *et al*.[24] found that nephrotoxic drugs were the most common cause of AKI in the medical ward. Shah *et al*.[25] found urogenital infection to be the most common source of sepsis in patients who developed AKI. In a study by Kashinkunti M *et al*,[20] the etiology of ARF was multifactorial and included sepsis (51.3%), hypotension (33.7%) and volume depletion (18.9%).

Outcome

Eighty-two per cent of patients were discharged after recovering while 18% of patients expired during their stay in hospital. In another study by Eswarappa M *et al*,[18] 37.6% of patients expired. In a study by Kashinkunti M *et al*,[20] mortality occurred in 28.3% of patients. In a study by Suresh V *et al*,[21] mortality was 9%. In a study by Kumar S *et al*,[26] overall mortality was 29.2%.

Association Between Biochemical Indices and Outcome

In the present study, a comparative analysis of biochemical indices based on clinical outcome demonstrated significantly worse renal parameters among patients who expired compared with those who were discharged. The mean blood urea level was markedly higher in the expired group (104.08 ± 8.65 mg/dL) than in the discharged group (93.72 ± 5.31 mg/dL), with the difference being statistically significant ($p = 0.001$). Similarly, mean serum creatinine levels were significantly elevated among patients who expired (4.40 ± 0.49 mg/dL) compared to those who were discharged (2.58 ± 0.68 mg/dL) ($p = 0.001$). Conversely, the average estimated glomerular filtration rate (eGFR) was significantly lower in the expired group (37.29 ± 4.40 ml/min/1.73 m²) as compared to the discharged group (42.86 ± 7.77 ml/min/1.73 m²), and this difference was also statistically significant ($p = 0.004$). In a study by Saxena A *et al*,[27] patients who did not survive had significantly worse kidney function and metabolic status compared to those who survived. Both serum creatinine (at baseline and follow-up) and blood urea levels were significantly higher among nonsurvivors, indicating more severe renal impairment in this group. Similarly, serum bicarbonate levels were significantly lower in nonsurvivors, suggesting a greater degree of metabolic acidosis. These differences were statistically significant, highlighting their association with poor outcome. In contrast, haemoglobin and serum sodium levels were comparable between survivors and nonsurvivors, with no statistically significant difference, indicating that anaemia and sodium levels were not major determinants of mortality in that study.

Association Between Sepsis and Outcome

In the present study, the comparison between sepsis and clinical outcome revealed a significant association between the presence of sepsis and mortality. Among patients who expired, 77.8% were diagnosed with sepsis, whereas only 39% of patients who were discharged had sepsis. This difference was statistically significant, indicating a strong association between sepsis and adverse outcome ($p = 0.003$). In a study by Jia H *et al*,[28] sepsis significantly increased attributable hospital mortality in AKI, with an attributable mortality of sepsis for AKI of 9.1% (95% CI 4.8–13.3%). In yet another study by Wang D *et al*,[29] AKI in sepsis was independently associated with 30-day mortality; stage 3 AKI was an independent predictor of death, with a 30-day attributable mortality of 6.6%.

Association Between Oliguria and Outcome

In the present study, the comparison between oliguria and clinical outcome demonstrated a strong and statistically significant association. All patients who expired (100%) had oliguria, whereas 59.8% of patients who were discharged exhibited oliguria. This marked difference underscores the significant association between oliguria and adverse outcome, which was statistically significant ($p = 0.001$). In a study by Macedo E *et al*,[30] oliguric patients (even without creatinine rise) had significantly higher ICU mortality (8.8%).

Limitations

- The study was conducted at a single tertiary care centre, which may limit the generalizability of the findings to other settings or populations.
- The relatively small sample size may reduce the statistical power to detect weaker associations and limits subgroup analysis.
- Being an observational study, causal relationships between risk factors and outcomes could not be established.
- Biomarkers beyond routine laboratory parameters were not evaluated, which may have provided additional prognostic insight.

Conclusion

This study demonstrates that acute kidney injury predominantly affects older adults, with a male predominance and a high burden of comorbidities such as diabetes, hypertension, and coronary artery disease. Sepsis and oliguria were common and showed a strong, statistically significant association with mortality, identifying them as key clinical predictors of poor outcome in AKI patients.

Patients who expired had significantly higher serum urea and creatinine levels and lower eGFR, reflecting more severe renal dysfunction and underscoring the prognostic importance of biochemical markers of kidney injury. Despite treatment, a considerable proportion of patients experienced in-hospital mortality, highlighting the serious nature of AKI. Overall, the findings emphasize that early identification of high-risk patients—particularly those with sepsis, oliguria, and markedly deranged renal parameters—along with prompt and aggressive management, is essential to improve outcomes and reduce mortality in acute kidney injury.

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