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CLINICAL SEVERITY AND PROGRESSION OF ACUTE KIDNEY INJURY IN HOSPITALIZED PATIENTS

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Abstract

Introduction: Acute Kidney Injury (AKI) represents a significant clinical condition in hospitalized patients, characterized by abrupt renal function decline. AKI is associated with considerable morbidity, mortality, and longterm risk of chronic kidney disease (CKD). Its incidence is rising due to aging populations, comorbidities, and exposure to nephrotoxic agents, particularly in intensive care settings.

Aim and Objective: This study aimed to evaluate the clinical severity and progression of AKI among hospitalized patients. Specific objectives included assessing AKI staging, identifying risk factors, analyzing etiological patterns, and determining the need for dialysis and related outcomes.

Materials & Methods: This prospective observational study was conducted over 18 months in the Department of General Medicine at Shivamogga Institute of Medical Sciences. One hundred adult inpatients diagnosed with AKI, per KDIGO criteria, were enrolled. Data on demographics, comorbidities, laboratory findings, AKI staging, and outcomes were collected. Patients were followed during hospitalization for disease progression, dialysis needs, and clinical outcomes. Statistical analysis was performed using SPSS.

Results: The most affected age group was 45–60 years (42%). Males constituted 65% of cases. Comorbidities included diabetes (6%), hypertension (8%), and chronic liver disease (10%). Most patients (69%) presented with Stage 3 AKI. Sepsis (26%) was the leading cause, followed by snake bite (14%) and gastrointestinal conditions (12%). Dialysis was required in 48% of patients, mainly due to combined metabolic acidosis and anuria (56.25%). Inhospital dialysis was common, with a subset requiring continued therapy post-discharge.

Conclusion: AKI in hospitalized patients exhibits a high severity spectrum, frequently requiring dialysis and intensive care. Early recognition, risk stratification, and targeted management are critical to improving outcomes and reducing progression to CKD.

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Keywords: Acute Kidney Injury; KDIGO; Sepsis; Dialysis; Hospitalized Patients;

INTRODUCTION

Acute Kidney Injury (AKI) is a frequent and serious clinical condition that arises from a rapid decline in renal function, typically indicated by an increase in serum creatinine and/or a decrease in urine output. It commonly develops in hospitalized patients and is associated with adverse short- and long-term outcomes. The burden of AKI is particularly high in critical care settings, where its presence significantly increases the risk of mortality, length of hospital stay, and progression to chronic kidney disease (CKD). The clinical course of AKI varies considerably, depending on its underlying causes, severity at presentation, associated comorbidities, and the timeliness of interventions. Understanding the trajectory and clinical severity of AKI in hospitalized individuals is essential for developing effective management strategies, improving prognostication, and reducing the risk of irreversible kidney damage (1).

Over recent decades, the incidence of AKI has increased globally, due in part to a combination of factors such as rising life expectancy, greater prevalence of comorbid illnesses, and the widespread use of nephrotoxic medications and contrast agents. Current data suggest that AKI affects between 10% and 20% of all hospitalized patients, with rates exceeding 50% in intensive care units (ICUs). This rise has prompted greater efforts to refine diagnostic criteria, improve early recognition, and stratify risk in vulnerable populations. The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines have become the most widely used framework for diagnosing and staging AKI, classifying it into three stages based on changes in serum creatinine and urine output. These stages help in predicting the severity of the condition and guiding clinical management (2).

AKI can result from a range of mechanisms, traditionally grouped into prerenal, intrinsic, and postrenal causes. Prerenal AKI is usually due to reduced renal perfusion without structural kidney damage and may be reversible if promptly corrected. Intrinsic AKI, such as acute tubular injury or necrosis, reflects direct injury to renal tissues often from ischemia, toxins, or inflammation. Postrenal AKI arises from obstruction to urine flow and is less frequent but can rapidly impair kidney function if not addressed. These pathophysiological mechanisms are not mutually exclusive and often coexist, especially in complex hospitalized patients. Moreover, systemic conditions like sepsis, heart failure, liver dysfunction, and surgical stress are frequent contributors to the development and progression of AKI(3).

The severity of AKI is not solely determined by peak serum creatinine levels or oliguria but also by the duration and pattern of renal dysfunction. Evidence suggests that persistent AKI— where renal function remains impaired for several days—carries a significantly worse prognosis compared to transient AKI that resolves quickly. Similarly, non-oliguric AKI may be overlooked initially, despite carrying similar risks for complications. The identification of AKI subtypes, such as subclinical AKI detected via biomarkers before changes in serum creatinine become apparent, has further refined the understanding of disease progression. These evolving concepts highlight the need for more sensitive diagnostic tools and personalized treatment approaches (4).

Multiple factors influence the clinical severity and evolution of AKI in hospitalized settings. Elderly patients, individuals with diabetes mellitus, pre-existing CKD, and those undergoing major surgeries are at heightened risk. ICU patients are particularly vulnerable due to frequent exposure to hemodynamic instability, vasopressors, mechanical ventilation, and

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systemic infections. Postoperative AKI is common after major procedures, especially cardiac and abdominal surgeries, and is often associated with worse postoperative outcomes. In medical patients, nephrotoxic drugs, dehydration, and infections remain prominent etiological factors. Once AKI sets in, it can perpetuate a cycle of systemic inflammation and organ dysfunction, thereby compounding clinical severity (5).

Timely identification of patients at risk for severe or progressive AKI remains a cornerstone of effective management. Predictive risk models incorporating clinical, laboratory, and hemodynamic parameters have been developed, although their widespread clinical application remains limited. In recent years, kidney injury biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), interleukin-18 (IL-18), and kidney injury molecule-1 (KIM-1) have shown promise for early detection and risk stratification. These markers can provide insight into structural kidney injury even before functional decline becomes apparent. However, further validation is needed before they can be routinely incorporated into clinical protocols (6).

From a therapeutic standpoint, management of AKI primarily involves supportive care and elimination of the precipitating factors. Adequate volume status, avoidance of nephrotoxins, and hemodynamic optimization are essential initial steps. In severe cases, especially when complications like fluid overload, acidosis, or uremia develop, renal replacement therapy (RRT) becomes necessary. The timing and modality of RRT in AKI remain areas of ongoing research, with debates continuing over early versus delayed initiation and the choice between continuous and intermittent dialysis (7).

Despite improved diagnostics and supportive care, acute kidney injury (AKI) in hospitalized patients continues to carry a guarded prognosis. Mortality remains high, especially in ICU patients with Stage 3 AKI requiring dialysis, where it can exceed 50%. Survivors often face incomplete renal recovery and are at risk of progressing to chronic kidney disease or end-stage renal disease. AKI also independently increases the risk of cardiovascular events, contributing to long-term morbidity and healthcare costs. Its clinical course is shaped by multiple factors, underscoring the need for early detection, personalized treatment, structured follow-up, and integration into long-term nephrology care to improve outcomes (8).

The aim of this study is to assess the clinical severity and progression of Acute Kidney Injury (AKI) in hospitalized patients. Objectives include evaluating AKI incidence and staging, identifying associated risk factors and comorbidities, analyzing patterns of progression, determining short- and longterm outcomes such as mortality and chronic kidney disease, and exploring the role of biomarkers and clinical indicators in predicting AKI severity to improve early detection, management, and patient outcomes.

MATERIALS AND METHODS

This prospective observational study was conducted over 18 months in the Department of General Medicine at Shivamogga Institute of Medical Sciences. One hundred adult inpatients diagnosed with Acute Kidney Injury (AKI) per KDIGO criteria were enrolled consecutively. Data on demographics, comorbidities, clinical signs, biochemical parameters, and AKI staging were collected at admission. AKI severity was assessed using KDIGO staging, and the need for dialysis or inotropic support was recorded. Patients were monitored during hospitalization for progression, clinical outcomes, and need for renal replacement therapy. Descriptive statistics were applied using SPSS to analyze the severity and acute progression of AKI.

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RESULTS

Table-1: Comparison between distributions according to Age

Age	No. of cases	Percentage
18-30	12	12.00%
30-45	36	36.00%
45-60	42	42.00%
>60	10	10.00%

The age-wise distribution of cases, with the highest incidence (42%) observed in the 45–60 age group, followed by 36% in the 30–45 age group. Younger adults aged 18–30 accounted for 12% of cases, while those above 60 years represented only 10%. This suggests that middle-aged individuals are the most affected population in this study.

Table 2: Demographics, Clinical History & Physical Examination

Category	Parameter	Value
Demographics	Age (mean ± SD)	44.52 ± 11.3 years
	Gender	Female: 35 (35.00%) Male: 65 (65.00%)
Medical Conditions	Chronic Liver Disease	10 (10.00%)
	Chronic Obstructive Pulmonary Disease	4 (4.00%)
	Diabetes Mellitus	6 (6.00%)
	Hypertension	8 (8.00%)
	Diabetes + Hypertension	12 (12.00%)
	Heart Failure	4 (4.00%)
	Hypothyroid	1 (1.00%)
	No co-morbidities	55 (55.00%)
Drug History	NSAIDs	6 (6.00%)
	ACEIs/ARBs	2 (2.00%)
	Herbal/Native	1 (1.00%)
	No Medications	91 (91.00%)

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Physical Findings	Hypotension	28 (28.00%)
	Edema	11 (11.00%)
	Jaundice	3 (3.00%)
	Pallor	2 (2.00%)
	Raised JVP	1 (1.00%)
	Oliguria	1 (1.00%)
	Anuria	1 (1.00%)

The baseline characteristics of study participants, showing an average age of 44.52 years with a male predominance (65%). Common medical conditions include diabetes, hypertension, and chronic liver disease. Most had no medication history or comorbidities. Physical findings frequently observed were hypotension, edema, and jaundice, indicating varied clinical presentations.

Table 3: Laboratory Findings, AKI Stage & Support Needs

Category	Parameter	Value
On Admission	Mean Hemoglobin (g/dL)	12.75 ± 1.6
	Mean TLC	18,500 ± 9030
	Mean Serum Creatinine (mg/dL)	3.9 ± 1.79
Urine Analysis	Urine Spot PCR - Normal	38
	Urine Spot PCR - 0.1 to 3	36
	Urine Spot PCR - >3	24
	RBCs in Sediment	14
	WBCs in Sediment	10
AKI Staging	Stage 1	9 (9.00%)
	Stage 2	22 (22.00%)
	Stage 3	69 (69.00%)
Support Needs	Ventilation - Yes	13 (13.00%)
	Ventilation - No	87 (87.00%)
	Inotropes - Yes	23

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	(23.00%)
Inotropes - No	77 (77.00%)

The clinical and laboratory data at admission, with elevated TLC and serum creatinine indicating infection and renal dysfunction. Most patients had Stage 3 AKI (69%). Urine analysis varied, with many showings' proteinuria. A small proportion required ventilation (13%) and inotropes (23%), reflecting varying degrees of critical illness.

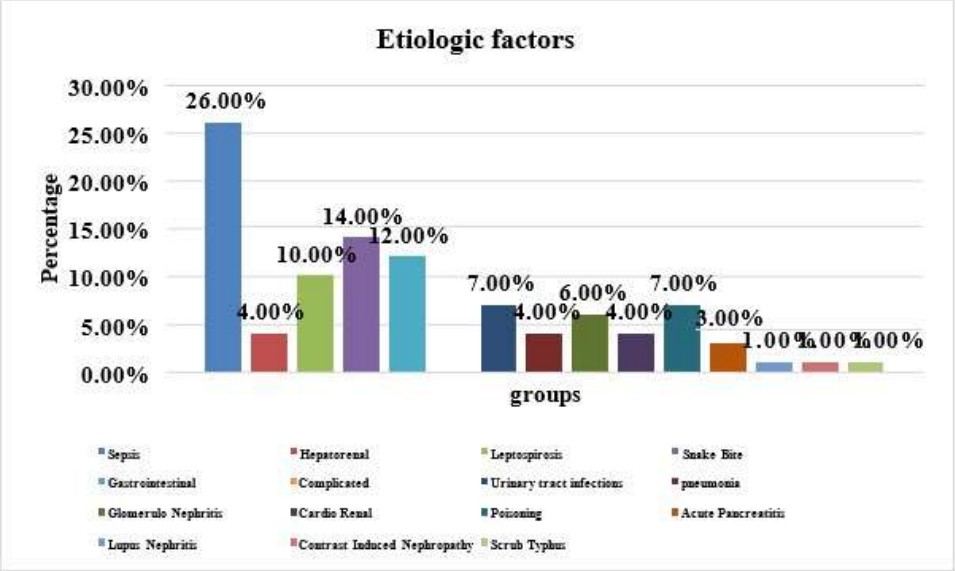

Figure 1: Distribution according to etiological factor

The sepsis as the leading etiologic factor for AKI, accounting for 26% of cases. Snake bite (14%), gastrointestinal causes (12%), and leptospirosis (10%) also contributed significantly. Other notable causes included complicated urinary tract infections and poisoning (7% each), and glomerulonephritis (6%). Less common etiologies were lupus nephritis, contrastinduced nephropathy, and scrub typhus (1% each), indicating a diverse spectrum of triggers with infections and envenomation being predominant contributors.

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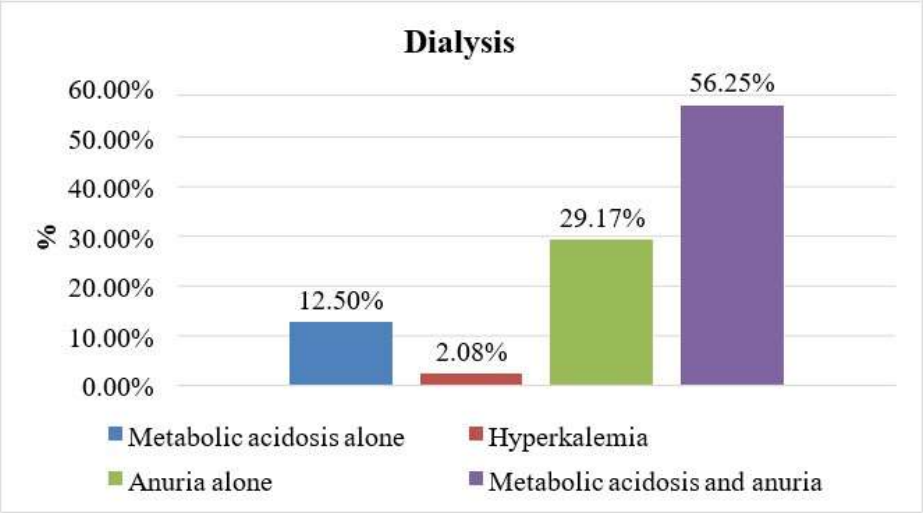

Figure 2: Distribution according to indication for dialysis

Dialysis was mainly required due to the combined presence of metabolic acidosis and anuria (56.25%). Anuria alone accounted for 29.17% of cases, while metabolic acidosis alone was seen in 12.50%. Hyperkalemia was the least common indication, observed in only 2.08% of patients, highlighting the predominance of severe renal dysfunction as the primary trigger for dialysis.

Table 4: Distribution according to Management of AKI

Management of AKI	No. of cases	Percentage
Hemodialysis	48	48.00%
Medical Management	52	52.00%

The management approach for Acute Kidney Injury (AKI), with 52% of patients managed conservatively through medical treatment, while 48% required hemodialysis. This near-equal distribution highlights that while many patients could be stabilized without dialysis, a significant proportion still progressed to severe renal dysfunction necessitating renal replacement therapy, underscoring the variable severity and clinical course of AKI in the studied population.

DISCUSSION

In the present study, the 45–60 age group was most affected by AKI (42%), followed by 18–30 years (12%). NS Deshpande et al. (9) also reported higher cases in the 50–60 age group (15.88%) and the least in 13–20 years (4.7%). Conversely, Ibrahim A et al. (10) found 42.4% of cases in the 18–30 age group. Older adults (≥65 years) represented 9.76% in this study, supporting findings from Igiraneza G et al. (11) linking aging with increased AKI risk.

In the present study, males comprised 65% and females 35%, closely matching Abebe et al. (12) (60% males), but differing from Singh D et al. (13) (52.7% males). Chronic liver disease was found in 10%, similar to Bhattacharya PK et al. (14) (10.67%) and Chetlapalli AK et al. (15) (13%). COPD was 4% here, compared to 13% and 5.33%, respectively. Diabetes with

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hypertension affected 12%, versus 6.67% in Bhattacharya. NSAID use (6%) was higher than Mathew et al. (16) (2.7%), with ACEIs/ARBs (2%) and herbal drugs (1%) being comparable. Medication-free patients were 91%, lower than Mathew et al. (93.3%) but higher than Jang SM et al. (17) (45%), highlighting demographic and clinical variability across studies.

In the present study, hypotension (28%) and edema (11%) were slightly lower than Bhattacharya PK et al. (14) (38.67% and 14.67%). Jaundice (3%) and pallor (2%) were also lower than 4% and 2.67% reported by Bhattacharya. Oliguria was significantly less (1%) compared to 25.3% in their study. Mean hemoglobin was 12.75 ± 1.6 g/dL, higher than 9.25 ± 2.46 g/dL in Abinet et al. (12), while mean TLC was 18,500 vs. 11.22 $\times 10^3$ in their findings. Serum creatinine averaged 3.9 ± 1.79 mg/dL, aligning with Schmidt EA et al. (18) (6.5 ± 5.1 mg/dL). Urine PCR patterns and sediment findings (RBCs and WBCs) were consistent with Nayak R et al. (19), who reported a spot PCR of 2.99 ± 2.71 mg/dL, highlighting proteinuria variability and renal impairment severity.

In the present study, Stage 3 AKI was most common (69%), closely aligning with 80% in S. Pawar et al. (20), while Stage 2 affected 22%, lower than 46.7% in Prasad G et al. (21). Stage 1 was least reported (9%) compared to 2% in S. Pawar et al. (20) and 20% in Prasad G et al. (21). Ventilation was required in 13% of patients here, higher than 5% in Sharanabasappa et al., suggesting more severe cases. Inotrope use was 23%, lower than 30% in Sharanabasappa et al. (22) and significantly less than in Chetlapalli AK et al. (15), who reported use in 109 patients, highlighting differences in treatment needs and patient severity across studies.

In the present study, sepsis was the leading cause of AKI, affecting 26% of participants—higher than 10% in Ananth PV et al. (23), close to 27.1% in Pillai VSN et al. (24), and above 17.3% in Mathew et al. [79]. Leptospirosis accounted for 10%, compared to 4% (Ananth PV et al. (15)), 7.1% (Pillai VSN et al.), and 21.3% (Mathew et al.). Snake bites were noted in 14%, higher than Ananth PV et al. (5%) and Mathew et al.(16) (0.7%), but closer to Pillai VSN et al. [85] (7.1%). Gastrointestinal causes (12%) and poisoning (7%) showed moderate variations. Igiraneza G et al. (12) reported pneumonia and sepsis equally at 3.7%, with hypertension (41.5%), diabetes (20.4%), and CKD (7.3%) as major noncommunicable contributors. These differences highlight the influence of geography, healthcare access, and diagnostic practices on AKI etiology in various studies.

The most common indication for dialysis in both studies was the combination of metabolic acidosis and anuria, seen in 56.25% of cases in the present study, closely aligning with 52.9% in Mathew et al., highlighting the critical role of these factors in initiating dialysis. Metabolic acidosis alone was the second most frequent cause, affecting 12.50% of patients in this study and 13.2% in Mekha et al., showing comparable prevalence. Anuria alone was reported in 29.17% of cases in the current study, slightly lower than 32.4% in Mekha et al. Hyperkalemia was the least common reason, affecting 2.08% of patients here versus 1.5% in Mekha et al. (16).

CONCLUSION

This study highlights the significant clinical burden and varied etiologies of acute kidney injury (AKI) in hospitalized patients, with sepsis emerging as the most common cause. A high prevalence of Stage 3 AKI, frequent dialysis requirement, and substantial morbidity underscore the need for early detection and aggressive management. Middle-aged males were predominantly affected, with hypotension and comorbidities contributing to severity. Despite supportive care, outcomes remained guarded, particularly in critically ill patients. These

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findings emphasize the importance of prompt risk stratification, use of biomarkers, and individualized care strategies to reduce AKI progression, enhance recovery, and improve both short- and long-term patient outcomes.

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