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Clinical Profile, Etiological Spectrum, Kidney Disease: Improving Global Outcomes Staging, and Predictors of Outcome in Acute Kidney Injury: A Prospective Study from a Medical Intensive Care Unit in Central India

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Abstract

Background: Acute kidney injury (AKI) in critically ill patients carries high morbidity and mortality. Regional variations in etiology and outcomes remain underreported in central India. This study evaluated the etiological causes, Kidney Disease: Improving Global Outcomes (KDIGO) stages, clinical outcomes, and prognostic determinants of AKI in a medical intensive care unit (MICU). **Materials and Methods:** A prospective observational study was conducted on 120 adult patients with AKI admitted to the MICU of a tertiary care teaching hospital over 21 months (June 2024–February 2026). AKI was staged according to KDIGO criteria. Patients were monitored throughout their hospital stay and followed for 3 months to evaluate renal recovery, progression to chronic kidney disease (CKD), and in-hospital mortality. **Results:** Among 120 patients, 53.3% (n = 64) were male. The modal age group was 18–30 years (25.0%), followed by 31–40 and 51–60 years (17.5% each). Stage III AKI predominated at presentation (63.3%, n = 76), whereas Stage II and Stage I accounted for 31.7% (n = 38) and 5.0% (n = 6), respectively. Intrinsic renal causes constituted 59.1% (n = 71), pre-renal causes 27.5% (n = 33), and post-renal obstructive causes 13.3% (n = 16). Sepsis was the leading individual etiology (43.3%, n = 52), driven primarily by urinary tract infections (13.3%), and pneumonia (9.1%). Overall, 57.5% (n = 69) achieved complete renal recovery, 17.5% (n = 21) developed CKD (11.7% conservative, 5.8% hemodialysis-dependent), and 25.0% (n = 30) died. Pre-renal AKI showed the highest resolution rate (90.9%, n = 30/33), whereas post-renal AKI had the highest mortality (50.0%). Hypertension (P = 0.042), diabetes mellitus (P = 0.001), advancing age (P < 0.05), and requirement of renal replacement therapy (RRT; mortality 80.6%, P = 0.001) were statistically significant predictors of poor outcome. **Conclusion:** AKI in central Indian MICUs presents predominantly in advanced stages (Stage III), with sepsis as the leading etiology. While younger patients and pre-renal AKI demonstrate favorable recovery rates, elderly patients, pre-existing metabolic comorbidities, and the requirement for RRT independently predict adverse outcomes and high mortality.

Keywords: Acute kidney injury, Central India, Kidney disease: Improving global outcomes criteria, Medical intensive care unit, Mortality predictors, Renal replacement therapy, Sepsis

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NTRODUCTION

Acute kidney injury (AKI) represents one of the most critical and challenging syndromes encountered in modern critical care nephrology. Characterized by an abrupt decline in glomerular filtration rate, accumulation of nitrogenous waste products (blood urea nitrogen and serum creatinine), and failure of fluid, electrolyte, and acid-base homeostasis, AKI exerts profound multiorgan systemic effects. Historically termed acute renal failure, the evolution of consensus definitions – from the RIFLE criteria (2004) and AKI network (2007) to the unified Kidney Disease: Improving Global Outcomes (KDIGO) criteria (2012) – has underscored that renal impairment exists across a wide spectrum of injury, from subclinical tubular damage to total functional failure.[1-3]

Globally, AKI affects an estimated 13.3 million individuals annually, with approximately 85% of cases occurring in low- and middle-income countries. In intensive care units (ICUs), the incidence ranges between 20% and 67%, carrying crude mortality rates exceeding 50% among patients who require renal replacement therapy (RRT). Critically ill patients are subject to multiple simultaneous insults, including severe systemic hypotension, invasive mechanical ventilation, multidrug nephrotoxicity, and sepsis-induced microvascular dysfunction.

Despite this enormous global burden, critical epidemiological gaps persist regarding AKI within central India. While significant data have emerged from metropolitan tertiary centers in southern and northern India, the central Indian demographic (encompassing Madhya Pradesh and contiguous regions) exhibits distinct geographical, socioeconomic, and etiological features. These include a high agrarian population density, frequent exposure to acute agricultural toxins (such as organophosphates and aluminum phosphide), seasonal infectious diseases, and widespread referral delay from rural primary health centers to urban tertiary ICUs.[4-6] This prospective study was therefore

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conducted to evaluate the etiological profile, KDIGO staging distribution, clinical outcomes, and independent predictors of mortality and progression to chronic kidney disease (CKD) in a tertiary care medical ICU in central India.

Materials and Methods

Study Design, Setting, and Duration

This study was designed as a prospective observational clinical investigation carried out in the Department of General Medicine and the associated Medical ICU (MICU) at L.N. Medical College and Research Centre and J.K. Hospital, Bhopal, Madhya Pradesh, India. The study spanned 21 months, from June 2024 to February 2026.

Selection Criteria

Adult patients aged ≥18 years admitted to the MICU who met the standardized KDIGO definition for AKI were consecutively enrolled. Patients aged <18 years, those with documented pre-existing (CKD stage 3b–5 or baseline serum creatinine [sCr] >2.0 mg/dL prior to current acute illness), and patients or legally authorized representatives unwilling to participate in follow-up were excluded.

Diagnostic and Staging Criteria (KDIGO)

AKI diagnosis and severity staging were established strictly according to the KDIGO clinical practice guidelines:

- Stage 1: sCr 1.5–1.9 times baseline OR absolute increase ≥0.3 mg/dL within 48 h; or urine volume <0.5 mL/kg/h for 6–12 h.
- Stage 2: sCr 2.0–2.9 times baseline; or urine volume <0.5 mL/kg/h for ≥12 h.
- Stage 3: sCr 3.0 times baseline OR increase in sCr to ≥4.0 mg/dL OR initiation of RRT; or urine volume <0.3 mL/kg/h for ≥24 h or anuria for ≥12 h.[7-9]

Clinical Workup, Data Management, and Follow-up

A standardized proforma captured demographic information, detailed clinical history, comorbid states (hypertension [HTN], diabetes mellitus [DM], hypothyroidism), vital signs, and physical examination findings. Baseline and serial laboratory investigations included complete blood counts, comprehensive renal and liver function panels, serum electrolytes (Na^+, K^+, Ca^{2+}, PO_4^{3-}), arterial blood gases, inflammatory biomarkers (erythrocyte sedimentation rate, C-reactive protein), urinalysis, and ultrasonography of the kidneys, ureters, and bladder (ultrasound of the kidneys, ureters, and bladder) to assess renal dimensions, echogenicity, corticomedullary differentiation, and obstructive uropathy. All patients were closely monitored throughout their ICU and hospital course. Clinical outcomes – classified as complete renal recovery, CKD on conservative management, CKD requiring maintenance RRT (hemodialysis), or in-hospital death – were recorded at discharge and verified at 3-month follow-up.[10-12]

Statistical Analysis

Data were systematically curated in Microsoft Excel and analyzed using IBM Statistical Package for the Social Sciences Statistics version 29.0 (IBM Corp., Armonk, NY). Categorical variables were presented as absolute numbers (*n*) and percentages (%). Continuous variables were expressed as mean ± standard deviation. Qualitative associations between categorical variables (e.g., comorbidity vs. outcome, RRT vs. mortality, etiology vs. stage) were tested using the Pearson Chi-square (χ^2) test or Fisher's exact test as appropriate. A two-tailed $P < 0.05$ was considered statistically significant.[13-16]

Ethical Approval

The study protocol received ethical clearance from the Institutional Ethics Committee of L.N. Medical College and Research Centre, Bhopal. Written informed consent was obtained from all patients or their legal surrogates prior to study enrollment in full compliance with the Declaration of Helsinki.

Results

Baseline Demographics and Comorbidity Profile

A total of 120 adult patients with AKI admitted to the MICU were evaluated. The cohort comprised 64 males (53.3%) and 56 females (46.7%), demonstrating a slight male predominance. The age distribution demonstrated a notable peak in young adults aged 18–30 years (25.0%, *n* = 30), followed by 31–40 years (17.5%, *n* = 21) and 51–60 years (17.5%, *n* = 21). Regarding comorbidities, 37.5% of patients (*n* = 45) were previously healthy with no documented medical conditions. HTN was the most common chronic comorbidity (24.2%, *n* = 29), followed closely by DM (23.3%, *n* = 28). Both diabetes and HTN coexisted in 14.2% (*n* = 17), whereas hypothyroidism was present in 15.0% (*n* = 18).

KDIGO Staging at Presentation

A key finding was that the vast majority of patients presented with advanced renal injury upon MICU admission. Stage III AKI was diagnosed in 63.3% of patients (*n* = 76), whereas Stage II AKI accounted for 31.7% (*n* = 38). Only 5.0% of patients (*n* = 6) presented in Stage I. Furthermore, BUN/creatinine ratio analysis revealed that 59.2% (n = 71) had a ratio <10:1 (characteristic of intrinsic renal AKI), 27.5% (n = 33) had a ratio >20:1 (pre-renal azotemia), and 13.3% (n = 16) exhibited a ratio between 10:1 and 20:1 (normal or post-renal obstruction).

Etiological Spectrum of AKI

Intrinsic renal parenchymal disease constituted the primary etiological class (59.1%, *n* = 71), followed by pre-renal causes (27.5%, *n* = 33) and post-renal obstructive uropathy (13.3%, *n* = 16). Among intrinsic causes, sepsis-associated AKI predominated overwhelmingly (43.3% of the total cohort, *n* = 52). The leading infectious sources were urinary tract infections (13.3%, *n* = 16), pneumonia (9.1%, *n* = 11), acute severe pancreatitis (8.4%, *n* = 10), liver abscesses (5.0%, *n* = 6), soft-tissue infections (4.2%, *n* = 5), and obstetric/other infections (3.3%, *n* = 4). Toxic acute tubular necrosis (ATN) secondary to chemical poisonings (aluminum phosphide/celphos and organophosphorus compounds) constituted 7.5% (*n* = 9), whereas hepatorenal syndrome accounted for 8.4% (*n* = 10). Pre-renal AKI was driven by acute

gastroenteritis-induced dehydration (13.3%, *n* = 16) and acute decompensated heart failure/cardiogenic shock (14.2%, *n* = 17). Post-renal obstructive AKI was attributed to advanced pelvic/abdominal malignancies (10.0%, *n* = 12) and obstructive bilateral/solitary nephrolithiasis (3.3%, *n* = 4) (Table 1).

Clinical Outcomes and Prognostic Determinants

Overall clinical outcome evaluation at 3 months revealed that 57.5% of patients (*n* = 69) achieved complete renal resolution with normal sCr. Progression to CKD occurred in 17.5% (*n* = 21), including 11.7% (*n* = 14) who were managed conservatively and 5.8% (*n* = 7) who remained dependent on maintenance hemodialysis. The overall in-hospital mortality rate was 25.0% (*n* = 30) (Tables 2 and 3).

Table 1: Baseline demographic and clinical characteristics of the study cohort (*n*=120)

Parameter	*Subcategory*	*Patient count (n)*	*Percentage*
Age group (Years)	18–30	30	25.0
	31–40	21	17.5
	41–50	16	13.3
	51–60	21	17.5
	61–70	19	15.8
	>70	13	10.9
Gender	Male	64	53.3
	Female	56	46.7
Comorbidities	None (Previously healthy)	45	37.5
	HTN	29	24.2
	DM	28	23.3
	Coexisting DM+HTN	17	14.2
	Hypothyroidism	18	15.0

HTN: Hypertension, DM: Diabetes mellitus

Etiological determinants

Pre-renal AKI carried an excellent prognosis, with 90.9% (*n* = 30/33) achieving complete resolution and 0% mortality. Intrinsic renal AKI resulted in 62.0% resolution (*n* = 44/71) and 25.4% mortality (*n* = 16/71). In sharp contrast, post-renal obstructive AKI was associated with the poorest outcomes: only 6.3% (*n* = 1/16) recovered completely, 31.2% (*n* = 5/16) progressed to CKD, and 50.0% (*n* = 8/16) died ($P < 0.001$).

Age determinants

Complete resolution was achieved in 83.3% of young patients aged 18–30 years, with a low mortality of 6.7%. Conversely, patients aged >70 years experienced a mortality rate of 46.1% and a 38.4% rate of progression to hemodialysis-dependent CKD.

Comorbidity impact

HTN was significantly associated with adverse outcomes ($P = 0.042$); only 37.9% of hypertensive patients achieved complete resolution compared to 63.7% without HTN. DM exhibited a highly significant correlation with adverse outcomes ($P = 0.001$): 65.2% of non-diabetics achieved complete resolution versus only 32.1% of diabetics, with 85.7% (*n* = 6/7) of all dialysis-dependent CKD cases occurring in diabetics. Hypothyroidism was not statistically significant ($P = 0.154$).

RRT

Of the 120 patients, 36 (30.0%) required RRT (all originating from KDIGO Stage III). The requirement for RRT was an extremely strong predictor of fatal outcome ($P = 0.001$): mortality among dialyzed

Table 2: Comprehensive etiological spectrum of acute kidney injury in the medical intensive care unit (*n*=120)

Broad class	*Specific etiological category*	*Patient count (n)*	*Subgroup (%)*
Pre-renal (*n*=33, 27.5%)	Acute gastroenteritis (Dehydration/Hypovolemia)	16	13.3
	Acute heart failure/Cardiogenic compromise	17	14.2
Intrinsic renal (*n*=71, 59.1%)	Sepsis: Urinary tract infection	16	13.3
	Sepsis: Pneumonia/Severe respiratory infection	11	9.1
	Sepsis: Acute pancreatitis	10	8.4
	Sepsis: Liver abscess	6	5.0
	Sepsis: Soft-tissue infection	5	4.2
	Sepsis: Meningoencephalitis/Pelvic inflammatory disease/Septic abortion	4	3.3
	Toxic acute tubular necrosis (Celphos/Organophosphate ingestion)	9	7.5
	Hepatorenal syndrome in decompensated cirrhosis	10	8.4
Post-renal (*n*=16, 13.3%)	Obstructive uropathy: Malignancy (Cervical/Prostate/Bladder)	12	10.0
	Obstructive uropathy: Urolithiasis/Renal calculi	4	3.3
Total cohort	All combined etiologies	120	100.0

Table 3: Cross-tabulation of comorbidities, staging, and RRT with AKI outcomes (*n*=120)

Parameter	*Category*	*Complete recovery (%)*	*CKD conservative (%)*	*CKD+RRT (%)*	*Death (%)*	*Total (n)*	*P-value*
Hypertension	Absent	58 (63.7)	10 (11.0)	3 (3.3)	20 (22.0)	91	0.042*
	Present	11 (37.9)	4 (13.8)	4 (13.8)	10 (34.5)	29	
Diabetes mellitus	Absent	60 (65.2)	11 (12.0)	1 (1.1)	20 (21.7)	92	0.001*
	Present	9 (32.1)	3 (10.7)	6 (21.4)	10 (35.7)	28	
Hypothyroidism	Absent	63 (61.8)	11 (10.8)	5 (4.9)	23 (22.5)	102	0.154
	Present	6 (33.3)	3 (16.7)	2 (11.1)	7 (38.9)	18	
RRT requirement	No	69 (82.1)	14 (16.7)	0 (0.0)	1 (1.2)	84	0.001*
	Yes	0 (0.0)	0 (0.0)	7 (19.4)	29 (80.6)	36	
Overall total	All patients	69 (57.5)	14 (11.7)	7 (5.8)	30 (25.0)	120	—

RRT: Renal replacement therapy, AKI: Acute kidney injury, CKD: Chronic kidney disease. *Statistically significant at $P<0.05$

patients was 80.6% (*n* = 29/36), whereas the mortality among non-dialyzed patients was only 1.2% (*n* = 1/84).

Discussion

In this prospective observational investigation conducted in a tertiary care Medical ICU in central India, several key epidemiological, etiological, and prognostic features of AKI were characterized. A paramount finding of this study is the marked skew toward advanced disease at the time of specialized admission: 63.3% of patients presented in KDIGO Stage III, whereas Stage I comprised only 5.0%. This distribution reflects substantial delays in AKI recognition at peripheral primary healthcare centers, late clinical referral, and the aggressive pathophysiology of sepsis-induced renal damage. These observations closely parallel those of Mathew and Radha (2019), who reported a 64.7% prevalence of Stage III AKI upon medical ICU admission in southern India.

Demographically, AKI demonstrated a unique bimodal distribution. While older age groups had predictable comorbidity-associated renal vulnerability, the single largest demographic subgroup was young adults aged 18–30 years (25.0%). In this young cohort, AKI was overwhelmingly triggered by acute community-acquired infections, acute gastroenteritis, toxic ingestions, and obstetric emergencies. Encouragingly, young patients displayed outstanding regenerative capacity, achieving an 83.3% complete recovery rate when provided timely resuscitation and critical care support. Conversely, elderly patients (>70 years) suffered a 46.1% mortality rate and high rates of progression to end-stage renal disease, underscoring that aging kidneys possess diminished functional reserve, microvascular rarefaction, and reduced regenerative tubular capacity.[17-20]

Etiologically, intrinsic renal AKI predominated (59.1%), with sepsis identified as the single leading cause (43.3% of total cases). Sepsis-induced AKI involves a complex pathophysiological cascade encompassing systemic arterial vasodilation, intrarenal microvascular flow redistribution, endothelial injury, inflammatory cytokine surges, reactive oxygen species generation, and mitochondrial metabolic shutdown in tubular epithelial cells. Our findings corroborate major ICU studies across India, such as Reddy *et al*. (sepsis 38.6%) and Saxena and Meshram (sepsis 24.5% in rural central India). In addition, toxic ATN accounted for 7.5% of cases, highlighting the ongoing public health threat posed by agricultural pesticide and rodenticide (celphos) poisoning in central India.

Regarding clinical outcomes, our observed in-hospital mortality rate of 25.0% compares favorably with historical ICU studies from developing nations (where mortality frequently ranges between 35% and 55%, e.g., Bhadade *et al*. 51.9%, Priyamvada *et al*. 52.5%). This lower mortality reflects proactive hemodynamic optimization, standardized antibiotic protocols, and timely dialysis intervention. However, the finding that 17.5% of surviving patients progressed to CKD (11.7% conservative, 5.8% hemodialysis-dependent) is of profound clinical relevance. It reinforces the contemporary paradigm that AKI and CKD are not distinct binary entities, but rather an interconnected pathological continuum where ATN precipitates progressive interstitial fibrosis, capillary rarefaction, and nephron loss.

Prognostic analysis clearly demonstrated that pre-existing metabolic comorbidities significantly worsen AKI outcomes. DM emerged as the strongest comorbidity predictor of adverse prognosis (*P* = 0.001), with diabetic patients exhibiting only a 32.1% complete recovery rate and an 85.7% risk of progressing to dialysis-dependent CKD. Pre-existing diabetic glomerulosclerosis, arteriolar hyalinosis, and chronic oxidative stress severely restrict the kidney's compensatory reserve. Finally, the requirement for RRT functioned as a powerful prognostic marker of multiorgan dysfunction syndrome: Dialyzed patients experienced an 80.6% mortality rate compared to only 1.2% in non-dialyzed patients (*P* = 0.001). This stark disparity indicates that the need for RRT in the ICU reflects profound multisystem collapse and extreme illness severity.

Strengths and Limitations

The major strengths of this study include its prospective design, standardized application of KDIGO consensus criteria, comprehensive tracking of biochemical and clinical parameters, and structured 3-month outcome surveillance. Limitations include a single-center design, modest sample size (*n* = 120), lack of novel renal biomarkers (such as neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, or cystatin C) to detect subclinical Stage I injury, and absence of multiyear follow-up to evaluate long-term cardiovascular and end-stage renal outcomes.

Conclusion

AKI in the medical ICU is a major clinical challenge in central India, characterized by a predominance of late presentations (KDIGO Stage III) and intrinsic sepsis-induced etiology. Although overall renal recovery reaches 57.5%, AKI carries a 25.0% mortality rate and precipitates CKD in nearly one-fifth of patients. Advancing age, pre-existing HTN, DM, post-renal obstructive etiologies, and the requirement for RRT represent potent independent predictors of adverse clinical outcomes and mortality. Enhancing peripheral awareness for early AKI detection, aggressive management of sepsis, and establishing long-term post-AKI nephrology follow-up clinics are vital strategies to reduce both immediate mortality and the burgeoning long-term burden of CKD.

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