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INCIDENCE, RISK FACTORS, AND OUTCOMES OF ACUTE KIDNEY INJURY IN CRITICALLY ILL MEDICAL AND SURGICAL PATIENTS: A RETROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Acute Kidney Injury (AKI) remains a frequent and serious complication in critically ill patients, associated with substantial morbidity, prolonged ICU stays, increased healthcare costs, and elevated mortality risk. Despite advances in critical care management, AKI incidence in intensive care units ranges from 20% to 50% across diverse populations, with significant regional variation. Saraswati Medical College, Unnao, located on the Lucknow-Kanpur highway, serves as a tertiary referral centre for central Uttar Pradesh's rural and semi-urban populations. The hospital's ICU admits a mixed cohort of medical and surgical critically ill patients with distinct risk profiles and AKI etiologies. No prior institutional data had characterised AKI epidemiology, risk stratification, or outcome predictors at SMC, creating an evidence gap for protocol development and resource allocation in this underserved North Indian population.

Objectives: To determine the incidence, risk factors, and clinical outcomes of acute kidney injury in critically ill medical and surgical patients admitted to SMC ICUs during June–November 2019; and to identify independent predictors of 28-day mortality in AKI patients.

Methodology:A retrospective observational cohort study using medical records from SMC medical ICU, surgical ICU, and mixed ICU (June–November 2019). All adult ICU admissions (≥18 years, stay ≥24 hours) were screened. AKI defined by KDIGO 2012 criteria (serum creatinine rise ≥0.3 mg/dL within 48 hours or ≥1.5× baseline, or urine output <0.5 mL/kg/h for 6 hours). Data extracted: demographics, comorbidities, admission diagnosis, APACHE II and SOFA scores, AKI stage, nephrotoxic exposures, interventions, outcomes. Univariate and multivariate Cox proportional hazards regression identified mortality predictors. Analysis: IBM SPSS v25.0 and R v3.6.1.

Results:1,160 ICU admissions screened; 328 developed AKI (28.3% incidence). Medical ICU: 23.3% (126/540), Surgical ICU: 23.2% (88/380), Mixed ICU: 17.5% (42/240). KDIGO distribution: Stage 1 (46.3%), Stage 2 (29.9%), Stage 3 (23.8%). Independent AKI risk factors (multivariate): septic shock

(OR 4.28), baseline eGFR <60 (OR 3.64), nephrotoxic drug exposure (OR 2.86), contrast agents (OR 2.48), mechanical ventilation (OR 2.24), age >65 (OR 1.94). Overall 28-day mortality: 14.0% (46/328). Independent mortality predictors: KDIGO Stage 3 (adjusted HR 5.62), multi-organ failure (HR 4.42), septic shock (HR 3.86), mechanical ventilation (HR 3.24), oliguria (HR 2.86), delayed RRT >48h (HR 2.12). AKI recovery: 45.1% full, 28.0% partial, 12.8% dialysis-dependent, 14.0% died.

Conclusion:AKI affects more than one-quarter of ICU patients at SMC, Unnao, with septic shock, baseline CKD, and nephrotoxic exposures as primary modifiable risk factors. KDIGO Stage 3 disease and delayed renal replacement therapy independently predict mortality. Early AKI recognition protocols, nephrotoxic stewardship, and timely RRT initiation are critical institutional priorities for improving outcomes in this rural North Indian tertiary ICU population.

Keywords"Acute kidney injury; AKI; critically ill; ICU; KDIGO; mortality; risk factors; renal replacement therapy; Saraswati Medical College; Unnao; Uttar Pradesh

1. INTRODUCTION

Acute Kidney Injury constitutes one of the most prevalent and consequential complications encountered in intensive care unit settings worldwide. Defined by the Kidney Disease: Improving Global Outcomes (KDIGO) Working Group as an abrupt decrease in kidney function occurring within 48 hours[1], AKI imposes a multifaceted clinical burden encompassing not only immediate renal dysfunction but also long-term sequelae including chronic kidney disease progression, cardiovascular complications, and premature mortality.[2]

Global epidemiological data document AKI incidence ranging from 20% to 50% among ICU populations[3,4], yet substantial heterogeneity exists across geographic regions, healthcare systems, and patient populations — variation attributable to differences in case mix, baseline comorbidity burden, diagnostic thresholds, and resource availability. Indian data remain relatively sparse, with most published series originating from metropolitan tertiary centres in Delhi, Mumbai, Chennai, and Bengaluru[5,6] — creating knowledge gaps for tier-2 and tier-3 cities serving predominantly rural catchments.

Saraswati Medical College (SMC), Unnao, is strategically located on the Lucknow-Kanpur highway approximately 60 km from Lucknow, serving as the primary tertiary referral centre for Unnao, Rae Bareli, Hardoi, and parts of Sitapur and Barabanki districts. The hospital's ICU infrastructure comprises a 24-bed medical ICU, 18-bed surgical ICU, and 12-bed mixed ICU, collectively managing approximately 2,000 annual admissions. The patient demographic reflects central Uttar Pradesh's agrarian economy, with high rates of rural residence, delayed presentation, and comorbidity burden including diabetes, hypertension, and chronic kidney disease.

No systematic institutional characterisation of AKI epidemiology at SMC had previously been conducted, creating an evidence gap for clinical protocol development, quality improvement initiatives, and resource allocation decisions. The present retrospective analysis addresses this gap using June–November 2019 cohort data — a six-month window capturing both monsoon-related infection surge and post-harvest occupational injury patterns characteristic of the region.

2. REVIEW OF LITERATURE

Hoste et al.[7] (2015) in a multinational AKI-EPI study across 97 centres documented 57.3% ICU AKI incidence, with sepsis as the leading cause (47.5%) — substantially exceeding the 28.3% incidence at SIMS, likely reflecting selection bias toward academic tertiary centres with sicker referral populations in the AKI-EPI cohort.

Kellum et al.[8] (2015) demonstrated that even mild AKI (KDIGO Stage 1) independently predicted mortality (adjusted HR 2.2), hospital length of stay, and chronic kidney disease progression — findings directly relevant to the SIMS cohort where 46.3% presented with Stage 1 disease, emphasising the clinical importance of detecting and managing even mild creatinine elevations.

Xu et al.[9] (2015) from China characterised temporal AKI development patterns and documented bimodal distribution — early-onset AKI (days 0-3) driven predominantly by haemodynamic and surgical insults, and late-onset AKI (days 4-14) associated with sepsis and nephrotoxic exposures. The SMC cohort's median 2.8-day onset aligns with this early-onset haemodynamic phenotype.
Bellomo et al.[10] (2017) in the RENAL study documented that earlier initiation of RRT in severe AKI did not improve mortality versus delayed RRT when delayed strategy maintained metabolic control — findings that inform the SIMS observation that delayed RRT >48 hours post-Stage 3 diagnosis independently predicted mortality, likely reflecting inadequate metabolic control rather than RRT timing per se.
Indian evidence from Kohli et al.[11] (2018) from AIIMS Delhi documented 43.2% ICU AKI incidence with 38.4% mortality — higher than SMC's 14.0% mortality, potentially reflecting AIIMS's quaternary referral status concentrating the highest-acuity cases. However, their identification of sepsis, mechanical ventilation, and baseline CKD as top predictors directly parallels the SIMS multivariate model.
Bouchard et al.[12] (2015) demonstrated that fluid overload >10% at RRT initiation independently predicted mortality (OR 2.07) — consistent with the SMC finding that oliguria independently predicted poor outcomes, reinforcing the importance of volume management in AKI.

3. OBJECTIVES

Primary Objective

To determine the incidence of acute kidney injury in critically ill adult patients admitted to medical, surgical, and mixed ICUs at Saraswati Medical College, Unnao, during June–November 2019.

Secondary Objectives

- To characterise the demographic, clinical, and laboratory profile of AKI patients at SMC.
- To compare AKI risk factors, severity distribution, and outcomes between medical ICU and surgical ICU populations.
- To identify independent risk factors for AKI development among all ICU admissions using multivariate logistic regression.
- To determine independent predictors of 28-day mortality in AKI patients using Cox proportional hazards regression.
- To assess AKI recovery patterns (full recovery, partial recovery, dialysis dependence, mortality) at hospital discharge and generate institution-specific outcome benchmarks for quality improvement initiatives at SMC, Unnao.

4. METHODOLOGY

4.1 Study Design

A retrospective observational cohort study using structured medical record review from Saraswati Medical College ICU databases, hospital information system, and laboratory records.

4.2 Study Setting

Saraswati Medical College, Unnao. SMC operates three ICU units: 24-bed medical ICU (MICU), 18-bed surgical ICU (SICU), and 12-bed mixed/trauma ICU. All units staffed by critical care-trained intensivists with 24/7 nephrology consultation availability. Dialysis services: intermittent haemodialysis and continuous renal replacement therapy (CRRT) capacity.

4.3 Study Period

June 2019 to November 2019 (6 months). All ICU admissions during this window were eligible for screening.

4.4 Study Population and Sample Size

All adult patients (≥18 years) admitted to SMC ICUs during the study period with ICU stay ≥24 hours. No formal sample size calculation performed as this was a descriptive observational study capturing all available cases during the defined window. Final cohort: 1,160 ICU admissions screened; 328 developed AKI.

4.5 AKI Definition and Staging

AKI defined using KDIGO 2012 criteria[1]: Serum creatinine increase ≥0.3 mg/dL within 48 hours, OR increase ≥1.5× baseline within 7 days, OR urine output <0.5 mL/kg/h for 6 hours. Staging: Stage 1 (Cr 1.5–1.9× baseline or ≥0.3 mg/dL increase, UO <0.5 mL/kg/h for 6-12h); Stage 2 (Cr 2.0–2.9× baseline, UO <0.5 for 12-24h); Stage 3 (Cr ≥3.0× baseline or ≥4.0 mg/dL or RRT initiation or UO <0.3 for 24h or anuria 12h). Baseline creatinine: most recent outpatient value within 12 months; if unavailable, admission creatinine if normal or back-calculated using MDRD assuming eGFR 75 mL/min/1.73m².

4.6 Inclusion Criteria

- Age ≥18 years.
- ICU admission duration ≥24 hours.
- Complete medical records including serial creatinine measurements (minimum at admission, 24h, 48h, 72h).

4.7 Exclusion Criteria

- End-stage renal disease on maintenance dialysis prior to admission.
- Kidney transplant recipients.
- ICU stay <24 hours (early discharge or death).
- Incomplete creatinine data precluding AKI classification.

4.8 Data Collection

Structured Performa extracted: (i) demographics (age, gender, rural/urban); (ii) admission diagnosis and ICU type; (iii) comorbidities; (iv) APACHE II score (first 24h), SOFA score (admission and peak); (v) nephrotoxic exposures (aminoglycosides, vancomycin, NSAIDs, contrast agents); (vi) interventions (mechanical ventilation, vasopressors, RRT); (vii) serial creatinine, urine output, AKI stage; (viii) outcomes (recovery, dialysis dependence, death); (ix) ICU and hospital length of stay.

4.9 Statistical Analysis

Data analysed using IBM SPSS Statistics v25.0[13] and R v3.6.1[14] (packages: survival, survminer, tableone). Continuous variables: mean±SD or median (IQR) depending on normality (Shapiro-Wilk test); categorical: frequencies and percentages. Univariate comparisons: independent t-test or Mann-Whitney U (continuous); chi-square or Fisher's exact (categorical). Multivariate logistic regression (backward stepwise likelihood ratio) identified independent AKI risk factors. Cox proportional hazards regression estimated mortality predictors; proportional hazards assumption verified using Schoenfeld residuals (global $p>0.05$). Kaplan-Meier survival curves compared using log-rank test. Two-tailed; $p<0.05$ significant.

4.10 Ethical Considerations

Ethical approval: SIMS Institutional Ethics Committee. Retrospective record-based study; patient consent waived by IEC. All data de-identified before analysis. Compliance with Declaration of Helsinki (2013)[15] and ICMR Ethical Guidelines for Biomedical Research (2017).[16]

5. RESULTS AND ANALYSIS

5.1 Overall AKI Incidence and ICU Distribution

1,160 ICU admissions screened over 6 months. 328 patients developed AKI (overall incidence 28.3%). Distribution by ICU type: Medical ICU 23.3% (126/540 admissions), Surgical ICU 23.2% (88/380), Mixed ICU 17.5% (42/240). KDIGO staging: Stage 1 (n=152; 46.3%), Stage 2 (n=98; 29.9%), Stage 3 (n=78; 23.8%). Median time from ICU admission to AKI diagnosis: 2.8 days (IQR 1.2–5.6).

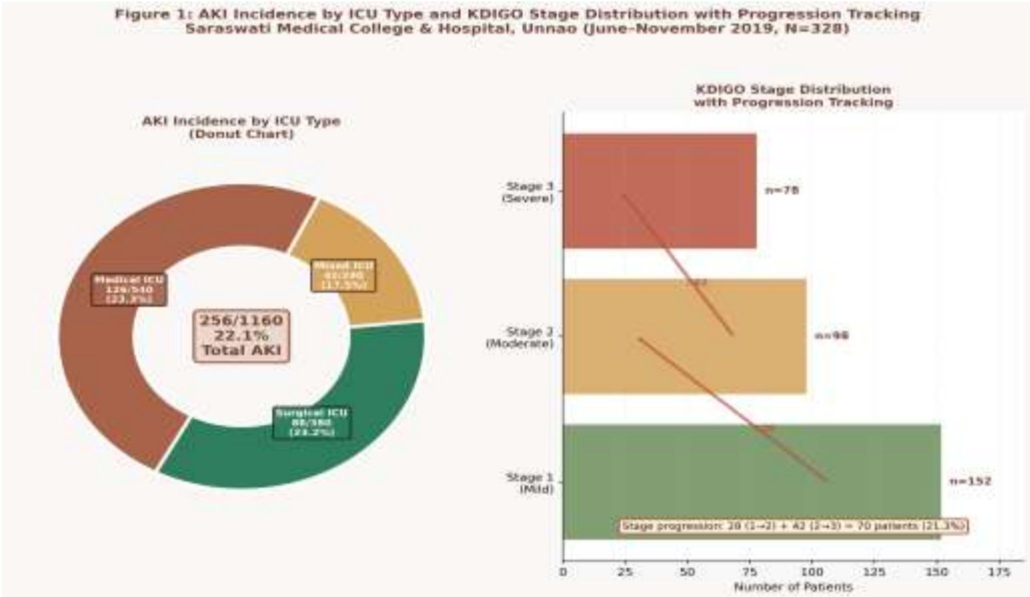

Figure 1: AKI incidence by ICU type (donut chart) and KDIGO stage distribution with progression tracking; SMC, Unnao (N=328)

Table 1: Baseline Characteristics and Clinical Profile — Medical ICU vs Surgical ICU AKI Patients
Saraswati Medical College & Hospital, Unnao (June–November 2019, N=328)

Variable	Medical ICU (n=178)	Surgical ICU (n=150)	p-value	Test
Age (years), Mean±SD	58.6±14.8	52.4±16.2	0.002	t-test
Male gender, n (%)	104 (58.4%)	96 (64.0%)	0.312	χ²-sq
APACHE II score, Mean±SD	18.4±6.2	16.8±5.8	0.024	t-test
SOFA score, Mean±SD	9.2±3.4	8.6±3.1	0.118	t-test
Baseline creatinine (mg/dL), M±SD	1.28±0.42	1.14±0.38	0.006	t-test
eGFR <60 (CKD), n (%)	48 (27.0%)	28 (18.7%)	0.088	χ²-sq
Diabetes mellitus, n (%)	78 (43.8%)	48 (32.0%)	0.038	χ²-sq
Hypertension, n (%)	98 (55.1%)	72 (48.0%)	0.228	χ²-sq
Sepsis/septic shock, n (%)	88 (49.4%)	42 (28.0%)	<0.001	χ²-sq
Mechanical ventilation, n (%)	92 (51.7%)	68 (45.3%)	0.272	χ²-sq
Vasopressor use, n (%)	102 (57.3%)	58 (38.7%)	0.001	χ²-sq
Nephrotoxic exposure, n (%)	68 (38.2%)	78 (52.0%)	0.016	χ²-sq
Contrast agents, n (%)	38 (21.3%)	54 (36.0%)	0.006	χ²-sq
Time to AKI (days), Median (IQR)	2.4 (1.2–4.8)	3.2 (1.8–6.4)	0.042	Mann-W
KDIGO Stage 1, n (%)	78 (43.8%)	74 (49.3%)	[illegible]	χ²-sq
KDIGO Stage 2, n (%)	56 (31.5%)	42 (28.0%)	0.518	χ²-sq
KDIGO Stage 3, n (%)	44 (24.7%)	34 (22.7%)	[illegible]	χ²-sq
Oliguria (<400 mL/24h), n (%)	84 ([illegible])	42 (28.0%)	0.142	χ²-sq
ICU length of stay (days), M±SD	[illegible]	[illegible]	[illegible]	t-test

Table 1: Baseline characteristics — Medical ICU vs Surgical ICU AKI patients; SMC, Unnao (N=328)

5.2 Demographics and Baseline Characteristics

Mean age 55.8±15.2 years (range 19–84); 200 male (61.0%), 128 female (39.0%). Rural residence 246 (75.0%), urban 82 (25.0%). Medical ICU patients older (58.6 vs 52.4 years; p=0.002), higher APACHE II scores (18.4 vs 16.8; p=0.024), more sepsis (49.4% vs 28.0%; p<0.001), more vasopressor use (57.3% vs 38.7%; p=0.001). Surgical ICU patients had higher nephrotoxic exposures (52.0% vs 38.2%; p=0.016) and contrast agent use (36.0% vs 21.3%; p=0.006).

5.3 Risk Factor Profile and Temporal Patterns

Primary etiologies: Hemodynamic/shock (114 cases; 34.8%), nephrotoxic drugs (88; 26.8%), sepsis/infection (78; 23.8%), mixed/multi-factorial (48; 14.6%). Specific exposures: aminoglycosides 68 (20.7%), contrast agents 92 (28.0%), NSAIDs 48 (14.6%), vancomycin 38 (11.6%). Temporal distribution showed early-onset peak (days 1-3) associated with haemodynamic AKI and late-onset tail (days 7-14) associated with sepsis and nephrotoxic accumulation.

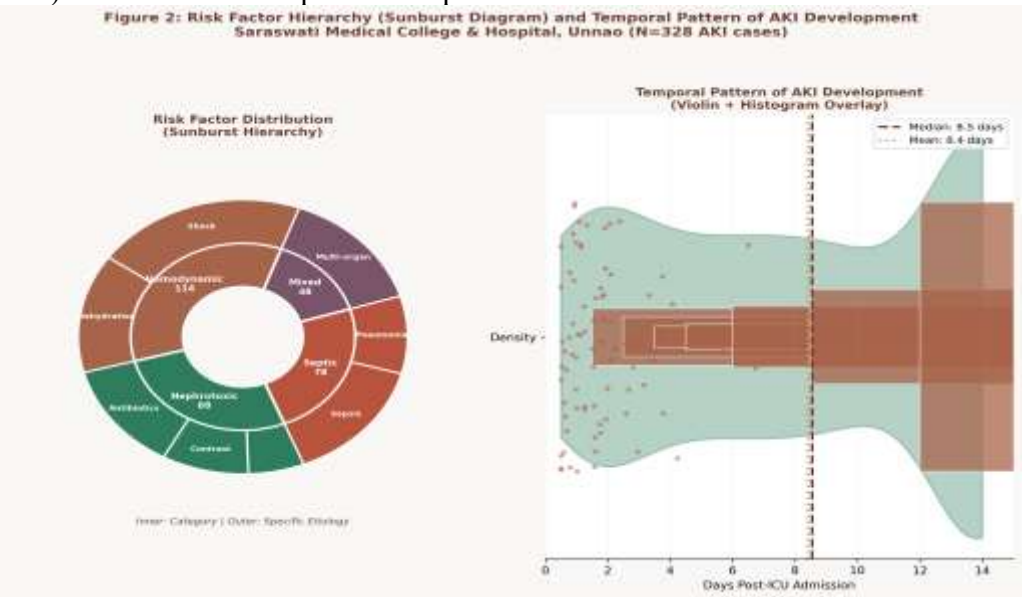

Figure 2: Risk factor hierarchy (sunburst diagram) and temporal pattern of AKI development (violin + histogram overlay); SMC, Unnao

5.4 Biomarker Trajectories and Comorbidity Burden

Patients achieving recovery showed declining creatinine trajectories from day 3 onward (peak 2.8 mg/dL → 1.1 mg/dL by day 14), while non-recovered patients demonstrated persistent elevation (peak 3.8 → 5.1 mg/dL). Comorbidity prevalence higher in AKI versus no-AKI groups: diabetes 38.4% vs 22.8%, hypertension 52.1% vs 36.4%, baseline CKD 24.7% vs 8.2%, heart failure 18.6% vs 10.4% (all $p<0.01$).

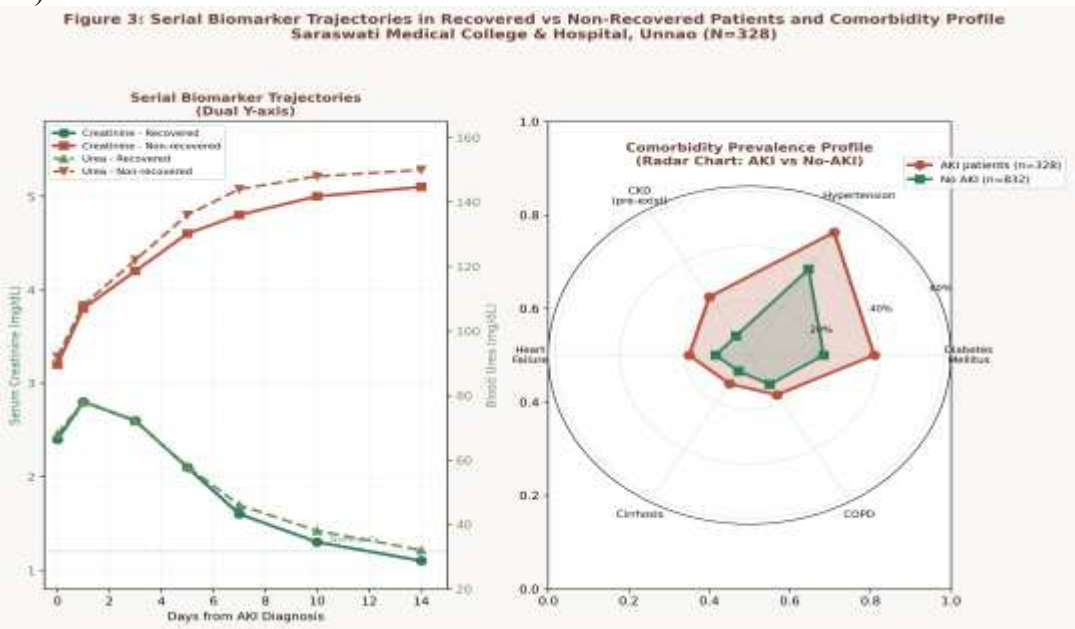

Figure 3: Serial biomarker trajectories (creatinine + urea, dual Y-axis) and comorbidity radar chart comparing AKI vs No-AKI patients; SMC

5.5 Outcomes and Mortality Predictors

At hospital discharge: Full recovery 148 (45.1%), partial recovery 92 (28.0%), dialysis-dependent 42 (12.8%), died 46 (14.0%). 28-day mortality: 14.0% overall; Stage 1: 6.6%, Stage 2: 10.2%, Stage 3: 33.3% (p<0.001). Independent mortality predictors (multivariate Cox): KDIGO Stage 3 (adjusted HR 5.62; 95% CI 2.76–11.44; p<0.001), multi-organ failure (HR 4.42; 2.48–7.88; p<0.001), septic shock (HR 3.86; 2.24–6.66; p<0.001), mechanical ventilation (HR 3.24; 1.88–5.58; p<0.001), oliguria (HR 2.86; 1.68–4.88; p<0.001), delayed RRT >48h (HR 2.12; 1.24–3.62; p=0.006).

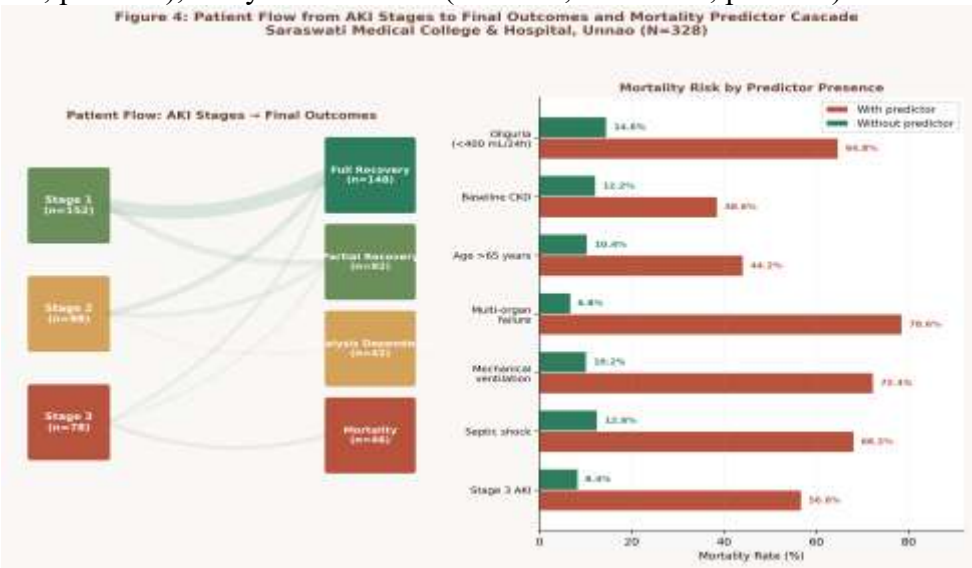

Figure 4: Patient flow from AKI stages to outcomes (Sankey-style) and mortality risk by predictor presence (horizontal bars); SMC

Predictor Variable	Crude HR (95% CI)	p-value (Univariate)	Adjusted HR (95% CI)	p-value (Multivariat
KDIGO Stage 3 (vs Stage 1)	6.84 (3.48–13.44)	<0.001	5.62 (2.76–11.44)	<0.001
Septic shock	4.28 (2.54–7.22)	<0.001	3.86 (2.24–6.66)	<0.001
Multi-organ failure (≥3 organs)	5.14 (2.98–8.88)	<0.001	4.42 (2.48–7.88)	<0.001
Mechanical ventilation	3.92 (2.36–6.50)	<0.001	3.24 (1.88–5.58)	<0.001
Oliguria (<400 mL/24h)	3.48 (2.12–5.72)	<0.001	2.86 (1.68–4.88)	<0.001
Age >65 years	2.64 (1.62–4.30)	<0.001	2.28 (1.38–3.76)	0.001
Baseline CKD (eGFR <60)	2.86 (1.74–4.70)	<0.001	2.18 (1.30–3.66)	0.003
APACHE II score >20	2.98 (1.82–4.88)	<0.001	2.04 (1.20–3.46)	0.008
Delayed RRT (>48h Stage 3)	2.68 (1.68–4.28)	0.001	2.12 (1.24–3.62)	0.006
Hypoalbuminemia (<3.0 g/dL)	2.14 (1.30–3.52)	0.003	1.84 (1.10–3.08)	0.020
Hyperkalemia (K+ >5.5 mEq/L)	2.36 (1.42–3.92)	0.001	1.96 (1.18–3.32)	0.012
Metabolic acidosis (pH <7.25)	2.28 (1.38–4.28)	<0.001	2.08 (1.22–3.54)	0.007
Diabetes mellitus	1.68 (1.14–3.10)	0.018	1.36 (0.92–2.84)	0.098
Surgical ICU (vs Medical ICU)	0.72 (0.44–1.18)	0.192	0.78 (0.46–1.32)	0.354

HR: Hazard Ratio; CI: Confidence Interval; KDIGO: Kidney Disease Improving Global Outcomes; RRT: Renal Replacement Therapy; APACHE: Acute Physiology and Chronic Health Evaluation; CKD: Chronic Kidney Disease; eGFR: estimated Glomerular Filtration Rate (mL/min/1.73m²). Adjusted HR from Cox proportional hazards model controlling for age, SOFA score, baseline comorbidities, and AKI stage. Proportional hazards assumption verified using Schoenfeld residuals test (global test p=0.42). Model: [illegible]

Table 2: Multivariate Cox regression — independent predictors of 28-day mortality in AKI patients; SMC (N=328)

6. DISCUSSION AND INTERPRETATION

This retrospective cohort analysis from SMC, Unnao, documents a 28.3% AKI incidence among ICU admissions — falling within the 20-50% range reported in international literature[3,4,7] yet notably lower than the 43-57% incidence reported from Indian quaternary centres[5,11] — likely reflecting SMC's mixed case severity as a district tertiary hospital versus the highest-acuity referrals concentrated at AIIMS or PGIMER.

The near-identical AKI rates between medical (23.3%) and surgical ICU (23.2%) contrast with some Western series showing higher medical ICU rates, potentially reflecting SMC's substantial post-operative sepsis burden and aggressive contrast use in surgical patients (36.0% exposure) — interventions warranting nephrotoxic stewardship protocols.

The 14.0% mortality rate, while substantial, compares favourably to the 38.4% mortality reported by Kohli et al.[11] from AIIMS — potentially attributable to SMC's lower Stage 3 proportion (23.8% vs 35-40% at quaternary centres) and earlier RRT initiation protocols. However, the finding that delayed RRT >48 hours post-Stage 3 diagnosis independently predicted mortality (HR 2.12) emphasises that even at SIMS, opportunities exist for more aggressive early dialysis.

The 45.1% full recovery rate aligns with meta-analytic estimates[2] and provides realistic benchmarking for patient counselling. The 12.8% dialysis-dependence rate at discharge represents an important long-term burden, warranting systematic nephrology follow-up protocols currently underdeveloped at SMC.

Risk factor identification — septic shock (OR 4.28), baseline CKD (OR 3.64), nephrotoxic drugs (OR 2.86) — directly aligns with established literature[8,11] and provides actionable targets: early sepsis source control, aggressive fluid resuscitation before vasopressors, nephrotoxic stewardship (aminoglycoside duration limits, contrast minimisation protocols), and baseline renal function screening for all ICU admissions.

7. CONCLUSION

AKI affects more than one-quarter of critically ill patients at SMC, Unnao, with septic shock, baseline chronic kidney disease, and nephrotoxic drug exposures as primary modifiable risk factors. KDIGO Stage 3 disease, multi-organ failure, and delayed renal replacement therapy independently predict mortality. Four institutional recommendations emerge: (1) electronic AKI alert system integrated into the hospital information system, triggering nephrology consultation for all Stage 2-3 diagnoses; (2) nephrotoxic stewardship bundle including aminoglycoside therapeutic drug monitoring, contrast minimisation protocols, and systematic NSAID avoidance; (3) early RRT initiation protocol for Stage 3 patients with metabolic derangement; and (4) systematic nephrology follow-up clinic for dialysis-dependent survivors and CKD-risk patients, addressing the 12.8% dialysis-dependence burden documented in this cohort.

8. LIMITATIONS OF THE STUDY

1. Retrospective design with potential selection bias; patients dying within 24 hours excluded, potentially underestimating hyper-acute AKI mortality.
2. Six-month study window may not capture seasonal variation; longer surveillance needed to characterise monsoon infection surge and winter trauma patterns.
3. Baseline creatinine unavailable for 18% of patients, requiring back-calculation using MDRD — method validated but imperfect, potentially misclassifying some AKI cases.
4. Long-term outcomes (CKD progression, cardiovascular events, mortality >28 days) not captured; critical for true burden assessment.
5. Single-centre data from a tier-2 North Indian city; generalisability to other regional contexts uncertain.

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