Section: Paediatrics

Original Research Article

STUDY OF THE INCIDENCE OF ACUTE KIDNEY INJURY AND OUTCOMES IN CHILDREN ADMITTED TO A PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE HOSPITAL

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Received : 10/03/2026
Received in revised form : 26/04/2026
Accepted : 13/05/2026

Keywords:
Acute kidney injury, incidence, pediatric intensive care, outcomes, renal angina index.

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DOI: 10.47009/jamp.2026.8.3.49

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (3); 271-274

ABSTRACT

Background: Acute kidney injury (AKI) is a common and serious complication in critically ill children, leading to increased morbidity and mortality. Worldwide, roughly one-quarter of children admitted to pediatric intensive care units (PICUs) develop AKI. There is limited data from India on AKI incidence and outcomes in PICUs. **Materials and Methods:** We conducted a prospective observational study over 18 months (Oct 2022–Mar 2024) in the PICU of Andhra Medical College, Visakhapatnam. Children aged 1 month to 12 years, admitted for ≥48 hours, were enrolled (n=360). Nineteen were excluded (short stay or known chronic kidney disease), leaving 341 cases for analysis. AKI was defined and staged by KDIGO criteria. Data collected included demographics, diagnoses, laboratory values, and renal angina index (RAI) at 24 hours. We recorded AKI incidence, severity, etiologies, need for renal replacement therapy (RRT), and outcomes. Statistical comparisons between AKI and non-AKI groups were performed (chi-square test, $p<0.05$ significant). **Results:** Of 341 children, 118 (34.6%) developed AKI. The incidence was higher than pooled international estimates (26%), possibly reflecting our PICU population. AKI patients were slightly older on average and had a non-significant male preponderance (M:F 1.3:1 in AKI). AKI incidence varied by age group ($p=0.001$), highest in 4–8 years (34.7%) and lowest in 1–4 years (13.6%) (Table 1). Stage 1 AKI was most common (54/118, 45.8%), followed by Stage 3 (39, 33%) and Stage 2 (25, 21%). Higher AKI stage was associated with greater mortality (Stage 1 death 11.1%, Stage 3 death 33.3%; overall AKI mortality 19.5%) (Table 2). Sepsis and other infections accounted for the majority of cases (60.2%), while nephrotoxic exposures were the leading non-infectious cause (24.6%). RAI at 24h strongly predicted AKI: of 116 children with RAI≥8 ("renal angina"), 97 (83.6%) developed AKI (sensitivity 82.2%, specificity 91.5%). RRT was required in 42 of 118 AKI patients (35.6%). Mortality was significantly higher in those requiring RRT (40.5%) versus not (9.2%; $p<0.001$). **Conclusion:** In this PICU cohort, AKI affected one-third of admissions. Infections and nephrotoxins were common causes. Many patients had transient AKI (Stage 1) with good recovery, but higher stages and need for RRT portended much worse outcomes. The 19.5% mortality among AKI cases underscores its severity. Early risk stratification with tools like the renal angina index and prompt supportive care (including timely RRT) are essential. These findings highlight the need for vigilance and early intervention to reduce AKI morbidity and mortality in critically ill children.

INTRODUCTION

Acute kidney injury (AKI) – an abrupt decline in renal function – is a frequent complication in pediatric critical illness. Children are particularly vulnerable due to developmental physiology and exposure to sepsis, surgery, and nephrotoxins in intensive care.[1,2] AKI in this setting is associated with prolonged hospital stays, need for renal replacement therapy (RRT), and higher mortality. Global data indicate roughly 20–30% of PICU patients develop AKI by KDIGO criteria. For

ISSN (O): 2687-5365; ISSN (P): 2753-6556

example, the AWARE study (4683 children worldwide) found AKI in 26.9% of first-week PICU admissions. However, rates vary widely by setting. A recent meta-analysis of pediatric AKI (2023) reported a pooled incidence of 26% (CI 22–29%), but studies from low-income countries suggest even higher burden and mortality. In India, sparse data exist: one tertiary center reported 19.3% AKI incidence (KDIGO) with nearly half of AKI patients dying, while another center (using RIFLE criteria) found 17.1% incidence and significant excess mortality in AKI children.[3,4]

AKI etiologies differ between regions.[5,6] In resource-limited settings, community-acquired causes (dehydration, infections, tropical diseases, toxins) are common, whereas in high-income countries iatrogenic or post-surgical AKI predominates. Early risk stratification tools (e.g. Renal Angina Index) and standardized definitions (KDIGO 2012) have improved consistency in AKI research. Nonetheless, local data are needed to guide practice.[7] We therefore studied the incidence, risk factors, and outcomes of AKI among children in our tertiary-care PICU. Our objectives were to determine AKI incidence, describe severity and causes, assess the predictive value of the renal angina index (RAI), and evaluate clinical outcomes, including the need for dialysis and mortality.

MATERIALS AND METHODS

A prospective observational study was conducted in the PICU of Andhra Medical College, Visakhapatnam, over 18 months (October 2022–March 2024). Children aged 1 month–12 years admitted to the PICU were eligible. Inclusion criteria were PICU stay ≥48 hours; every 5th eligible admission was enrolled by systematic sampling until the target of 360 was reached (based on an assumed AKI incidence of ~33.7% and 5% precision). Exclusion criteria were known chronic kidney disease or inadequate consent. Institutional ethics committee approval was obtained.

Demographic and clinical data were recorded at admission. This included age, sex, primary diagnosis, vital signs, hydration status, need for ventilation or vasopressors, and Pediatric Risk of Mortality (PRISM) III score. Laboratory data (daily serum creatinine for 3 days or until discharge, electrolytes, complete blood count) and urine output (6-hourly monitoring) were collected. AKI was defined according to KDIGO 2012 criteria (an increase in serum creatinine ≥0.3 mg/dL within 48h, or ≥1.5× baseline, or urine output <0.5 mL/kg/h for 6h). Baseline creatinine was taken as the admission value (no chronic kidney disease patients were included). AKI stage (1–3) was assigned per KDIGO. The Renal Angina Index (RAI) was calculated at 24 hours using published criteria (points for risk factors and injury), with RAI ≥8 considered "renal angina." Patients were followed for development of AKI by day 3. All AKI patients were managed supportively; RRT (peritoneal dialysis or hemodialysis) was provided when indicated by fluid overload or refractory metabolic derangements.

Statistical analysis was performed using SPSS v25.0. Continuous variables are reported as mean±SD or median (IQR) and categorical as counts (%). AKI incidence was the primary outcome. Comparisons between the AKI and non-AKI groups used chi-square or Fisher's exact tests for categorical variables, and t-test or Mann–Whitney U for continuous variables. A p-value <0.05 was considered significant. We also calculated sensitivity and specificity of RAI for predicting AKI. Outcomes (need for RRT, survival) were analyzed descriptively and by chi-square between groups.

RESULTS

Patient Characteristics and AKI Incidence

During the study period, 360 children were enrolled (Figure 1). Nineteen were excluded (14 had PICU stay <48 h, 5 had pre-existing CKD), leaving 341 for analysis. The mean age was 5.7±3.2 years; 182 (53.4%) were male. The overall AKI incidence was 34.6% (118 of 341). Thus 118 children developed AKI (Group AKI) and 223 did not (Group No AKI). The AKI incidence in our PICU is higher than pooled international rates (~26%) and also higher than some other Indian centers (e.g. 17–19%), possibly reflecting case-mix or local factors.

(Table 1 shows demographic breakdown.) There was no statistically significant sex difference: AKI occurred in 67/182 (36.8%) of males and 51/159 (32.1%) of females (p=0.42). Children with AKI tended to be older, with 41 of 118 (34.7%) in the 4–8 year age range, compared to only 16/118 (13.6%) in the 1–4 year group. The age distribution differed between AKI and non-AKI (χ^2 test p=0.001). In particular, infants (≤1 yr) had AKI in 32/110 cases (29.1%), whereas the 1–4 year group had AKI in only 16/77 (20.8%). The 4–8 year group had the highest AKI proportion. [See Table 1]

Table 1: Demographics of PICU Patients by AKI Status

Characteristic	AKI (n=118)	No AKI (n=223)	Total (n=341)
Age group			
≤1 year	32 (27.1%)	78 (35.0%)	110 (32.2%)
1–4 years	16 (13.6%)	61 (27.4%)	77 (22.6%)
4–8 years	41 (34.7%)	50 (22.4%)	91 (26.7%)
8–12 years	29 (24.6%)	34 (15.3%)	63 (18.5%)
Gender			
Male	67 (56.8%)	115 (51.6%)	182 (53.4%)
Female	51 (43.2%)	108 (48.4%)	159 (46.6%)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

AKI Severity and Renal Angina

Among the 118 AKI cases, the distribution of KDIGO stage was: Stage 1: 54 (45.8%), Stage 2: 25 (21.2%), and Stage 3: 39 (33.0%). As shown in Table 2, the majority had mild AKI, but one-third developed severe (Stage 3) AKI. The mean peak serum creatinine among AKI patients was 2.3±1.4 mg/dL.

The Renal Angina Index (RAI) proved a strong predictor: 116 of 341 children (34.0%) had RAI ≥8 at 24h. Of these 116 "RA+" children, 97 (83.6%) went on to develop AKI by day 3. In contrast, among the 225 RA– children (RAI<8), only 21 (9.3%) developed AKI ($p<0.00001$). This corresponds to RAI sensitivity 82.2%, specificity 91.5%, and positive predictive value 83.6% for AKI in our cohort. In practice, RAI assessment at admission could identify most children who later developed AKI.

Table 2: AKI Stage and Outcomes

AKI Stage	n (%) of AKI	Deaths (n, % of stage)
Stage 1	54 (45.8%)	6 (11.1%)
Stage 2	25 (21.2%)	4 (16.0%)
Stage 3	39 (33.0%)	13 (33.3%)
Total	118 (100%)	23 (19.5% of AKI)

Etiology of AKI

Infection was the most common precipitant (71 of 118 AKI cases, 60.2%). Among these, sepsis (culture-positive or -negative) accounted for 23 cases (19.5%), pneumonia 18 (15.3%), gastroenteritis 15 (12.7%), meningitis 5 (4.2%), urinary tract infection 4 (3.4%), and other infections 6 (5.1%). Non-infectious causes were seen in 47 cases (39.8%), led by nephrotoxic exposures (drugs or poisons) in 29 children (24.6%). Other non-infectious etiologies included central nervous system diseases (3 cases), hemolytic uremic syndrome (2), diabetic ketoacidosis (2), multisystem inflammatory syndrome in children (MIS-C) (2), and snake envenomation (1). These findings echo prior reports that sepsis and nephrotoxins are leading causes of pediatric AKI in similar settings.

Outcomes: RRT and Mortality

Overall mortality among AKI patients was 19.5% (23/118), compared with 2.6% (6/223) in non-AKI children ($p<0.001$). Mortality rose with AKI severity (Table 2) – Stage 3 patients had a one-third mortality rate. All 23 deaths occurred in AKI patients.

Renal replacement therapy (RRT) was required in 42 AKI cases (35.6%). Of these, 25 survived and 17 died (40.5% mortality in RRT group). In contrast, among 76 AKI patients managed without dialysis, 69 survived and 7 died (mortality 9.2%). The difference was significant (χ^2, $p<0.001$). (See Figure 2 for survival by RRT status.) This indicates that children needing dialysis had a substantially higher risk of death. Notably, dialysis (mostly peritoneal) was initiated early when indicated; 61.9% of AKI survivors recovered full renal function by discharge. The majority of AKI patients (95/118; 80.5%) ultimately survived, often with complete recovery of kidney function.

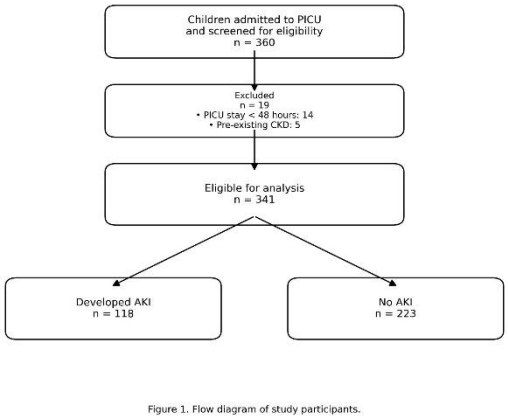

Figure 1. Flow diagram of study participants.

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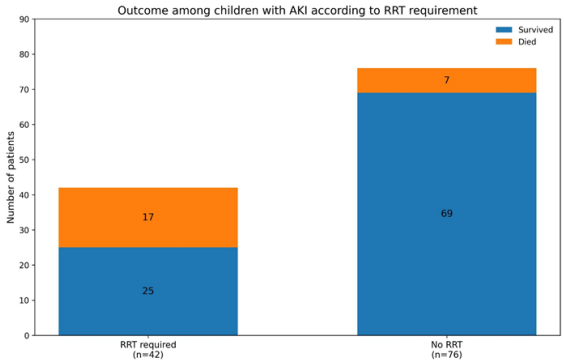

Figure 2. Stacked bar chart showing survival and mortality by RRT status.

Figure 2: Outcome among children with acute kidney injury according to renal replacement therapy requirement

DISCUSSION

This study found that over one-third (34.6%) of critically ill children in our PICU developed AKI, a rate higher than many reports.[8,9] For comparison, a recent meta-analysis of 94 pediatric studies worldwide reported a pooled AKI incidence of 26%, and the multicenter AWARE study found 26.9%. Our higher incidence may reflect local factors: resource-

ISSN (O): 2687-5365; ISSN (P): 2753-6556

limited settings often have more severe illness at admission and a higher burden of tropical infections and delayed referrals. A tertiary Indian PICU (Pune) reported 19.3% AKI and another (Guntur) 17.1%; we exceed these, perhaps due to different inclusion (we used KDIGO, they used RIFLE) or patient severity. Our findings align with the view that pediatric AKI incidence is rising in low- and middle-income countries.

The age distribution showed the highest AKI proportion in the 4–8 year group, and a significant association between age and AKI. The reasons are unclear; it may reflect exposure patterns (more infections or interventions in school-aged children) or underlying disease mix. We did not find gender differences, similar to other series. Most AKI cases were mild (Stage 1, 46%); still, one-third were severe (Stage 3). This severity mix is comparable to other PICU studies.

Our etiological pattern (60% infectious causes, 24.6% nephrotoxin) mirrors global trends in similar settings.[10,11] Sepsis-induced AKI is well-recognized. The substantial nephrotoxin-associated AKI underlines the need for cautious drug use and monitoring. Prevention strategies should target hydration and prompt infection control, as suggested by prior authors.

The renal angina index (RAI) was highly predictive: RAI≥8 had sensitivity 82% and specificity 92% for future AKI. This supports its utility as an early risk stratifier. Previous multicenter studies also found RAI to reliably identify children at high risk for severe AKI. In practice, measuring RAI on admission could help clinicians intensify monitoring and preventive measures in those at risk.

The overall mortality among AKI children was 19.5%, significantly higher than in non-AKI patients, as expected. This is within the range reported internationally (AKI-associated mortality ~11% overall, but up to 18–22% in low-resource settings). In our study, only 5.4% of survivors required dialysis, indicating many had reversible AKI. However, those requiring RRT had a much higher mortality (40.5% vs 9.2%, $p<0.001$), consistent with studies showing poor outcomes in dialyzed pediatric AKI. Dialysis likely identified the sickest children (often with multiorgan failure), yet timely RRT may have salvaged many otherwise fatal cases. Our RRT rate (35% of AKI) was higher than in some centers (e.g. 3% in another Indian series), possibly due to differing practices or thresholds. This suggests a need to develop pediatric RRT protocols suited to local resources.

Limitations: This was a single-center study with convenience sampling (every 5th admission), which may limit generalizability. We relied on admission creatinine as baseline and did not have long-term follow-up for chronic outcomes. Also, while we calculated RAI, other novel biomarkers were not assessed. Despite these, our study provides current data on PICU-AKI in an Indian tertiary hospital.

CONCLUSION

AKI affected one-third of PICU admissions in our center and was associated with substantial morbidity and mortality. Early-stage AKI predominated, but severe AKI and dialysis requirement carried high risk of death. The renal angina index was a useful early predictor of AKI. Our findings emphasize the importance of vigilant monitoring of kidney function in all critically ill children, judicious use of fluids and medications, and prompt renal support when indicated. Multicenter studies in low-resource settings are needed to further define risk factors and refine management protocols to improve outcomes in pediatric AKI.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556