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Original Research Article

ASSESSING OUTCOME AND RISK FACTORS OF ACUTE KIDNEY INJURY IN CHILDREN ADMITTED TO THE PAEDIATRIC INTENSIVE CARE UNIT (PICU)

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ABSTRACT

Background: Acute kidney injury (AKI) is a common and serious complication among critically ill children admitted to the Paediatric Intensive Care Unit (PICU). It contributes significantly to morbidity, mortality, prolonged hospitalization, and long-term renal sequelae. Early identification of risk factors and timely intervention are essential to improve clinical outcomes.
Aim: To assess the outcomes and risk factors associated with acute kidney injury among children admitted to the Paediatric Intensive Care Unit.
Materials and Methods: This prospective observational study was conducted in the PICU of Indira Gandhi Institute of Child Health, Bengaluru, over 18 months from October 2022. A total of 250 children aged 1 month to 18 years diagnosed with AKI according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria were enrolled. Demographic, clinical, laboratory, treatment, and outcome data were collected. Survivors were followed for three months to assess renal recovery. Statistical analysis was performed using R version 4.4.0, with $p<0.05$ considered statistically significant.
Results: Among the 250 children, 138 (55.2%) were aged ≤5 years and 148 (59.2%) were males. Pneumonia (23.2%) and acute gastroenteritis (16.4%) were the leading causes of AKI. Stage 3 AKI was observed in 153 (61.2%) children, while prerenal AKI accounted for 60.4% of cases. Metabolic acidosis (74.8%), nephrotoxic drug exposure (50.8%), and mechanical ventilation (48.4%) were common. Overall mortality was 33.6%. Non-survivors had significantly higher rates of shock, oliguria, hyperkalaemia, metabolic acidosis, nephrotoxic drug exposure, mechanical ventilation, inotropic support, and dialysis ($p<0.05$). At discharge, 77.1% of survivors had normal renal function, increasing to 97.0% at three-month follow-up.
Conclusion: Severe AKI is frequent among critically ill children and is associated with high mortality. Early recognition of high-risk patients, prompt management of modifiable risk factors, and structured post-discharge renal follow-up are essential to improve survival and long-term renal outcomes.
Keywords: Acute kidney injury, children, KDIGO, Paediatric Intensive Care Unit, mortality, renal recovery, risk factors.

INTRODUCTION

Acute kidney injury (AKI) is a common and potentially life-threatening complication among critically ill children admitted to the Paediatric Intensive Care Unit (PICU). It is characterized by an abrupt decline in renal function, resulting in disturbances of fluid, electrolyte, and acid–base balance. AKI contributes substantially to morbidity, mortality, prolonged hospitalization, and increased healthcare utilization. Early identification and timely intervention are therefore essential to prevent progression and improve outcomes.[1]

The incidence of AKI in PICUs varies according to the study population, underlying illness, and diagnostic criteria used. The Kidney Disease:

Improving Global Outcomes (KDIGO) criteria provide a standardized definition based on changes in serum creatinine and urine output. According to KDIGO, AKI is diagnosed by an increase in serum creatinine of ≥0.3 mg/dL within 48 hours, an increase to ≥1.5 times baseline within seven days, or urine output <0.5 mL/kg/hour for at least six hours.[1]

The multinational AWARE study reported AKI in nearly 27% of critically ill children, with severe AKI in approximately 12%. Severe AKI was independently associated with increased mortality, prolonged mechanical ventilation, and longer PICU stay.[2,3] The pathogenesis of AKI in critically ill children is multifactorial and may involve renal hypoperfusion, sepsis, systemic inflammation, ischemic injury, oxidative stress, and nephrotoxic drug exposure. Common risk factors include shock, pneumonia, dehydration, acute gastroenteritis, multiorgan dysfunction, mechanical ventilation, vasoactive support, and the use of nephrotoxic medications.[2,4]

Early recognition of high-risk children is important because increasing AKI severity is associated with a greater need for renal replacement therapy, incomplete renal recovery, and death. Although novel biomarkers and renal angina scoring systems have been investigated, serum creatinine and urine output remain the most practical and widely available diagnostic tools.[4,5]

AKI is no longer considered entirely reversible. Survivors may develop persistent proteinuria, hypertension, reduced glomerular filtration rate, recurrent AKI, or chronic kidney disease. Therefore, post-discharge monitoring of renal function and blood pressure is essential, particularly in children with severe AKI.[6,7]

Data on the causes, risk factors, outcomes, and renal recovery of pediatric AKI remain limited in many Indian settings. Hence, the present study was undertaken to assess the outcomes and associated risk factors of AKI among children admitted to the PICU at Indira Gandhi Institute of Child Health, Bengaluru.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based prospective observational study was conducted in the Paediatric Intensive Care Unit (PICU) of **Indira Gandhi Institute of Child Health, Bengaluru**, a tertiary care referral center.

Study Duration

The study was carried out over **18 months**, from **October 2022**.

Study Population

Children aged **1 month to 18 years** admitted to the PICU and diagnosed with acute kidney injury (AKI) according to the **Kidney Disease: Improving Global Outcomes (KDIGO)** criteria were included.

Sample Size

The sample size was calculated using the single population proportion formula:

$$n = Z^2P(1-P)/d^2$$

Assuming an expected prevalence of 18%, 95% confidence level, and 5% absolute precision, the calculated sample size was **227**. After adding 10% for possible dropouts, the final sample size was **250 children**.

Inclusion Criteria

- Children aged 1 month to 18 years.
- AKI diagnosed according to KDIGO criteria.
- Written informed consent obtained from parents or legal guardians.

Exclusion Criteria

- Pre-existing chronic kidney disease.
- Congenital or acquired structural renal disease.
- Previous renal transplantation.
- Known medical or surgical kidney disorders.

Study Procedure

All eligible children admitted to the PICU were screened daily for AKI using KDIGO criteria. After obtaining informed consent, demographic details, clinical history, examination findings, and admission diagnosis were recorded in a structured proforma.

Baseline investigations included complete blood count, C-reactive protein, blood urea, serum creatinine, serum electrolytes, urine routine examination, and urine spot protein-to-creatinine ratio (SPCR). Serum creatinine was estimated by the **Jaffe method** at admission and repeated every 24 hours until recovery or discharge. Urine output was monitored hourly, and estimated glomerular filtration rate (eGFR) was calculated using the **modified Schwartz formula**.

Diagnosis and Staging of AKI

AKI was diagnosed according to **KDIGO criteria**, defined by any of the following:

- Increase in serum creatinine ≥0.3 mg/dL within 48 hours.
- Increase in serum creatinine to ≥1.5 times baseline within seven days.
- Urine output ≤0.5 mL/kg/hour for at least six hours.

AKI was classified as:

- **Stage 1:** Creatinine 1.5–1.9 times baseline or urine output ≤0.5 mL/kg/hour for 6–12 hours.
- **Stage 2:** Creatinine 2.0–2.9 times baseline or urine output ≤0.5 mL/kg/hour for >12 hours.
- **Stage 3:** Creatinine ≥3 times baseline, serum creatinine ≥4 mg/dL, eGFR <35 mL/min/1.73 m², urine output ≤0.5 mL/kg/hour for >24 hours, or anuria for >12 hours.

Data Collection and Follow-up

Clinical variables including etiology, shock, sepsis, metabolic acidosis, electrolyte abnormalities, nephrotoxic drug exposure, mechanical ventilation, inotropic support, peritoneal dialysis, duration of PICU and hospital stay, survival, and mortality were documented.

Children discharged from the hospital were followed up after **three months** with assessment of serum creatinine, eGFR, urine SPCR, and blood pressure to evaluate renal recovery. Patients with persistent renal abnormalities were referred to the Pediatric Nephrology Unit.

Outcome Measures

The primary outcomes were mortality and renal recovery. Secondary outcomes included identification of risk factors for AKI, association between AKI stage and clinical outcome, need for renal replacement therapy, duration of PICU and hospital stay, and renal status at three-month follow-up.

Ethical Considerations

The study was approved by the Institutional Ethics Committee of Indira Gandhi Institute of Child Health, Bengaluru. Written informed consent was obtained from the parents or legal guardians of all participants before enrolment.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using **R statistical software version 4.4.0**. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean ± standard deviation or median (interquartile range). Normality was assessed using the **Shapiro–Wilk test**. Associations between categorical variables were analysed using the **Chi-square test** or **Fisher's exact test**. Continuous variables were compared using the **Mann–Whitney U test** or **Kruskal–Wallis test** with **Dunn's post hoc test** where appropriate. A **p-value <0.05** was considered statistically significant.

RESULTS

A total of 250 children diagnosed with acute kidney injury according to the KDIGO criteria were enrolled and analysed. The mean age of the study population was 5.63 years, and more than half of the children were aged five years or younger. Males constituted 59.2% of the study population. Stage 3 AKI was the most frequently observed stage, and prerenal AKI was more common than intrinsic renal AKI. Overall mortality was 33.6%.

Table 1: Demographic and etiological profile of children with AKI

Variable	Number (N=250)	Percentage
Age group		
≤5 years	138	55.2
6–10 years	60	24.0
11–15 years	42	16.8
16–18 years	10	4.0
Sex		
Male	148	59.2
Female	102	40.8
Major causes of AKI		
Pneumonia	58	23.2
Acute gastroenteritis	41	16.4
Meningitis/encephalitis	18	7.2
Dengue	14	5.6
Infective hepatitis	11	4.4
Sepsis	11	4.4
Hematological causes	11	4.4
Trauma	11	4.4
Metabolic crisis	10	4.0
Congenital heart disease	9	3.6
Other causes	56	22.4

Among the 250 children, 138 (55.2%) were aged ≤5 years, followed by 60 (24.0%) aged 6–10 years. The mean age was 5.63 years. Males predominated, with a male-to-female ratio of 1.45:1. Pneumonia was the most common underlying cause of AKI, accounting for 58 (23.2%) cases, followed by acute gastroenteritis in 41 (16.4%) cases and meningitis or encephalitis in 18 (7.2%) cases. Acute gastroenteritis was the most frequent cause among children aged ≤5 years, whereas pneumonia was more commonly observed in children aged 6–10 years and 11–15 years. Poisoning and meningitis/encephalitis were relatively more frequent among adolescents aged 16–18 years.

Table 2: Clinical, laboratory, and renal profile of children with acute kidney injury (n = 250)

Parameter	n (%) / Mean ± SD
Clinical and laboratory abnormalities	
Metabolic acidosis	187 (74.8)
Shock	88 (35.2)
Hyperkalaemia	70 (28.0)
Oliguria	61 (24.4)
Hyponatraemia	61 (24.4)
Hypernatraemia	46 (18.4)
Culture-proven sepsis	42 (16.8)

Hypertension	14 (5.6)
Type of AKI	
Prerenal AKI	151 (60.4)
Intrinsic renal AKI	99 (39.6)
KDIGO stage	
Stage 1	46 (18.4)
Stage 2	51 (20.4)
Stage 3	153 (61.2)
Renal parameters	
Blood urea at admission (mg/dL)	**61.64 ± 51.31**
Serum creatinine at admission (mg/dL)	**1.02 ± 1.07**
Highest serum creatinine (mg/dL)	**1.59 ± 1.41**
Lowest eGFR (mL/min/1.73 m²)	**40.05 ± 26.04**

Metabolic acidosis was the most common biochemical abnormality, affecting 187 (74.8%) children, followed by hyperkalaemia in 70 (28.0%) and oliguria in 61 (24.4%). Hyponatraemia and hypernatraemia were observed in 61 (24.4%) and 46 (18.4%) children, respectively. Stage 3 AKI was the predominant category (61.2%), followed by Stage 2 (20.4%) and Stage 1 (18.4%), while prerenal AKI accounted for 60.4% of cases. The mean highest serum creatinine was 1.59 ± 1.41 mg/dL and the mean lowest eGFR was 40.05 ± 26.04 mL/min/1.73 m². The mean age decreased significantly with increasing AKI severity (7.02 years in Stage 1, 6.46 years in Stage 2, and 4.93 years in Stage 3; p=0.0139), indicating that severe AKI occurred more frequently in younger children.

Table 3: Treatment profile and its distribution according to AKI stage

Treatment parameter	Total N (%)	Stage 1 N (%)	Stage 2 N (%)	Stage 3 N (%)
Mechanical ventilation	121 (48.4)	16 (13.2)	30 (24.8)	75 (62.0)
Nephrotoxic drug exposure	127 (50.8)	21 (16.5)	22 (17.3)	84 (66.1)
Inotropic support	88 (35.2)	7 (7.9)	22 (25.0)	59 (67.1)
Peritoneal dialysis	39 (15.6)	2 (5.1)	3 (7.7)	34 (87.2)

More than half of the children had been exposed to nephrotoxic medications, while 121 (48.4%) required mechanical ventilation. Inotropic support was administered to 88 (35.2%) children, and 39 (15.6%) underwent peritoneal dialysis. The requirement for intensive supportive treatment increased with AKI severity. Among mechanically ventilated children, 75 (62.0%) had Stage 3 AKI. Similarly, 59 (67.1%) children receiving inotropes and 34 (87.2%) children undergoing peritoneal dialysis had Stage 3 AKI. These findings demonstrated that children with severe AKI had greater requirements for respiratory, circulatory and renal replacement support.

Table 4: Comparison of clinical, laboratory and treatment characteristics between survivors and non-survivors

Variable	Survivors (n=166)	Non-survivors (n=84)	P-value
Mean age, years	5.53 ± 4.97	5.81 ± 5.36	0.8096
Male sex	100 (60.2%)	48 (57.1%)	0.6953
Oliguria	25 (15.1%)	36 (42.9%)	<0.001
Hypertension	10 (6.0%)	4 (4.8%)	0.7941
Shock	22 (13.3%)	66 (78.6%)	<0.001
Hyperkalaemia	38 (22.9%)	32 (38.1%)	0.0114
Metabolic acidosis	110 (66.3%)	77 (91.7%)	<0.001
Mechanical ventilation	45 (27.1%)	76 (90.5%)	<0.001
Peritoneal dialysis	15 (9.0%)	24 (28.6%)	<0.001
Nephrotoxic drug exposure	74 (44.6%)	53 (63.1%)	0.0057
Inotropic support	22 (13.3%)	66 (78.6%)	<0.001
Highest creatinine, mg/dL	1.42 ± 1.18	1.94 ± 1.72	0.0081
Lowest eGFR, mL/min/1.73 m²	43.21 ± 26.97	33.81 ± 22.99	0.0066
Hospital stay, days	15.90 ± 12.05	10.32 ± 8.23	<0.001
PICU stay, days	6.90 ± 6.15	7.75 ± 5.38	0.048

Of the 250 children, 166 (66.4%) survived and 84 (33.6%) died. Age and sex were not significantly associated with mortality. Non-survivors had significantly higher rates of oliguria (42.9% vs. 15.1%), shock (78.6% vs. 13.3%), metabolic acidosis (91.7% vs. 66.3%), and hyperkalaemia (38.1% vs. 22.9%) (all p<0.05), while serum sodium abnormalities and hypertension showed no significant differences.

Non-survivors also had significantly higher peak serum creatinine (1.94 vs. 1.42 mg/dL) and lower minimum eGFR (33.81 vs. 43.21 mL/min/1.73 m²) (p<0.01). Mechanical ventilation (90.5% vs. 27.1%), inotropic support (78.6% vs. 13.3%), peritoneal dialysis (28.6% vs. 9.0%), and nephrotoxic medication exposure were significantly associated with mortality (p<0.001). Survivors had a longer mean hospital stay (15.9 vs. 10.3 days), whereas non-survivors had a slightly longer PICU stay (7.75 vs. 6.90 days).

Table 5: AKI stage, mortality and renal recovery

Outcome parameter	Number	Percentage
Overall outcome		
Survived/discharged	166	66.4
Death	84	33.6
AKI stage among survivors (n=166)		
Stage 1	37	22.3
Stage 2	33	19.9
Stage 3	96	57.8
AKI stage among non-survivors (n=84)		
Stage 1	9	10.7
Stage 2	18	21.4
Stage 3	57	67.9
Renal function at discharge among survivors (n=166)		
Normal renal function	128	77.1
Abnormal renal function	38	22.9
Renal function at 3-month follow-up (n=166)		
Normal renal function	161	97.0
Abnormal renal function	5	3.0

Mortality increased with AKI severity, with Stage 3 AKI accounting for the majority of deaths (67.9%), followed by Stage 2 (21.4%) and Stage 1 (10.7%). Severe AKI was also associated with delayed renal recovery. At discharge, 77.1% of survivors had normal renal function, while 22.9% had persistent abnormalities, most of whom (84.2%) had Stage 3 AKI. By the three-month follow-up, renal function had normalized in 97.0% of survivors, with only 3.0% showing persistent renal impairment requiring continued nephrology follow-up.

DISCUSSION

Acute kidney injury (AKI) remains one of the most serious complications among critically ill children admitted to the Paediatric Intensive Care Unit (PICU), contributing significantly to morbidity, mortality, prolonged hospitalization, and increased healthcare utilization. The present prospective observational study evaluated 250 children with AKI and demonstrated that younger age, severe infection, Stage 3 AKI, haemodynamic instability, metabolic abnormalities, and the need for intensive organ support were the major determinants of adverse outcomes.

More than half of the children (55.2%) were aged ≤5 years, and younger age was significantly associated with greater AKI severity. This finding is consistent with previous studies showing that younger children are more susceptible to AKI because of immature renal physiology, limited renal reserve, and increased vulnerability to dehydration and severe infections. Raina et al. reported that younger age and illness severity significantly influence both the occurrence and prognosis of pediatric AKI.[9] A male predominance (59.2%) was observed, although gender was not significantly associated with mortality, similar to previous reports.

Renal recovery among survivors was encouraging. Normal renal function was observed in 77.1% of children at discharge and improved to 97% after three months of follow-up. Nevertheless, children with severe AKI remain at risk of persistent renal dysfunction, hypertension, proteinuria, and chronic kidney disease. Greenberg et al. emphasized the importance of long-term renal surveillance following pediatric AKI, particularly in patients with severe disease or incomplete recovery.[10] Pneumonia (23.2%) was the most common underlying illness, followed by acute gastroenteritis (16.4%), reflecting the predominance of infectious diseases in developing countries. Severe infections may lead to AKI through hypoperfusion, systemic inflammation, sepsis, and nephrotoxic drug exposure. Sethi et al. identified sepsis, respiratory infections, multiorgan dysfunction, and nephrotoxic medications as important contributors.[11] while Mehta et al. highlighted infection and volume depletion as the principal causes of pediatric AKI in resource-limited settings,[12]

Prerenal AKI accounted for 60.4% of cases, suggesting that renal hypoperfusion remained the predominant mechanism of injury. However, persistent hypoperfusion may progress to intrinsic renal damage if not corrected promptly. Gameiro J et al. described AKI as a continuum from reversible functional impairment to irreversible structural injury when renal ischemia persists.[13]

A notable finding of the present study was the high proportion of Stage 3 AKI (61.2%). Advanced AKI was associated with oliguria, shock, metabolic acidosis, hyperkalaemia, higher serum creatinine, lower eGFR, mechanical ventilation, inotropic support, and dialysis requirement. Previous studies have consistently demonstrated that increasing AKI severity is associated with higher mortality and greater utilization of intensive care resources.[9,11,14]

Metabolic acidosis (74.8%) and hyperkalaemia (28%) were the most frequent biochemical abnormalities. These disturbances result from impaired renal excretion and tissue hypoperfusion and are recognized indicators of severe AKI requiring urgent management. Similarly, nephrotoxic medications were administered to 50.8% of children and were significantly more common among non-survivors. Contemporary

guidelines recommend minimizing unnecessary nephrotoxic exposure and adjusting drug doses according to renal function to reduce further kidney injury.[14-16]

Mechanical ventilation, inotropic support, and peritoneal dialysis were all significantly associated with mortality. These interventions primarily reflect severe multiorgan dysfunction rather than acting as independent causes of death. Selewski et al. demonstrated that fluid overload and the need for kidney replacement therapy are closely related to poor outcomes in critically ill children with AKI.[8]

The overall mortality rate was 33.6%, with significantly higher mortality among children with Stage 3 AKI, shock, oliguria, metabolic acidosis, hyperkalaemia, elevated serum creatinine, and reduced eGFR. These findings are consistent with international studies demonstrating that severe AKI independently predicts mortality in critically ill children.[9,11,14,15]

The strengths of the present study include its prospective design, relatively large sample size, standardized KDIGO-based diagnosis, and three-month follow-up. However, its single-centre design, absence of novel renal biomarkers, and limited follow-up period may restrict the generalizability of the findings. Overall, this study highlights the importance of early recognition of high-risk children, prompt correction of haemodynamic instability, avoidance of preventable nephrotoxicity, and structured post-discharge nephrology follow-up to improve both survival and long-term renal outcomes.

CONCLUSION

Acute kidney injury is a major cause of morbidity and mortality among critically ill children admitted to the PICU. Younger age, severe infections, Stage 3 AKI, shock, oliguria, metabolic acidosis, hyperkalaemia, nephrotoxic medication exposure, and the need for mechanical ventilation, inotropic support, or dialysis were associated with poor outcomes. Although most survivors showed satisfactory renal recovery within three months, continued follow-up is essential to detect persistent renal dysfunction. Early diagnosis using KDIGO criteria, timely management of modifiable risk factors, and structured nephrology follow-up can improve both survival and long-term renal outcomes.

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