Section: Paediatrics

Original Research Article

PREDICTION OF ACUTE KIDNEY INJURY AND ITS OUTCOME USING ACUTE RENAL ANGINA INDEX SCORE IN CRITICALLY ILL CHILDREN ADMITTED IN PAEDIATRIC INTENSIVE CARE UNIT AT A TERTIARY CARE CENTRE

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ABSTRACT

Background: Acute kidney injury (AKI) is a frequent complication in critically ill children, associated with prolonged intensive care stay and increased mortality. The Acute Renal Angina Index (ARAI) integrates clinical risk factors and early renal injury markers to predict AKI before biochemical deterioration. This study aimed to evaluate the utility of the ARAI for early AKI prediction and its association with short-term outcomes in critically ill children. **Materials and Methods:** This prospective observational cohort study was conducted 225 children in the PICU of the Government Chengalpattu Medical College and Hospital in India. Children aged 2 months to 12 years were enrolled over 12 months, and ARAI was calculated at admission, with AKI assessed at 48 h using the modified KDIGO criteria. **Result:** We included 225 children. Ninety children (40%) had positive ARAI scores (≥8) at admission. AKI developed in 58 children (25.8%) within 48 h, with stage 1 being the most common (12.0%). The incidence of AKI was significantly higher in ARAI-positive children than in ARAI-negative children (48.9% vs. 10.4%, $p < 0.001$). The ARAI demonstrated good diagnostic performance, with a sensitivity of 75.9%, specificity of 72.5%, and negative predictive value of 89.6%. Children with AKI had a longer PICU stay, higher need for mechanical ventilation, greater requirement for renal replacement therapy, and increased mortality. ARAI positivity, sepsis, fluid overload, and mechanical ventilation were independent AKI predictors. **Conclusion:** ARAI is a useful early risk stratification tool for predicting AKI and adverse outcomes in critically ill paediatric patients. Its application may support timely interventions and improve outcomes in resource-limited PICU settings in the future.

INTRODUCTION

Acute kidney injury (AKI) is a common complication in the Paediatric Intensive Care Unit (PICU). Children admitted to the PICU often present with shock, sepsis, respiratory failure, and multiple comorbid conditions.[1] The paediatric kidney is immature and sensitive to hypoperfusion, inflammation, and nephrotoxic exposure. Even brief episodes of renal stress can lead to a decline in renal function. Early identification of children at risk is essential because serum creatinine levels rise only after significant injury. Delayed recognition affects the opportunity for preventive measures.[2] Children with AKI commonly require mechanical ventilation and renal replacement therapy (RRT).[3] Mortality is higher in children who develop AKI during critical illness. A multinational study involving 4,683 PICU patients across 32 centres reported a fourfold increase in mortality among children with severe AKI compared to those without AKI (11% vs. 2.5%).[4] A modern tool for early prediction of AKI is important because the clinical deterioration can become well established by the time AKI is diagnosed using conventional criteria.

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The Acute Renal Angina Index (ARAI) is a bedside scoring system developed for this purpose. It includes risk factors such as mechanical ventilation, vasoactive drug use, and fluid overload with minor increases in serum creatinine to estimate AKI risk within 48 h of PICU admission.[5] ARAI identifies children who are vulnerable to AKI during the early phase of critical illness. Early recognition allows clinicians to closely monitor renal function and adjust management before the injury progresses. An ARAI score of ≥8 at admission predicts severe AKI on Day 2, with negative predictive values ranging from 80% to 95%. This allows the exclusion of low-risk children while focusing attention on those at higher risk. The addition of urinary biomarkers, such as neutrophil gelatinase–associated lipocalin, improves predictive specificity, reaching values as high as 0.97.[6] However, biomarker testing is costly and not routinely available in many PICUs.

Paediatric AKI differs from adult AKI because of differences in renal physiology, fluid balance, and electrolyte handling. Earlier paediatric studies relied on adult diagnostic thresholds, leading to delayed diagnosis in children.[7] Even transient AKI episodes in childhood have been associated with long-term renal sequelae, including chronic kidney disease and hypertension.[8] ARAI was developed specifically for paediatric populations to address these limitations and has been increasingly applied in PICU settings. Fluid overload is a common problem in critically ill children and is a key component of ARAI. It independently predicts prolonged ventilation, higher AKI incidence, and increased mortality, especially in Stage 3 AKI.[9] Early identification of AKI risk using ARAI supports fluid management decisions and timely initiation of RRT.[10]

In India, delayed referrals, high rates of critical illness, and limited nephrology access worsen the outcomes of AKI. Existing case reports link higher admission ARAI scores with Day-2 AKI, prolonged ventilation, and mortality; however, prospective data are limited. Therefore, this study aimed to assess the ability of the ARAI at PICU admission to predict AKI within 48 h and evaluate its association with clinical outcomes in critically ill children.

MATERIALS AND METHODS

This prospective observational cohort study included 225 children aged 2 months to 12 years admitted to the Paediatric Intensive Care Unit of Government Chengalpattu Medical College and Hospital, and was conducted from May 2024 to May 2025. The study received Institutional Ethics Committee approval, included only children with written parental consent, and maintained ethical standards and patient confidentiality throughout.

Inclusion and Exclusion Criteria

The study included children between 2 months and 12 years of age who required admission to the PICU for the management of haemodynamic instability. Children were excluded if they had chronic kidney disease, congenital or acquired structural kidney abnormalities, evidence of AKI at admission, or a history of RRT.

Methods: Data were collected at PICU admission using a pre-validated, standardised case record form. The information was categorised into demographic, clinical, physiological, and laboratory variables. Demographic data included age, sex, nutritional status, immunisation history, and family history of renal disease. The clinical details recorded included presenting symptoms, admission diagnosis, comorbid conditions, sepsis, shock, systemic illness, and exposure to nephrotoxic drugs. Physiological parameters measured at admission included heart rate, respiratory rate, blood pressure, oxygen saturation, capillary refill time, and the Glasgow Coma Scale. Laboratory investigations included complete blood count, serum creatinine, serum electrolytes, arterial blood gas analysis, liver function tests, urine examination, and blood or urine cultures when clinically indicated.

Serum creatinine was measured on day 0 of PICU admission and repeated at 48 h. Baseline creatinine was estimated, and when height data were unavailable, a height-independent method, such as the Normsmax method, was used. The Acute Renal Angina Index was calculated on day 0 using the product of the risk strata score and injury score. The risk strata included sepsis or septic shock, transplant or oncologic diagnosis, and the requirement of a fluid bolus >40 mL/kg or emergency department intubation. Injury scoring was based on fluid overload or an increase in serum creatinine levels from the baseline. An ARAI score ≥ 8 was considered positive. Children with positive ARAI results were prospectively followed up. AKI was diagnosed and staged at 48 h using the modified KDIGO criteria. The outcomes assessed included the duration of mechanical ventilation, need for RRT, and length of PICU stay.

Statistical analysis: Data were presented as mean, standard deviation, frequency, and percentage. Continuous variables were compared using the independent sample t-test. Categorical variables were compared using the Pearson chi-square test. Significance was defined by p-values <0.05 using a two-tailed test. Data analysis was performed using IBM-SPSS version 25.0 (IBM-SPSS Science Inc., Chicago, IL).

RESULTS

Most children admitted to the PICU were aged 1–5 years (45.3%), and the majority were male (58.2%). Normal nutritional status was observed in 44% of the children, and moderate malnutrition was observed in 38.7% [Table 1].

Sepsis or septic shock was the most common admission diagnosis (28.4%), followed by pneumonia with respiratory failure (21.8%). Most

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children had no comorbidities (58.7%), while congenital heart disease (11.6%) was the most frequent comorbidity [Table 2].

Table 1: Baseline demographic and nutritional characteristics

Variable	Category	N (%)
Age group (years)	1 month – <1 year	38 (16.9%)
	1 – 5 years	102 (45.3%)
	6 – 12 years	85 (37.8%)
Gender	Male	131 (58.2%)
	Female	94 (41.8%)
Nutritional status	Normal	99 (44%)
	Moderate malnutrition	87 (38.7%)
	Severe malnutrition	15 (6.7%)
	Overweight / Obese	24 (10.6%)

Table 2: Clinical profile and comorbid conditions at PICU admission

Variable	Category	N (%)
Primary diagnosis	Sepsis / Septic shock	64 (28.4%)
	Pneumonia with respiratory failure	49 (21.8%)
	Seizure disorder/status epilepticus	32 (14.2%)
	Acute gastroenteritis with dehydration	27 (12.0%)
	Dengue / viral haemorrhagic fevers	21 (9.3%)
	Encephalopathy/meningitis	14 (6.2%)
	Snakebite/envenomation	10 (4.4%)
	Others (trauma, poisoning, burns)	8 (3.6%)
Comorbidities	None	132 (58.7%)
	Congenital heart disease	26 (11.6%)
	Developmental delay / cerebral palsy	23 (10.2%)
	Tuberculosis / chronic infections	18 (8.0%)
	Haematological malignancies	14 (6.2%)
	Others (genetic syndromes)	12 (5.3%)

At admission, tachycardia and tachypnoea were common, with a mean heart rate of 138.2 ± 23.5 beats/min and a respiratory rate of 38.4 ± 8.2/min. Delayed capillary refill time was noted in 47 children (20.9%), while anaemia was the most frequent laboratory abnormality (32.9%), followed by leucocytosis (27.1%) [Table 3].

Table 3: Admission physiological and laboratory parameters

Parameter	Mean ± SD	Abnormal / N (%)
Heart rate (beats/min)	138.2 ± 23.5	-
Respiratory rate (/min)	38.4 ± 8.2	-
Systolic BP (mmHg)	94.1 ± 12.3	-
Diastolic BP (mmHg)	57.3 ± 10.6	-
SpO_2 (%) on room air	92.8 ± 5.4	-
Capillary refill time >3 s	-	47 (20.9%)
Haemoglobin (g/dL)	9.7 ± 1.6	74 (32.9%)
Total leukocyte count (/mm³)	12,540 ± 4,620	61 (27.1%)
Serum creatinine (mg/dL)	0.64 ± 0.21	-
Serum sodium (mEq/L)	133.4 ± 5.8	59 (26.2%)
Serum potassium (mEq/L)	4.6 ± 0.7	19 (8.4%)
Arterial pH	7.29 ± 0.12	51 (22.7%)

At PICU admission, 40% of children were classified as ARAI-positive, while 60% were negative. Among ARAI-positive patients, the most common risk component was intubation or large fluid bolus >40 mL/kg (44.4%), followed by sepsis/shock (31.1%) and oncologic/post-transplant status (24.4%). Serum creatinine rises ≥1.5× baseline were noted in about 37.8%. Most patients had no AKI (74.2%), followed by Stage 1 (12%), 2 (8.4%), and 3 (5.4%) AKI. Within 48 hours, 25.8% developed AKI overall. AKI occurred more often in the ARAI-positive group (48.9%) compared to the ARAI-negative group (10.4%) [Table 4].

Table 4: Distribution of ARAI scores and incidence of AKI

Variable	Category	N (%)
ARAI score category	< 8 (Negative)	135 (60%)
	≥ 8 (Positive)	90 (40%)
ARAI risk score components (ARAI-positive, n = 90)	Sepsis/shock (1)	28 (31.1%)
	Oncologic / post-transplant (3)	22 (24.4%)
	Intubation or fluid bolus >40 mL/kg (5)	40 (44.4%)

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Injury score (n = 90)	No Creatinine Rise (1)	30 (33.3%)
	1.0–1.5 × Baseline (2)	26 (28.9%)
	1.5–2.0 × Baseline (4)	20 (22.2%)
	>2.0 × Baseline (8)	14 (15.6%)
AKI status at 48 hours (KDIGO)	No AKI	167 (74.2%)
	Stage 1	27 (12.0%)
	Stage 2	19 (8.4%)
	Stage 3	12 (5.4%)
AKI incidence by ARAI status	ARAI ≥ 8 with AKI	44 (48.9%)
	ARAI < 8 with AKI	14 (10.4%)
Total AKI cases		58 (25.8%)

The ARAI showed high sensitivity (75.9%) and a strong negative predictive value (89.6%) for AKI prediction. Children who developed AKI had longer PICU stays (6.9 ± 2.8 days), higher need for mechanical ventilation (60.3%), RRT requirement (15.5%), and higher mortality (20.7%) (all $p < 0.001$). ARAI-positive children had significantly higher AKI development (48.9%), longer PICU stay (6.3 ± 2.6 days), greater ventilation requirement (58.9%), and increased mortality (16.7%) (all $p < 0.001$). On univariate analysis, significant risk factors for AKI included ARAI ≥ 8, sepsis, fluid overload >10%, nephrotoxic drug exposure, mechanical ventilation, and severe malnutrition (all $p < 0.05$) [Table 5].

Table 5: Diagnostic performance of ARAI and clinical outcomes by AKI and ARAI status

Analysis domain	Variable	Category / Group	Value / n (%)	p-value
Diagnostic performance of ARAI (≥8)	Sensitivity	-	75.90%	-
	Specificity	-	72.50%	-
	Positive predictive value	-	48.90%	-
	Negative predictive value	-	89.60%	-
	Accuracy	-	73.30%	-
Clinical outcomes based on the presence of AKI	Mean PICU stay (days)	Present	6.9 ± 2.8	<0.001
		Absent	4.1 ± 1.9	
	Mechanical ventilation	Present	35 (60.3%)	<0.001
		Absent	41 (24.5%)	
	RRT required	Present	9 (15.5%)	<0.001
		Absent	0 (0%)	
	Mortality	Present	12 (20.7%)	<0.001
		Absent	8 (4.8%)	
Clinical outcomes based on ARAI scores	AKI development	≥8	44 (48.9%)	<0.001
		<8	14 (10.4%)	
	Mean PICU stay (days)	≥8	6.3 ± 2.6	<0.001
		<8	4.3 ± 2.1	
	Mechanical ventilation	≥8	53 (58.9%)	<0.001
		<8	23 (17.0%)	
	Mortality	≥8	15 (16.7%)	<0.001
		<8	5 (3.7%)	
Univariate risk factors for AKI	ARAI ≥8	AKI present	44 (75.9%)	<0.001
		AKI absent	46 (27.5%)	
	Sepsis	AKI present	28 (48.3%)	<0.001
		AKI absent	36 (21.6%)	
	Fluid overload >10%	AKI present	22 (37.9%)	<0.001
		AKI absent	11 (6.6%)	
	Nephrotoxic drug use	AKI present	17 (29.3%)	0.004
		AKI absent	21 (12.6%)	
	Mechanical ventilation	AKI present	35 (60.3%)	<0.001
		AKI absent	41 (24.5%)	
	Severe malnutrition	AKI present	9 (15.5%)	0.003
		AKI absent	6 (3.6%)	

Multivariate analysis identified ARAI ≥8 (adjusted odds ratio [aOR] 6.42, $p < 0.001$), fluid overload >10% (aOR 5.34, $p < 0.001$), sepsis (aOR 2.89, $p = 0.005$), and mechanical ventilation (aOR 2.17, $p = 0.037$) as independent predictors of AKI [Table 6].

Table 6: Multivariate predictors of AKI

Predictor Variable	aOR	95% Confidence Interval	p-value
ARAI ≥8	6.42	3.02 – 13.64	<0.001
Sepsis	2.89	1.38 – 6.08	0.005
Fluid overload >10%	5.34	2.02 – 14.12	<0.001
Mechanical ventilation	2.17	1.04 – 4.52	0.037

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DISCUSSION

Acute kidney injury is a common and serious complication among critically ill children admitted to the PICU. This study evaluated the effectiveness of ARAI at PICU admission for early prediction of AKI and its association with short-term clinical outcomes. The findings demonstrated that ARAI positivity was strongly associated with subsequent AKI development, longer PICU stay, increased requirement for mechanical ventilation and renal replacement therapy, and higher mortality, highlighting its value in guiding timely preventive strategies and improving outcomes in resource-limited critical care settings.

The study found that AKI was common among critically ill children admitted to the PICU, particularly in the 1–5 year age group (45.3%), with a male predominance (58.2%). Similarly, the AWAKEN study by Jetton et al. reported a high prevalence of AKI among neonates and young children in low- and middle-income countries, which was associated with delayed presentation and infectious illnesses.[11] Thus, indicating that younger children are more vulnerable to AKI because of their immature renal physiology and limited compensatory mechanisms.

In our study, many children were malnourished, with moderate and severe malnutrition present in 45.4% of the cases. Alobaidi et al., in a study, identified malnutrition as an important risk factor for AKI and adverse outcomes in critically ill children, consistent with our observations.[12] Malnutrition reduces the renal reserve and increases susceptibility to inflammatory injury. These findings highlight the importance of including nutritional assessment in AKI risk stratification.

Our study showed that sepsis and respiratory failure were the most common primary diagnosis, responsible for more than half of the PICU admissions. Zarbock et al. described sepsis as a major contributor to AKI development and poor outcomes in pediatric critical care, findings that closely align with our cohort.[13] Alobaidi et al. found fluid overload, often associated with sepsis management, further increases the risk of AKI.[12] Sepsis-associated AKI results from systemic inflammation, endothelial injury and microvascular dysfunction. Therefore, early identification of high-risk children and careful fluid stewardship, hemodynamic optimisation, and renal monitoring are essential to reduce AKI incidence and improve clinical outcomes in critically ill paediatric patients.

In our study, haemodynamic instability was frequent at admission, with a mean heart rate of 138.2 ± 23.5 bpm (tachycardia) and delayed capillary refill observed in 20.9% of children. Although the mean serum creatinine values were within normal limits at admission, metabolic abnormalities such as anaemia, hyponatraemia, and acidosis were common. Similarly, Raina et al. emphasise that hemodynamic instability and fluid imbalance (tachycardia, poor perfusion, fluid overload) are common in paediatric ICU patients and are associated with organ dysfunction, including AKI. Fluid imbalances can arise from both disease severity and resuscitation efforts, and they frequently precede or exacerbate AKI.[14] Therefore, early hemodynamic optimisation, careful fluid balance, and close monitoring of perfusion and electrolyte status are essential to identify high-risk children promptly and prevent progression to AKI.

In this study, 40% of the children (90 of 225) were ARAI-positive at admission, from which 48.9% (44 of 90) developed AKI. Basu et al. reported similar ARAI positivity rates in critically ill pediatric populations, particularly among septic and mechanically ventilated patients.[5] Goldstein et al. described ARAI as a tool that integrates clinical risk and early injury markers, allowing early identification of vulnerable children before overt renal dysfunction develops.[15] Kaddourah et al. reported an AKI incidence of approximately 27% in critically ill children.2 Selewski et al. confirmed the applicability of KDIGO criteria for AKI staging in pediatric intensive care, supporting the staging approach used in this study.[16] Basu et al. show AKI occurred significantly more often in ARAI-positive children than in ARAI-negative children (48.9% vs. 10.4%), and they also demonstrated strong predictive performance and high negative predictive value of ARAI.[5] Thus, suggesting that ARAI helps in the early identification of children at risk for kidney injury. Routine ARAI assessment at PICU admission may facilitate earlier monitoring, targeted preventive strategies, and timely interventions to reduce AKI incidence and improve outcomes in critically ill children.

In this study, children who developed AKI had longer PICU stays, higher need for mechanical ventilation, increased requirement for RRT, and higher mortality. Similarly, Kaddourah et al. show that associations between AKI and adverse clinical outcomes have been reported in a large paediatric population.[2] Ronco et al. emphasised that early recognition and timely intervention may reduce AKI-related morbidity and mortality, and supported the clinical relevance of early risk identification.[17] Thus, the delayed diagnosis of AKI can prolong PICU stay, increase the need for mechanical ventilation and RRT, and result in higher mortality.

Our study showed that in the multivariate analysis, ARAI ≥8, fluid overload greater than 10%, sepsis, and mechanical ventilation were independent predictors of AKI. Similarly, Basu et al. reported a comparable magnitude of risk associated with ARAI positivity in multinational paediatric studies.[5] Keneni et al. found that sepsis and mechanical ventilation were independently associated with the development of AKI in critically ill children. In multivariable logistic regression, children admitted with sepsis or infection had significantly higher odds of developing AKI, and mechanically ventilated

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children also had greater AKI risk compared to those who were not ventilated. These findings confirm the utility of ARAI as an early, bedside risk stratification tool for predicting AKI and guiding preventive strategies in resource-limited PICU settings.[18] Thus, indicating that routine ARAI assessment at PICU admission, combined with fluid management and treatment of sepsis and respiratory failure, may enable earlier monitoring, targeted preventive strategies, and timely interventions to reduce AKI incidence and improve clinical outcomes in critically ill children.

Limitations: The single-centre design limits generalisability. Short follow-up captured only early AKI. Urine output data were also excluded. Biomarkers were not available. Residual confounding from unmeasured illness severity may have persisted in this study population.

CONCLUSION

Acute kidney injury is common in critically ill children and is associated with increased morbidity and mortality. The ARAI reliably identified children at a high risk of early AKI. Its strong negative predictive value supports the safe exclusion of low-risk cases. Early ARAI-based risk stratification may guide timely fluid management and provide renal support. Future multicentre studies with longer follow-up periods are recommended. The integration of biomarkers may further improve early prediction.

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