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Original Research Article

Renal angina index versus serum creatinine for early prediction of acute kidney injury in critically ill children: a prospective observational study

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ABSTRACT

Background: Acute kidney injury (AKI) is common in critically ill children, while serum creatinine is a delayed marker of renal dysfunction. The Renal Angina Index (RAI) combines clinical risk and early signs of renal injury and may permit earlier identification of children likely to develop severe AKI.
Methods: This prospective observational study included 150 children aged 1 month-12 years admitted to a tertiary-care pediatric intensive care unit. RAI was calculated 12-24 hours after admission; a score ≥8 was considered positive. The primary outcome was severe AKI (KDIGO stage 2 or 3) by Day 3. Diagnostic performance of RAI was compared with Day-1 serum creatinine using receiver operating characteristic analysis.
Results: AKI occurred in 55/150 (36.7%) children and 52/150 (34.7%) were RAI positive. Mean RAI was higher in children with AKI than without AKI (9.65±3.23 vs 3.05±1.78; p<0.001). RAI had sensitivity 81.8%, specificity 92.6%, positive predictive value 86.5%, negative predictive value 89.8%, and accuracy 88.7%. Its area under the ROC curve was 0.928 (95% CI 0.887-0.969), compared with 0.307 (95% CI 0.208-0.405) for Day-1 serum creatinine.
Conclusions: RAI was a strong early predictor of AKI in critically ill children and substantially outperformed Day-1 serum creatinine. As a low-cost bedside score using routinely available data, it may facilitate early risk stratification in resource-limited PICUs.

Keywords: Acute kidney injury, Renal angina index, Pediatric intensive care, Serum creatinine, KDIGO, Early prediction

INTRODUCTION

Acute kidney injury (AKI) is a frequent and clinically important complication in critically ill children. It is associated with prolonged hospitalization, greater ventilator dependence, increased healthcare utilization, mortality, and subsequent risk of chronic kidney disease.[1-3] Standardized systems including RIFLE, AKIN and Kidney Disease: Improving Global Outcomes (KDIGO) define AKI using changes in serum creatinine and urine output. However, serum creatinine is an imperfect early biomarker: its rise may lag behind renal injury and it is influenced by age, muscle mass, hydration and other clinical factors.[4] Several biomarkers, including neutrophil gelatinase-associated lipocalin, kidney injury molecule-1 and interleukin-18, have been investigated for earlier AKI detection. Their routine use, however, may be constrained by cost, availability and laboratory requirements, particularly in resource-limited settings.[5] The renal angina index (RAI) was developed to improve early risk stratification by combining a patient's clinical risk with early evidence of renal injury, including fluid overload and changes in renal function.[6] A positive score may therefore identify high-risk patients before overt

creatinine-defined AKI develops. This approach is attractive in pediatric intensive care because it uses readily available bedside information and can guide intensified monitoring and renal-protective measures. The present study evaluated the ability of RAI assessed within the first 24 hours of pediatric intensive care unit (PICU) admission to predict severe AKI by day 3 and compared its predictive performance with day-1 serum creatinine.

METHODS

Study design and setting

This was a prospective observational study conducted in the PICU of a tertiary-care teaching hospital over 18 months. A total of 150 children were enrolled. The calculated minimum sample size was 147, based on an expected 10% prevalence of severe AKI, an alpha error of 0.05 and allowance for 5% attrition.

Participants

Consecutive children aged 1 month to 12 years who remained in the PICU for at least 8 hours and had documented body weight and intake-output data were eligible. Children with known chronic renal disease, severe AKI (KDIGO stage 2 or 3) at presentation, or ongoing dialysis were excluded.

Data collection

Data were recorded using a predesigned case-record form and included age, sex, weight, primary diagnosis, vital signs, hemodynamic status, respiratory and inotropic support, serum creatinine, blood urea, electrolytes, urine output and fluid balance. Serum creatinine and urine output were monitored through day 3.

Renal angina index

RAI was calculated 12-24 hours after PICU admission as the product of a renal risk score and a renal injury score. The risk component reflected severity of illness, including mechanical ventilation and/or inotropic support; the injury component incorporated percentage fluid overload during the early admission period or decline in estimated creatinine clearance from baseline. An RAI ≥8 was considered renal-angina positive and <8 renal-angina negative.

Outcome

The primary outcome was development of severe AKI (KDIGO stage 2 or 3) by Day 3. AKI diagnosis and staging were based on KDIGO serum creatinine and urine-output criteria.

Statistical analysis

Analysis was performed using SPSS version 23. Continuous variables were summarized as mean±standard deviation and categorical variables as frequency and percentage. Normality was assessed using the Kolmogorov–Smirnov test. Independent-samples t test, one-way ANOVA and chi-square test were used as appropriate. Receiver operating characteristic (ROC) analysis assessed predictive performance. A p value ≤0.05 was considered statistically significant.

Ethics

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from parents or guardians before enrollment.

RESULTS

The study included 150 critically ill children with a mean age of 5.21±3.15 years; 90 (60%) were male. Dengue fever (36%), sepsis (11.3%) and bronchopneumonia (8%) were the leading admission diagnoses (Table 1).

Table 1: Baseline characteristics and principal outcomes of the study population.

Predictors	AUC	95% CI	P value
Day-1 serum creatinine	0.307	0.208–0.405	<0.001
Renal Angina Index	0.928	0.887–0.969	<0.001

AKI developed in 55 (36.7%) children, while 95 (63.3%) did not develop AKI. In children who developed AKI, mean serum creatinine increased from 0.35±0.055 mg/dl at baseline to 0.53±0.117 mg/dl on day 1 and 1.08±0.314 mg/dl on day 3 (p<0.001). In the non-AKI group, creatinine increased transiently from 0.39±0.034 mg/dl at baseline to 0.60±0.086 mg/dl on Day 1 and decreased to 0.43±0.041 mg/dl on Day 3 (Table 2).

Table 2: Serum creatinine trends according to AKI status.

Measures	Value
Sensitivity	81.8%
Specificity	92.6%
Positive predictive value	86.5%
Negative predictive value	89.8%
Accuracy	88.7%

Fifty-two (34.7%) participants were RAI positive and 98 (65.3%) were RAI negative. Mean RAI was significantly higher among children who developed AKI than among those who did not (9.65±3.23 vs 3.05±1.78; p<0.001). RAI positivity was strongly associated with AKI (p<0.001): 45 of 52 RAI-positive children developed AKI, whereas 10 of 98 RAI-negative children developed AKI (Table 3). RAI yielded a sensitivity of 81.8%,

specificity of 92.6%, positive predictive value of 86.5%, negative predictive value of 89.8% and overall diagnostic accuracy of 88.7% (Table 4). ROC analysis showed excellent discrimination for RAI (AUC 0.928; 95% CI 0.887–0.969; p<0.001), whereas Day-1 serum creatinine showed poor discrimination (AUC 0.307; 95% CI 0.208–0.405; p<0.001) (Figures 1 and 2) (Table 5).

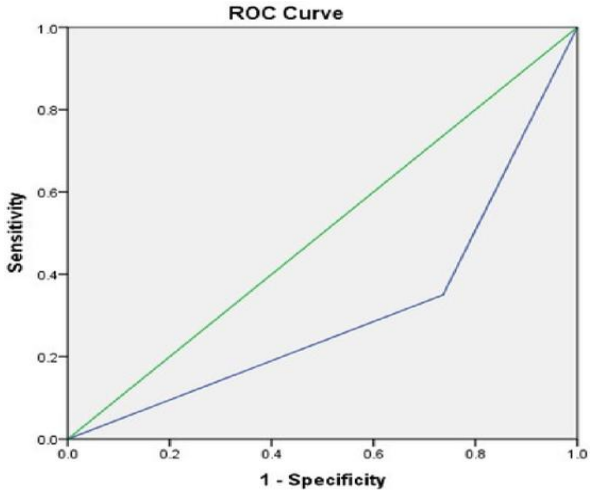

Figure 1: ROC analysis of day-1 mean serum creatinine levels for acute kidney injury.

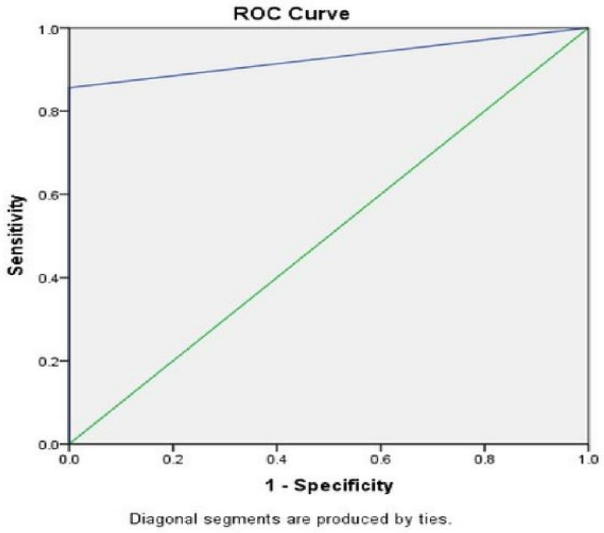

Figure 2: ROC analysis of renal angina index levels for acute kidney injury.

Table 3: Association between renal angina index and acute kidney injury.

Time points	AKI present, mean±SD (mg/dl)	AKI absent, mean±SD (mg/dl)	P value
Baseline	0.34±0.055	0.39±0.033	<0.001
Day 1	0.52±0.117	0.60±0.085	<0.001
Day 3	1.08±0.313	0.43±0.041	<0.001

Table 4: Diagnostic performance of renal angina index for acute kidney injury.

Time point	AKI present, mean±SD (mg/dl)	AKI absent, mean±SD (mg/dl)	P value
Baseline	0.34±0.055	0.39±0.033	<0.001
Day 1	0.52±0.117	0.60±0.085	<0.001
Day 3	1.08±0.313	0.43±0.041	<0.001

Table 5: ROC analysis comparing day-1 serum creatinine and renal angina index.

Characteristics	Value
Participants	150
Age, mean±SD (years)	5.21±3.15
Male sex	90 (60%)
Dengue fever	54 (36%)
Sepsis	17 (11.3%)
Bronchopneumonia	12 (8%)
Developed AKI	55 (36.7%)
RAI positive (≥8)	52 (34.7%)

DISCUSSION

In this prospective cohort of critically ill children, RAI measured during the first 24 hours of PICU admission demonstrated strong predictive performance for subsequent AKI. Approximately one-third of the cohort developed AKI, and children with AKI had substantially higher RAI scores than those without AKI. The central finding was the marked difference in discrimination between RAI and day-1 serum creatinine: RAI achieved an AUC of 0.928, whereas day-1 creatinine had an AUC of only 0.307.

The observed AKI prevalence of 36.7% is similar to that reported by Kumar et al and Mehta et al although pediatric studies have reported widely varying rates depending on illness severity, case mix and AKI definitions.[29] Our cohort was characterized by a high burden of infectious and hemodynamically stressful illnesses, particularly dengue and sepsis, which may partly explain the substantial AKI burden.

Serum creatinine remained useful for documenting progression of established renal dysfunction. In the AKI group it rose progressively through day 3, while the Day-1 increase in the non-AKI group was largely transient. This illustrates an important limitation of isolated early creatinine measurements: short-term changes may reflect hydration and hemodynamic factors, while a diagnostically meaningful rise may occur only after renal injury is established.

RAI was positive in 34.7% of participants and showed sensitivity of 81.8%, specificity of 92.6%, PPV of 86.5%, NPV of 89.8% and accuracy of 88.7%. These estimates closely resemble those reported by Kumar et al who found sensitivity 83.33%, specificity 92.47%, PPV

86.54%, NPV 90.53% and accuracy 89.12%.[28] Sankannavar et al also reported high specificity and NPV, although sensitivity was lower.[1] Such differences may reflect patient characteristics, disease severity, timing of RAI assessment and AKI definitions.

The excellent AUC for RAI in our study is consistent with previous pediatric validation work. Gawadia et al reported an AUC of approximately 0.90, while other studies have similarly shown that RAI improves early identification of children at risk for severe AKI.[2] Basu et al demonstrated the value of integrating clinical risk with early renal injury to enrich the population in whom severe AKI subsequently develops.[24] Collectively, these observations support the concept that risk stratification can be more informative early in critical illness than waiting for a delayed rise in serum creatinine.

RAI has practical advantages for resource-constrained PICUs. It uses routinely available clinical and laboratory information, requires no specialized biomarker assay, and can be calculated early after admission. A positive RAI can therefore prompt closer fluid and urine-output monitoring, review of nephrotoxic exposures and optimization of hemodynamics. Importantly, RAI should be regarded as a risk-prediction tool rather than a replacement for KDIGO diagnostic criteria.

The strengths of this study include its prospective design, standardized KDIGO outcome assessment, early RAI calculation and serial assessment of renal function. Limitations include the single-center design and sample size of 150, which may restrict generalizability. Children with pre-existing chronic renal disease or severe AKI at presentation were excluded, and follow-up was limited to early AKI through Day 3; long-term renal outcomes were not evaluated. RAI calculation may also be affected by the accuracy of fluid-balance and clinical measurements.

CONCLUSION

In conclusion, RAI assessed within the first 24 hours of PICU admission was a strong early predictor of AKI and showed substantially better discrimination than Day-1 serum creatinine. Its simplicity, low cost and reliance on routinely available bedside data make it a potentially useful tool for early AKI risk stratification, particularly in resource-limited pediatric critical care settings. Larger multicenter studies should evaluate its performance across broader case mixes and its effect on clinically important outcomes.

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