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ROLE OF RENAL ANGINA INDEX IN PREDICTING THE SEVERITY AND OUTCOME OF ACUTE KIDNEY INJURY IN NEONATES

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Abstract

Introduction: The diagnosis of AKI is a problem, as diagnosis depends on two functional defects; increase in serum creatinine and decrease in urine output (oliguria) especially in neonates. Both are late outcomes of the injury and not predictors of the injury. Regardless of appropriate treatment, the mortality rate may range from 25-50%, while survivors may suffer from problems in the long run. Recognizing early parameters that are able to detect kidney injury before the rise of serum creatinine, has been studied in recent decades to improve patient management and patient outcome. Renal angina is a method used clinically to predict AKI in critically ill neonates.

Materials and methods: An Institutional based Retrospective study was conducted in all critically ill neonates receiving level II and III care in the NICU. The data will be collected from the medical records department of the study institution. All the case records of term neonates requiring level II and III care in the NICU of the study institution will be reviewed and the" renal angina index" will be calculated on the day 1 of admission. These neonates will be followed up for the development of acute kidney injury until 7 days of life or post admission. The sensitivity and specificity of the renal angina index in predicting the risk of development of acute kidney injury in critically ill neonates will be assessed. A cut off value of renal angina index will be calculated for predicting the risk of acute kidney injury in neonates requiring level II and III care.

Results: 90 patients admitted to the NICU during the study period based on inclusion criteria. In present study 56.66% (n=51) were male, 39 (43.33%) were females. The majority of children aged 2-3 days 34(37.77%). The mean age of the participants was 3.8 days (SD=4.05). The median length of the neonates was 40 cm (QR=4 cm) and median weight was 2.7 kg (QR=0.7). Body weight Less than 2 kg were in 6 (6.66%), 2-3 kg were in 56 (62.22%), 3-4 kg were 28 (31.11%).

Conclusion: The RAI is a simple yet reliable predictor of the development of AKI in critically ill children. A positive RAI score emerges as a superior tool to help in the early reorganization of development AKI. This score is more useful in developing countries because it requires fewer investigations and can be easily applied in resource settings. The discriminative accuracy of RAI surpasses that of traditional creatinine-based renal injury parameters. Additionally, RAI has an independent predictive value for AKI severity, morbidity, and mortality in critically ill children.

Key Words: AKI, renal angina index, morbidity, and mortality, NICU.

INTRODUCTION

The diagnosis of AKI is a problem, as diagnosis depends on two functional defects; increase in serum creatinine and decrease in urine output (oliguria) especially in neonates. Both are late outcomes of the injury and not predictors of the injury. Regardless of appropriate treatment, the mortality rate may range from 25– 50%, while survivors may suffer from problems in the long run.[1] Recognizing early parameters that are able to detect kidney injury before the rise of serum creatinine, has been studied in recent decades to improve patient management and patient outcome. Renal angina is a method used clinically to predict AKI in critically ill neonates. For prediction of infants who are at high risk of developing severe AKI, Renal angina index can be used which can be calculated in an easy way. It is the product of scores for the risk of AKI and clinical signs of injury. The score ranges between 1and 40 and score more than 8 is considered a cut off value that predicts renal angina.[2]

Despite increasing awareness of the prevalence and significance of AKI, effective therapies for this condition are lacking. This, at least in part, results from a failure to recognize AKI before a significant degree of renal damage has already occurred. The inability to diagnose AKI expeditiously follows from the fact that the currently accepted definitions of AKI rely on changes in serum creatinine (SCr) and urine output.[2]

Increasing AKI severity, characterized by serum creatinine (SCr) and urine output (UOP) based stratifications of AKI, is associated with increased mortality in adults and children.[3] Even small increase in SCr (0.3 mg/dl) reflects significant kidney damage and is associated with poor patient outcomes. The well- recognized limitations of SCr for real-time accurate AKI diagnosis have prevented timely therapeutic interventions.[4]

Extensive research has targeted the discovery of biomarkers to disclose AKI prior to elevations in serum creatinine. Till date, a number of promising urinary AKI biomarkers have emerged, of which clinical studies indicate that urinary Neutrophil Gelatinase-associated Lipocalin (NGAL), Kidney Injury Molecule-1 (KIM-1), Interleukin 18 (IL-18) and Liver-type Fatty Acid Binding Protein (L-FABP) all predict AKI in children following cardiopulmonary bypass where demographic homogeneity, lack of comorbidities, and a known onset and duration of ischemic injury prior to changes in serum creatinine.[5]

AIMS AND OBJECTIVES

1. To know the sensitivity and specificity of "Renal Angina Index" in detecting the risk of occurrence of acute kidney injury in critically ill neonates

2.To know the incidence, risk factors and outcome of acute kidney injury in neonates under level II and III care in NICU , department of pediatrics, Kurnool medical college.

MATERIALS AND METHODS

Study setting: Institutional based

Study Design: Retrospective study

Study Population: All critically ill neonates receiving level II and III care in the NICU.

Study period: 6 months

Inclusion criteria:

1. All term neonates receiving level II and III care in the NICU of pediatric department, Kurnool medical college, Kurnool.

Exclusion criteria:

1. All term neonates requiring level I care.

2. All preterm neonates.

3. Neonates with preexisting genitourinary anomalies.

Data collection procedure:

The data will be collected from the medical records department of the study institution. All the case records of term neonates requiring level II and III care in the NICU of the study institution will be reviewed and the" renal angina index" will be calculated on the day 1 of admission. These neonates will be followed up for the development of acute kidney injury until 7 days of life or post admission. The sensitivity and specificity of the renal angina index in predicting the risk of development of acute kidney injury in critically ill neonates will be assessed. A cut off value of renal angina index will be calculated for predicting the risk of acute kidney injury in neonates requiring level II and III care.

Statistical Analysis:

Statistical Package of Social Science (SPSS) Software program will be used for data analysis. Mean and standard deviation will be used to express Quantitative data (if parametric) while median and interquartile ranges will be used to express quantitative data (if nonparametric).

Frequencies and percentages will be used to express qualitative data. Group comparison was performed using independent sample t-test and one-way ANOVA test (if parametric) and Chi square or Fisher's exact test for qualitative ones. Construction of ROC curves will be done for the renal angina index with area under the curve analysis. Significant P-value was less than 0.05.

RESULTS

90 patients admitted to the NICU during the study period based on inclusion criteria. In present study 56.66% (n=51) were male, 39 (43.33%) were females. The majority of children aged 2-3 days 34(37.77%). The mean age of the participants was 3.8 days (SD=4.05). The median length of the neonates was 40 cm (QR=4 cm) and median weight was 2.7 kg (QR=0.7). Body weight Less than 2 kg were in 6 (6.66%), 2-3 kg were in 56 (62.22%), 3-4 kg were 28 (31.11%).

S.No	Age group	N (%)
1	Less than 1 day	22 (24.44%)
2	2-3 days	34 (37.77%)
3	3-5 days	13 (14.44%)
4	6-10 days	21 (23.33%)

Tablet 1: Age distribution of patients

S.No	Gender	N (%)
1	Male	51 (56.66%)
2	Female	39 (43.33%)

Tablet 2: Gender distribution of patients

S.No	Body weight	N (%)
1	Less than 2 kg	6 (6.66%)
2	2-3 kg	56 (62.22%)
3	3-4 kg	28 (31.11%)

Table 3: Body weight (in kg)

S.No	Gestational age at birth	N (%)
1	37 weeks	15 (16.66%)
2	38 weeks	19 (21.11%)
3	39 weeks	38 (34.44%)
4	40 weeks	18 (20%)

Table 4: Gestational age at birth

15 (16.66%) has 37 weeks of Gestational age at birth, 19 (21.11%) has 38 weeks of Gestational age at birth, 38 (34.44%) has 39 weeks of Gestational age at birth, 18 (20%) has 39 weeks of Gestational age at birth.

S.No	Baby on mechanical ventilation	N (%)
1	Yes	9 (10%)
2	No	81 (90%)

Table 5: baby on mechanical ventilation

9 (10%) of babies were on mechanical ventilation, 81 (90%) are not on Ventilation.

S.No	Sepsis	N (%)
1	Yes	64 (71.11%)
2	No	26 (28.88%)

Table 6: Sepsis

64 (71.11%) case had sepsis, 6 (6.66%) had Peritoneal dialysis.

S.No	P.D (peritoneal dialysis)	N (%)
1	Yes	6 (6.66%)
2	No	84(93.33%)

Table 7: P.D (peritoneal dialysis)

S.No	Complications	N (%)
1	Medullary nephrocalcinosis uremic encephalopathy SSVT(Superior sagittal sinus thrombosis)	3 (3.33%)
2	Medullary nephrotic edema	6 (6.99%)
3	Perinatal asphyxia	24 (26.66%)
4	Perinatal asphyxia and Coagulase neg s aureus	3 (3.33%)
5	Perinatal asphyxia and MRSA	3 (3.33%)
6	No complications	51 (56.66%)

Tablet 8: Complications

S.No	Percentage fluid	N (%)
1	3-4	6 (6.99%)
2	4-5	6 (6.99%)
3	5-6	7 (7.77%)
4	6-7	18 (20%)
5	7-8	15 (16.66%)
6	9-10	15 (16.66%)

7	10-11	14 (15.55%)
8	11-12	9 (10%)

Table 9: Percentage fluid

S.No	Risk Score	N (%)
1	1	60 (66.66%)
2	6	30 (33.33%)

Table 10: Risk Score

S.No	RIA	N (%)
1	3-5	15 (16.66%)
2	6-7	24 (26.66%)
3	7-8	15 (16.66%)
4	8-9	6 (6.66%)
5	9-10	9 (10%)
6	12	3 (3.33%)

Table 11: RIA

S.No	Serum creatinine	Mean	SD	Median	Quartile range
1	Serum creatinine at day 0	0.40	0.18	0.37	0.25
2	Serum creatinine at day 3	0.57	0.56	0.36	0.34
3	Z value	1.4680			
4	P value	0.1421			

Table 12: Compression of serum creatinine levels on day 0 and day 3

S.No	Statistic	Value	95% CI for OR
1	Sensitivity	65.38%	44.33% to 82.79%
2	Specificity	88.06%	77.82%-94.70%
3	Positive likelihood ratio	5.48%	2.70 to 11.11
4	Negative likelihood ratio	0.39%	0.23-0.67
5	Positive predictive value	68%	51.15%-81.18%
6	Negative predictive	86.76%	79.33%-91.80%

	value		
7	Accuracy	81.72%	72.35% to 88.98%

Table 13: Sensitivity, specificity, accuracy, and positive and negative predictive values of RAI

S.No	Outcome	N (%)
1	Recovered	90 (100%)
2	No recovered	0

Table 14: Outcome

90 (100%) patients were recovered from acute kidney injury in neonates**.**

DISCUSSION

The present study is a hospital-based retrospective study. Results showed that a positive RAI obtained at admission was useful in predicting the development of AKI by day 3. RAI outperformed conventional baseline SCr level predicting AKI. The study, which included children with a similar range of disease severity (as assessed using PIM-3 score at admission), had a notably higher proportion of younger children in the RAI-positive group than in the RAI-negative group.[6]

In the present study, 22.58% of children admitted to the NICU developed AKI on day 3 admission. Compared with previous studies done by Gawadia et al.1, Basu et al.2, Mehta et al.13 and Naik et al.4, the incidences of AKI were 70%, 13.6%, 36.1%, and 90%, respectively. This variation in the incidence of AKI may be attributed to several factors, such as diagnosis at admission, presence of FO, use of nephrotoxic drugs, presence of sepsis, and MODS. The incidence of AKI increases with increasing severity of illness. Severe AKI was observed in 80.77% of RAI-positive children.[7]

These study results are in line with the studies conducted by Gawadia et al.[1] and Menon et al.[14], where severe AKI was observed in 72% and 80% respectively. In present study, approximately 80% of children with positive RAI developed AKI on day 3 of admission. These results are in good agreement with the studies of Gawadia et al.[1] and Basu et al.[2] However, predictive value was lower in studies by Sethi et al. and Kaur et al. The predictive ability of RAI in the development of severe AKI on day 3 was AUC 0.76 with 95% confidence interval (CI) of 0.72-0.88, which was similar to the studies by Basu et al. (AUC=0.86, with 95% CI of 0.75-0.86) and Sethi et al.16 (AUC =0.73, 95% CI of 0.61-0.82).[8]

In the present study, 26 children were RAI positive, among whom 10 (38.46%) developed ARDS, 24 (92.3%) required mechanical ventilation, and 18 (69.23%) required one or more inotropic supports. The study by Gawadia et al. showed that among children with positive RAI,

71% required mechanical ventilation and 78% needed inotropic support. In another study by Menon et al.14 showed 1.68% required mechanical ventilation, and 8.6% had a prolonged hospital stay among RAI-positive children.[9]

The present study observed that there was zero mortality among RAI-positive children, These results correlate with the study by Gawadia et al and Menon et al. where the mortality rates were 24% and 18.3% respectively.[10]

CONCLUSION

The RAI is a simple yet reliable predictor of the development of AKI in critically ill children. A positive RAI score emerges as a superior tool to help in the early reorganization of development AKI. This score is more useful in developing countries because it requires fewer investigations and can be easily applied in resource settings. The discriminative accuracy of RAI surpasses that of traditional creatinine-based renal injury parameters. Additionally, RAI has an independent predictive value for AKI severity, morbidity, and mortality in critically ill children.

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