e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(1); 214-217

Original Research Article

Predictors of Outcomes of Neonatal Acute Kidney Injury in Tertiary Care Hospital

Akash Parashar[1], Sunita Khandelwal[2], Anjali Singh[3], Jai Singh[4]

[1]Junior Resident, Department of Pediatrics JK loan Hospital, Kota, Rajasthan, India
[2]Associate Professor, Department of Pediatrics, JK loan Hospital, Kota, Rajasthan, India
[3]Medical officer, District Hospital Nadbai, Rajasthan, India
[4]Junior Resident, Department of Pediatrics, JK loan Hospital, Kota, Rajasthan, India

Received: 01-10-2025 / Revised: 15-11-2025 / Accepted: 21-12-2025
Corresponding author: Dr. Akash Parashar
Conflict of interest: Nil

Abstract

Background: Acute kidney injury (AKI) is a common clinical syndrome in hospitalized children and it imposes heavy burden of mortality and morbidity. Acute kidney injury is an acute and reversible increment in serum creatinine (SCr) levels with a reduction in urine output oliguria, or anuria.

Objective: To Study the Etiology, Clinical Profile and Outcome of Acute Kidney Injury (AKI) In Neonates Admitted in NICU of JK Lon Hospital Kota.

Materials and Methods: A prospective cross-sectional study was conducted in NICU JK loan with 255 neonates. Neonates (≤28 days) having acute kidney injury according to AKI criteria were included.

Results: Among 255 neonates, mortality was 16.9%. Low birth weight, sex, gestational age, and mode of delivery showed no significant association with outcome. Sepsis was the most common etiology, while asphyxia and higher HIE grades, especially grade 3, were strongly linked to mortality. Significant predictors of death included metabolic acidosis, elevated urea and creatinine levels, and AKI stage 3. Most cases occurred in summer, but deaths were more common during monsoon. Overall, severe metabolic and renal abnormalities were key determinants of poor outcome.

Conclusion: Severity of illness, hypoxic injury, metabolic acidosis, and advanced AKI stage are the primary determinants of mortality in neonatal AKI, rather than demographic factors.

Keywords: Acute Kidney Injury; Infant, Newborn; Hypoxic-Ischemic Encephalopathy; Metabolic Acidosis; Neonatal Mortality.

DOI: 10.25258/ijcpr.18.1.34

Introduction

Acute kidney injury (AKI) is a common clinical syndrome in hospitalized children and it imposes heavy burden of mortality and morbidity. More than 13 million people suffer from AKI each year and 85% of them live in developing countries [1]. Further, it is estimated that 1.7 million deaths occur each year from AKI and 60% of AKI survivors may experience residual renal abnormalities: proteinuria, hypertension, and reduced glomerular filtration rate (GFR) [2].

Incidence, etiology, and outcome of pediatric AKI are quite variable and depend upon age, geographic region, and clinical setting. Global data report mortality rates between 11% and 63% among children with AKI [3,4,5] oliguria/anuria(83.6%), hypertension (37.1%), and severe anemia (17.2%) to be the most common presentations among children with AKI while post-infectious glomerulonephritis (PIGN) and crescentic glomerulonephritis in primary renal, obstructive urolithiasis in postrenal, and sepsis in prerenal were the most common etiologies[6]. Acute kidney injury (AKI) is common among sick neonates admitted to an NICU. Studied indicate that AKI is an independent risk factor for morbidity as well as mortality. The incidence of AKI varies widely across studies from 1% to 56% depending upon the type of study population. Among the neonates with AKI, more than 65% are within 7 days of life. Acute kidney injury (AKI) is defined by an acute reduction of kidney function, resulting in uremia, altered fluid balance, and disturbed electrolytes homeostasis. In critically ill neonates, AKI is relatively common and occurs mainly in the first days of life, secondary to hypovolemia, hypotension, and ischemia, and less frequently to primary kidney disease [1,7].

Methods

This prospective observational cross-sectional study was conducted in the Neonatal Intensive Care Unit of Government Medical College and attached J.K. Lon Hospital, Kota, over a period of one year.

A total of 255 neonates aged ≤28 days diagnosed with acute kidney injury were enrolled after obtaining informed parental consent and institutional ethical clearance.

Neonates who died within the first 48 hours of life or had major congenital malformations incompatible with life were excluded. AKI was diagnosed according to standard neonatal AKI criteria.

A structured proforma was used to collect demographic data, antenatal and perinatal history, mode of delivery, gestational age, birth weight, and postnatal clinical details including sepsis, perinatal asphyxia, hypoxic-ischemic encephalopathy (HIE), dehydration, nephrotoxic drug exposure, and urine output.

All neonates underwent detailed clinical examination and laboratory investigations including complete blood count, C-reactive protein, serum urea, serum creatinine, serum electrolytes, and arterial blood gas analysis.

Statistical analysis was performed using SPSS version 24. Categorical variables were analyzed using Chi-square or Fisher's exact test. Continuous variables were compared using Student's t-test. A p-value <0.05 was considered statistically significant.

Observation and Results

Table 1:

Weight on admission	Outcome						P-value
	Death		Improved		Total		
	N	%	N	%	N	%	
< 2.5 kg	27	62.8%	148	69.8%	175	68.6%	0.803
≥ 2.5 kg	16	37.2%	64	30.2%	80	31.4%	0.803
Total	43	100.0%	212	100.0%	255	100.0%	0.803
SEX							
Female	13	30.2%	77	36.3%	90	35.3%	0.557
Male	30	69.8%	135	63.7%	165	64.7%	0.557
Total	43	100.0%	212	100.0%	255	100.0%	0.557
Mode of Delivery							
LSCS	10	23.3%	62	29.2%	72	28.2%	0.542
NVD	33	76.7%	150	70.8%	183	71.8%	
Total	43	100.0%	212	100.0%	255	100.0%	
Gestational age							
Preterm	15	34.9%	74	34.9%		34.9%	>0.05
Term	28	65.1%	138	65.1%	166	65.1%	
Total	43	100.0%	212	100.0%	255	100.0%	
Etiology							
Asphyxia	25	58.1%	58	27.4%	83	32.5%	<0.001
Dehydration	8	18.6%	33	15.6%	41	16.1%	
Haemorrhage	2	4.7%	0	0.0%	2	.8%	
Sepsis	8	18.6%	121	57.1%	129	50.6%	
Total	43	100.0%	212	100.0%	255	100.0%	
HIE							
HIE 1	3	7.0%	44	20.7%	47	18.5%	<0.001
HIE 2	5	11.6%	20	9.5%	25	9.8%	
HIE 3	19	44.2%	4	1.9%	23	9.0%	
No	16	37.2%	144	67.9%	160	62.7%	
Total	43	100.0%	212	100.0%	255	100.0%	
Nephrotoxic drug exposure							
No	36	83.7%	164	77.4%	200	78.4%	0.474
Yes	7	16.30%	48	22.6%	55	21.6%	
Total	43	100.0%	212	100.0%	255	100.0%	
Season							
Monsoon	11	25.6%	18	8.5%	29	11.4%	0.003
Summer	17	39.5%	123	58.0%	140	54.9%	
Winter	15	34.9%	71	33.5%	86	33.7%	

Total	43	100.0%	212	100.0%	255	100.0%	
Mean hematological values							
Hb	14.76	3.89	15.81	7.46	15.64	6.99	0.370
WBC	12.53	7.72	13.02	7.09	12.94	7.19	0.687
PLT	138.90	97.19	155.43	94.27	152.65	94.78	0.298
Mean electrolyte values							
Na+	144.19	18.32	142.17	12.40	142.51	13.56	0.374
K+	5.43	1.36	5.38	1.08	5.39	1.13	0.807
Cl-	105.51	9.97	105.08	7.73	105.15	8.13	0.752
Mean metabolic values							
pH	7.18	0.12	7.27	0.08	7.26	0.09	<0.001
pCO2	51.82	11.00	42.73	5.62	44.26	7.61	<0.001
BICARBONATE	18.70	3.32	20.72	2.62	20.38	2.85	<0.001
Mean RFT values							
Urea D1	71.09	35.79	58.10	28.64	60.29	30.28	0.010
Urea D3	174.56	73.69	140.84	53.23	146.52	58.41	<0.001
Creatinine D1	1.08	0.75	0.73	0.60	0.79	0.64	<0.001
Creatinine D3	2.13	1.01	1.63	0.60	1.71	0.71	<0.001

Table 2:

Outcome	**N**	**%**
Death	43	16.9
Improved	212	83.1
Total	255	100.0

Discussion

In the present cross-sectional observational study of 255 neonates with acute kidney injury (AKI), mortality was higher among infants weighing less than 2.5 kg compared to those weighing ≥2.5 kg; however, this difference was not statistically significant. The mean birth weight was comparable between the death and improvement groups, suggesting that birth weight alone may not independently predict outcome. Similar observations were reported by Dario Gallo and Karen A. de Bijl-Marcus et al., who demonstrated increasing mortality with advancing AKI severity, while the majority of neonates survived acute kidney disease [8]

Male predominance was observed in both death and improvement groups without a significant difference in outcome. This finding is consistent with previous studies and has been attributed to hormonal influences, genetic susceptibility, and higher rates of perinatal complications among male neonates [9]. Mode of delivery, parity, and gestational age did not show a statistically significant association with outcome in the present study. Although more deaths were observed among term neonates, similar findings have been reported previously, suggesting that severity of illness rather than gestational maturity may be a more important determinant of prognosis [9]

Etiology showed a significant association with outcome. Asphyxia was the leading cause of mortality, whereas sepsis predominated among survivors, highlighting the role of hypoxic injury in severe renal and multi-organ dysfunction. The strong association between hypoxic-ischemic encephalopathy (HIE) severity and mortality observed in the present study is consistent with findings by Robertsson Grossmann K and Bárány P et al., who demonstrated that AKI severity correlates with HIE grade and neonatal mortality[10]. Oliguria was the most common clinical presentation, reflecting the severity of renal dysfunction. Hematological and electrolyte parameters did not differ significantly between outcome groups; however, metabolic acidosis was significantly more severe among non-survivors, characterized by lower pH, higher pCO_2, and reduced bicarbonate levels. This finding supports the established bidirectional relationship between metabolic acidosis and AKI, wherein acidosis worsens renal perfusion and contributes to adverse outcomes.

Serum urea and creatinine levels were significantly higher among non-survivors, and mortality increased with advancing AKI stage, with stage 3 accounting for the majority of deaths. These findings reinforce that severity of renal injury and associated metabolic derangements, rather than demographic factors, are the principal determinants of outcome in neonatal AKI.

Summary

This cross-sectional study of 255 neonates evaluated factors influencing outcomes in neonatal acute kidney injury (AKI). Mortality was 16.9%,

with no significant association found between outcomes and birth weight, sex, gestational age, mode of delivery, or parity. Asphyxia and severe hypoxic-ischemic encephalopathy (HIE) were strongly linked to mortality, while sepsis was more common among survivors. Significant metabolic abnormalities—such as low pH, high pCO_2, low bicarbonate—and elevated urea and creatinine levels were major predictors of poor outcome. Seasonal variation showed higher mortality during the monsoon. The study concludes that hypoxic injury and biochemical severity, rather than demographic factors, primarily determine prognosis in neonatal AKI.

Conclusion

In this study, neonatal AKI outcomes were primarily influenced by clinical and biochemical severity rather than demographic or obstetric factors. Asphyxia and severe HIE emerged as the strongest predictors of mortality, along with significant metabolic acidosis and elevated serum urea and creatinine levels. Birth weight, sex, parity, gestational age, and mode of delivery showed no significant association with outcomes. Seasonal variation also affected mortality, with higher deaths during the monsoon season. These findings highlight the importance of early recognition of hypoxic injury and metabolic derangements to reduce mortality in neonatal AKI. Strengthening perinatal care and rapid intervention in high-risk infants may substantially improve clinical outcomes.

What This Study Adds

- Identifies perinatal asphyxia and severe HIE as the strongest predictors of mortality in neonatal AKI
- Demonstrates that biochemical severity (acidosis, elevated urea and creatinine) is more predictive of outcome than demographic factors
- Highlights seasonal variation in mortality, with increased deaths during monsoon months
- Provides region-specific data from a tertiary care NICU in a resource-limited setting

References

1. Lameire NH, Bagga A, Cruz D, et al.: Acute kidney injury: an increasing global concern. Lancet. 2013, 382:170-9. 10.1016/S0 1406736 (13)606479
2. Mehta RL, Cerdá J, Burdmann EA, et al.: International Society of Nephrology's 0by25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology. Lancet. 2015, 385:2616-43. 10.1016/S0140-6736(15)60126-X
3. Podoll AS, Kozar R, Holcomb JB, Finkel KW: Incidence and outcome of early acute kidney injury in criticallyill trauma patients. PLoS One. 2013, 8:e77376. 10.1371/journal.pone. 0077376
4. Cleto-Yamane TL, Gomes CL, Suassuna JH, Nogueira PK: Acute kidney injury epidemiology in pediatrics. J Bras Nefrol. 2019, 41:275-83. 10.1590/2175-8239-JBN-201 8-0127
5. Andreoli SP: Acute kidney injury in children .PediatrNephrol. 2009, 24:253-63. 10.1007/s0 0467-008-1074-9
6. Tresa V, Yaseen A, Lanewala AA, Hashmi S, Khatri S, Ali I, Mubarak M: Etiology, clinical profile and shortterm outcome of acute kidney injury in children at a tertiary care pediatric nephrology center in Pakistan. Ren Fail. 2017, 39:26-31. 10.1080/0886022X.2016.1244074 1 Selewski DT, Charlton JR, Jetton JG, Guillet R, Mhanna MJ, Askenazi DJ, et al.
7. Gouyon JB, Guignard JP. Management of acute renal failure in newborns. Pediatr Nephrol. 2000 Sep; 14(10–11): 1037–44.
8. Dario Galloa Karen A. de Bijl-Marcusa, Early Acute Kidney Injury in Preterm and Term Neonates: Incidence, Outcome, and Associated Clinical Features, Neonatology 2021;118:174–179 DOI: 10.1159/000513666
9. Satvik Chaitanya Bansal, Clinical Profile and Outcome of Newborns with Acute Kidney Injury in a Level 3 Neonatal Unit in Western India, Journal of Clinical and Diagnostic Research. 2017 Mar, Vol-11(3): SC01-SC04.
10. Robertsson Grossmann K, Bárány P, Blennow M, Chromek M. Acute kidney injury in infants with hypothermia-treated hypoxic-ischaemic encephalopathy: An observational population-based study. ActaPaediatr.2022 Jan;111(1):86-92. doi: 10.1111/apa.16078. Epub 2021 Aug 30. PMID: 34431538.