Section: Pediatrics

Original Research Article

TO STUDY ACUTE KIDNEY INJURY IN NEONATES ADMITTED IN NEONATAL INTENSIVE CARE CENTRE: A CROSS-SECTIONAL STUDY

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Received *: 15/11/2025*
Received in revised form *: 07/01/2026*
Accepted *: 24/01/2026*

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DOI: 10.70034/ijmedph.2026.1.125

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 705-709

ABSTRACT

Background: Acute kidney injury (AKI is a frequent and clinically relevant complication among neonates admitted to the neonatal intensive care unit (NICU), contributing substantially to morbidity and prolonged hospital stay. Early recognition of etiological factors, severity patterns, and associated biochemical abnormalities is critical for timely intervention and outcome improvement. **Objective:** To identify the etiology of AKI in NICU-admitted neonates, evaluate clinical and laboratory predictors, classify AKI types, and assess immediate outcomes.

Materials and Methods: This descriptive cross-sectional study included 100 NICU-admitted neonates aged 0–28 days, was conducted in Dept of Pediatrics, Rohilkhand Medial College and Hospital Bareilly, UP, India during period of 1 year.

Results: AKI predominantly affected male neonates and was commonly identified in the early neonatal period. Sepsis was the leading etiology, followed by hypoxic-ischemic encephalopathy. Prerenal and intrinsic renal AKI constituted most cases, with the majority classified as early-stage disease. Elevated serum creatinine, inflammatory markers, electrolyte abnormalities, and culture positivity showed significant associations, and higher creatinine levels correlated with prolonged NICU stay.

Conclusion: Neonatal AKI in the NICU is largely infection-driven, detected early in most cases, and associated with biochemical derangements and extended hospitalization, highlighting the importance of early diagnosis and focused management strategies.

Keywords: Neonatal acute kidney injury; NICU; Sepsis; Serum creatinine; KIDGO criteria.

INTRODUCTION

Acute kidney injury (AKI) in neonates is increasingly recognized as a significant contributor to morbidity and mortality in neonatal intensive care units (NICUs). Advances in neonatal care have improved survival of preterm and critically ill neonates; however, these vulnerable infants remain at high risk for renal dysfunction due to physiological renal immaturity and exposure to multiple insults during the early postnatal period.[1] Neonatal AKI is associated with prolonged hospitalization, increased need for ventilatory and inotropic support, and adverse short-term outcomes. The reported incidence of neonatal AKI varies widely across studies, largely depending on population characteristics, diagnostic criteria, and availability of renal monitoring.[2] Recent cohort studies have highlighted that neonatal AKI is not uncommon and frequently under-diagnosed in routine clinical practice.[3] Conditions such as sepsis, perinatal asphyxia, prematurity, low birth weight, and exposure to nephrotoxic medications have been consistently identified as major risk factors.[3,4] A major challenge in neonatal AKI research has been the lack of uniform diagnostic definitions. Several classification systems, including

pRIFLE, AKIN, and KDIGO, have been evaluated in pediatric and neonatal populations, with modified KDIGO criteria currently being the most widely accepted for neonates.[4,5] Large multicentre studies, such as the AWAKEN cohort, have provided robust epidemiological data and demonstrated a clear association between AKI and increased neonatal mortality.[6] Subsequent refinements in AKI definitions during the first postnatal week have further improved diagnostic accuracy.[7] Emerging evidence also suggests that neonatal AKI has implications beyond the acute phase. AKI has been linked to neurological complications, including intraventricular hemorrhage, particularly in premature infants,[8] and adverse long-term neurodevelopmental outcomes in conditions such as hypoxic-ischemic encephalopathy.[9,10] Despite growing awareness, data from Indian NICU settings remain limited. In this context, the present cross-sectional study aims to evaluate the occurrence of acute kidney injury in neonates admitted to a neonatal intensive care centre and to analyze associated clinical characteristics and outcomes, thereby contributing region-specific evidence to this evolving field.[1,2]

MATERIALS AND METHODS

This descriptive cross-sectional study was conducted after clearance from Board of Studies and Ethical committee in the Neonatal Intensive Care Unit of Department of Paediatrics at Rohilkhand Medical College and Hospital, Bareilly (U.P.).

Sample size: 100

P4 = expected proportion of AKI in a newborn admitted in NICU.

Q = 100-p

L = absolute allowable error (5%)

N = 4pq/L2

= 4 x 6.5x (100-6.5) /25

= 97.24

= 100

Inclusion Criteria

- Neonates whose parents gave consent.
- Neonates who are admitted in NICU.

Exclusion Criteria

- Antenatally diagnosed renal congenital anomaly.
- Preterm neonates less than 24weeks.

Methodology

All newborns' taking part in the study, written informed consent was taken from their parents /guardians in a language they can understand. All newborns admitted in NICU was make up the study group. Detailed history taking, complete physical examination and investigation was done. All cases enrolled in the study was subjected to the following: Detailed maternal history like gravida, peripartum infections, history of drug in take during pregnancy, foul smelling liquor. Maternal factors were also recorded, detailed neonatal history included oliguria or anuria, colour of urine, presence of swelling or edema, poor feeding, lethargy, vomiting, abdominal distension, exposure to nephrotoxic drugs, respiratory distress, perinatal asphyxia, and evidence of birth trauma. Detailed General and Physical examination to be done. Investigations (Complete Blood Count, Serum, Electrolytes, Kidney function test, Urine routine and microscopy, C-reactive protein, Blood culture, Fractional excretion of sodium FENa). Therefore, Kidney function test (KFT) was done in newborn at 48 hrs of life and was repeated after 24 hrs to see the S. Creatinine level and assess the stage of AKI.

If newborn is > 2 days then kidney function test was done at the time of admission and was repeated after 24 hrs. Imaging – Ultrasound of whole abdomen and Computed tomography if required. Therefore, KDIGO Classification was used to define and stage AKI. Identify risk factors and etiology, identification of potential causes of AKI, such as infections (e.g., sepsis, urinary tract infections), perinatal asphyxia, dehydration, hypoxia, nephrotoxic drugs (e.g., antibiotics, diuretics), and obstructive uropathy.

Risk: Serum creatinine >1.5 times baseline or urine output <0.5 mL/kg/h for 24 hours.

Injury: Serum creatinine >2 times baseline or urine output <0.5 mL/kg/h for 24-48 hours.

Failure: Serum creatinine >3 times baseline or urine output <0.3 mL/kg/h for 24 hours, or anuria for 12 hours.

Loss: Persistent AKI for more than 4 weeks.

End-stage renal disease (ESRD): Persistent renal failure for more than 3 months.

The primary outcome (dependent) variable was the development of AKI which was determined by measuring S.Cr and defined using the modified neonatal KIDGO AKI criteria. The secondary study outcomes were length of hospital stay in days and mortality. The Independent Variables assessed were the age of the neonate in days at admission, the sex of the neonate (male/female), the gestation age of the neonate at delivery (Using the date of the Last Normal Menstrual Period), and the age of the mother. Other independent variables noted were the birth weight in kilograms, the Apgar score at 5 minutes, the mode of delivery (either by spontaneous vaginal delivery, by cesarean section, or by vacuum extraction), and where the delivery occurred (at home, at clinic, or at UTH). The drugs administered during the course of admission were noted as was the diagnosis at admission to NICU. Any other diagnosis during course of the admission was considered as secondary diagnosis. Other treatments noted were the need for mechanical ventilation and the need for vasopressor support. The laboratory variables included the hemoglobin, platelets, and total white cell count.

Statistical Analysis: Data were entered, coded, and compiled using Microsoft Excel and analysed with SPSS version 23.0. Descriptive statistics were applied, appropriate tests used based on data

distribution, results tabulated, and $p < 0.05$ considered statistically significant.

RESULTS

In this study, neonates were evaluated for acute kidney injury, there was a statistically significant male predominance (62% male vs 38% female; p=0.032). The mean postnatal age at assessment was 5.3 ± 2.1 days, placing most infants within the early neonatal period; the age distribution was non-uniform and statistically significant (p=0.017). Together, these baseline characteristics suggest that analyses of AKI risk and outcomes should account for sex and early postnatal timing as potential confounders or effect modifiers.

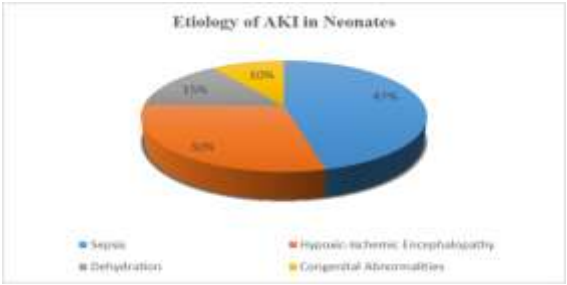

Figure 1: Etiology of AKI in Neonates

Etiological analysis of neonatal acute kidney injury, as delineated in Figure 1, identified sepsis as the predominant causative determinant, accounting for 47% of incidents and demonstrating a statistically robust association (p=0.001). Hypoxic-ischemic encephalopathy constituted the second most prevalent etiology, implicated in 30% of cases and also exhibiting a significant correlation (p=0.025). Dehydration states and congenital anomalies were identified in 15% (p=0.039) and 10% (p=0.048) of the cohort, respectively. These findings collectively underscore a multifactorial pathogenesis for neonatal AKI, wherein infectious processes, perinatal hypoxic insults, fluid imbalances, and developmental malformations represent the principal convergent pathways to renal compromise.

Figure 2: Type of AKI

As demonstrated in Figure 2, the pathophysiological categorization of acute kidney injury revealed a predominance of the prerenal variant, constituting 47% of cases and demonstrating a statistically significant correlation (p=0.012). The intrinsic renal subtype was observed in 43% of neonates, also exhibiting statistical significance (p=0.004). Postrenal AKI, while representing a comparatively infrequent etiology at 10% of the cohort, maintained a statistically significant association (p=0.035). This nosocomial distribution substantiates that hemodynamically mediated and parenchymal injuries represent the principal mechanisms of renal compromise in neonates, whereas obstructive uropathies, though less prevalent, remain a clinically consequential diagnostic consideration.

Figure 3: Stages of AKI (KDIGO Classification)

The stratification of acute kidney injury severity according to the "kidney disease: Improving Global Outcomes (KDIGO)" classification system, as presented in Figure 3, revealed a distinct distribution across the diagnostic spectrum. Stage 1 AKI was the most frequently observed classification, present in 50% of cases and demonstrating statistical significance (p=0.015). Stage 2 affliction was documented in 35% of neonates, a finding that also attained statistical significance (p=0.029). The most severe manifestation, Stage 3 AKI, was identified in 15% of the cohort, a proportion that was statistically significant (p=0.048). This graduated distribution of disease severity indicates that the majority neonatal AKI presentations are identified in their incipient phases (Stages 1 and 2), while the most advanced stage, though less prevalent, constitutes a clinically grave subgroup with substantial implications for therapeutic intervention and prognostic outcomes.

Table 1: Investigations in AKI Patients

Investigation	Frequency (%)	p-value
High CRP	60%	0.005
Abnormal Electrolytes	50%	0.019
Blood Culture Positive	30%	0.027
Elevated Serum Creatinine	80%	0.003

The investigation findings for newborns with acute kidney injury (AKI) are shown in Table 1. There was a statistically significant correlation (p=0.005) between high C-reactive protein (CRP) levels and 60% of cases. Fifty percent of newborns had abnormal electrolytes, which was statistically significant (p=0.019). Thirty percent of cases had positive blood cultures, with a significant p-value of 0.027. The most frequent finding was elevated serum creatinine, which was found in 80% of newborns and had a statistically significant correlation (p=0.003). According to these findings, higher serum creatinine, positive blood cultures, aberrant electrolytes, and high CRP are important indicators of AKI in newborns.

Table 2: Laboratory Parameters in AKI

Parameter	Value	p-value
Sodium (Mean ± SD)	135.6 ± 5.2	0.022
Potassium (Mean ± SD)	4.8 ± 0.9	0.034
Creatinine (Mean ± SD)	2.3 ± 0.2	0.019
Urea (Mean ± SD)	45 ± 12	0.041

Table 2 presents the laboratory parameters in neonates with Acute Kidney Injury (AKI). The mean sodium level was 135.6 ± 5.2 mEq/L, with a statistically significant association (p=0.022). Potassium levels had a mean of 4.8 ± 0.9 mEq/L, showing a significant difference (p=0.034). Serum creatinine levels were elevated, with a mean of 2.3 ± 0.2 mg/dL, and this difference was statistically significant (p=0.019). Urea levels had a mean of 45 ± 12 mg/dL, also demonstrating a significant association (p=0.041). These findings suggest that disturbances in sodium, potassium, creatinine, and urea are significant laboratory markers in AKI in neonates.

Table 3: Correlation Between Serum Creatinine and NICU Stay Duration

	Creatinine levels			
NICU Stay (days)	0.54–1.0	2.01–3.0	3.01–3.49	Grand Total
<7	3	23	6	32
7–14	11	29	3	43
15–21	6	10	1	17
>21	0	6	2	8
Grand Total	20	68	12	100
Correlation Coefficient (r)			0.78	p = 0.001

In Table 3, the distribution of serum creatinine levels across NICU stay categories shows that most neonates had creatinine between 2.01–3.0 mg/dL (68%) and stayed predominantly 7–14 days (43%), while those with the highest creatinine levels (3.01–3.49 mg/dL; 12%) were more concentrated in the longer-stay groups (15–21 days and >21 days; together 10 cases). In contrast, neonates with lower creatinine values (0.54–1.0 mg/dL; 20%) were mainly clustered in the shorter-stay category (<7 days; 3 cases) and 7–14 days. Overall, the strong positive correlation between serum creatinine and NICU stay duration (r = 0.78, p = 0.001) indicates that higher degrees of renal dysfunction are associated with progressively prolonged hospitalization in neonates with AKI.

DISCUSSION

The present NICU-based study demonstrated a significant male predominance (62% vs 38%, p=0.032) and early postnatal presentation (mean 5.3 ± 2.1 days, p=0.017), findings that are concordant with Indian multicentric observations from the TINKER registry, which reported a higher burden of AKI among male neonates and early neonatal onset in hospitalized cohorts Agrawal et al., 2021).[1] The predominance of sepsis as the leading etiology (47%, p=0.001) closely aligns with the epidemiological profile described in TINKER, where sepsis emerged as the principal driver of neonatal AKI and adverse outcomes Agrawal et al., (2021).[1] The substantial contribution of hypoxic-ischemic encephalopathy (30%, p=0.025) is comparable to cohorts evaluating HIE-associated AKI and adverse outcomes Cavallin et al., (2020),[10] Gupta et al., 2016.[9] Stoops et al., (2019),[8] Starr et al., (2020),[11] while the relatively lower frequency of postrenal AKI (10%, p=0.035) represents a distinct observation within this cohort. The predominance of early-stage disease in this cohort, with Stage 1 and Stage 2 AKI accounting for 50% (p=0.015) and 35% (p=0.029) respectively, mirrors the pattern reported in large neonatal cohorts where most AKI cases are detected before progression to advanced stages, emphasizing early identification Agrawal et al., 2021.[1] The smaller yet

clinically significant proportion of Stage 3 AKI (15%, p=0.048) is comparable to observations in critically ill neonates, in whom severe AKI confers higher morbidity and prolonged recovery Stoops et al., (2019).[8] The strong association of elevated serum creatinine (80%, p=0.003), raised CRP (60%, p=0.005), electrolyte disturbances (50%, p=0.019), and positive blood cultures (30%, p=0.027) aligns with infection- and inflammation-driven renal injury described in Indian and international cohorts Agrawal et al., 2021,[1] Gupta et al., (2016).[9] The significant correlation between rising creatinine levels (mean 2.3 ± 0.2 mg/dL, p=0.019) and prolonged NICU stay (r = 0.78, p = 0.001) reinforces prior evidence linking AKI severity with extended hospitalization, while the graded creatinine–stay relationship represents a distinct, cohort-specific observation.

CONCLUSION

This study demonstrates that neonatal acute kidney injury is a frequent and clinically significant complication among NICU admissions, with a clear predominance in the early neonatal period and a statistically significant male preponderance, underscoring the influence of early postnatal vulnerability. Sepsis emerged as the leading etiological factor, followed by hypoxic-ischemic encephalopathy, highlighting infection-driven systemic inflammation and perinatal hypoxia as dominant pathways for renal injury in neonates. The predominance of prerenal and intrinsic renal AKI emphasizes the role of hemodynamic instability and parenchymal damage rather than obstructive causes. Most neonates were identified in KDIGO Stage 1 and 2, indicating that AKI is often recognized in its early phases, although a smaller subset progressed to severe disease with important prognostic implications. Elevated serum creatinine, electrolyte disturbances, raised CRP, and positive blood cultures were significant laboratory correlates of AKI. The strong positive correlation between serum creatinine levels and prolonged NICU stay confirms that increasing renal dysfunction is directly associated with extended hospitalization and greater clinical burden in affected neonates.

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