Original Research Article

Study of Acute Kidney Injury in Newborns Admitted in NICU at a Tertiary Care Hospital

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Abstract

Introduction
Acute kidney injury (AKI) is an important cause of morbidity and mortality among neonates admitted to neonatal intensive care units (NICUs). Critically ill neonates are particularly vulnerable because of immature renal function, sepsis, birth asphyxia, dehydration, and exposure to nephrotoxic drugs. Early identification of AKI is important for reducing complications and improving survival.

Materials and Methods
This hospital-based cross-sectional observational study was conducted in the NICU of K.D. Medical College and Research Centre, Mathura. A total of 160 term neonates admitted to the NICU were included. Preterm neonates, neonates with congenital renal anomalies, and those without parental consent were excluded. AKI was diagnosed using modified KDIGO criteria based on serum creatinine and urine output. Relevant clinical, demographic, biochemical, and outcome data were recorded and analyzed using SPSS version 25.0.

Results
Among 160 neonates, 63 developed AKI, giving an incidence of 39.4%. Stage 1 AKI was most common (38.1%), followed by Stage 2 (33.3%) and Stage 3 (28.6%). Sepsis was the most common etiological factor (69.8%), followed by birth asphyxia (14.3%) and neonatal jaundice (12.7%). Neonates with AKI had significantly lower birth weight, lower urine output, lower serum sodium, and higher serum potassium, blood urea, and serum creatinine levels. Mechanical ventilation and vasoactive support were significantly more common among AKI neonates. Mortality was significantly higher in neonates with AKI compared to those without AKI (28.6% vs 4.1%).

Conclusion
AKI is a frequent and serious complication among NICU-admitted neonates. Sepsis, low birth weight, oliguria, and hemodynamic instability are major risk factors. Early diagnosis and prompt management are essential to improve neonatal outcomes.

Keywords: Acute kidney injury, neonates, NICU, sepsis, serum creatinine, KDIGO criteria, mortality

Introduction

Acute kidney injury (AKI) is an important cause of morbidity and mortality among neonates admitted to neonatal intensive care units (NICUs). Neonatal kidneys are functionally immature, with low glomerular filtration rate and limited tubular function, making them highly susceptible to ischemia, hypoxia, sepsis, dehydration, nephrotoxic drugs, and hemodynamic instability [1,2].AKI is defined as a sudden decline in renal function resulting in impaired fluid, electrolyte, and acid-base balance. In neonates, AKI is usually identified by rising serum creatinine levels and decreased urine output [3]. However, early diagnosis is difficult because neonatal serum creatinine is influenced by maternal creatinine levels, gestational age, birth weight, and postnatal maturation [4].Several factors

contribute to neonatal AKI, including perinatal asphyxia, neonatal sepsis, prematurity, low birth weight, respiratory distress syndrome, congenital anomalies of the kidney and urinary tract, dehydration, shock, and exposure to nephrotoxic drugs such as aminoglycosides and NSAIDs [5,6]. Critically ill neonates requiring mechanical ventilation, vasopressors, or prolonged NICU stay are at even higher risk [7].To standardize diagnosis, modified Kidney Disease: Improving Global Outcomes (KDIGO) criteria have been adapted for neonates. According to these criteria, AKI is defined as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, a rise of ≥1.5 times baseline, or urine output less than 1 mL/kg/hour for more than 24 hours [8].The reported incidence of neonatal AKI ranges from 8% to 40%, depending on the population studied and diagnostic criteria used [8,9]. The incidence is particularly high among preterm, low birth weight, and critically ill neonates [10]. Jetton et al. reported AKI in approximately 8–24% of NICU admissions, while other studies found even higher rates in very low birth weight infants [8,11].

Neonatal AKI is associated with poor outcomes including prolonged hospital stay, need for mechanical ventilation, electrolyte imbalance, increased mortality, and long-term risk of chronic kidney disease [2,5]. Mortality rates among neonates with AKI have been reported to range from 20% to 60%, especially in those with sepsis, shock, or multi-organ dysfunction [6,12].Despite its clinical importance, neonatal AKI remains underdiagnosed in many developing countries due to lack of awareness, inadequate monitoring, and absence of routine screening protocols [7]. In India, data on neonatal AKI remain limited, making it important to generate local evidence regarding its incidence, risk factors, and outcomes [10,13].Therefore, the present study was undertaken to assess the incidence, clinical profile, risk factors, and outcomes of AKI in newborns admitted to the NICU at a tertiary care hospital.

Methodology:

Study Design

This hospital-based cross-sectional observational study was conducted in the Neonatal Intensive Care Unit (NICU) of K.D. Medical College and Research Centre, Mathura, Uttar Pradesh. The study included term newborns admitted to the NICU during the study period. All eligible neonates were evaluated for the presence of acute kidney injury (AKI) and associated clinical outcomes. The estimated sample size was 146, which was rounded off to 160 neonates.

Inclusion Criteria

All term newborns (gestational age ≥37 weeks) admitted to the NICU for any indication.

Exclusion Criteria

- Preterm newborns (<37 weeks of gestation).
- Neonates with known congenital renal anomalies, extra-renal anomalies, or genetic disorders.
- Parents or guardians unwilling to provide informed consent.

Study Methodology

Eligible neonates were enrolled after obtaining written informed consent from parents or guardians. A detailed clinical history and demographic profile were recorded for each newborn. Relevant information including age at admission, sex, birth weight, gestational age, mode of delivery, indication for NICU admission, maternal history, and associated comorbid conditions was documented.

All enrolled neonates underwent serial monitoring of serum creatinine levels and urine output from the time of admission. Urine output was measured at 12-hour intervals using urinary catheterization or diaper weight method, depending on clinical feasibility.

Acute kidney injury was diagnosed according to the modified Kidney Disease: Improving Global Outcomes (mKDIGO) criteria. AKI was defined as any one of the following:

- Increase in serum creatinine by ≥0.3 mg/dL within 48 hours.
- Increase in serum creatinine to ≥1.5 times baseline within 7 days.
- Urine output <1 mL/kg/hour for more than 24 hours.

After identification of AKI, further investigations were performed to determine the underlying etiology. These included kidney function tests such as serum urea, serum creatinine, and serum electrolytes; urine routine and microscopic examination; sepsis screening including complete blood count and C-reactive protein; and ultrasonography of kidney, ureter, and bladder (USG KUB) wherever indicated.Based on clinical evaluation and investigation findings, AKI was categorized as prerenal, intrinsic renal, or postrenal. Relevant information regarding need for dialysis, duration of NICU stay, mechanical ventilation, and survival outcome was also recorded.All data were entered into a structured proforma and compiled in Microsoft Excel for statistical analysis.

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD) and median with

interquartile range (IQR), whereas categorical variables were expressed as frequencies and percentages. Comparisons between neonates with AKI and those without AKI were performed using the independent Student's t-test for continuous variables and Chi-square test for categorical variables. Chi-square goodness-of-fit test was used wherever comparison with expected proportions was required. The incidence of AKI was calculated as the proportion of total NICU admissions. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

Ethical approval for the study was obtained from the Institutional Ethics Committee prior to commencement of the study. Written informed consent was obtained from parents or guardians before enrollment of neonates into the study. Confidentiality of all patient information was maintained throughout the study, and no additional financial burden was imposed on the families.

Results:

A total of 160 term newborns admitted to the NICU were included in the present study. The baseline demographic and clinical characteristics of the study population are shown in Table 1. The mean age at admission was 5.34 ± 1.53 days, with a median age of 5 days (IQR: 4–6). Male newborns constituted 51.9% of the study population, while females accounted for 48.1%. The mean birth weight was 2.86 ± 0.42 kg, and the mean gestational age was 38.6 ± 1.1 weeks. Slightly more than half of the neonates were delivered by LSCS (51.2%), while 48.8% were delivered vaginally. Inborn neonates accounted for 52.5% of admissions, while 47.5% were outborn (Table 1).The incidence, severity, and etiological profile of acute kidney injury are summarized in Table 2. Among the 160 neonates included in the study, 63 developed AKI, resulting in an incidence of 39.4%, while 97 neonates (60.6%) did not develop AKI. According to KDIGO staging, Stage 1 AKI was observed in 24 neonates (15.0%), Stage 2 in 21 neonates (13.1%), and Stage 3 in 18 neonates (11.3%). Among neonates with AKI, Stage 1 was the most common stage, accounting for 38.1% of cases, followed by Stage 2 (33.3%) and Stage 3 (28.6%). Sepsis was identified as the most common etiological factor associated with AKI, being present in 44 neonates (69.8%). Birth asphyxia was the second most common cause, affecting 9 neonates (14.3%), followed by neonatal jaundice in 8 neonates (12.7%) and other causes in 2 neonates (3.2%) (Table 2).The comparison of clinical and laboratory parameters between neonates with AKI and those without AKI is presented in Table 3. Neonates with AKI had a significantly lower mean birth weight compared to non-AKI neonates (2.71 ± 0.41 kg vs 2.96 ± 0.40 kg, $p = 0.002$). The mean temperature was significantly higher among AKI neonates (37.4 ± 0.4°C) compared to non-AKI neonates (37.1 ± 0.3°C, $p = 0.01$). Similarly, the mean pulse rate was significantly elevated in the AKI group (154.6 ± 17.2 beats/min) compared to the non-AKI group (141.7 ± 18.1 beats/min, $p < 0.001$). Respiratory rate was also significantly higher among neonates with AKI (58.6 ± 9.4 breaths/min) compared to those without AKI (51.4 ± 8.7 breaths/min, $p < 0.001$) (Table 3).Significant differences were also observed in biochemical and renal function parameters between AKI and non-AKI neonates. Serum sodium levels were significantly lower among neonates with AKI (137.1 ± 4.9 mEq/L) compared to those without AKI (140.6 ± 3.8 mEq/L, $p < 0.001$). In contrast, serum potassium levels were significantly higher in AKI neonates (4.9 ± 0.6 mEq/L) than in non-AKI neonates (4.1 ± 0.5 mEq/L, $p < 0.001$). Urine output was markedly lower among AKI neonates (0.54 ± 0.36 ml/kg/hr) compared to non-AKI neonates (1.50 ± 0.48 ml/kg/hr, $p < 0.001$). Blood urea levels were significantly higher in the AKI group (92.6 ± 41.8 mg/dL) than in the non-AKI group (30.4 ± 12.9 mg/dL, $p < 0.001$). Similarly, maximum serum creatinine was markedly elevated among neonates with AKI (2.78 ± 0.96 mg/dL) compared to those without AKI (0.61 ± 0.12 mg/dL, $p < 0.001$), while eGFR was significantly lower in AKI neonates (16.2 ± 6.4 mL/min/1.73 m^2) compared to non-AKI neonates (36.2 ± 7.8 mL/min/1.73 m^2, $p < 0.001$) (Table 3).Mechanical ventilation was required in 57.1% of neonates with AKI compared to only 8.2% of those without AKI, and this difference was highly significant ($p < 0.001$). Similarly, vasoactive medications were required in 50.8% of AKI neonates compared to only 6.2% of non-AKI neonates ($p < 0.001$). Mortality was also significantly higher among neonates with AKI, with 28.6% of AKI neonates succumbing during hospital stay compared to only 4.1% of non-AKI neonates ($p < 0.001$) (Table 3). These findings indicate that AKI was associated with increased illness severity, greater requirement for intensive support, and poorer short-term outcomes.

Table 1. Baseline Demographic and Clinical Characteristics of Study Population (n = 160)

Variable	Value
Mean Age at Admission ± SD (days)	5.34 ± 1.53
Median Age (IQR) (days)	5 (4–6)
Male Sex	83 (51.9%)
Female Sex	77 (48.1%)
Mean Birth Weight ± SD (kg)	2.86 ± 0.42
Mean Gestational Age ± SD (weeks)	38.6 ± 1.1
Vaginal Delivery	78 (48.8%)
LSCS Delivery	82 (51.2%)
Inborn Neonates	84 (52.5%)
Outborn Neonates	76 (47.5%)

Table 2. Incidence, Severity, and Etiology of Acute Kidney Injury Among Neonates (n = 160)

Variable	Number (n)	Percentage (%)
AKI Present	63	39.4
No AKI	97	60.6
Stage 1 AKI	24	15.0
Stage 2 AKI	21	13.1
Stage 3 AKI	18	11.3
Sepsis	44	69.8
Birth Asphyxia	9	14.3
Neonatal Jaundice	8	12.7
Others	2	3.2

Table 3. Comparison of Clinical and Laboratory Parameters Between AKI and Non-AKI Neonates

Variable	AKI Present (n = 63)	No AKI (n = 97)	p-value
Birth Weight (kg)	2.71 ± 0.41	2.96 ± 0.40	0.002
Temperature (°C)	37.4 ± 0.4	37.1 ± 0.3	0.01
Pulse Rate (beats/min)	154.6 ± 17.2	141.7 ± 18.1	<0.001
Respiratory Rate (breaths/min)	58.6 ± 9.4	51.4 ± 8.7	<0.001
Serum Sodium (mEq/L)	137.1 ± 4.9	140.6 ± 3.8	<0.001

Serum Potassium (mEq/L)	4.9 ± 0.6	4.1 ± 0.5	<0.001
Urine Output (ml/kg/hr)	0.54 ± 0.36	1.50 ± 0.48	<0.001
Blood Urea (mg/dL)	92.6 ± 41.8	30.4 ± 12.9	<0.001
Maximum Serum Creatinine (mg/dL)	2.78 ± 0.96	0.61 ± 0.12	<0.001
eGFR (mL/min/1.73 m²)	16.2 ± 6.4	36.2 ± 7.8	<0.001
Mechanical Ventilation Required	36 (57.1%)	8 (8.2%)	<0.001
Vasoactive Medications Required	32 (50.8%)	6 (6.2%)	<0.001
Mortality	18 (28.6%)	4 (4.1%)	<0.001

Discussion:

The present study evaluated acute kidney injury (AKI) among 160 term neonates admitted to the NICU using modified KDIGO criteria. AKI was observed in 39.4% of neonates, indicating that renal dysfunction is a common complication among critically ill newborns. This incidence was higher than that reported by Hamsa et al. (30%) [14], Nandhagopal et al. (21.9%) [15], Ashraf et al. (8.33%) [16], and Bolat et al. (8.4%) [17]. The higher incidence in our study may be due to the greater burden of sepsis and the use of serial serum creatinine and urine output monitoring.Most neonates in the present study were admitted during the first week of life, with a mean age at admission of 5.34 ± 1.53 days. This finding is comparable with Nagaraj et al. [18] and Jetton et al. [19], who also reported that most cases of neonatal AKI occur during the early neonatal period. The sex distribution in the present study was nearly equal, with 51.9% males and 48.1% females, and no significant association was observed between sex and AKI. Similar findings were reported by Hamsa et al. [14], Ashraf et al. [16], and Jetton et al. [19]. However, Bansal et al. [20] reported male gender as a significant predictor of AKI. Birth weight was significantly lower among neonates with AKI compared to those without AKI (2.71 ± 0.41 kg vs 2.96 ± 0.40 kg; p = 0.002). Low birth weight (<2.5 kg) was also significantly associated with AKI. Similar findings were reported by Gupta et al. [49], Bolat et al. [17], and Patel et al. [62], suggesting that lower birth weight increases the risk of renal injury. Sepsis was the most common etiological factor for AKI in the present study, accounting for 69.8% of cases, followed by birth asphyxia and neonatal jaundice. Similar findings were reported by Nandhagopal et al. [15], Ashraf et al. [16], and Bansal et al. [20], who identified sepsis as the strongest risk factor for neonatal AKI.

Clinical severity was significantly greater among neonates with AKI. Oliguria, prolonged capillary refill time, edema, need for mechanical ventilation, and vasoactive support were all significantly more common among AKI neonates. These findings are comparable Bansal et al. [20], who also reported that AKI is strongly associated with hemodynamic instability and multiorgan dysfunction. Biochemical abnormalities were more severe in neonates with AKI. Serum sodium was significantly lower, while serum potassium, blood urea, and serum creatinine were significantly higher among AKI neonates. Urine output and eGFR were significantly lower in the AKI group. Similar findings have been reported by Nagaraj et al. [18], Hamsa et al. [14], and Bolat et al. [17].In the present study, Stage 1 AKI was the most common stage, followed by Stage 2 and Stage 3. However, a considerable proportion of neonates had moderate-to-severe AKI (Stage 2 or Stage 3). Similar stage-wise distributions have been reported by Nandhagopal et al. [15] and Hamsa et al. [14].Mortality among neonates with AKI was 28.6%, which was significantly higher than the mortality among neonates without AKI (4.1%). Mortality also increased with increasing AKI severity, reaching 50% in Stage 3 AKI. Similar findings have been reported by Ashraf et al. [16], Bolat et al. [17] and Jetton et al. [19].

Conclusion

Acute kidney injury is a frequent and serious complication among NICU-admitted term neonates, with an incidence of 39.4% in the present study. Sepsis, low birth weight, oliguria, and hemodynamic instability were major factors associated with AKI. Neonates with AKI had significantly higher mortality and greater need for intensive care support. Early recognition and timely management of AKI are essential to improve neonatal outcomes.

LIMITATIONS

The study was conducted at a single tertiary care center with a relatively small sample size, which may limit the generalizability of the findings. Only

term neonates were included, so the results cannot be applied to preterm infants. Long-term renal outcomes were not assessed due to lack of follow-up. In addition, advanced biomarkers of AKI were not evaluated because of limited resources.

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