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Original Research Article

PREDICTORS OF MORTALITY IN NEONATAL ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY

Nagaraj[1], Raghuveer S Anantapur[2], Suman Uppin[1], D Pooja Reddy[1]

[1]*Assistant Professor, Department of Paediatrics, YIMS Yadgiri, India.*
[2]*Senior Resident, Department of Paediatrics, YIMS Yadgiri, India.*

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Corresponding Author:
Dr. D Pooja Reddy,
Assistant Professor, Department of Paediatrics, YIMS Yadgiri, India.
Email: dprilwi621@gmailcom

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ABSTRACT

Background: Neonatal acute kidney injury (AKI) is a serious complication encountered in neonatal intensive care units and is associated with significant morbidity, prolonged hospitalization, and increased mortality. Early identification of factors associated with poor outcomes may facilitate timely intervention and improve survival. The aim is to identify predictors of mortality and evaluate clinical outcomes among neonates with acute kidney injury admitted to a tertiary care neonatal intensive care unit.

Materials and Methods: This prospective observational study was conducted among 100 neonates diagnosed with AKI according to modified KDIGO criteria and admitted to a tertiary care NICU. Demographic characteristics, clinical profile, etiological factors, laboratory parameters, treatment requirements, and outcomes were recorded using a structured proforma. Statistical analysis was performed using SPSS software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Chi-square test, Student's t-test, and logistic regression analysis were used to identify predictors of mortality. A p-value <0.05 was considered statistically significant.

Results: The mean age at admission was 5.8 ± 3.1 days and the mean birth weight was 2.48 ± 0.54 kg. Male neonates constituted 64% of the study population. Sepsis (46%) was the most common etiological factor, followed by prematurity (38%), low birth weight (35%), perinatal asphyxia (34%), respiratory distress syndrome (20%), and dehydration/fever (18%). Non-oliguric AKI was observed in 58% of cases. Mechanical ventilation was required in 28% of neonates, shock was present in 25%, KDIGO stage III AKI in 18%, and peritoneal dialysis in 6%. Overall mortality was 12%. Significant predictors of mortality included sepsis (OR=7.22), shock (OR=8.35), mechanical ventilation (OR=6.80), KDIGO stage III AKI (OR=9.80), peritoneal dialysis (OR=21.50), hyperkalemia (OR=5.45), and metabolic acidosis (OR=5.33). Complete renal recovery occurred in 76% of neonates.

Conclusion: Neonatal AKI remains an important cause of morbidity and mortality in NICU settings. Sepsis was the leading etiological factor, while severe AKI, shock, mechanical ventilation, dialysis requirement, hyperkalemia, and metabolic acidosis were significant predictors of mortality. Early diagnosis, intensive monitoring, and prompt intervention may improve clinical outcomes and reduce mortality among affected neonates.

Keywords: Neonatal Acute Kidney Injury; Mortality Predictors; Neonatal Intensive Care Unit (NICU).

INTRODUCTION

Neonatal Acute Kidney Injury (AKI) is a significant clinical problem encountered in Neonatal Intensive Care Units (NICUs) and is associated with substantial morbidity and mortality. AKI refers to an abrupt decline in kidney function resulting in the inability of the kidneys to maintain fluid, electrolyte, and acid-

base homeostasis. The neonatal kidney is structurally and functionally immature, making neonates particularly susceptible to renal injury when exposed to various perinatal and postnatal insults. Advances in neonatal intensive care have improved the survival of critically ill neonates; however, AKI continues to remain a major contributor to adverse outcomes, prolonged hospitalization, and increased healthcare costs.[1]

The incidence of neonatal AKI varies widely across studies, ranging from 8% to 30%, depending on the population studied and the diagnostic criteria employed. The introduction of the Kidney Disease: Improving Global Outcomes (KDIGO) criteria has enabled more uniform diagnosis and staging of neonatal AKI. Several risk factors have been implicated in the development of neonatal AKI, including perinatal asphyxia, neonatal sepsis, respiratory distress syndrome, prematurity, dehydration, congenital heart disease, and exposure to nephrotoxic medications. Among these, sepsis and perinatal asphyxia are consistently reported as the leading etiological factors in developing countries.[2]

The pathophysiology of neonatal AKI is multifactorial and involves renal hypoperfusion, ischemia-reperfusion injury, inflammatory responses, oxidative stress, and direct nephrotoxic effects. Neonates with AKI often present with oliguria, fluid overload, electrolyte disturbances, metabolic acidosis, and elevated serum creatinine levels. Early identification remains challenging because serum creatinine may not accurately reflect renal function during the first days of life, as neonatal levels are influenced by maternal creatinine.[3]

Mortality among neonates with AKI remains high despite improvements in intensive care practices. Previous studies have demonstrated that severe AKI, requirement of mechanical ventilation, shock, dialysis dependence, and associated multiorgan dysfunction significantly increase the risk of death. Furthermore, survivors of neonatal AKI are at increased risk of developing chronic kidney disease, hypertension, and long-term renal dysfunction later in life. Therefore, recognizing predictors of mortality is crucial for identifying high-risk neonates, optimizing monitoring strategies, and initiating timely interventions.[4]

Although several studies have evaluated the incidence and etiological profile of neonatal AKI, data regarding predictors of mortality in Indian NICU settings remain limited. Understanding the clinical and laboratory factors associated with poor outcomes can help clinicians improve risk stratification and management protocols. Hence, the present prospective observational study was undertaken to identify predictors of mortality and evaluate the clinical outcomes among neonates diagnosed with acute kidney injury in a tertiary care hospital.[5]

Aim

To identify predictors of mortality and evaluate clinical outcomes among neonates with acute kidney injury admitted to a tertiary care NICU.

Objectives

1. To study the clinical profile and etiological factors associated with neonatal acute kidney injury.
2. To identify clinical and laboratory predictors of mortality among neonates with acute kidney injury.
3. To assess short-term clinical outcomes, including recovery, complications, duration of hospitalization, and mortality in neonates with acute kidney injury.

MATERIALS AND METHODS

Source of Data: The data were collected from neonates admitted to the Neonatal Intensive Care Unit (NICU) of the Department of Pediatrics at a tertiary care teaching hospital. Clinical, demographic, maternal, laboratory, and outcome-related information were obtained from hospital records, bedside assessments, and laboratory investigations using a structured data collection proforma.

Study Design: A hospital-based prospective observational study was conducted.

Study Location: The study was conducted in the Neonatal Intensive Care Unit (NICU) of a tertiary care teaching hospital.

Study Duration: The study was carried out over a period of 24 months.

Sample Size: A total of 100 neonates diagnosed with acute kidney injury were included in the study.

Inclusion Criteria

1. Neonates aged 0–28 days admitted to the NICU.
2. Neonates fulfilling modified neonatal KDIGO criteria for acute kidney injury.
3. Neonates with oliguria (urine output <1 mL/kg/hour) or elevated serum creatinine suggestive of AKI.
4. Parents or legal guardians providing written informed consent.

Exclusion Criteria

1. Neonates with congenital anomalies of the kidney and urinary tract (CAKUT).
2. Neonates with known chromosomal abnormalities affecting renal function.
3. Neonates with inherited metabolic disorders associated with renal dysfunction.
4. Neonates discharged against medical advice before completion of evaluation.
5. Neonates whose parents refused consent.

Procedure and Methodology: After obtaining approval from the Institutional Ethics Committee and written informed consent from parents or guardians, all eligible neonates admitted to the NICU were screened for AKI. Diagnosis and staging of AKI were established according to the modified neonatal KDIGO criteria based on serum creatinine levels and urine output measurements.

A detailed maternal history including maternal age, antenatal complications, pregnancy-induced hypertension, diabetes mellitus, premature rupture of

membranes, and maternal infections was recorded. Neonatal details including gestational age, birth weight, sex, mode of delivery, APGAR scores, and birth status (inborn/outborn) were documented.

Clinical evaluation was performed daily to identify etiological factors such as sepsis, perinatal asphyxia, respiratory distress syndrome, dehydration, shock, and exposure to nephrotoxic drugs. Urine output was monitored hourly using diaper weight measurements or urinary catheterization when indicated.

All neonates underwent laboratory investigations including complete blood count, blood culture, serum creatinine, blood urea nitrogen, serum electrolytes, arterial blood gas analysis, and urine examination. Radiological investigations and ultrasonography of kidneys were performed whenever clinically indicated.

Patients were managed according to standard NICU protocols. Information regarding requirement of mechanical ventilation, inotropic support, dialysis, duration of NICU stay, complications, and final outcome was recorded. Neonates were followed until discharge or death.

Sample Processing: Venous blood samples were collected under aseptic precautions. Approximately 1–2 mL of blood was obtained for biochemical analysis. Serum creatinine, blood urea nitrogen, sodium, potassium, chloride, bicarbonate, calcium, and other relevant biochemical parameters were measured using automated analyzers in the central laboratory.

Blood culture samples were processed according to standard microbiological procedures. Urine samples were collected aseptically and analyzed for routine microscopy and culture whenever indicated. All laboratory investigations were performed in accordance with established quality-control protocols.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) version 25.0.

1. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) depending on data distribution.
2. Categorical variables were expressed as frequencies and percentages.
3. Student's t-test or Mann–Whitney U test was used for comparison of continuous variables.
4. Chi-square test or Fisher's exact test was used for comparison of categorical variables.
5. Univariate logistic regression analysis was performed to identify factors associated with mortality.
6. Variables showing statistical significance in univariate analysis were entered into multivariate logistic regression analysis to determine independent predictors of mortality.
7. Odds ratios (OR) with 95% confidence intervals (CI) were calculated.
8. A p-value <0.05 was considered statistically significant.

Data Collection: Data were collected prospectively using a predesigned and pretested case record form. Information recorded included:

- Maternal demographic and obstetric details.
- Neonatal demographic characteristics.
- Gestational age and birth weight.
- Clinical presentation and etiological factors of AKI.
- Laboratory investigations and KDIGO staging.
- Requirement of mechanical ventilation, inotropes, and dialysis.
- Duration of NICU stay.
- Complications during hospitalization.
- Clinical outcome (recovered, discharged, referred, or died).

All collected data were verified for completeness and accuracy before statistical analysis.

RESULTS

Table 1: Predictors of Mortality and Clinical Outcomes among Neonates with AKI Admitted to Tertiary Care NICU (N=100)

Variable	n (%) / Mean ± SD	95% CI	Test value	P value
Age at admission (days)	5.8 ± 3.1	5.2–6.4	t=18.71	<0.001
Birth weight (kg)	2.48 ± 0.54	2.37–2.59	t=45.93	<0.001
Male	64 (64.0)	54.2–72.8	χ^2=7.84	0.005
Female	36 (36.0)	27.2–45.8		
Term neonates	62 (62.0)	52.2–70.9	χ^2=5.76	0.016
Preterm neonates	38 (38.0)	29.1–47.8		
Inborn	58 (58.0)	48.2–67.2	χ^2=2.56	0.110
Outborn	42 (42.0)	32.8–51.8		
Mechanical ventilation required	28 (28.0)	20.1–37.5	χ^2=9.45	0.002
Shock present	25 (25.0)	17.5–34.3	χ^2=8.12	0.004
KDIGO stage III	18 (18.0)	11.7–26.7	χ^2=12.96	<0.001
Peritoneal dialysis required	6 (6.0)	2.8–12.5	χ^2=16.42	<0.001
Improved / discharged	88 (88.0)	80.2–93.1	χ^2=57.76	<0.001
Death	12 (12.0)	6.9–19.8		

[Table 1] presents the baseline characteristics, predictors of mortality, and overall clinical outcomes among 100 neonates diagnosed with acute kidney injury (AKI) and admitted to the tertiary care NICU.

The mean age at admission was 5.8 ± 3.1 days (95% CI: 5.2–6.4 days), while the mean birth weight was 2.48 ± 0.54 kg (95% CI: 2.37–2.59 kg), both demonstrating statistically significant distributions

(p<0.001). Male neonates constituted the majority of the study population (64%), which was significantly higher than females (36%) (χ^2=7.84, p=0.005). Term neonates accounted for 62% of cases compared to 38% preterm neonates, indicating a significant predominance of term births among AKI cases (χ^2=5.76, p=0.016). More than half of the neonates were inborn (58%), while 42% were outborn; however, this difference was not statistically significant (p=0.110). Regarding severity indicators, 28% of neonates required mechanical ventilation, 25% developed shock, 18% were classified as KDIGO stage III AKI, and 6% required peritoneal dialysis. All these variables showed statistically significant associations, highlighting their potential role as markers of severe disease (p<0.01 for all). Clinical outcomes revealed that 88% of neonates improved and were discharged successfully, whereas mortality was observed in 12% of cases. The significantly higher proportion of survivors compared to deaths (χ^2=57.76, p<0.001) indicates favorable outcomes in the majority of patients, although a substantial burden of mortality persisted among severe AKI cases.

Table 2: Clinical Profile and Etiological Factors Associated with Neonatal AKI (N=100)

Variable	n (%) / Mean ± SD	95% CI	Test value	P value
Oliguria	32 (32.0)	23.7–41.7	χ^2=19.52	<0.001
Non-oliguric AKI	58 (58.0)	48.2–67.2		
Anuria	10 (10.0)	5.5–17.4		
Sepsis	46 (46.0)	36.6–55.7	χ^2=15.84	<0.001
Perinatal asphyxia	34 (34.0)	25.5–43.7	χ^2=4.24	0.039
Respiratory distress syndrome	20 (20.0)	13.3–28.9	χ^2=36.00	<0.001
Dehydration / fever	18 (18.0)	11.7–26.7	χ^2=40.96	<0.001
Prematurity	38 (38.0)	29.1–47.8	χ^2=5.76	0.016
Low birth weight	35 (35.0)	26.4–44.7	χ^2=9.00	0.003
Nephrotoxic drug exposure	22 (22.0)	15.0–31.1	χ^2=31.36	<0.001
Serum creatinine (mg/dL)	1.82 ± 0.64	1.69–1.95	t=28.43	<0.001
Blood urea (mg/dL)	64.5 ± 18.7	60.8–68.2	t=34.49	<0.001
Serum potassium (mEq/L)	5.6 ± 0.9	5.4–5.8	t=62.22	<0.001

[Table 2] summarizes the clinical presentation, etiological factors, and laboratory profile of neonates with acute kidney injury. Among the study participants, non-oliguric AKI was the most common presentation, observed in 58% of neonates, followed by oliguria in 32% and anuria in 10%, with the distribution being highly significant (χ^2=19.52, p<0.001). Sepsis emerged as the leading etiological factor, affecting 46% of neonates (95% CI: 36.6–55.7%), followed by prematurity (38%), low birth weight (35%), and perinatal asphyxia (34%). Respiratory distress syndrome and dehydration/fever were present in 20% and 18% of cases, respectively. Exposure to nephrotoxic drugs was documented in 22% of neonates. All these factors showed statistically significant associations with neonatal AKI, emphasizing their importance in disease development. Laboratory evaluation demonstrated a mean serum creatinine level of 1.82 ± 0.64 mg/dL (95% CI: 1.69–1.95 mg/dL), mean blood urea level of 64.5 ± 18.7 mg/dL (95% CI: 60.8–68.2 mg/dL), and mean serum potassium concentration of 5.6 ± 0.9 mEq/L (95% CI: 5.4–5.8 mEq/L), all of which were highly significant (p<0.001). These findings indicate that sepsis, prematurity, low birth weight, and perinatal asphyxia were the predominant contributors to neonatal AKI, accompanied by marked biochemical evidence of renal dysfunction.

Table 3: Clinical and Laboratory Predictors of Mortality among Neonates with AKI (N=100)

Predictor	Survivors n=88	Deaths n=12	95% CI / OR	Test value	P value
Age at admission (days), Mean ± SD	5.5 ± 2.9	7.9 ± 3.6	MD=2.4; 0.6–4.2	t=2.78	0.007
Birth weight (kg), Mean ± SD	2.54 ± 0.49	2.05 ± 0.61	MD=0.49; 0.18–0.80	t=3.25	0.002
Sepsis	36 (40.9)	10 (83.3)	OR=7.22; 1.49–34.99	χ^2=7.56	0.006
Perinatal asphyxia	27 (30.7)	7 (58.3)	OR=3.16; 0.93–10.71	χ^2=3.65	0.056
Shock	17 (19.3)	8 (66.7)	OR=8.35; 2.25–30.99	χ^2=12.52	<0.001
Mechanical ventilation	20 (22.7)	8 (66.7)	OR=6.80; 1.85–25.00	χ^2=10.29	0.001
KDIGO stage III	11 (12.5)	7 (58.3)	OR=9.80; 2.61–36.79	χ^2=14.52	<0.001
Peritoneal dialysis	2 (2.3)	4 (33.3)	OR=21.50; 3.37–137.17	Fisher exact	<0.001
Serum creatinine (mg/dL), Mean ± SD	1.68 ± 0.51	2.85 ± 0.72	MD=1.17; 0.75–1.59	t=7.09	<0.001
Blood urea (mg/dL), Mean ± SD	59.8 ± 15.4	99.2 ± 21.5	MD=39.4; 28.3–50.5	t=7.94	<0.001
Hyperkalemia	18 (20.5)	7 (58.3)	OR=5.45; 1.54–19.27	χ^2=8.29	0.004
Metabolic acidosis	24 (27.3)	8 (66.7)	OR=5.33; 1.48–19.18	χ^2=8.01	0.005

[Table 3] compares survivors (n=88) and non-survivors (n=12) to identify factors associated with mortality in neonatal AKI. Neonates who died had a significantly higher mean age at admission (7.9 ± 3.6 days) compared to survivors (5.5 ± 2.9 days) (p=0.007). They also had significantly lower birth weight (2.05 ± 0.61 kg vs. 2.54 ± 0.49 kg; p=0.002). Sepsis was present in 83.3% of non-survivors compared to 40.9% of survivors and was associated with a seven-fold increased risk of death (OR=7.22; p=0.006). Although perinatal asphyxia was more common among deaths (58.3%) than survivors (30.7%), the association did not reach statistical significance (p=0.056). Clinical severity indicators

showed strong associations with mortality. Shock increased the odds of death more than eight times (OR=8.35; p<0.001), while mechanical ventilation increased mortality risk nearly seven-fold (OR=6.80; p=0.001). KDIGO stage III AKI was a particularly strong predictor, increasing mortality risk almost ten-fold (OR=9.80; p<0.001). Peritoneal dialysis requirement emerged as the strongest predictor, with a 21.5-fold increase in mortality risk (p<0.001). Laboratory parameters were also significantly worse among non-survivors, who had markedly elevated serum creatinine (2.85 ± 0.72 mg/dL) and blood urea levels (99.2 ± 21.5 mg/dL) compared to survivors (p<0.001). Hyperkalemia and metabolic acidosis were significantly associated with mortality, increasing the odds of death by 5.45 and 5.33 times, respectively. These findings indicate that advanced AKI, hemodynamic instability, need for intensive support, and severe biochemical derangements are major determinants of mortality in neonatal AKI.

Table 4: Short-Term Clinical Outcomes among Neonates with AKI (N=100)

Outcome Variable	n (%) / Mean ± SD	95% CI	Test value	P value
Complete renal recovery	76 (76.0)	66.8–83.3	χ^2=27.04	<0.001
Partial renal recovery	12 (12.0)	6.9–19.8		
Death	12 (12.0)	6.9–19.8		
Duration of NICU stay (days)	9.6 ± 4.2	8.8–10.4	t=22.86	<0.001
Duration of AKI recovery (days)	5.4 ± 2.1	5.0–5.8	t=25.71	<0.001
Mechanical ventilation	28 (28.0)	20.1–37.5	χ^2=19.36	<0.001
Inotropic support	25 (25.0)	17.5–34.3	χ^2=25.00	<0.001
Peritoneal dialysis	6 (6.0)	2.8–12.5	χ^2=77.44	<0.001
Electrolyte imbalance	42 (42.0)	32.8–51.8	χ^2=2.56	0.110
Hyperkalemia	25 (25.0)	17.5–34.3	χ^2=25.00	<0.001
Metabolic acidosis	32 (32.0)	23.7–41.7	χ^2=12.96	<0.001
Fluid overload	18 (18.0)	11.7–26.7	χ^2=40.96	<0.001
Discharged successfully	88 (88.0)	80.2–93.1	χ^2=57.76	<0.001
Mortality rate	12 (12.0)	6.9–19.8		

[Table 4] depicts the short-term outcomes and complications among neonates with acute kidney injury. Complete renal recovery was achieved in 76% of neonates, while 12% experienced partial recovery and 12% died during hospitalization. The predominance of complete recovery was statistically significant (χ^2=27.04, p<0.001), indicating favorable short-term renal outcomes in most cases. The mean duration of NICU stay was 9.6 ± 4.2 days (95% CI: 8.8–10.4 days), and the mean duration required for AKI recovery was 5.4 ± 2.1 days (95% CI: 5.0–5.8 days), both showing highly significant results (p<0.001). Mechanical ventilation and inotropic support were required in 28% and 25% of neonates, respectively, reflecting the substantial burden of critical illness among affected infants. Peritoneal dialysis was required in 6% of cases, indicating severe renal impairment in a subset of neonates. Electrolyte imbalance was observed in 42% of patients, although this was not statistically significant (p=0.110). Hyperkalemia, metabolic acidosis, and fluid overload were documented in 25%, 32%, and 18% of neonates, respectively, and all showed significant associations (p<0.001). Overall, 88% of neonates were discharged successfully, whereas the mortality rate remained 12%. These findings suggest that despite considerable morbidity and the need for intensive supportive care, the majority of neonates with AKI experienced recovery and favorable short-term outcomes when managed appropriately in a tertiary care NICU setting.

DISCUSSION

In the present prospective observational study of 100 neonates with acute kidney injury (AKI), the mean age at admission was 5.8 ± 3.1 days and the mean birth weight was 2.48 ± 0.54 kg. Male neonates constituted 64% of cases, showing male predominance. Similar male predominance was reported by Mathur et al,[1] (2006) Mortazavi et al,[2] (2009) and AlGadeeb et al,[3] (2021) who observed that neonatal AKI was more frequent among male neonates admitted to NICU. In the present study, 62% were term and 38% were preterm neonates. This finding is comparable with Meshram et al,[4] (2021) who also reported a significant burden of AKI among term neonates, especially when associated with sepsis, asphyxia, and shock. However, Jetton et al. (2017),[5] and Selewski et al (2015),[6] reported higher AKI frequency among preterm and critically ill neonates, probably due to immature renal function, nephrotoxic exposure, and prolonged intensive care support.

In the present study, non-oliguric AKI was the most common clinical pattern, seen in 58% of cases, followed by oliguria in 32% and anuria in 10%. This observation is consistent with studies by Agras et al. (2004),[7] and Momtaz et al (2014),[8] who reported that non-oliguric AKI is common in neonates and may be missed if urine output alone is used for diagnosis. The presence of anuria in 10% of neonates in the present study reflected severe renal dysfunction and was clinically important, as anuric neonates are more likely to require renal replacement therapy and have poor outcomes.

Sepsis was the leading etiological factor in the present study, affecting 46% of neonates. This finding is in agreement with Mathur et al (2006),[1] Jayashree et al (1991),[9] and Chirico et al. (2024),[10] who reported neonatal sepsis as one of the most important causes of AKI. Sepsis-induced AKI occurs

due to renal hypoperfusion, inflammatory cytokine release, endothelial dysfunction, microcirculatory impairment, and multiorgan involvement. Perinatal asphyxia was present in 34% of cases in the present study, which was comparable with Kaur et al. (2011),[11] and Selewski et al. (2013),[12] who reported AKI in a substantial proportion of neonates with hypoxic ischemic encephalopathy. Respiratory distress syndrome was observed in 20% of cases, while dehydration/fever was found in 18%. Similar findings were reported by Mortazavi et al (2009),[2] and Airede et al (1997),[13] who identified asphyxia, sepsis, dehydration, and respiratory illness as important contributors to neonatal renal dysfunction. The present study found that low birth weight was present in 35% of neonates and prematurity in 38%. Hu et al. (2021),[14] in a meta-analysis of critically ill neonates, reported that lower gestational age and lower birth weight were significantly associated with neonatal AKI. Low birth weight and prematurity increase susceptibility to AKI because of reduced nephron number, immature tubular function, poor autoregulation of renal blood flow, and vulnerability to hypoxic and nephrotoxic injury. Nephrotoxic drug exposure was observed in 22% of neonates in the present study, similar to findings by Rhone et al. (2014),[15] who emphasized that aminoglycosides, vancomycin, and other nephrotoxic medications are important modifiable risk factors for AKI in NICU settings.

In the present study, mechanical ventilation was required in 28% of neonates, shock was present in 25%, KDIGO stage III AKI was seen in 18%, and peritoneal dialysis was required in 6%. These findings indicate that a subset of neonates had severe systemic illness. Similar observations were reported by Meshram et al. (2021),[4] AlGadeeb et al. (2021),[3] and Gohiya et al (2022),[16] who found that shock, mechanical ventilation, higher AKI stage, and dialysis requirement were strongly associated with poor outcomes. The AWAKEN study by Jetton et al. (2017),[5] also showed that neonatal AKI was associated with increased mortality, longer hospital stay, and greater need for intensive care support.

Mortality in the present study was 12%, while 88% of neonates improved and were discharged. This mortality rate was lower than that reported in some earlier studies by Airede et al. (1997),[13] Mortazavi et al (2009),[2] and Momtaz et al (2014),[8] where mortality was higher among neonates with severe AKI. The relatively lower mortality in the present study may be attributed to early recognition, better NICU monitoring, timely correction of electrolyte imbalance, and availability of supportive care. However, mortality remained significantly associated with severe AKI and systemic complications.

Comparison between survivors and non-survivors revealed that neonates who died had significantly higher mean age at admission, lower birth weight, higher serum creatinine, and higher blood urea levels. Sepsis was present in 83.3% of deaths compared to 40.9% of survivors, with an odds ratio of 7.22. Similar findings were observed by Mathur et al (2006),[1] and Jayashree et al (1991),[9] who reported high mortality among neonates with septicemia-associated renal dysfunction. Shock was another strong predictor of mortality in the present study, increasing the odds of death by 8.35 times. This is comparable with Meshram et al,[4] (2021) and Youssef et al,[17] (2015) who reported that hemodynamic instability and inotropic requirement were significant predictors of mortality.

Mechanical ventilation was required in 66.7% of non-survivors compared to 22.7% of survivors and was significantly associated with mortality. Similar findings were reported by AlGadeeb et al,[3] (2021) and Hu et al,[14] (2021), who identified mechanical ventilation as an important risk factor associated with AKI and adverse outcomes. KDIGO stage III AKI was present in 58.3% of deaths compared to 12.5% of survivors, with an odds ratio of 9.80. This finding supports the prognostic value of KDIGO staging, as also reported by Jetton et al,[5] (2017) Selewski et al,[6] (2015) and Samuel et al,[18] (2026). Peritoneal dialysis showed the highest odds ratio for mortality (OR=21.50), suggesting that dialysis requirement reflected advanced renal injury and severe systemic illness.

Laboratory parameters also showed strong association with mortality. Non-survivors had significantly higher serum creatinine and blood urea levels than survivors. Hyperkalemia was present in 58.3% of deaths compared to 20.5% of survivors, while metabolic acidosis was present in 66.7% of deaths compared to 27.3% of survivors. Similar biochemical predictors of mortality were reported by Meshram et al,[4] (2021) Youssef et al (2015),[17] and Mortazavi et al (2009).[2] Hyperkalemia and metabolic acidosis indicate severe renal impairment and impaired homeostatic regulation, requiring urgent recognition and management.

Short-term outcomes in the present study showed complete renal recovery in 76%, partial recovery in 12%, and mortality in 12%. The mean duration of NICU stay was 9.6 ± 4.2 days, and the mean duration of AKI recovery was 5.4 ± 2.1 days. These findings are comparable with AlGadeeb et al (2021),[3] and Coleman et al (2022),[19] who reported that neonatal AKI is associated with prolonged hospitalization and increased intensive care requirement. Electrolyte imbalance was observed in 42%, hyperkalemia in 25%, metabolic acidosis in 32%, and fluid overload in 18% of neonates. These complications reflect the systemic impact of AKI and emphasize the need for close biochemical monitoring.

CONCLUSION

The present prospective observational study evaluated the clinical profile, etiological factors, predictors of mortality, and short-term outcomes among neonates with acute kidney injury admitted to

a tertiary care NICU. The study demonstrated that neonatal AKI remains a significant contributor to neonatal morbidity and mortality despite advances in neonatal intensive care. Sepsis emerged as the most common etiological factor, followed by perinatal asphyxia, prematurity, low birth weight, respiratory distress syndrome, and exposure to nephrotoxic medications. Non-oliguric AKI was the predominant clinical presentation, emphasizing the importance of routine biochemical monitoring for early diagnosis.

The overall mortality rate was 12%, while the majority of neonates (88%) were successfully discharged following appropriate management. Several clinical and laboratory variables were identified as significant predictors of mortality. Neonates with shock, requirement of mechanical ventilation, KDIGO stage III AKI, and need for peritoneal dialysis had significantly higher risk of death. Elevated serum creatinine and blood urea levels, hyperkalemia, and metabolic acidosis were also strongly associated with adverse outcomes. Lower birth weight and delayed presentation further increased mortality risk.

The study highlights that severe AKI is often a manifestation of underlying systemic illness and multiorgan dysfunction. Early recognition of high-risk neonates, prompt management of sepsis and shock, careful monitoring of renal function, avoidance of nephrotoxic medications whenever possible, and timely initiation of renal replacement therapy are essential to improve survival. Identification of these mortality predictors can help clinicians stratify risk, optimize resource utilization, and improve neonatal outcomes. Overall, early diagnosis and aggressive management of neonatal AKI remain crucial strategies for reducing mortality and improving short-term clinical outcomes in NICU settings.

Limitations of the study

1. The study was conducted at a single tertiary care center, which may limit the generalizability of the findings to other healthcare settings.
2. The sample size of 100 neonates, although adequate for analysis, may not fully represent the diverse spectrum of neonatal AKI.
3. Long-term renal outcomes and neurodevelopmental outcomes were not assessed after discharge.
4. Follow-up of survivors beyond the hospital stay was not performed, preventing evaluation of chronic kidney disease risk.
5. Biomarkers of early kidney injury such as NGAL, cystatin C, and KIM-1 were not measured.
6. The observational study design limited the ability to establish causal relationships between risk factors and mortality.
7. Variability in treatment protocols and supportive care measures may have influenced clinical outcomes.
8. Maternal and antenatal risk factors were not evaluated in extensive detail.
9. Some neonates had multiple overlapping etiological factors, making it difficult to determine the independent contribution of each factor.
10. The study relied primarily on serum creatinine and urine output-based KDIGO criteria, which may underestimate early or subclinical renal injury.
11. The small number of deaths may have limited the precision of mortality risk estimates for certain variables.
12. Advanced renal imaging and detailed hemodynamic monitoring were not uniformly available for all participants.

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