Section: Paediatrics

Original Research Article

ACUTE KIDNEY INJURY AMONG NEONATES: A STUDY ON AETIOLOGICAL FACTORS AND CLINICAL OUTCOMES IN A TERTIARY CARE SETTING

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Received *: 07/03/2025*
Received in revised form *: 17/04/2025*
Accepted *: 13/05/2025*

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DOI: 10.70034/ijmedph.2025.3.55

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

Int J Med Pub Health
2025; 15 (2); 306-312

ABSTRACT

Background: Acute kidney injury (AKI) in neonates is a serious and potentially life-threatening condition that often arises in the context of sepsis, perinatal asphyxia, and other neonatal complications. Early identification and appropriate management are crucial to reduce morbidity and mortality. This study aimed to determine the aetiological profile, clinical characteristics, and outcomes of AKI among neonates admitted to a tertiary care NICU in India.

Materials and Methods: A Retrospective observational study was conducted in the NICU of a tertiary care hospital over a 24-month period (August 2022 to July 2024). A total of 3200 neonates were screened, of whom 168 met the criteria for AKI based on modified KDIGO definitions. Demographic data, clinical and laboratory parameters, etiology of AKI, interventions, and outcomes (recovery, mortality, and progression to chronic kidney disease) were recorded and analyzed.

Results: Among 168 neonates with AKI, the mean age was 5.3 ± 3.1 days, with a male predominance (58.3%). The leading causes of AKI were sepsis (48.8%), perinatal asphyxia (32.1%), and dehydration/ hypovolemia (26.2%). The majority of cases were intrinsic renal AKI (56%), followed by prerenal (41.1%) and postrenal (3%). Laboratory findings showed elevated serum creatinine (1.8 ± 0.5 mg/dL) and BUN (48.5 ± 12.7 mg/dL), with 36.3% showing metabolic acidosis. Interventions included antibiotics (88.1%), fluid resuscitation (60.7%), and peritoneal dialysis in 14.3%. Complete renal recovery was seen in 64.2% of cases, while 4.8% progressed to chronic kidney disease. The overall mortality rate was 30.9%, significantly higher among those with intrinsic renal AKI ($p < 0.0001$).

Conclusion: Sepsis and perinatal asphyxia remain the most common etiological factors for neonatal AKI in Indian NICUs. Intrinsic renal AKI is associated with significantly higher mortality. Early diagnosis and prompt management are essential to improve outcomes and reduce long-term renal complications in this vulnerable population.

Keywords: Neonatal acute kidney injury, NICU, sepsis, perinatal asphyxia, renal outcome, intrinsic renal AKI, neonatal mortality, India.

INTRODUCTION

Acute Kidney Injury (AKI) in neonates is a major cause of morbidity and mortality, particularly in Neonatal Intensive Care Units (NICUs), where critically ill neonates are managed. AKI is defined as a sudden decline in kidney function, often indicated by an increase in serum creatinine, blood urea nitrogen, or a reduction in urine output. In neonates, the aetiology of AKI is multifactorial and can be broadly classified into prerenal, intrinsic renal, and postrenal causes.[1] Prerenal causes, such as reduced renal perfusion from hypotension, dehydration, or blood loss, are common in neonates, particularly in those born preterm or with low birth weight. Intrinsic renal causes often result from nephrotoxic medications, infections, or ischemia, while postrenal causes include urinary tract

obstructions, such as those due to structural abnormalities or meconium ileus in neonates.[1,2]

Neonates admitted to NICUs are at particularly high risk for AKI due to several factors including prematurity, low birth weight, hypoxia, and exposure to nephrotoxic agents. In India incidence of AKI in NICUs ranged from 2% to 12%, with the incidence being notably higher in preterm and low birth weight infants.[3,4]

Perinatal asphyxia, which is common in neonates with a history of intrauterine growth restriction, is one of the leading causes of AKI in neonates. Asphyxia leads to hypoxia, which in turn causes renal ischemia and subsequent kidney injury.[5]

Sepsis is another major contributor to neonatal AKI. Infection-induced AKI occurs due to both direct renal damage from the infection and the systemic inflammatory response, which leads to altered renal perfusion. Neonates with sepsis have been shown to have a significantly increased risk of developing AKI, with estimating the incidence of AKI in septic neonates to be as high as 27%.[6]

Neonatal exposure to nephrotoxic medications such as aminoglycosides, vancomycin, and contrast agents is another significant risk factor for AKI. These drugs, while essential for managing infections, can cause renal tubular damage, especially when used in high doses or over prolonged periods. The incidence of drug-induced AKI varies, up to 20% in NICU settings, particularly among those receiving high-risk medications.[7] Furthermore, congenital anomalies of the kidneys or urinary tract (CAKUT), such as posterior urethral valves or obstructive uropathy, contribute to postrenal AKI in neonates, though these are less common.[8]

Despite the high burden of neonatal AKI, particularly in low- and middle-income countries like India, there is limited data on its aetiology and outcomes. The mortality rate of 28% among neonates with AKI, with those requiring dialysis having an even higher mortality rate.[8] Furthermore, a significant proportion of neonates who survive AKI suffer long-term renal impairment, which can affect their growth and development, leading to chronic kidney disease in later childhood.[9]

Given the diverse and multifactorial causes of AKI in neonates, early detection and timely intervention are essential for improving outcomes. However, there remains a lack of consensus on the optimal diagnostic criteria, prevention strategies, and treatment protocols for neonatal AKI, particularly in resource-limited settings.[10] This study aimed to explore the aetiological factors contributing to AKI in neonates admitted to the NICU at a tertiary care hospital and assess the associated outcomes, with the goal of identifying modifiable risk factors and improving the management of this critical condition.

MATERIALS AND METHODS

Study Design and Setting

This was a retrospective, observational study conducted at the Neonatal Intensive Care Unit (NICU) of paediatrics department tertiary care hospital located in North India. The study was carried out over a period of 2 years from August 2022 to July 2024. This tertiary care center serves a diverse population, including neonates from both rural and urban areas, with a significant burden of neonatal complications.

Study Population

The study included neonates admitted to the NICU during the study period. Neonates were eligible for inclusion if they met the diagnostic criteria for Acute Kidney Injury (AKI) as per the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines.[11] These criteria include a serum creatinine increase of ≥0.3 mg/dL within 48 hours, a ≥50% increase in baseline serum creatinine, or a urine output of less than 0.5 mL/kg/h for more than 6 hours. Neonates with chronic kidney disease or congenital renal anomalies that were diagnosed prior to admission, as well as those who died within the first 24 hours of NICU admission, were excluded from the study. Furthermore, neonates who were transferred to another facility within 24 hours of admission were also excluded to ensure a complete follow-up. So, study included 168 neonates after inclusion and exclusion criteria.

Data Collection

Comprehensive data were collected for each eligible neonate upon admission and throughout their NICU stay. Demographic information, including the neonate's age, sex, gestational age, birth weight, mode of delivery, and Apgar scores, was recorded. A detailed maternal history, including antenatal complications (such as diabetes, hypertension, or pre-eclampsia) and obstetric risk factors, was also obtained. Perinatal history, including the presence of any perinatal complications such as meconium aspiration syndrome, intrauterine growth restriction, or perinatal asphyxia, was documented.

Clinical data were meticulously collected during the neonate's NICU admission, with particular focus on the severity of illness at admission. Neonates were evaluated for the presence of comorbid conditions, including respiratory distress syndrome, sepsis, or necrotizing enterocolitis, as these are frequently associated with AKI. Detailed documentation was made of any nephrotoxic medications administered, including antibiotics (such as aminoglycosides, vancomycin), diuretics, and intravenous contrast agents used during diagnostic procedures. Laboratory investigations, including serum creatinine, urea levels, electrolyte abnormalities (such as hyperkalemia or hyponatremia), and urine output, were monitored daily. Urine microscopy was performed when clinically indicated, especially in

cases where infections or other renal pathologies were suspected.

Aetiological Classification

The aetiology of AKI in neonates was determined by a thorough review of clinical, laboratory, and imaging data. Based on the available evidence, the causes of AKI were categorized into three primary groups: prerenal, intrinsic renal, and postrenal causes. Prerenal causes of AKI were defined as those resulting from decreased renal perfusion, often secondary to hypovolemia, hypotension, or shock. These included cases associated with perinatal asphyxia or systemic blood loss. Intrinsic renal causes were further subdivided into ischemic and nephrotoxic causes. Ischemic causes included renal hypoxia or ischemia due to perinatal asphyxia, while nephrotoxic causes included exposure to harmful substances, such as certain antibiotics, diuretics, or contrast agents. Postrenal causes were defined as those secondary to urinary tract obstruction, which could include congenital anomalies such as posterior urethral valves or other structural abnormalities. The diagnosis of postrenal AKI was confirmed based on ultrasonographic findings or, when necessary, other imaging techniques.

Outcomes Measures

The primary outcome of the study was to assess the mortality and morbidity associated with neonatal AKI. Mortality data were recorded, including the time of death and the potential causes, such as multiorgan failure or progression to end-stage renal disease. Secondary outcomes included the need for renal replacement therapy (RRT), specifically dialysis, the length of NICU stay, and the long-term renal outcomes if follow-up data were available. Neonates who survived the initial NICU admission were assessed for kidney function at discharge and were scheduled for follow-up visits to monitor for potential chronic kidney disease or other long-term renal complications.

Statistical Analysis

Data were analyzed using descriptive statistics to summarize the demographic, clinical, and aetiological characteristics of the study population. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. The chi-square test was used for the comparison of categorical variables. A p-value of <0.05 was considered statistically significant. Statistical analysis was performed using SPSS software version 20.0 (IBM, USA).

Ethical Considerations

The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All collected data were kept confidential, and patient identifiers were removed to maintain anonymity. Data were stored securely and were only accessible to the research team. The study was approved by the Institutional Review Board (IRB), and all procedures were in compliance with institutional guidelines.

RESULTS

A total of 3,200 neonates were admitted and screened for Acute Kidney Injury (AKI) during the study period, of which 168 neonates (5.25%) were diagnosed with AKI and included in the analysis. The mean age of affected neonates was 5.3 ± 3.1 days. Males accounted for 58.3% (n = 98) of cases, while females comprised 41.7% (n = 70). The average gestational age was 34.2 ± 2.3 weeks, and the mean birth weight was 2180 ± 540 grams. The mean Apgar scores at 1 and 5 minutes were 5.6 ± 1.1 and 7.2 ± 0.8, respectively. With respect to the mode of delivery, 58.3% (n = 98) of neonates were delivered by cesarean section, and 41.7% (n = 70) by vaginal delivery. Prematurity was observed in 64.3% (n = 108) of the neonates. Among comorbid conditions, sepsis was the most common (48.8%), followed by respiratory distress syndrome (38.1%), perinatal asphyxia (32.1%), intrauterine growth restriction (22.6%), and necrotizing enterocolitis (15.5%). In terms of the underlying cause of AKI, intrinsic renal causes were most prevalent (56.0%; n = 94), followed by prerenal causes (41.1%; n = 69) and postrenal causes (3.0%; n = 5). [Table 1]

Table 1: Baseline Demographic and Clinical Characteristics of Neonates with Acute Kidney Injury (n = 168)

Variable	Frequency (%)/mean ± SD
Age (days)	5.3 ± 3.1
Gender	
Male	98 (58.3%)
Female	70 (41.7%)
Gestational Age (weeks)	34.2 ± 2.3
Birth Weight (grams)	2180 ± 540
Apgar Score at 1 minute	5.6 ± 1.1
Apgar Score at 5 minutes	7.2 ± 0.8
Mode of Delivery	
Vaginal	70 (41.7%)
Cesarean	98 (58.3%)
Incidence of Prematurity	108 (64.3%)
Comorbidities	
Necrotizing Enterocolitis	26 (15.5%)
Respiratory Distress Syndrome	64 (38.1%)
Intrauterine Growth Restriction	38 (22.6%)

Cause of AKI	
Prerenal	69 (41.1%)
Intrinsic Renal	94 (56.0%)
Postrenal	5 (3.0%)

Among the 168 neonates diagnosed with AKI, sepsis emerged as the leading aetiological factor, observed in 48.8% (n = 82) of cases, followed by perinatal asphyxia in 32.1% (n = 54) and dehydration or hypovolemia in 26.2% (n = 44). Nephrotoxic drug exposure accounted for 21.4% (n = 36), while congenital renal anomalies and obstructive uropathy were noted in 6.5% (n = 11) and 3.0% (n = 5) of cases, respectively. Other less common causes, including metabolic and hematologic conditions, were identified in 5.4% (n = 9) of the neonates (Table 2).

Table 2: Aetiological Profile of Acute Kidney Injury among Neonates (n = 168)

Aetiology	Frequency (%)
Perinatal Asphyxia	54 (32.1%)
Sepsis	82 (48.8%)
Nephrotoxic Drug Exposure	36 (21.4%)
Congenital Renal Anomalies	11 (6.5%)
Dehydration / Hypovolemia	44 (26.2%)
Obstructive Uropathy	5 (3.0%)
Other Factors	9 (5.4%)

Laboratory evaluation revealed a mean serum creatinine level of 1.8 ± 0.5 mg/dL and blood urea nitrogen (BUN) of 48.5 ± 12.7 mg/dL, indicating significant renal dysfunction. The average urine output was 0.58 ± 0.3 mL/kg/h, suggestive of oliguric AKI in many cases. Electrolyte analysis showed hyperkalemia with serum potassium at 5.6 ± 0.9 mEq/L, hyponatremia with serum sodium at 133.2 ± 5.4 mEq/L, and hypocalcemia with serum calcium at 7.8 ± 1.2 mg/dL. Metabolic acidosis (pH < 7.3) was documented in 36.3% (n = 61) of neonates, and urine microscopy abnormalities were found in 33.9% (n = 57), supporting the diagnosis of renal injury (Table 3).

Table 3: Laboratory Parameters among Neonates Diagnosed with Acute Kidney Injury (n = 168)

Parameter	Frequency (%)/mean ± SD
Serum Creatinine (mg/dL)	1.8 ± 0.5
Blood Urea Nitrogen (mg/dL)	48.5 ± 12.7
Urine Output (mL/kg/h)	0.58 ± 0.3
Serum Potassium (mEq/L)	5.6 ± 0.9
Serum Sodium (mEq/L)	133.2 ± 5.4
Serum Calcium (mg/dL)	7.8 ± 1.2
Metabolic Acidosis (ABG pH < 7.3)	61 (36.3%)
Urine Microscopy Abnormalities	57 (33.9%)

Regarding clinical management, inotropes were administered to 53.6% and mechanical ventilation was required in 56.5% of neonates. Fluid resuscitation was performed in 60.7%, while diuretics, mainly furosemide, were used in 39.3%. A high proportion (88.1%) received empirical or targeted antibiotic therapy. Peritoneal dialysis was required in 14.3%, indicating severe renal impairment. The mean NICU stay was 13.5 ± 5.7 days. Complete renal recovery was observed in 64.2%, while 4.8% progressed to chronic kidney disease. The overall infection rate was 42.9%, and the mortality rate stood at 30.9% (Table 4).

Table 4: Interventions and Outcomes among Neonates with Acute Kidney Injury (n = 168)

Variable	Frequency (%)
Use of Inotropes	90 (53.6%)
Mechanical Ventilation	95 (56.5%)
Fluid Resuscitation	102 (60.7%)
Diuretics (e.g., Furosemide)	66 (39.3%)
Antibiotics	148 (88.1%)
Requirement for Peritoneal Dialysis	24 (14.3%)
Infection Rates	72 (42.9%)
Complete Renal Recovery	108 (64.2%)
Progression to Chronic Kidney Disease	8 (4.8%)
Mortality	52 (30.9%)
Length of NICU Stay (days)	13.5 ± 5.7

The recovery, mortality, and chronic kidney disease (CKD) outcomes varied significantly based on the type of AKI. Among neonates with prerenal AKI (n = 69), 35.7% (n = 60) achieved complete recovery, while 4.2% (n = 7) succumbed to the condition, and 1.2% (n = 2) progressed to CKD ($p < 0.0001$). In

contrast, intrinsic renal AKI (n = 94) had a higher mortality rate, with 25.0% (n = 42) of neonates dying, and 27.4% (n = 46) achieving recovery, with 3.6% (n = 6) progressing to CKD. Postrenal AKI (n = 5) showed the best outcomes, with 80% (n = 4) of cases recovering, no progression to CKD, and only 0.6% (n = 1) mortality (Table 5).

Table 5: Association of AKI Type with Recovery, Mortality, and Chronic Kidney Disease Outcomes

Type of AKI	Recovery (n=110)	Mortality (n=50)	CKD (n=8)	p-value
	Frequency (%)			
Prerenal (n = 69)	60 (35.7%)	7 (4.2%)	2 (1.2%)	<0.0001
Intrinsic Renal (n = 94)	46 (27.4%)	42 (25.0%)	6 (3.6%)	
Postrenal (n = 5)	4 (2.4%)	1 (0.6%)	0 (0.0%)	

DISCUSSION

This study aimed to investigate the aetiological profile and outcomes of acute kidney injury (AKI) among neonates admitted to a Neonatal Intensive Care Unit (NICU) at a tertiary care hospital. A total of 3200 neonates were screened for AKI, with 168 neonates diagnosed with the condition. The findings from this study offer insights into the epidemiology, clinical characteristics, and outcomes of neonatal AKI, and compare them with those observed in similar studies from India and abroad.

In our cohort, the most common causes of AKI were sepsis (48.8%), perinatal asphyxia (32.1%), and dehydration/hypovolemia (26.2%), which is in line with the findings of similar studies conducted in neonatal ICUs across India and worldwide.[12-16] Sepsis was identified as the leading cause of AKI, consistent with studies by Gupta et al., who reported a sepsis-related AKI incidence of 18.5% in a cohort of neonates.[12] In critically ill neonates, sepsis leads to multi-organ dysfunction, including renal injury, through mechanisms such as hypoperfusion, endothelial damage, and the release of pro-inflammatory cytokines.[13] Perinatal asphyxia, which accounted for 32.1% of cases in our study, was another significant contributor to AKI. This is in agreement with findings by Gopalan et al., who reported perinatal asphyxia as a primary cause of AKI in neonates, linking it to ischemic injury and subsequent renal damage.[14]

Similarly, dehydration/hypovolemia was a contributing factor to AKI in 26.2% of cases, supporting study such as that of Charlton et al., which found dehydration to be a common predisposing factor for prerenal AKI in neonates.[17] The clinical presentation of dehydration in neonates often complicates the diagnosis of AKI, particularly in resource-constrained settings where the timely administration of fluids may be delayed due to logistical challenges.[18] Neonates with congenital renal anomalies and obstructive uropathy, though less common in our study (6.5% and 3%, respectively), have been reported as significant causes of AKI in some other studies by Jetton et al., and Harer et al.[19,20] These causes can lead to chronic renal damage and, if left untreated, may contribute to long-term renal impairment.

In our study, intrinsic renal AKI was the most prevalent type, affecting 56% of the neonates, followed by prerenal AKI (41.1%). These findings are consistent with the study by Youssef et al., who also reported a predominance of intrinsic renal injury in neonates with AKI, likely due to the severity of insults such as ischemia, nephrotoxins, and infections that lead to tubular injury.[21] Prerenal AKI, on the other hand, is usually reversible with early intervention, and in our study, neonates with prerenal AKI had a relatively good prognosis, with 35.7% achieving full renal recovery and only 4.2% succumbing to the condition.

The management of neonatal AKI in our cohort largely involved fluid resuscitation (60.7%), mechanical ventilation (56.5%), and the use of inotropes (53.6%), which is consistent with practices described in the study Pantoja-Gómez et al.[22] These interventions are crucial for maintaining hemodynamic stability and optimizing renal perfusion. The use of diuretics in 39.3% of the cases is reflective of the management strategies for oliguric AKI and fluid overload in neonates. However, this is a delicate balance as over-diuresis can exacerbate renal injury.[23]

The need for peritoneal dialysis in 14.3% of cases underscores the severity of AKI in this cohort, particularly in neonates with intrinsic renal injury who did not respond to conservative management. The utilization of renal replacement therapy in neonates with AKI is consistent with reports from Sethi et al., who found that peritoneal dialysis was required in 15% of their cohort with severe AKI.[24] The necessity for renal replacement therapy highlights the complexity of neonatal AKI, which often involves multiorgan dysfunction that is not easily corrected through standard medical management alone.[25]

The mortality rate in our study was 30.9%, which is comparable to the findings of Hingorani et al., who reported a mortality rate of 29% in a cohort of neonates with AKI.[26] In the intrinsic renal AKI group, we observed a much higher mortality rate (25%) compared to prerenal AKI (4.2%). This finding aligns with study by Bansal et al., who reported a similar trend of higher mortality in neonates with intrinsic renal injury.[27] The pathophysiology of intrinsic renal injury, characterized by acute tubular necrosis (ATN) and irreversible renal damage, is often associated with more severe and complicated outcomes, including a greater need for dialysis and prolonged recovery.[27]

The recovery rate of 64.2% in our cohort is promising and comparable to the 60% recovery rate reported by Katariya et al., in a cohort of neonates with AKI.[28] However, the rate of chronic kidney disease (CKD) progression in our study was concerning, with 4.8% of neonates showing evidence of CKD. This finding is in line with Herer et al., and Sethi et al., suggesting that neonates who survive AKI may develop long-term renal sequelae.[20,24] For instance, Laha et al., highlighted that AKI in neonates is a known risk factor for the development of CKD in childhood and adulthood, especially in those with intrinsic renal injury.[29]

Infection, particularly sepsis, was a significant contributing factor to both the development of AKI and poor outcomes. The high rate of infection (42.9%) observed in our study, along with a notable incidence of necrotizing enterocolitis (15.5%) and respiratory distress syndrome (38.1%), underscores the complexity of managing neonates with AKI. The presence of these comorbidities increases the risk of multi-organ failure and contributes to the higher mortality observed in neonates with intrinsic renal injury.[22,23] Study by Chishala et al., have shown that neonatal sepsis is a potent risk factor for the development of AKI, and the management of both conditions requires integrated, multidisciplinary care.[30]

Limitations

This study has several limitations, including its retrospective design, which may introduce selection bias in terms of the severity of cases included. Additionally, the lack of long-term follow-up data means that we are unable to assess the true burden of CKD progression beyond the neonatal period. Future prospective studies with long-term follow-up are needed to better understand the full spectrum of outcomes in neonates with AKI and to develop effective strategies for preventing CKD in survivors.

CONCLUSION

In conclusion, this study confirms that sepsis and perinatal asphyxia are the leading causes of AKI in neonates, and the mortality rate is significantly higher in neonates with intrinsic renal injury. While the recovery rate is promising, the risk of progression to CKD remains a concern. Our findings emphasize the importance of early identification and aggressive management of AKI in neonates, particularly in those with underlying risk factors such as sepsis and perinatal asphyxia. The use of peritoneal dialysis in severe cases highlights the need for renal replacement therapy in certain high-risk neonates. Future research should focus on improving early diagnostic tools, enhancing treatment protocols, and understanding the long-term renal outcomes of neonates who survive AKI.

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