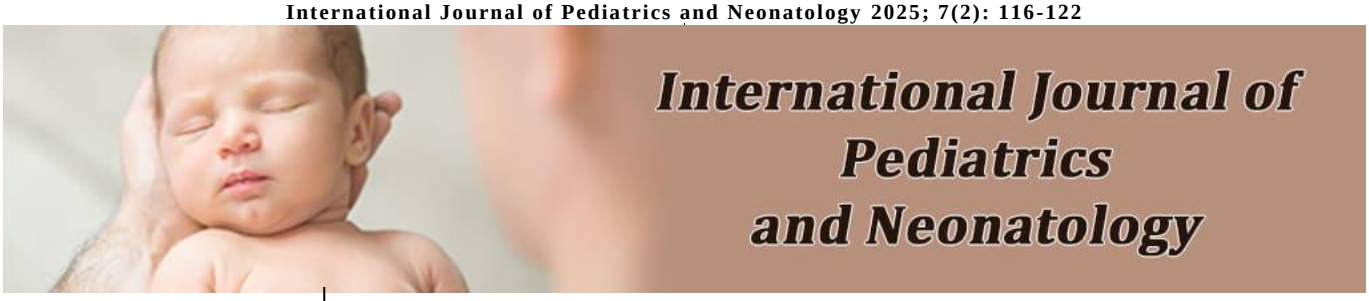

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A study of incidence of acute kidney injury in neonates admitted in neonatal ICU of tertiary health care hospital

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Abstract
Background: Acute Kidney Injury (AKI) in neonates is an increasingly recognized complication associated with significant morbidity and mortality. Early identification of risk factors and prompt management can improve neonatal outcomes. The present study was undertaken to find out incidence and related risk factors causing AKI in neonates admitted to the neonatal intensive care unit (NICU) of a tertiary health care hospital.
Methods: This prospective cross-sectional observational study was conducted in the Department of Pediatrics at a tertiary care center in Central India from January 2021 to December 2022. A total of 307 neonates admitted to the NICU were enrolled in the study. Serum creatinine and urine output were measured as per NICU protocol, and AKI was diagnosed using neonatal RIFLE (nRIFLE) criteria. Relevant clinical, demographic, and biochemical parameters were recorded and analyzed.
Results: The incidence of AKI was 10.42%. According to nRIFLE criteria, 53.12% of neonates were in the "Risk" group, 40.62% in the "Injury" group, and 6.25% in the "Failure" group. Significant risk factors associated with AKI were sepsis, shock, birth asphyxia, hypoxic ischemic encephalopathy (HIE), and maternal PIH. Neonates with AKI had a higher mean serum creatinine (1.31±0.15 mg/dL) and significantly higher mortality (43.75%) compared to those without AKI (16.36%).
Conclusion: The incidence of AKI among NICU-admitted neonates was substantial, with sepsis, shock, and asphyxia as key contributors. Early identification and management of high-risk neonates can reduce AKI-related morbidity and mortalitylevel.

Keywords: NICU, Maternal PIH, nRIFLE criteria, neonatal acute kidney injury, incidence, risk factors, sepsis, birth asphyxia, hypoxic ischemic encephalopathy, neonatal outcome

Introduction
Acute Kidney Injury (AKI) is defined as a sudden decline in kidney function within seven days, characterized by an increase in serum creatinine of ≥ 0.3 mg/dL, a ≥ 50% rise from baseline, or urine output < 1 mL/kg/hr over 24 hours [1, 2]. It results in the accumulation of nitrogenous wastes, leading to metabolic disturbances and potential multi-organ dysfunction. The burden of AKI is rising globally, particularly in developing countries, yet its true incidence remains uncertain. Studies across different populations show variable rates depending on age and clinical condition. In neonates, the reported incidence of AKI in NICUs ranges from 6% to 24%, [3] while in preterm infants it varies from 3.4% to 24% across countries [4]. Recent studies suggest that 3-8% of all NICU admissions are affected, highlighting the growing clinical importance of early recognition and management of AKI in this vulnerable population [5].
The most common type of AKI in neonates is prerenal failure, caused by renal hypoperfusion or ischemia. If not promptly corrected, it can progress to intrinsic renal damage. Neonatal kidneys are particularly prone to hypoperfusion due to physiological factors such as high renal vascular resistance, elevated plasma renin activity, low glomerular filtration rate, reduced cortical perfusion, and limited sodium reabsorption during the first few days of life. Consequently, newborns are vulnerable to acute tubular or cortical necrosis. The etiology of neonatal AKI is often multifactorial, with perinatal asphyxia and sepsis being the most common contributors.

Other associated risk factors include very low birth weight (< 1500 g), respiratory distress syndrome, dehydration, congestive heart failure, nephrotoxic drugs, mechanical ventilation at birth, and maternal use of NSAIDs or antibiotics [6, 7].

There are different guidelines for the diagnosis and classification of AKI, with RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease) and Kidney Disease Improving Global Outcomes (KDIGO) are among two of the most notable guidelines [8]. The pediatric-modified RIFLE (pRIFLE) criteria emphasize urine output and creatinine clearance. Prevention remains the best management approach, highlighting the importance of understanding AKI risk factors [9, 10].

However, the short-term outcome of neonatal AKI depends on its underlying cause, associated multi-organ involvement, and access to renal replacement therapy, with mortality higher in cases of multi-organ failure. Studies indicate that AKI affects 18-70% of critically ill neonates, leading to poor outcomes, prolonged hospital stays, and increased mortality [11]. The incidence and etiology may vary across postnatal periods due to different exposures during NICU stay and stages of renal development. More than 30% of hospitalized neonates develop AKI, with higher mortality rates reported. However, most available data come from developed countries, where healthcare systems and neonatal care differ significantly from developing nations. Hence, the present study was undertaken to determine the incidence and associated risk factors of AKI among neonates admitted to the NICU of a tertiary health care hospital.

Materials and Methods

After obtaining Institutional Ethical Committee (IEC) approval and written informed consents from parents/ guardian, this prospective cross-sectional observational study was conducted in the Department of Pediatrics at Tertiary Care Centre in central India during a period of 2 years from January 2021 to 31st December 2022. A total of 307 neonates admitted in NICU were included in the study. Neonates with congenital renal abnormalities, multiple congenital anomalies, chromosomal anomalies, antenatally diagnosed hydronephrosis, whose mothers with acute or chronic renal disease and whose parents were not willing to give consent were excluded from the study.

Serum Creatinine levels were measured in all clinically suspected cases of acute kidney injury or as a part of routine screening per unit protocol (those admitted before 72hr of life were screened at 72 hr. of life and those admitted after 72 hr. were screened at admission). Urine output measured in all patients admitted in NICU. Detailed demographic details including sex, gestational, age, mode of delivery, birth and admission weights, day of life at admissions and day of life at diagnosis of acute kidney injury were recorded. The neonates were classified as appropriate for GA(AGA), small for GA(SGA) and large for GA(LGA) in accordance with revised Fenton growth charts. (14) Sample collected from all the neonates admitted in NICU.

Blood sample of neonates collected in plain bulb and sent to biochemical laboratory for investigation of serum creatinine, BUN, serum sodium, potassium, calcium, complete blood count, C-reactive protein, arterial blood gases, urine sodium and urine creatinine. Daily Urine output monitored and on clinical judgment based on urine output and condition of neonates repeat sample of serum creatinine was sent. On the basis of serum creatinine value and urine output of that neonate diagnosis of acute kidney injury was made.

Diagnosis of acute kidney injury is made on the basis of following classification

Table 1: Kidney disease improving global outcomes

Stage	Serum Creatinine	Urine Output
0	No Change in Sr.Cr or less than 0.3mg/dl	≥ 1ml/kg/hr
1	SrCr rise ≥ 0.3mg/dl within 48hr or SrCr rise ≥ 1.5-1.9 × reference SrCr within 7 days	≥ 0.5ml/kg/hr and less than 1ml/kg/hr
2	SrCr rise ≥ 2-2.9 reference SrCr	≥ 0.3ml/kg/hr and less than 0.5ml/kg/hr
3	SrCr rise ≥ 3 × 3 reference SrCr or SrCr ≥ 2.5mg/dl or receipt dialysis	Less than 0.3ml/kg/hr

Table 2: nRIFLE

	Urine Output criteria
Risk	U/O < 1.5ml/kg/hr for 24 hr.
Injury	U/O <1ml/kg/hr for 24 hr.
Failure	U/O < 0.7ml/kg/hr for 24 hr. or anuric for 12 hr.
Loss of function	Persistent failure > 4 weeks
End stage	Persistent failure > 3 months

Statistical Analysis

The collected data were tabulated and statistically analyzed using a computer and presentation of data were made using the Microsoft Excel & SPSS software version 21. All quantitative data were expressed as mean±standard deviation. The chi square test was used for the comparison of the frequency of mortality between the groups. A p value < 0.05 was considered as being significant.

Incidence = Number of New Positive Cases / Number of Population Examined × 100.

Observations and Results

During the study period, 307 neonates were enrolled, of which 32 (10.42%) developed AKI. A male predominance was observed in both groups. The mean gestational age of neonates with AKI was 37.44±2.2 weeks, compared to 37.98±2.4 weeks in those without AKI. Among neonates with AKI, most had a birth weight between 1500-2499 g (34.37%), followed by ≥ 2500 g (31.25%). The mean birth weight was lower in the AKI group (1981±615 g) than in the non-AKI group (2241±580 g) (Table 1).

Table 3: Comparison of demographic characteristics between neonates with and without AKI

Characteristics		Neonates with AKI	Neonates without AKI	P-Value
Gender	Male	18 (56.25%)	140 (50.90%)	0.234
	Female	14 (43.75%)	135 (49.09%)	
Gestational age groups (weeks)	≤ 32	03 (9.37%)	15 (5.45%)	0.341
	33 to 34	05 (15.62%)	49 (17.81%)	
	35 to 36	10 (31.25%)	65 (23.63%)	
	≥ 37	14 (43.75%)	146 (53.09%)	
Birth weight (gm)	<1000	03 (9.37%)	10 (3.63%)	0.213
	1000-1499	08 (25.0%)	49 (17.81%)	
	1500-2499	11 (34.37%)	73 (26.54%)	
	≥2500	10 (31.25%)	143 (52.0%)	

In the AKI group, 50% of neonates were delivered by LSCS, 34.37% by normal vaginal delivery (NVD), and 15.62% by instrumental delivery. In contrast, among neonates without AKI, the majority (60.36%) were born by NVD, followed by 29.45% by LSCS and 10.18% by instrumental delivery (Figure 1).

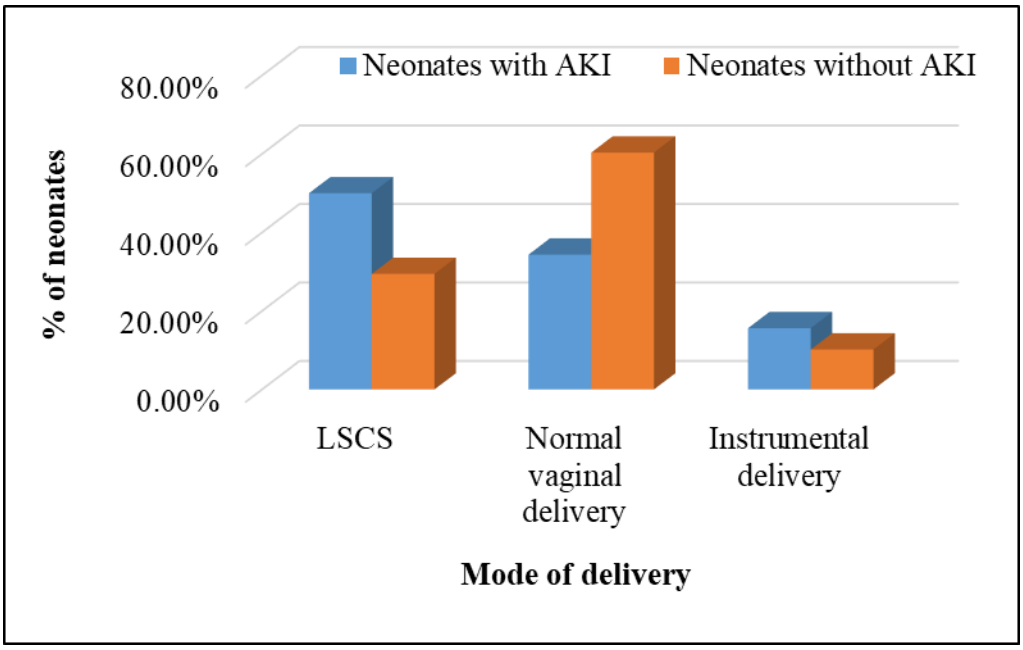

Fig 1: Comparison of mode of delivery between neonates with and without AKI

Most mothers in both groups were aged 21-30 years, followed by 31-40 years. Primipara mothers comprised 51.14%, while multipara mothers made up 48.85%. Both groups were comparable regarding parity (P=0.417). However, a statistically significant difference was observed between the groups concerning maternal PIH (P=0.024). Other maternal risk factors such as GDM, hypothyroidism, and PROM showed no significant difference (p>0.05) (Table 4).

Table 4: Comparison of obstetric characteristics and maternal risk factors between neonates with and without AKI

Parameters		Neonates with AKI	Neonates without AKI	P-Value
Mother age in years	18 to 20	03 (9.37%)	08 (2.90%)	0.214
	21 to 30	22 (68.75%)	192 (69.81%)	
	31 to 40	07 (21.87%)	75 (27.27%)	
Parity of mothers	Primipara	17 (53.12%)	140 (50.90%)	0.417
	Multipara	15 (46.87%)	135 (49.09%)	
Maternal risk factors	GDM	03 (9.37%)	15 (5.45%)	0.512
	PIH	05 (15.62%)	28 (10.18%)	0.024*
	Hypothyroidism	03 (9.37%)	11 (4.0%)	0.234
	PROM	02 (6.25%)	15 (5.45%)	1.000

There was no significant statistical difference between the studied groups as regarding clinical features of AKI except poor perfusion (P=0.033) and oligouria (P=0.018) which shows significant statistical difference as shown in Table 5.

Table 5: Comparison of clinical features between neonates with and without AKI

Clinical features	Neonates with AKI	Neonates without AKI	P-Value
Grunting	6 (18.75%)	36 (13.09%)	0.078
Poor perfusion	08 (25%)	29 (10.54%)	0.033*
Oligouria	14 (43.75%)	32 (11.63%)	0.018*
Fever	12 (4.36%)	38 (13.81%)	0.087
Vomiting	04 (12.5%)	26 (9.45%)	0.527
Tachypnoea	18 (56.25%)	152 (55.27%)	0.678
Chest Retractions	13 (40.62%)	136 (49.45%)	0.652
Poor feeding	6 (18.75%)	45 (16.36%)	0.234
Lethargy	05 (15.62%)	42 (15.27%)	1.00
Seizures	03 (9.37%)	36 (13.09%)	0.068
Abdominal distension	4 (12.5%)	32 (11.63%)	0.557

Among neonates with AKI, 68.75% required nasal oxygen, 31.25% were managed with CPAP, and 34.37% needed mechanical ventilation. In contrast, among neonates without AKI, 58.90% received nasal oxygen, 20.36% required CPAP, and 28.36% were on mechanical ventilation. Although the need for CPAP and ventilation was higher in the AKI group, the differences were not statistically significant (p>0.05). No significant difference was observed between the groups regarding oxygen delivery (Figure 2).

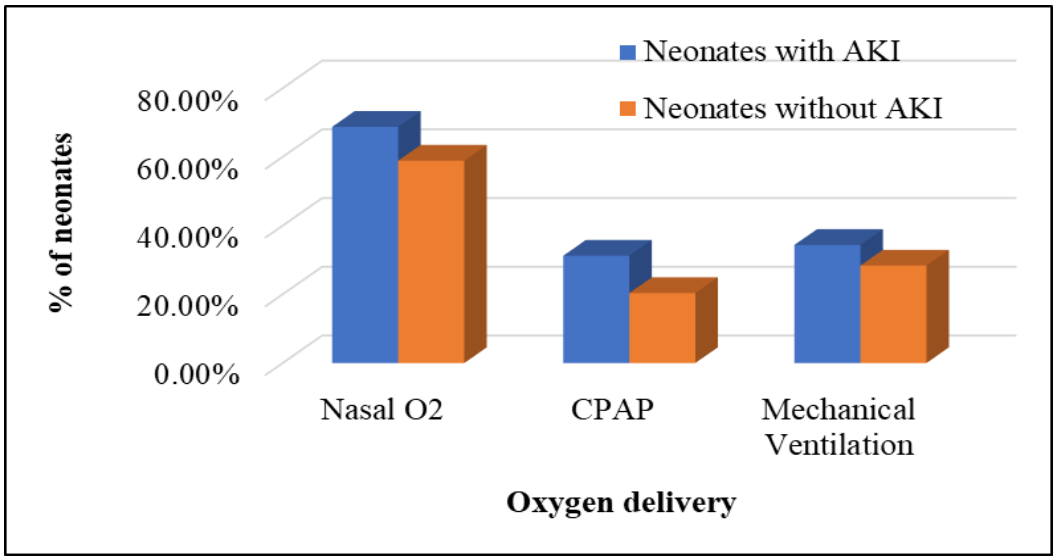

Fig 2: Comparison of oxygen delivery between neonates with and without AKI

In the AKI group, 68.75% of neonates had serum creatinine > 1.2 mg/dL, while 31.25% had levels between 1-1.2 mg/dL. All neonates without AKI had serum creatinine < 1.0 mg/dL. The mean serum creatinine was 1.31±0.15 mg/dL in the AKI group and 0.74±0.10 mg/dL in the non-AKI group. There was a highly significant difference in blood urea nitrogen (p<0.001), and significant differences in pH (P=0.030) and potassium (P=0.005) between the groups. None of the neonates with AKI required dialysis or continuous renal replacement therapy, (Table 6).

Table 6: Laboratory findings of the studied neonates

Parameters		Neonates with AKI	Neonates without AKI	P-Value
Serum creatinine level	<1.0	00 (0.0%)	275 (100.0%)	-
	>1 to 1.2	10 (31.25%)	00 (0.0%)	-
	>1.2	22 (68.75%)	00 (0.0%)	-
Laboratory findings	BUN (mg/dL)	38.2±18.3	23.7±14.2	< 0.001
	Na+ (mg/dL)	143.5±8.4	141.7±5.0	0.204
	K+ (mg/dL)	5.4±1.2	4.7±0.9	0.005

HCO_3: Bicarbonate, PO_2: Oxygen tension in blood, PCO_2: Carbon dioxide tension in blood, BE: Base excess, Na: Sodium, K: Potassium, BUN: Blood urea nitrogen

Out of 307 NICU admissions 32 neonates were admitted with AKI which accounts to 10.42%. Hence the incidence of AKI in neonates admitted NICU of tertiary health care hospital was 10.42%, (Figure 3). The majority of patients were classified under the Risk category, accounting for 17 cases (53.12%), with a mean urine output of 1.33±0.221 ml/kg/hour. The Injury category was observed in 13 cases (40.62%) with a lower mean urine output of 0.89±0.468 ml/kg/hour. The least number of cases fell under the Failure category, comprising 2 cases (6.25%), who had the lowest mean urine output of 0.41±0.315 ml/kg/hour. This indicates a progressive decline in urine output with increasing severity of AKI.

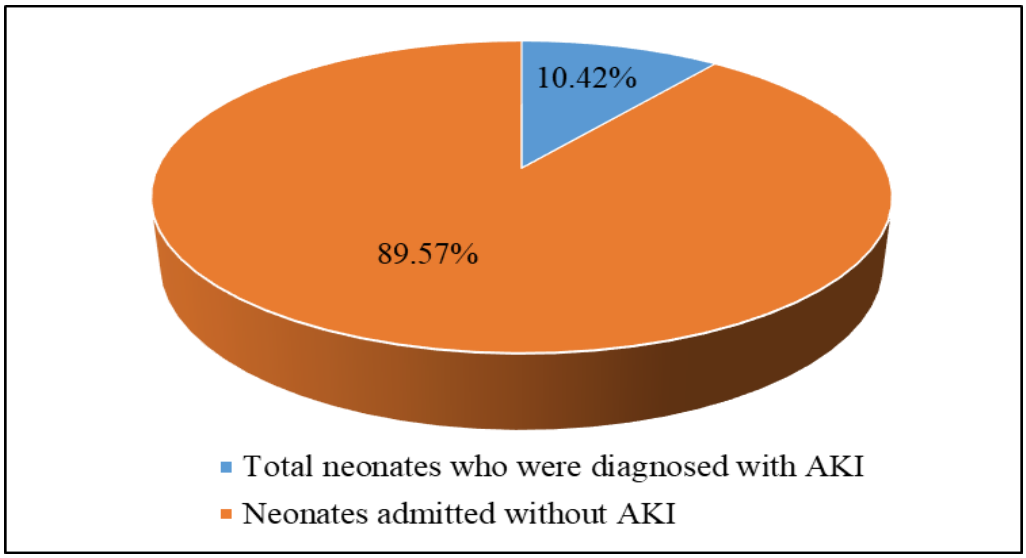

Fig 3: Incidence of acute kidney injury in neonates

The sepsis, shock and birth asphyxia were the most common risk factors causing AKI in neonates admitted in NICU. Table 16 shows that there was no significant statistical difference between the studied groups as regarding risk factors of AKI except sepsis, shock, birth asphyxia and HIE which shows significant statistical difference, (Figure 7).

Table 7: Related risk factors causing AKI in neonates admitted in NICU

Risk factor	Neonates with AKI	Neonates without AKI	P-Value
Sepsis	24 (75%)	72 (26.18%)	0.001*
Prematurity	18 (56.25%)	135 (49.09%)	0.356
Birth asphyxia	15 (46.87%)	39 (14.18%)	0.001*
RDS	06 (18.75%)	38 (13.81%)	0.544
Shock	17 (53.12%)	85 (30.90%)	0.001*
IUGR	2 (6.25%)	18 (6.54%)	1.00
HIE	04 (12.5%)	22 (8.0%)	0.042*
IVH	04 (12.5%)	16 (5.81%)	0.652
NEC	03 (%)	13 (16.36)	0.234

RDS: Respiratory distress syndrome, CHD: Congenital heart disease, HIE: Hypoxic-ischemic encephalopathy, IUGR: Intrauterine growth retardation. Necrotizing enterocolitis (NEC); IVH: intraventricular hemorrhage

The overall in-hospital mortality was 19.21% (59/307), with significantly higher mortality in neonates with AKI (43.75%) compared to those without AKI (16.36%, P=0.017). Among the AKI survivors, 83.33% recovered renal function within 14 days, and 16.66% within 28 days. Most neonates with AKI (68.75%) had a hospital stay ≥ 14 days, whereas the majority without AKI (69.81%) stayed < 14 days. This difference was statistically significant (P=0.016), indicating that AKI was associated with prolonged hospitalization, (Figure 4).

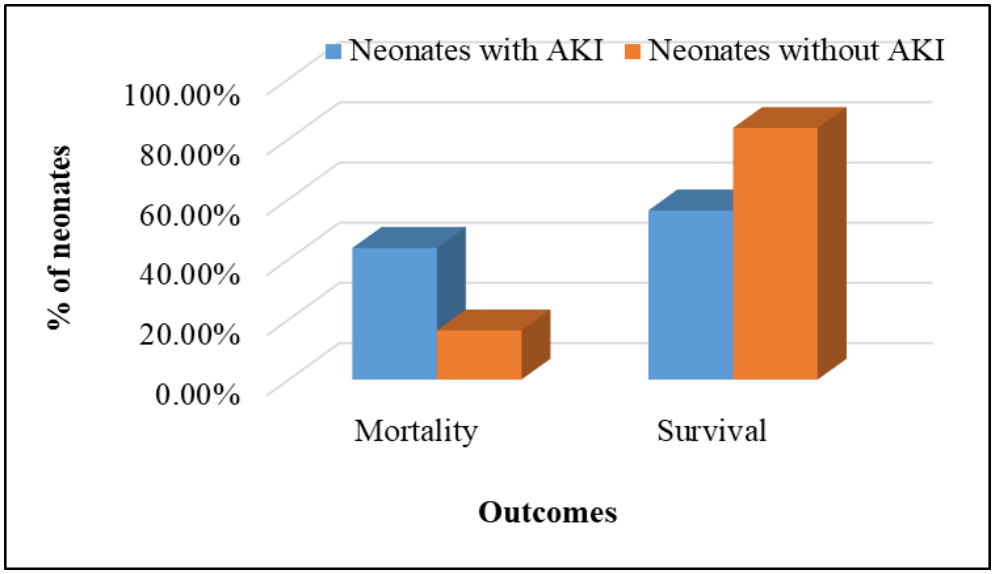

Fig 4: Outcome of neonates

Discussion

Neonatal kidney diseases present in various forms, with AKI being the most significant, ranging from mild dysfunction to complete anuric kidney failure. Several studies report that the incidence of AKI in NICUs ranges from 6-24% [12]. In the present study, male predominance was observed among AKI cases (male-to-female ratio 1.2:1). The mean gestational age was similar between AKI (37.44±2.2 weeks) and non-AKI neonates (37.98±2.4 weeks). Most AKI neonates had a birth weight of 1500-2499 g (34.37%), followed by ≥ 2500 g (31.25%), whereas non-AKI neonates were mostly ≥ 2500 g (52%). The mean birth weight was lower in the AKI group (1981.9±615.9 g vs. 2241.8±580.2 g). Regarding mode of delivery, 50% of AKI neonates were delivered by LSCS, 34.37% by NVD, and 15.62% by instrumental delivery, while most non-AKI neonates were born via NVD (60.36%). The higher LSCS rate in the AKI group may reflect fetal distress as a prenatal risk factor for AKI. These findings are in accordance with the study conducted by Nandhagopal N *et al.* [13] and Katariya KL *et al.* [14] and Ali *et al.* [15].

Most mothers in both groups were aged 21-30 years, followed by 31-40 years, with the mean maternal age was 26.96±4.51 years in the AKI group and 27.26±4.18 years in the non-AKI group, similar to Ghobrial EE *et al.* [10]. Primipara mothers comprised 51.14%, and multipara mothers 48.85%, with no significant difference between groups (P=0.417). Maternal PIH showed a statistically significant association with neonatal AKI (P=0.024), while other maternal risk factors, including GDM, hypothyroidism, and PROM, were comparable between groups (p>0.05), consistent with the study done by Ghobrial EE *et al.* [10].

There was no significant difference between groups regarding most clinical features of AKI, except for poor perfusion (P=0.033) and oliguria (P=0.018), which were significantly associated with AKI, often related to sepsis. In the AKI group, 68.75% of neonates had serum creatinine > 1.2 mg/dL, and 31.25% had levels between 1-1.2 mg/dL, while all neonates without AKI had creatinine < 1.0 mg/dL. The mean serum creatinine was 1.31±0.15 mg/dL in AKI neonates versus 0.74±0.10 mg/dL in non-AKI neonates (P=0.038). There was a highly significant difference in blood urea nitrogen (p<0.001), and significant differences in pH (P=0.030) and potassium (P=0.005) between groups. None of the AKI patients required dialysis or renal replacement therapy. These findings are consistent with previous studies conducted by Ghobrial EE *et al.* [10], Ali *et al.* [15] and Mortazavi *et al.* [16].

In current study, 68.75% of neonates with AKI required nasal oxygen, compared to 58.9% of neonates without AKI. CPAP was needed in 31.25% of AKI cases versus 20.36% in non-AKI, while mechanical ventilation was required in 34.37% of AKI neonates and 28.36% of non-AKI neonates. The use of CPAP and ventilation was largely related to coexisting conditions such as prematurity, birth asphyxia, HMD, and sepsis, and was comparable between both groups. Few studies have used urine output to define or classify AKI as per nRIFLE criteria. In current study, the incidence of AKI among NICU neonates was 10.42%, comparable to Youssef D *et al.* (10.8%) [17] and Ashraf *et al.* (8.33%) [18]. Among AKI cases, 53.12% were in the Risk (R) category, 40.62% in Injury (I), and 6.25% in Failure (F). Urine output decreased with increasing nRIFLE severity: 1.33±0.22 ml/kg/hr in Risk, 0.89±0.47 ml/kg/hr in Injury, and 0.41±0.32 ml/kg/hr in Failure (P=0.0423). These findings are comparable with the study conducted by Ghobrial EE *et al.* [10], Katariya KL *et al.* [14] and Mitharwal, *et al* [19].

The most common risk factors for AKI were comparable between groups, except sepsis, shock, birth asphyxia, and HIE, which were significantly associated with AKI. Sepsis was the most common risk factor, affecting 75% of AKI cases, likely due to hypotension, renal microvascular damage, shock, DIC, hemorrhage, and cardiac failure as similar to Mathur NB *et al.* [6], and Nillsen *et al.* [20] Contributing factors in developing countries, such as overcrowding and low nurse-to-patient ratios, may increase sepsis rates, highlighting the importance of strict infection control measures. Birth asphyxia was observed in 46.87% of AKI neonates, comparable to study done by Kaur *et al* (41.7%) [21]. Overall, sepsis, shock, and birth asphyxia were the predominant risk factors for neonatal AKI, consistent with prior research identifying low birth weight and shock as significant contributors [13, 14].

In the present study, the overall in-hospital mortality was 19.21%, with significantly higher mortality in neonates with AKI (43.75%) compared to those without AKI (16.36%, P=0.017), consistent with previous studies reporting mortality rates of 24-44.4% among AKI neonates.[16-18,22] Additionally, 68.75% of neonates with AKI had a hospital stay > 14 days, compared to 30.18% without AKI (P=0.016), aligning with findings from Ghobrial EE *et al.* [10], Nandhagopal N *et al.* [13] and Mitharwal *et al* [19]. The prolonged hospitalization in this study may be attributed to the tertiary referral nature of the center, with complex cases, late presentations, and a high incidence of sepsis contributing to longer stays.

The major limitations of current study include the lack of follow-up after discharge, preventing assessment of the long-term effects of AKI on growth, blood pressure, or renal function in the neonates. Additionally, as the study was conducted in a referral tertiary NICU that admits critically ill patients from the region, the findings may not be generalizable to other NICU levels, such as Level I units, where clinical practices and patient profiles may differ. Consequently, the results cannot be considered indicative of long-term outcomes.

Conclusion

Neonatal AKI is an increasing clinical burden, with an incidence of 10.42% in our NICU. The most common risk factors were sepsis, shock, birth asphyxia, and HIE, while maternal PIH was a notable maternal contributor. According to nRIFLE criteria, most affected neonates were in the risk category. Lower birth weight was associated with higher AKI incidence and mortality. AKI significantly prolongs hospital stay and increases mortality risk. Early recognition of risk factors and timely identification of AKI are crucial steps in prevention and improved neonatal outcomes.

Recommendations

High-risk neonates should be identified early, with regular monitoring of renal function and urine output to guide therapy. Prompt management of risk factors can reduce complications. Ensuring adequate antenatal care, managing maternal illnesses, and maintaining strict infection control are essential. Neonates with AKI require urgent care,

follow-up, and monitoring for nutrition, blood pressure, and renal function to prevent future chronic kidney disease. Large-scale studies are needed to refine AKI definitions, understand risk factors, and improve outcomes in neonatal renal disorders.

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