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Incidence, Risk Factors, and Outcome of AKI in Preterm Neonates Admitted to the NICU

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ABSTRACT

Objective: To determine the incidence, potential risk factors, and outcomes of acute kidney injury (AKI) among preterm neonates.

Methodology: This prospective cohort study was conducted in the neonatal intensive care units (NICUs) of Fatima Memorial Hospital, Lahore, from April 2024 to October 2024. Preterm neonates born between 28 and 36 weeks of gestation were eligible for inclusion. A total of 432 preterm neonates admitted to the NICU of this tertiary care hospital during the study period were enrolled. Neonates with major congenital anomalies or chromosomal abnormalities were excluded. AKI was diagnosed using the modified Kidney Disease: Improving Global Outcomes (KDIGO) criteria. The primary outcomes assessed were in-hospital mortality and duration of hospitalization.

Results: AKI was diagnosed in 178 preterm neonates (41.2%). Maternal anemia emerged as the most significant risk factor, increasing the risk of neonatal AKI by 5.8 times (487%), followed by hemodynamically significant patent ductus arteriosus (hsPDA) (2.89 times; 189%) and mechanical ventilation (1.7 times; 76.8%). In contrast, exposure to antenatal steroids was nephroprotective, reducing the risk of AKI by 63.6%. The mortality rate among neonates with AKI was 16.3% ($p < 0.001$), with stage 3 AKI demonstrating the highest mortality rate (30%). Kaplan–Meier survival analysis demonstrated that neonates with stage 3 AKI had a statistically significant decrease in survival (mean survival time 16.51 days).

Conclusion: Among 432 enrolled preterm neonates 178 (41.2%) were reported to have AKI. Key risk factors included maternal anemia, hsPDA, and need for mechanical ventilation, while antenatal steroid exposure was noted to had a protective effect. Preterm neonates with stage 3 AKI experienced hospital stays that were twice as long and demonstrated a 50% reduction in mean survival ($p < 0.001$).

Keywords: Acute kidney injury, hsPDA, Mechanical ventilation, Risk Factors.

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Introduction

AKI or acute kidney injury is an acute deterioration of renal functions. It results in uremia, altered fluid balance, along disturbed electrolyte homeostasis.[1] By approximately 34 weeks of gestation, nephrogenesis is typically complete.[2]

Preterm neonates are particularly susceptible to AKI due to incomplete nephrogenesis and the immaturity of renal regulatory mechanisms. Premature neonates have underdeveloped glomerular and tubular structures, leading to impaired glomerular filtration, sodium handling, and urinary concentration capacity. [2,3]

The immature neonatal kidney exhibits limited autoregulatory capacity, reduced renal blood flow, and a

low baseline glomerular filtration rate (GFR), rendering it highly vulnerable to ischemic and toxic insults. In preterm neonates, the renal cortex is more perfused than the medulla, predisposing the deeper nephron segments to hypoxic injury. Additionally, hormonal and hemodynamic control systems—such as the renin–angiotensin–aldosterone system and tubuloglomerular feedback—are not fully developed, further compromising renal resilience [3,4]

Several maternal and neonatal factors have been identified that increase the susceptibility of preterm infants to renal injury. These factors act during the antenatal, perinatal, and postnatal periods by disrupting normal nephrogenesis.[3,4] Maternal risk factors for neonatal AKI include antenatal steroid exposure, preterm prelabor rupture of membranes (PPROM), pregnancy-induced hypertension (PIH), antepartum hemorrhage (APH), and the use of nephrotoxic drugs like nonsteroidal anti-inflammatory drugs. Neonatal risk factors encompass perinatal stress related events, congenital anomalies of the kidney and urinary tract (CAKUT), use of positive inotropic agents, and shock. Additional contributors include hemodynamically significant patent ductus arteriosus (hsPDA), sepsis, intraventricular hemorrhage (IVH), disseminated intravascular coagulation (DIC), nephrotoxic medication exposure (e.g., aminoglycosides, vancomycin, and inotropes), necrotizing enterocolitis (NEC), and the placement of an umbilical venous catheter (UVC).[4-6]

Up to 50% of ELBW neonates develop AKI during NICU stay, contributing to higher morbidity and mortality.[1] Shalaby et al. reported an incidence of 56% in a prospective cohort study from Saudi Arabia [5]. Gul et al. observed AKI in 37.6% of preterm neonates in a tertiary NICU in Pakistan.[6]

However, there is a paucity of comprehensive data and universally accepted diagnostic criteria on AKI in preterm neonates are still lacking.

To address this gap, the KDIGO (Kidney Disease: Improving Global Outcomes) neonatal-modified criteria have been developed [7]. It aims to facilitate uniform diagnosis and staging of AKI in neonates. These criteria utilize serum creatinine (S. Cr) trends and urine output (UOP) measurements to identify and classify AKI into three stages of severity.[7]

- Stage 1: Increase in S. Cr ≥0.3 mg/dL within 48 hours or a rise to 1.5–1.9 times baseline, or UOP <0.5 mL/kg/hour for 6–12 hours.
- Stage 2: A S. Cr increase of 2.0–2.9 times baseline or UOP <0.5 mL/kg/hour for ≥12 hours.
- Stage 3: A ≥3.0-fold rise in S. Cr, a level ≥2.5 mg/dL, UOP <0.3 mL/kg/hour for ≥24 hours, or anuria for ≥12 hours.

National as well as international literature regarding AKI profile especially preterm neonates while using KDIGO guidelines is infrequent.[6] To bridge this gap, future research should utilize KDIGO guidelines in the diagnosis and staging of AKI in neonates accordingly. Our study while utilizing KDIGO guidelines aims to characterize the incidence, risk factors, staging, and outcomes of AKI among preterm neonates. This will contribute valuable insights into understanding of AKI in neonates, especially in ICU settings.

Methodology

A prospective cohort study was planned and conducted, with written informed consent obtained from parents or guardians before their neonates' participation. Ethical approval was granted by the Institutional Review Board IRB # FMH-06/04/2022-IRB-1044. The study included only preterm neonates born between 28 to 36+6 weeks of gestation who were admitted to the tertiary care neonatal ICU during the study period.

All preterm neonates with severe, life-incompatible congenital anomalies or disorders (e.g., anencephaly, bilateral renal agenesis, or complex cyanotic heart disease with no surgical option), as well as those who did not survive beyond 72 hours after birth, and cases where parental consent was declined were excluded from study. In our hospital, serum creatinine levels were measured using the C311 Rosch machine through cytometric analysis.

The sample size calculation was carried out using the OpenEpi online calculator, available at https://www.openepi.com/. A total sample size of 432 was determined, based on a 95% confidence level, a 10% margin of error, and a 5% significance level, with an estimated 56% prevalence of AKI among preterm neonates.[5]

The primary outcome variables included the incidence and severity of acute kidney injury (AKI), all-cause in-hospital mortality, and the duration of NICU stay, which was calculated from the time of admission until either discharge or death.

For all preterm neonates, reference point serum creatinine levels were the value that was measured within 24 hours

of study enrolment. Subsequent serum creatinine measurements were taken every 24 to 48 hours, as needed.

Acute Kidney Injury (AKI) was diagnosed and staged based on modified KDIGO (Kidney Disease: Improving Global Outcomes) criteria for neonates, using serum creatinine values. In our study, we have used only serum creatinine level for diagnosing and staging AKI. Stage 1 is defined by an increase in S. Cr ≥0.3 mg/dL within 48 hours or a rise to 1.5–1.9 times baseline. Stage 2 includes a S. Cr increase of 2.0–2.9 times baseline, while Stage 3 is defined by a ≥3.0-fold rise in S. Cr, a level ≥2.5 mg/dL.[7]

AKI was staged using modified KDIGO criteria based on relative increases in serum creatinine from baseline. It is noted that absolute serum creatinine values may vary due to individual baseline levels. Therefore, staging was based on the rise of S. Cr from baseline. Serum creatinine levels for AKI stages were presented as mean with interquartile ranges (IQRs). Maternal data including information on their parity, antepartum hemorrhage (APH), diabetes mellitus, hypertension, anemia, mode of delivery, and use of antenatal corticosteroids (ACS) during pregnancy.

Neonatal data includes gestation age, weight, length, and gender, mechanical ventilation, umbilical catheterization, medication use, hsPDA, IVH, DIC, NEC (modified Bell's stage II or III). Neonatal sepsis was diagnosed based on leukocytosis > 30 x 103, leukopenia < 5 x 103, or ANC <1500, elevated CRP, platelets <100, and/or positive blood cultures. For all preterm neonates' outcome was recorded as length of stay and either discharge home or death.

The Statistical Package for the Social Sciences (SPSS 20.0) was used for data analysis. Descriptive statistics and tests of significance were applied to all variables. The Pearson chi-square test or Fisher's exact test were used to assess proportional differences for categorical variables. Column proportions were compared using the z-test, with p-values adjusted using the Bonferroni correction method. The Shapiro-Wilk test was used to assess the normality of distribution for continuous variables. As all continuous variables were not normally distributed, they were expressed as medians with interquartile ranges (IQR) and compared using the Kruskal-Wallis test. A p-value of <0.05 was considered statistically significant.

Neonates were compared based on maternal and neonatal characteristics, with AKI used as the reference. Factors associated with AKI, with a p-value < 0.05 in the univariate analysis, were further evaluated using a logistic regression model to calculate the odds ratio (OR) with 95% confidence intervals (CI). Kaplan-Meier survival analysis was conducted for the entire cohort to explore the association between AKI severity and time-to-event outcomes for survival, with a significance level set at p<0.05.

Results

During the study period of 6 months (April to October 2024), total 470 preterm neonates were admitted to our tertiary care neonatal ICU. Of these, 38 were excluded due to parental refusal, incomplete data, or death within 72 hours of admission.

Consequently, 432 neonates (91.9%) had complete data and were finally analyzed. Out of total 432 preterm neonates, 178 (41.2%) were diagnosed with acute kidney injury (AKI) any stage. The distribution of AKI stages among these neonates was as 52 (12%), 106 (24.5%), and 20 (4.6%) stage 1, 2 and 3 respectively.

The stratification of our cohort based on gestation age revealed that all groups were comparable (p = 0.819). Specifically, 129 neonates (29.9%) had a gestational age of 28–32+6 weeks, 123 (28.5%) were 33–34+6 weeks, and 180 (41.7%) were 35–36+6 weeks. Table 1 presents the relationship between AKI severity, birth weight, and gestational age.

The univariate analysis revealed several neonatal risk factors for AKI that were statistically significant, including male gender, hemodynamically significant patent ductus arteriosus (hsPDA), necrotizing enterocolitis (NEC), use of inotropic agents, presence of central lines, intraventricular hemorrhage/intracranial hemorrhage/disseminated intravascular coagulation (IVH/ICH/DIC), exposure to nephrotoxic medications, and mechanical ventilation, all with p < 0.05 (Table I). Similarly, maternal risk factors associated with neonatal AKI were primigravida status, administration of antenatal steroids, maternal anemia, maternal diabetes mellitus, and mode of delivery via lower segment cesarean section (LSCS) (Table I).

Table I: Neonatal and maternal characteristics with and without AKI.

Characteristics		AKI (defined by KDIGO guidelines)				Univariate analysis p-value
		No AKI 254, n (%)	Stage 1 AKI 52, n (%)	Stage 2 AKI 106, n (%)	Stage 3 AKI 20, n (%)	
Neonatal						
Weight* (kg)	Median & IQR	1.7 (1.1 – 2.4)	1.6(0.98–2.2)	1.9 (1.1 – 2.4)	1.5(0.86-2.2)	<0.120[a]
	<1	12 (37.5%)	8 (25.0%)	5 (15.6%)	7 (21.9%)	
	1.1 –1.5	90 (58.1%)	23 (14.8%)	37 (23.9%)	5 (3.2%)	
	1.6 –2.5	126(61.2%)	17 (8.3%)	59 (28.6%)	4 (1.9%)	
	>2.5	26 (66.7%)	4 (10.3%)	5 (12.8%)	4 (10.3%)	
Gestation age*(weeks)	Median & IQR	34 (32 – 35)	34 (33 – 35)	33 (32 – 36)	35 (28 – 35)	0.819[a]
	28 – 32	76 (58.9%)	11 (8.5%)	33 (25.6%)	9 (7.0%)	
	33 – 34	63 (51.2%)	21 (17.1%)	39 (31.7%)	0 (0.0%)	
	35 – 36	115(63.9%)	20 (11.1%)	34 (18.9%)	11(6.1%)	
Length*(cm)		42 (38 – 44)	40 (37 – 43)	42 (39 – 44)	40 (34 – 43)	<0.002[a]
Gender (male)		151 (59.4)	34 (65.4)	59 (55.7)	20 (100)	0.002[b]
Birth asphyxia		11 (4.3)	0 (0.0)	9 (8.5)	2 (10.0)	0.085[b]
Serum creatinine (mg/dl)		0.89 (0.76-0.96)	1.2 (0.85 –1.4)	1.1 (0.95 – 1.6)	2.15 (2-2.3)	<0.01[a]
GFR		100 (45.4-136)	71.2 (38.6-94)	62.6 (36.7 –75.0)	16.3 (14.7-28)	<0.01[a]
Mechanical ventilation		137 (53.9)	36 (69.2)	89 (84.0)	10 (50.0)	<0.001[b]
Central lines		40 (15.7)	19 (36.5)	26 (24.5)	2 (10.0)	0.003[b]
Nephrotoxic drugs		222 (87.4)	33 (63.5)	100 (94.3)	14 (70.0)	<0.001[b]
Inotropic agents		37 (14.6)	17 (32.7)	19 (17.9)	4 (20)	0.021[b]
Sepsis		106 (41.7)	24 (46.2)	53 (50.0)	11 (55)	0.391 [b]
NEC		19 (7.5)	11 (21.2)	19 (17.9)	5 (25.0)	0.002[b]
IVH/ICH/DIC		32 (12.6)	19 (36.5)	6 (5.7)	6 (30.0)	<0.001[b]
hsPDA		7 (2.8)	16 (30.8)	26 (24.5)	8 (40.0)	<0.001[b]
Duration of stay (days)		6 (5 – 9)	11 (7 – 19)	9.5 (7 – 15)	14 (7 – 18)	<0.01[a]
Mortality		5 (2.0)	8 (15.4)	15 (14.2)	6 (30)	<0.001[b]
Maternal						
Primigravida		147 (57.9)	17 (32.7)	66 (62.3)	10 (55.6)	0.003[b]
Antenatal steroids		49 (19.3)	4 (7.7)	11 (10.4)	0 (0.0)	0.01[b]
Hypertension		72 (28.3)	14 (26.9)	31 (29.2)	7 (35.0)	0.919[b]
Diabetes Mellitus		11 (4.3)	7 (13.5)	8 (7.5)	0 (0.0)	0.05[b]
Maternal anemia		234 (92.1)	51 (98.1)	105 (99.1)	20 (100)	0.019[b]
Antepartum hemorrhage		219 (86.2)	45 (86.5)	95 (89.6)	18 (90.0)	0.816[b]
LSCS		215(84.6%)	41 (78.8%)	97 (91.5%)	20 (100%)	0.034 [b]

* Median and inter quartile range,[a] Kruskal-Wallis test, [b] Chi Square test

Table II summarizes all independent risk factors for neonatal AKI. Among these, maternal anemia emerged as the most significant risk factor, increasing the likelihood of AKI by 5.8 times (487%). This was followed by hsPDA, which increased the risk by 2.89 times (189%), and mechanical ventilation, which raised the risk by 1.7 times (76.8%). Conversely, antenatal steroids demonstrated a protective effect, reducing the risk of AKI by 63.6%.

After adjusting all significant factors like primigravida, NEC, mechanical ventilation, IVH/ICH/DIC, central lines, nephrotoxic drugs, hsPDA, and inotropes in a multivariable logistic regression model the most statistically significant risk factor associated with mortality was renal failure (AOR 8.298, 95%CI 3.015 – 22.836, $p < 0.001$). All clinically relevant maternal and neonatal variables—including antepartum hemorrhage, diabetes mellitus, hypertension, birth asphyxia, and sepsis—were included in the univariate analysis. Only variables with $p < 0.05$ were retained in the final multivariate logistic regression model to identify independent predictors of AKI. Variables not reaching statistical significance or demonstrating multicollinearity were excluded from the final model.

Mortality rates were higher among neonates with AKI compared to those in the non-AKI group. The prevalence of AKI stages and mortality (%) by gestation age has been summarized in Figure 1.

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Table II: Independent risk factors for AKI.

Independent Risk Factors	AOR	p value
Antenatal steroids	0.364 (0.182 - 0.725)	0.004
Maternal anemia	5.872 (1.228 – 28.086)	0.027
Mechanical ventilation	1.768 (1.076 – 2.905)	0.025
HsPDA	2.890 (1.537 – 5.435)	0.001
Primigravida	0.642 (0.411 – 1.001)	0.051

Among the 178 preterm neonates diagnosed with AKI, 16.3% succumbed to the condition ($p < 0.001$).

Mortality rates based on AKI severity were as 15.4% in stage 1, 14.2% in stage 2, and 30% in stage 3. In addition, neonates with AKI had prolonged hospital stays compared to those without AKI (Table I).

Kaplan-Meier survival analysis assesse the survival time of preterm neonates in the cohort (Figure 2). The estimated mean duration from admission to death was 32.02 days with normal renal function, whereas it was significantly shorter at 16.51 days for those with stage 3 AKI. Log-rank tests for pairwise comparisons confirmed that this difference was statistically significant ($p < 0.01$).

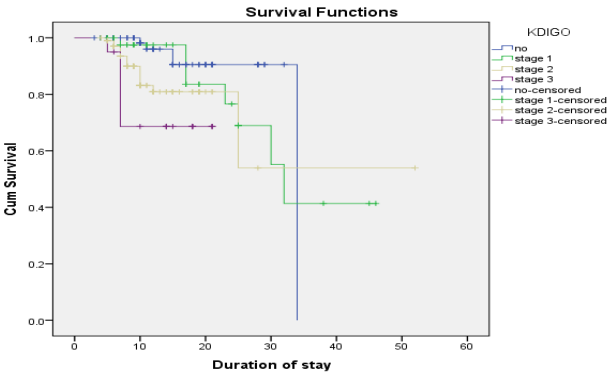
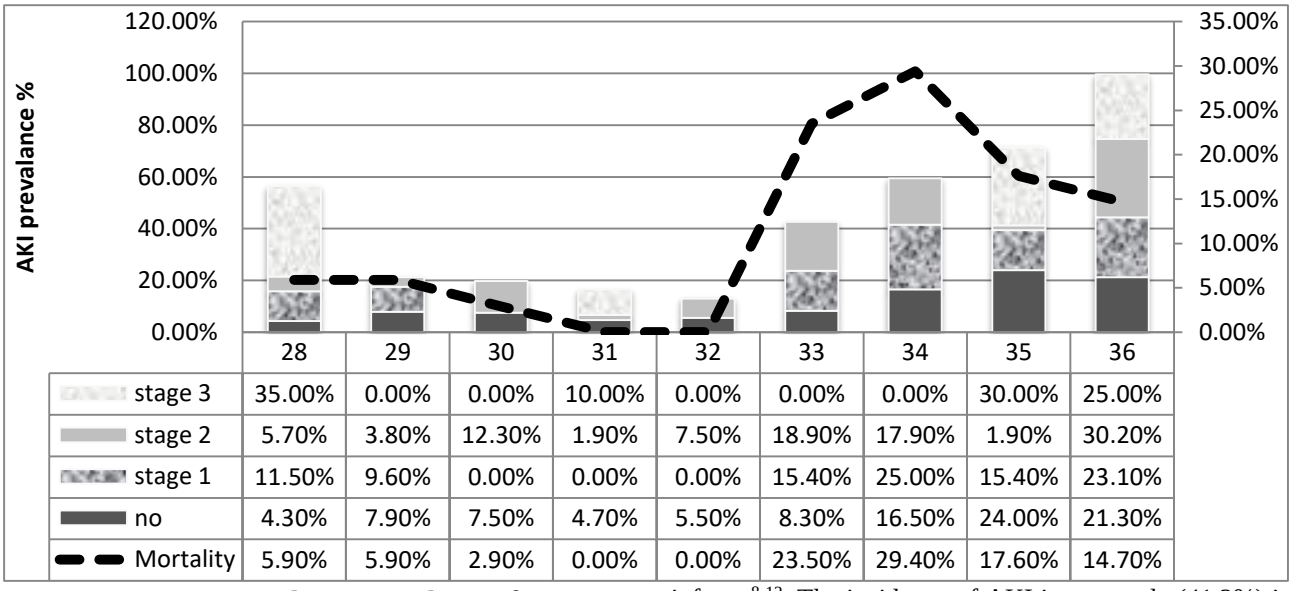


Figure 2: Kaplan-Meier curve (Survival curves).

The AKI incidence ranges from 11.6% to 63.3% and this wide range influenced by population characteristics and diagnostic criteria.

Elmas et al. reported an incidence of 37.6% in premature infants, while Carmody et al. observed AKI in approximately 48% of very low birth weight neonates. Similarly, the multinational AWAKEN study reported AKI rates as high as 63.3% in extremely low birth weight



	28	29	30	31	32	33	34	35	36
stage 3	35.00%	0.00%	0.00%	10.00%	0.00%	0.00%	0.00%	30.00%	25.00%
stage 2	5.70%	3.80%	12.30%	1.90%	7.50%	18.90%	17.90%	1.90%	30.20%
stage 1	11.50%	9.60%	0.00%	0.00%	0.00%	15.40%	25.00%	15.40%	23.10%
no	4.30%	7.90%	7.50%	4.70%	5.50%	8.30%	16.50%	24.00%	21.30%
Mortality	5.90%	5.90%	2.90%	0.00%	0.00%	23.50%	29.40%	17.60%	14.70%

Figure 1: Gestation age bases prevalence of AKI, its stages, and mortality. (%)

Discussion

Premature neonates are highly vulnerable to acute kidney injury (AKI), yet regional data remain limited. Our study evaluates the incidence, risk factors, and outcomes of AKI in preterm neonates using KDIGO criteria, with the aim of improving early detection, management, and outcomes in the NICU.

infants [8-12]. The incidence of AKI in our study (41.2%) is consistent with previously published data, particularly among preterm and very low birth weight neonates. These findings collectively support the reliability and external consistency of our results. Preterm neonates are at increased risk of acute kidney injury (AKI) due to renal immaturity. Up to two-thirds of nephrons develop during the third trimester, making gestational age an independent risk factor for AKI.[13] According to Brenner's hypothesis, preterm birth disrupts normal kidney development, resulting in reduced nephron endowment.[14] Postnatal

nephrogenesis is further impaired by additional risk factors, with prematurity being a primary contributor.[14-16]

Maternal anemia during prenatal period has been allied to adverse outcomes, including prematurity and related complications.[11,14,16] However, the association between maternal anemia and AKI in preterm neonates remains underreported. In our cohort, maternal anemia increased the risk of AKI by 5.8 times. One possible explanation is that fetal nephron development is closely tied to maternal nutritional and iron homeostasis[14]. Maternal malnutrition and anemia during pregnancy can influence the fetal epigenome through DNA methylation, thereby affecting fetal programming.[19,20-22]

Guillet et al. reported that among 30% of neonates with a PDA also developed AKI, a statistically significant finding (OR 3.74, 95% CI 2.17–6.44, $p < 0.0001$).[23] Similarly, Alaro demonstrated a strong association between AKI and PDA (AOR 4.3, 95% CI 2.25–8.07, $p < 0.001$)[9]. Our data indicates that hemodynamically significant PDA (hsPDA) increases the risk of renal insult in preterm neonates by 187% compared to those with no PDA. In the context of hsPDA, preterm neonates often experience ineffective intravascular volume, reduced renal blood flow, and heightened renal insult.[14] Preterm neonates typically exhibit delayed or absent natural PDA closure compared to term neonates. HsPDA leads to pulmonary over-circulation and systemic under-circulation, particularly affecting renal perfusion, which exacerbates renal injury and increases AKI risk.[24,25]

Mechanical ventilation is another significant risk factor for AKI in critically ill preterm neonates, with high-frequency ventilation support particularly increasing the risk ($p < 0.001$, AOR 3.4, 95% CI 1.78–6.67).[9] Similarly, Fan et al. found that the degree of prematurity and the usage of invasive mechanical ventilation were autonomous risk factors for AKI (p 0.035, OR 4.790, 95% CI 1.115–20.575).[17] Our study found that mechanically ventilated neonates had a 176% higher odds of developing AKI, consistent with findings from other studies.

Selewski et al. reported a 30% AKI incidence in NICU neonates, with mechanical ventilation as a key risk factor.[1] Gul et al. found a 37.6% AKI rate in a Pakistani cohort, similarly linked to invasive ventilation.[6] Elmas et al. observed a 37.6% incidence in preterm infants, many requiring ventilatory support.[10] Gallo et al. reported a 33.3% AKI incidence, particularly in ventilated neonates.[26]

Mechanical ventilation may initiate or exacerbate AKI through multiple mechanisms, including increased intrathoracic pressure, permissive hypercapnia, and high-pressure ventilation, all of which reduce renal perfusion. Additionally, endotracheal tube-induced local biotrauma and the release of systemic inflammatory mediators further contribute to renal injury.[27]

Antenatal corticosteroids reduced AKI risk by 63.6% in our cohort, supporting findings by Ustun et al. (AKI incidence 35.6%), Jetton and Askenazi , and Charlton et al. (30.6%), all of whom reported a protective effect.[11,16,28] Our cohort supports this finding, with antenatal corticosteroids reducing the risk of AKI in preterm neonates by 63.6%. Antenatal glucocorticoid therapy has been shown to increase mean arterial pressure, renal blood flow, and glomerular filtration rate (GFR) in preterm animal and human neonates, suggesting accelerated kidney maturation. Steroid exposure during pregnancy may also promote fetal kidney maturation.[12] However, Garg et al. reported the nephrotoxic role of antenatal steroids in neonates (AOR 3.9, 95% CI 1.1–8.9).[29] Despite this, international obstetrics and gynecology guidelines recommend antenatal steroids for fetal lung maturation in cases of prematurity. Animal studies have shown that antenatal steroids can affect fetal nephrogenesis by maturational arrest as it impairs nephron branching arrest.[30]

Jetton et al. and Selewski et al. reported that AKI increases mortality risk by over fourfold (AOR 4.6, 95% CI 2.5–8.3, $p < 0.0001$) and significantly prolongs NICU stay (adjusted 8.8 days, 95% CI 6.9–11.5, $p < 0.001$).[12, 14] The AWAKEN study similarly noted a 43% incidence of AKI in neonates <29 weeks, with markedly worse outcomes.[11] Comparable findings were reported by Selewski (38%), Gul (37.6%), and Gallo (33.3%), all linking AKI to increased ventilation needs, prolonged hospitalization, and poor prognosis.[12,6,26] Our study supports these trends, showing that AKI severity was significantly associated with longer hospital stay (OR 11.7, 95% CI 5.1–18.4) and higher mortality (AOR 4.0, 95% CI 1.4–11.5), reinforcing the substantial burden of AKI on preterm neonatal outcomes.

Our study further identified obstetric factors including primigravida status, LSCS delivery as they reflect underlying perinatal risk for AKI.[1,5,7] Similarly, male gender, low birth weight are known predictors of neonatal AKI and morbidity.[8,16] The central line placement, nephrotoxic drug exposure, IVH, and inotrope use. The association with invasive interventions underscores the renal vulnerability of critically ill preterm

neonates.[16 – 18] These findings align with prior studies highlighting the multifactorial nature of AKI.

Conclusion

In conclusion, AKI occurred in over 40% of preterm neonates, with severe AKI associated with high mortality and longer hospitalization. Maternal anemia, hsPDA, and mechanical ventilation were key risk factors, while antenatal corticosteroids were strongly protective. Early risk stratification and preventive strategies are essential to reduce AKI-related morbidity and mortality in preterm neonates.

The key strength of this study is that it represents the first South Asian cohort to demonstrate a six-fold increased risk of AKI in preterm neonates associated with maternal anemia. It is a novel and clinically significant finding with important public health implications for low-resource settings.

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