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Frequency of Acute Kidney Injury in Neonates with Perinatal Asphyxia

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ABSTRACT

Background: Acute Kidney Injury (AKI) is a common and severe complication in neonates with perinatal asphyxia, contributing to long-term morbidity. Identifying risk factors and ensuring early detection are crucial for improving outcomes in these vulnerable infants. **Objective:** To determine the prevalence of AKI in neonates with perinatal asphyxia. **Study Design:** Cross-sectional study. **Duration and Place of Study:** The study was conducted from February 2024 to August 2024 at the Paediatrics Department, POF Hospital Wah Cantt. **Methodology:** A total of 145 neonates with perinatal asphyxia, defined by an APGAR score of <7 at 5 minutes or delayed crying for ≥1 minute, were included. Neonates with congenital anomalies, maternal drug addiction, or oligohydramnios were excluded. The primary outcome, AKI, was diagnosed based on serum creatinine levels >133 µmol/l at 24, 48, and 72 hours' post-birth. **Results:** The mean maternal age was 28.71 ± 3.73 years, and the mean gestational age at birth was 38.67 ± 1.32 weeks. The mean APGAR score was 4.26 ± 0.89. Of the neonates, 40.7% developed AKI. Maternal age did not show a significant association with AKI ($p = 0.794$), while gestational age ≤39 weeks ($p < 0.001$), male gender ($p < 0.001$), and residential status ($p = 0.002$) were significantly associated with AKI. **Conclusion:** Our study concluded that AKI is a common complication of perinatal asphyxia, with male gender and gestational age being significant risk factors.

INTRODUCTION

Perinatal asphyxia is a condition in which there is a lack of oxygen and reduced blood supply to the baby or fetus at or close to the time of birth, leading to systemic hypoxia and metabolic acidosis.[1] Perinatal asphyxia can be caused by maternal conditions such as preeclampsia or placental abruption, prolonged labour, compression of the umbilical cord, or intrapartum conditions such as shoulder dystocia.[2] The lack of oxygen supply compromises cellular metabolism, particularly in highly metabolically active tissues such as brain, heart, and kidney.[4] Neonates who have experienced perinatal asphyxia can have clinical features such as low Apgar scores, respiratory distress, bradycardia, and hypotonia.[4] Perinatal asphyxia, if not managed early, can have severe short- and long-term consequences such as multi-organ failure, neurodevelopmental impairment, and even death.[5] Detection and early intervention like therapeutic hypothermia are crucial in achieving improved outcomes in affected neonates.[6]

Acute kidney injury (AKI) in the neonate is a complex and multifactorial disease resulting in sudden loss in kidney function, classically manifesting as a fall in urine output, electrolyte abnormalities, and elevation in serum creatinine level.[7] Neonate AKI is caused by a variety of etiologies, including hypoxic-ischemic injury, sepsis, congenital abnormalities in kidney and urinary tract (CAKUT), dehydration, or nephrotoxic medication.[8] The relative immaturity of the kidney in the neonate makes them vulnerable to injury resulting from insult to renal perfusion or direct injury to renal tissue.[9] Neonate AKI is associated with increased morbidity, increased length of stay, and increased risk for subsequent chronic kidney disease.[10] Initial recognition is based upon close observation for clinical presentation, laboratory parameters, and in some cases, imaging procedures. Treatment is targeted at addressing underlying etiology, fluid status correction, correction of electrolytes, and supportive care, including in severe cases, renal replacement therapy.[11]

Acute kidney injury in asphyxiated neonates is a serious complication of systemic hypoxic and ischemic injury and is reflective of increased susceptibility to

hypoxic injury in the kidneys.[12] Perinatal asphyxias is accompanied by reduced renal perfusion consequent upon systemic hypotension and oxidative injury leading to tubular necrosis and defective glomerular filtration.[13] The resulting manifestations are oliguria or anuria, metabolic acidosis, hyperkalemia, and fluid overload and contribute to worsening of the course in the neonate.[14] The interrelationship between asphyxia and AKI is self-perpetuating, as injury to the kidney exacerbates systemic acid-base disturbance and delays recovery from the initiating hypoxic injury.[15] It is challenging to detect AKI in this context in early stages but is essential as early interventions like fluid status correction, correction of electrolytes, and renal supportive interventions can dramatically alter outcomes.[16] Multidisciplinary expertise involving specialists in neonatology, nephrology, and intensive care is required in managing severely asphyxiated neonates and in preventing long-term renal and neurodevelopmental morbidities.[17]

A study revealed that the occurrence of Acute Kidney Injury (AKI) in neonates with Hypoxic Ischemic Encephalopathy (HIE) was 37.7%.[18] The risk of developing AKI was approximately 39 times higher in neonates with HIE stage III compared to those in HIE stage I. The mortality rate in this cohort was found to be 51.6%, with significantly higher mortality in stage III HIE. Additionally, neonates with AKI had a fivefold increased risk of mortality when compared to those with HIE but without AKI.[18]

Perinatal asphyxia is still a leading cause for neonatal morbidity and mortality, and AKI is one of the affected organs. Detection and early correction of AKI can dramatically improve outcomes in affected neonates, but prevalence and risk factors for it are still not adequately studied. The current study will contribute valuable information regarding the prevalence of AKI in asphyxiated neonates and will be beneficial for better decision-making in early interventions and in clinical practices for prevention of related long-term complications.

METHODOLOGY

The study was conducted in a cross-sectional design in the time period between February and August 2024 at the Paediatrics department, POF Hospital. The study involved a total of 145 neonates who had perinatal asphyxia. The calculation for required sample size utilized the WHO calculator for calculation of sample size and considered expected prevalence in cases of perinatal asphyxia for acute kidney injury (AKI) as being at 37.7%, confidence level at 95%, and level for absolute precision at 8%.[18]

Included patients age was less than 24 hours of both genders. Perinatal Asphyxia had been defined as any neonate who at birth had had an APGAR score below 7 at 5 minutes or had had delayed crying for ≥1 minute, as recorded at time of birth by treating clinician. Neonates who had at time of birth had an APGAR score between 7 and 10 had been classified as being in normal condition, between 4 and 6 as being in moderately depressed condition, and below 4 as being in severely depressed condition, in accordance with perinatal asphyxia. Exclusion criteria had consisted in neonates suffering from congenital malformations such as Down syndrome, maternal drug addiction, or kidney and urinary system congenital abnormalities recognized on antenatal ultrasound. Neonates who had a mother diagnosis as suffering from oligohydramnios were also excluded.

With permission from the ethical committee of the hospital, informed consent was obtained from the parents of the neonates. The maternal and neonatal information was recorded in a structured proforma by the investigator in charge. The neonates were afterwards admitted in department of neonatology in pediatrics for follow-up. The main goal in this research involved follow-up for presence of AKI in the neonates. The serum level of creatinine at 24, 48, and 72 hours after birth and follow-up at 3 months of age was evaluated.

Acute Kidney Injury (AKI) was defined as any asphyxiated neonate who had serum creatinine > 133 µmol/l. The data was analyzed on IBM SPSS version 23.0. Descriptive statistics have been utilized and for continuous variables such as mother's age, gestation at baby's time of birth, APGAR score, and serum creatinine level, values for mean and standard deviation have been computed. Frequencies and percentages for categorical variables such as gender, type of delivery, and presence or absence of AKI have been computed. The stratification was based on maternal age, gestation at baby's time of birth, sex, APGAR score, and type of delivery in order to consider confounding variables. A chi-square test for post-stratification analysis is used and p-value of ≤0.05 is considered significant. Logistic regression analysis for predicting AKI was also done.

RESULTS

As shown in Table-I, the mean maternal age was 28.71 ± 3.73 years, with a gestational age at birth of 38.67 ± 1.32 weeks. The mean APGAR score was 4.26 ± 0.89, and the serum creatinine levels at 24, 48, and 72 hours were 125.41 ± 23.27, 119.97 ± 23.14, and 115.06 ± 23.42, respectively. Gender distribution showed 73.1% male (n=106) and 26.9% female (n=39), with the mode of delivery being predominantly vaginal (71.7%, n=104) compared to C-section (28.3%, n=41).

Table I

Patient Demographics (n=145)

Demographics	Mean ± SD
Mothers Age (years)	28.710±3.73
Gestational Age at Birth (weeks)	38.669±1.32

APGAR Score		4.255±0.89
Serum Creatinine at 24 hours		125.414±23.27
Serum Creatinine at 48 hours		119.972±23.14
Serum Creatinine at 72 hours		115.055±23.42
Gender	Male n (%)	106 (73.1%)
	Female n (%)	39 (26.9%)
Mode of Delivery	Vaginal n (%)	104 (71.7%)
	C-Section n (%)	41 (28.3%)

Figure 1

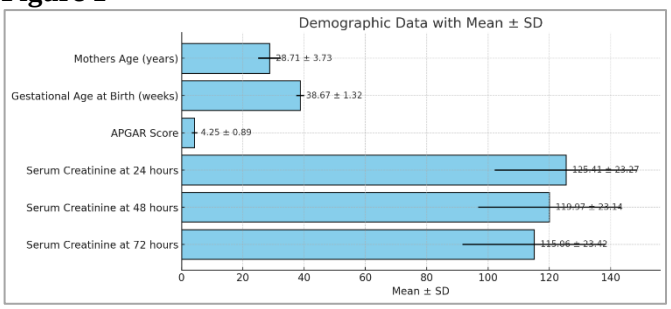


Table-II presents the occurrence of Acute Kidney Injury (AKI) in neonates with perinatal asphyxia, where 40.7% (n=59) developed AKI, and 59.3% (n=86) did not.

Table II

Acute Kidney Injury in neonate with perinatal asphyxia

Acute Kidney Injury	Frequency	% age
Yes	59	40.7%
No	86	59.3%
Total	145	100%

In Table-III, the association of AKI with demographic factors was assessed. Maternal age did not show a significant association with AKI (p = 0.794), with 41.4% of mothers aged ≤30 years and 39.1% of mothers aged >30 years having neonates with AKI. Gestational age at birth showed a significant relationship (p < 0.001), with 54.5% of neonates born at ≤39 weeks developing AKI, compared to just 9.1% in those born after 39 weeks. Gender was also significantly associated with AKI (p < 0.001), with a higher incidence in males (30.2%) compared to females (69.2%). Residential status was found to be significant (p = 0.002), with 53.5% of those living in larger homes (>4 rooms) developing AKI. The mode of delivery showed no significant association with AKI (p = 0.798), as the percentage of AKI was similar between vaginal (41.3%) and C-section deliveries (39%).

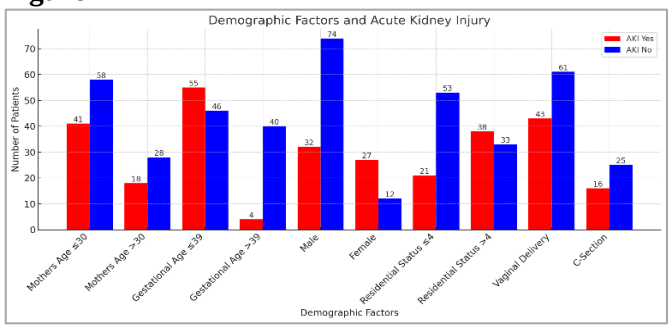
Table III

Association of Acute Kidney Injury with Demographic Factors

Demographic Factors		Acute Kidney Injury		p-value
		Yes n(%)	No n(%)	
Mothers Age (years)	≤30	41 (41.4%)	58 (58.6%)	0.794
	>30	18 (39.1%)	28 (60.9%)	
Gestational Age at Birth (weeks)	≤39	55 (54.5%)	46 (45.5%)	<0.001*
	>39	4 (9.1%)	40 (90.9%)	
Gender	Male	32 (30.2%)	74 (69.8%)	<0.001
	Female	27 (69.2%)	12 (30.8%)	
Residential Status	≤4	21 (28.4%)	53 (71.6%)	0.002
	>4	38 (53.5%)	33 (46.5%)	
Mode of Delivery	Vaginal Delivery	43 (41.3%)	61 (58.7%)	0.798
	C-Section	16 (39%)	25 (61%)	

Fischer Exact Test

Figure 2



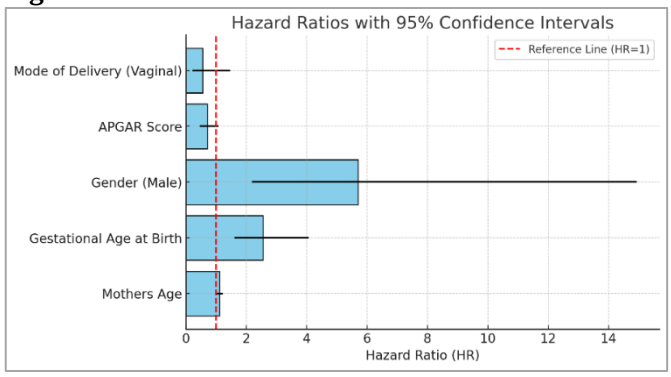
The logistic regression analysis indicates that gestational age at birth was a significant predictor, with neonates of higher gestational age having an increased risk of developing AKI (*HR* = 2.557, 95% CI: 1.599–4.072, $p < 0.001$). Additionally, male neonates had a significantly higher likelihood of AKI compared to females (*HR* = 5.696, 95% CI: 2.186–14.937, $p = 0.001$), suggesting a potential sex-related vulnerability. However, maternal age ($p = 0.087$), APGAR score ($p = 0.126$), and mode of delivery ($p = 0.264$) did not show statistically significant associations with AKI. The model fit was supported by a significant omnibus test ($\chi^2 = 56.410$, $p < 0.001$) and the Hosmer-Lemeshow test ($\chi^2 = 20.158$, $p = 0.010$), indicating a reliable predictive model as shown in Table-IV

Table IV

Logistic Regression Analysis for Predicting AKI

Characteristic	HR (95% CI)	P-value
Mothers Age	1.105 (0.993 - 1.230)	0.087
Gestational Age at Birth	2.557 (1.599 - 4.072)	< 0.001
Gender (Male)	5.696 (2.186 - 14.937)	0.001
APGAR Score	0.704 (0.453 - 1.081)	0.126
Mode of Delivery (Vaginal)	0.553 (0.210 - 1.469)	0.264

Figure 3



DISCUSSION

These results showed that 40.7% of the neonates with perinatal asphyxia developed AKI, pointing toward the prevalent occurrence of the condition within the at-risk population of these infants. The incidence of AKI with different demographic characteristics such as the gestational age and gender of the infants presents valuable insight into the likely risk factors for the condition. The much higher incidence of AKI within the population of infants with a gestational age of ≤39 weeks might be due to the intrinsic physiological immaturity of the preterm infant kidneys that increases the susceptibility of these infants' kidneys toward the insults of asphyxia-induced injury. The increased susceptibility of the male infant toward AKI, noted within the study population, conforms with the reported sex-differences within the renal function and susceptibility toward injury within the population of infants, with the males generally presenting with increased vulnerability toward the insult of renal damage within the infant population. The presentation of these results highlights the need for early detection and follow-up of the at-risk population of infants with increased susceptibility toward AKI within the context of the occurrence of perinatal asphyxia.

Our study showed that acute kidney injury (AKI) is a serious and prevalent complication of perinatal asphyxia with 40.7% of the neonates diagnosed with AKI. The 40.9% incidence reported by the study of Cheni et al. [19] for the development of AKI with the setting of perinatal asphyxia closely matches the current study. Even though the current study differs from the study of Memon et al. [20] with 13.3% incidence of AKI, the disparity may be attributed to the study population characteristics, study design, and the population under study.

In our study the maternal age did not show a significant relationship with AKI in our population ($p = 0.794$), consistent with the findings of the work of other researchers such as that of Essajee[21] and Cheni et al. [19] that the characteristics of the mother such as the mother's age did not carry much weight with the incidence of AKI. The reasons for the same are that while the health of the mother does contribute toward causing the perinatal asphyxia, the characteristics of the neonate such as the sex and the gestational age might directly influence the renal outcome of the infant.

Our study showed that the birth gestational age correlated with AKI strongly ($p < 0.001$) with increasing numbers of the neonates at birth at ≤39 weeks developing AKI. The findings are consistent with the study of Tounsa et al. [22] and Medani et al. [23] with the latter documenting increased incidence of AKI in the preterm infants or at younger gestational age. The preterm infants are more prone to renal insult owing to the underdeveloped nature of the kidneys and are likely more sensitive to the hypoxic insult of the perinatal asphyxia.

Our study similarly revealed that gender correlated strongly with AKI ($p < 0.001$), with higher incidence in males (30.2%) compared with that of the females (69.2%). The finding is consistent with that of previous studies such as that of Cheni et al. [19] in which AKI incidence was more probable in the males, and that of Tounsa et al. [22] with higher AKI incidence reported for the male neonates. The heightened susceptibility of the male neonates for AKI could be attributed to intrinsic imbalance between the genders for the development of the kidneys and susceptibility for the stressors and hormonal influences that might enhance the susceptibility of the latter for the nephrotoxic effect of the perinatal asphyxia.

Our analysis of residential status as a demographic factor revealed that it did predict AKI ($p = 0.002$), with AKI incidence being more for the households with more spacious dwellings (>4 rooms). The relationship between residential status and AKI incidence is novel with the present study but could be due to socioeconomic determinants of the health care-seeking ability or the quality of prenatal health care that indirectly affects the susceptibility of the neonates for AKI. Notably, delivery type did not differ substantially with AKI ($p = 0.798$) in the current study, contrary to the findings of Memon et al. [20] with delivery type reported by them to be correlated with AKI. The difference may be due to different study populations or practices of medicine under which the rates of the C-section are more for the infants at higher risk of complications including AKI.

Our logistic regression analysis revealed that higher gestational age at birth (HR = 2.557, $p < 0.001$) and male sex (HR = 5.696, $p = 0.001$) were predictors of AKI. The above are consistent with the outcome of Bhosgi et al.'s study[24] that of Essajee,[21] and that of Alaro et al.'s study[25] where male sex and gestational age were predictors of AKI. The heightened susceptibility of AKI among male infants and increased gestational age emphasizes the need for heightened vigilance within these populations subsequent to perinatal asphyxia.

The results of our study joined the increasing number of evidence that point toward the high incidence of acute kidney injury (AKI) in infants with perinatal asphyxia, with the predictors of male gender and gestational age being most important. Most of the studies are consistent with our results, pointing toward the importance of careful monitoring of at-risk infants such as males and infants with lower gestational ages.

There are, however, a few limitations to our study. It was a single-center study, and this may restrict the generalizability of the results to other populations or healthcare environments. The sample size was also modest, and this may influence the statistical power of

some of the associations. A larger multicenter study involving a more heterogeneous population would be more powerful and enable wider inferences to be made. The study also did not have long-term follow-up to evaluate the possibility of chronic kidney disease or other renal sequelae in AKI survivors. These aspects need to be considered in future studies in order to advance our knowledge in AKI in neonatal perinatal asphyxia.

CONCLUSION

Our study concluded that acute kidney injury (AKI) is a serious and prevalent complication of perinatal asphyxia in the neonate. We concluded that male sex and gestational age are crucial risk factors and that these were strongly correlated with the incidence of AKI. We reaffirm the importance of early detection and assessment of at-risk neonates for the improvement of outcomes and prevention of subsequent renal complications. We advocate that interventions need to be implemented promptly and that more effort should be placed toward the development of more accurate AKI prediction models for the perinatal asphyxia neonate.

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Dr. Yumna Zafar spearheaded the project design, manuscript preparation, and clinical information collection at the hospital site.
Dr Sohail Ashraf was instrumental in manuscript refinement, research framework development, and the statistical evaluation and contextual assessment of findings.

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