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Original article

The Effect of Neonatal Birth Asphyxia on Renal Function in the First Week of Life: A Prospective Cross-Sectional Study

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Abstract
Perinatal asphyxia remains one of the most significant causes of neonatal morbidity and mortality globally, particularly in developing countries. The kidneys are highly susceptible to hypoxic-ischemic injury, making acute kidney injury a common complication in asphyxiated neonates. This study aimed to determine the prevalence and patterns of renal function disturbance in asphyxiated newborns of varying severity during the early neonatal period. This prospective cross-sectional study was conducted at two tertiary care facilities over a one-year period in 2021. Ninety-eight neonates with perinatal asphyxia admitted to the neonatal intensive care unit were enrolled. Renal function was assessed through serum creatinine, blood urea, electrolytes, and urine output monitoring. Hypoxic-ischemic encephalopathy staging was performed using the Sarnat criteria. Among the 98 neonates, 52% were male, 78.6% were term infants, and 76.5% had normal birth weight. Renal dysfunction occurred in 34.7% of cases. HIE stages I, II, and III accounted for 27.6%, 42.9%, and 29.6% of neonates, respectively. Neonates with renal dysfunction had significantly higher serum creatinine (0.92 ± 0.57 vs. 0.58 ± 0.33 mg/dL; p=0.001) and blood urea levels (40.0 ± 30.38 vs. 11.0 ± 4.32 mg/dL; p=0.001). Mechanical ventilation was significantly associated with renal dysfunction (p=0.011). Mortality was 19.4% and was significantly higher among neonates with renal dysfunction (p=0.004). Perinatal asphyxia is a major cause of neonatal acute kidney injury. Early monitoring of renal function parameters is essential for timely intervention and improved outcomes in asphyxiated neonates.
Keywords: Perinatal Asphyxia, Renal Function, Acute Kidney Injury, Hypoxic-ischemic Encephalopathy, Neonates.

Introduction

Perinatal asphyxia is a critical condition characterized by impaired respiratory gas exchange leading to progressive hypoxemia, hypercapnia, and metabolic acidosis. The World Health Organization defines birth asphyxia as the failure to initiate and sustain breathing at birth, typically characterized by an Apgar score of less than 7 at one minute of life. This condition remains a leading cause of neonatal mortality and long-term morbidity worldwide, accounting for approximately 23% of all neonatal deaths globally [1, 2]. The global burden of perinatal asphyxia disproportionately affects low- and middle-income countries, with systematic reviews reporting pooled prevalence rates of 15.9% in East and Central Africa compared to rates of approximately 2.28 per 1,000 births in developed countries [3, 4]. Multiple studies from Ethiopia, Zimbabwe, and Libya have documented significant mortality risks associated with perinatal asphyxia in resource-limited settings [5-8].

The pathophysiology of perinatal asphyxia involves redistribution of cardiac output to maintain perfusion to vital organs, including the brain and heart, while perfusion to less critical organs such as the kidneys may be compromised. This compensatory mechanism renders the kidneys particularly vulnerable to injury. During hypoxic-ischemic events, renal tubular cells are susceptible to damage due to their high metabolic demands and limited regenerative capacity. The resulting acute kidney injury can manifest as oliguria, electrolyte imbalances, and elevated serum creatinine levels [9]. Experimental studies have demonstrated that perinatal asphyxia leads to acute kidney damage through increased expression of renal injury markers, proinflammatory cytokines, and fibrotic markers, with potential long-term consequences including increased renal susceptibility in adulthood [10]. Hypoxic-ischemic encephalopathy represents the neurological manifestation of perinatal asphyxia and is classified into three stages based on clinical severity. The Sarnat staging system categorizes HIE into mild (stage I), moderate (stage II), and severe (stage III) based on clinical features including level of consciousness, neuromuscular control, reflexes, and autonomic function. Transcranial ultrasound and color Doppler may provide additional insights into cerebral hemodynamics in affected neonates [11]. The severity of HIE has been shown to correlate with the extent of multi-organ dysfunction, including renal impairment.

Acute kidney injury in neonates with perinatal asphyxia is diagnosed using modified KDIGO criteria, which incorporate serum creatinine elevation and urine output reduction. The emergence of novel urinary biomarkers including neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, cystatin C, and interleukin-18 offers promise for earlier detection of AKI before serum creatinine elevation [12-14]. These biomarkers may enable earlier intervention and improved outcomes. This study was designed to determine the prevalence of renal function disturbance in asphyxiated newborns, to evaluate the relationship between the severity of hypoxic-ischemic encephalopathy and renal dysfunction, and to identify factors associated with adverse outcomes in this vulnerable population.

Methods

Study Design and Setting

This prospective cross-sectional study was conducted at two tertiary care facilities equipped with neonatal intensive care units. The study period extended from January 1, 2021, to December 30, 2021. During this period, 138 neonates were

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identified with perinatal asphyxia, of whom 98 met the inclusion criteria and had complete data available for analysis.

Study Population and Selection Criteria

Neonates were included if they met the following criteria: asphyxiated newborns with Apgar scores less than 7 at 5 minutes; newborns who required immediate resuscitation, including bag-and-mask ventilation or intubation; and newborns with abnormal neurological examination findings, including alteration of muscle tone, abnormal neonatal reflexes, or presence of seizures. Neonates were excluded if they had congenital kidney anomalies, evidence of sepsis or septic shock, inborn errors of metabolism, or congenital infections.

Data Collection

Following emergency care and resuscitation, data were collected using standardized forms. Demographic and clinical data included neonatal age, sex, gestational age, birth weight, mode of delivery, resuscitation requirements, presence of meconium-stained amniotic fluid, seizures, need for mechanical ventilation, Apgar scores, HIE severity, and outcomes.

Laboratory Investigations

Baseline venous blood samples were collected and analyzed for serum urea, serum creatinine, serum calcium, and blood glucose. Reference values for interpretation included serum creatinine of 0.8-1.0 mg/dl on day 1, less than 0.7-0.8 mg/dl on day 3, and less than 0.6 mg/dl on day 7; blood urea of 15-20 mg/dl; and urine output of 1-5 ml/kg/hour (Kliegman et al., 2020). Renal dysfunction was defined as serum creatinine exceeding the upper limit of normal for age or blood urea exceeding 20 mg/dl in conjunction with reduced urine output for 24 hours or more.

HIE Staging

The severity of hypoxic-ischemic encephalopathy was classified according to the Sarnat and Sarnat staging system, with stage I representing mild, stage II moderate, and stage III severe encephalopathy.

Ethical Considerations

The study protocol was approved by the institutional Ethics Committee. Written informed consent was obtained from parents or legal guardians of all enrolled neonates.

Statistical Analysis

Data were analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Associations were assessed using the chi-square test and independent-samples t-test. A p-value ≤0.05 was considered statistically significant.

Results

During the study period, 98 neonates with perinatal asphyxia were enrolled. The demographic characteristics are summarized in (Table 1). The gender distribution showed 51 (52%) males and 47 (48%) females. The mean age at presentation was 3.64 ± 1.7 days. The majority were term neonates (77, 78.6%), with 9 (9.2%) preterm and 12 (12.2%) post-term. The mean birth weight was 3.25 ± 0.58 kg, with 75 (76.5%) having normal birth weight.

Table 1. Demographic Characteristics of Asphyxiated Neonates (N=98)

Variable	n	%
Age (Days)		
≤ 4	66	67.3
≥ 5	29	29.6
Not Recorded	3	3.1
Gender		
Male	51	52.0
Female	47	48.0
Gestational Age		
Preterm	9	9.2
Full term	77	78.6
Post term	12	12.2
Birth Weight (g)		
< 2,500	13	13.3
2,500 - 3,999	75	76.5
≥ 4,000	9	9.2

The maternal characteristics and perinatal factors are presented in (Table 2). The mean maternal age was 28.2 ± 6.7

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years. Multigravidas comprised 71 (72.4%) of mothers, with pregnancy-related complications identified in 20 (21.4%).

Table 2. Maternal and Perinatal Characteristics (N=98)

Variable	n	%
Gravidity		
Primigravida	27	27.6
Multigravida	71	72.4
Maternal Complications		
None	78	79.6
Present	20	20.4
Mode of Delivery		
Vaginal	49	50.0
Cesarean section	49	50.0

Clinical characteristics and resuscitation requirements are shown in (Table 3). Delayed first cry was observed in 67 (68.4%) neonates, cyanosis in 74 (75.5%), and meconium-stained amniotic fluid in 29 (29.6%). Bag and mask ventilation was required in 68 (69.4%), intubation in 24 (24.5%), and mechanical ventilation in 38 (38.8%). Seizures occurred in 90 (91.8%) neonates.

Table 3. Clinical Characteristics and Resuscitation Requirements (N=98)

Variable	n	%
Delayed Cry at Birth	67	68.4
Cyanosis at Birth	74	75.5
Bag and Mask Ventilation	68	69.4
Required Intubation	24	24.5
Meconium-Stained Fluid	29	29.6
Seizures	90	91.8
Mechanical Ventilation	38	38.8

The distribution of Apgar scores and HIE staging is shown in (Table 4). The mean 1-minute Apgar score was 4.58 ± 2.84 and the 5-minute score was 7.66 ± 1.89. Regarding HIE staging, 27 (27.6%) had stage I, 42 (42.9%) had stage II, and 29 (29.6%) had stage III.

Table 4. Apgar Scores and HIE Staging (N=98)

Variable	n	%
1-Minute Apgar Score		
0-3	40	40.8
4-6	28	28.6
7-10	29	29.6
5-Minute Apgar Score		
2-6	24	24.5
7-10	73	74.5
HIE Stage		
Stage I (Mild)	27	27.6
Stage II (Moderate)	42	42.9
Stage III (Severe)	29	29.6

The biochemical parameters are summarized in (Table 5). Mean serum urea was 21.6 ± 19.51 mg/dl and serum creatinine was 0.69 ± 0.45 mg/dl. Mean urine output was 2.35 ± 3.4 ml/kg/hour.

Table 5. Biochemical Parameters in Asphyxiated Neonates

Parameter	Minimum	Maximum	Mean	SD
Serum urea (mg/dl)	2.0	109	21.6	19.51
Serum creatinine (mg/dl)	0.20	2.78	0.69	0.46
Serum calcium (mg/dl)	6.6	12.0	8.87	1.07
Urine output (ml/kg/hr)	0.10	15	2.35	3.4

The distribution of biochemical abnormalities is presented in (Table 6). Serum urea was elevated in 34 (36.6%), serum

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creatinine in 26 (27.1%), and hypocalcemia in 15 (16.9%). Renal dysfunction was identified in 34 (34.7%) of asphyxiated neonates.

Table 6. Distribution of Biochemical Abnormalities

Parameter	High n (%)	Normal n (%)	Low n (%)
Serum urea	34 (36.6)	59 (63.4)	-
Serum creatinine	26 (27.1)	70 (72.9)	-
Serum calcium	4 (4.5)	70 (78.7)	15 (16.9)

The associations between renal function and demographic variables are shown in (Table 7). No significant associations were found with sex (p=0.833), gestational age (p=0.069), or birth weight (p=0.505).

Table 7. Association Between Renal Function and Demographic Variables

Variable	Renal Dysfunction n (%)	Normal Function n (%)	p-value
Sex			
Male	17 (33.3)	34 (66.7)	0.833
Female	17 (36.2)	30 (63.8)	
Gestational Age			
Preterm	2 (22.2)	7 (77.8)	0.069
Full term	31 (40.3)	46 (59.7)	
Post term	1 (8.3)	11 (91.7)	
Birth Weight			
< 2,500 g	5 (38.5)	8 (61.5)	0.505
≥ 2,500 g	29 (34.5)	55 (65.5)	

The relationship between renal function and HIE staging is presented in (Table 8). Although renal dysfunction increased with HIE severity (22.2% in stage I, 33.3% in stage II, 48.3% in stage III), this did not reach statistical significance (p=0.119).

Table 8. Association Between Renal Function and HIE Staging

HIE Stage	Renal Dysfunction n (%)	Normal Function n (%)	p-value
Stage I	6 (22.2)	21 (77.8)	0.119
Stage II	14 (33.3)	28 (66.7)	
Stage III	14 (48.3)	15 (51.7)	

The association between mechanical ventilation and renal function is shown in (Table 9). Neonates requiring ventilation had significantly higher rates of renal dysfunction (50.0% versus 25.0%, p=0.011).

Table 9. Association Between Renal Function and Mechanical Ventilation

Ventilator Support	Renal Dysfunction n (%)	Normal Function n (%)	p-value
Yes	19 (50.0)	19 (50.0)	0.011
No	15 (25.0)	45 (75.0)	

Neonates with renal dysfunction had significantly higher serum urea (40.0 ± 30.38 versus 11.0 ± 4.32 mg/ dl, p=0.001) and creatinine (0.92 ± 0.57 versus 0.58 ± 0.33 mg/dl, p=0.001). The outcomes and their association with renal function are shown in (Table 10). The overall mortality rate was 19.4%. Among those who died, 63.2% had renal dysfunction compared to 27.8% of survivors (p=0.004).

Table 10. Association Between Renal Function and Outcome

Outcome	Renal Dysfunction n (%)	Normal Function n (%)	p-value
Discharged	22 (27.8)	57 (72.2)	0.004
Died	12 (63.2)	7 (36.8)	

Discussion

This study demonstrated that renal dysfunction is a common complication of perinatal asphyxia, occurring in 34.7% of affected neonates. This prevalence aligns with Memon et al. (2021) [15], who reported acute kidney injury in 37.93% of 145 term neonates with perinatal asphyxia, and with Gul et al. (2022) [16], who found AKI in 37.6% of NICU admissions with birth asphyxia identified as a significant risk factor. However, our finding is lower than Sood et al. (2022) [17], who reported 62% AKI prevalence, and substantially lower than Rajesh et al. (2022) [18], who found renal failure rates reaching 82% in

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HIE grade II and 100% in grade III. This variation likely reflects differences in diagnostic criteria, timing of assessment, and population severity distribution. Conversely, our prevalence exceeds the 25% reported by Rumpel et al. (2022) [14] in neonates receiving therapeutic hypothermia, suggesting that neuroprotective interventions may confer renal benefits, and the 16.9% reported by Phuljhele et al. (2024), possibly reflecting differences in case mix and definitions [19]. Our study population showed a male-to-female ratio of approximately 1.08:1, which is lower than the 1.90:1 ratio reported by Rajesh et al. (2022) [18] and the 73.1% male predominance found by Memon et al. (2021) [15]. However, our finding aligns closely with Yusuf et al. (2026) [20], who reported 52.6% males in their Ugandan cohort. The lack of significant association between sex and renal dysfunction in our study (p=0.833) is consistent with findings from Roy et al. (2020) [21] and Kumar et al. (2020) [22], suggesting that gender does not independently predict renal injury risk in asphyxiated neonates.

Although we observed a clear trend of increasing renal dysfunction with HIE severity (22.2% in stage I, 33.3% in stage II, 48.3% in stage III), this did not reach statistical significance (p=0.119). This finding contrasts with multiple studies demonstrating significant correlations. Rajesh et al. (2022) [18] found a highly significant association (p<0.05) with renal failure rates of 12% in grade I, 82% in grade II, and 100% in grade III. Chavda et al. (2026) [23] reported significant differences across HIE stages (p<0.001), and Phuljhele et al. (2024) [19] found a significant correlation between HIE III and AKI. Similarly, Roy et al. (2020) [21] and Kumar et al. (2020) [22] demonstrated significant relationships between HIE staging and renal parameters. The lack of statistical significance in our study, despite the clear trend, may be attributed to our smaller sample size and the relatively even distribution of HIE stages, which reduced statistical power to detect differences. Our biochemical findings showed significantly elevated serum creatinine (0.92 ± 0.57 versus 0.58 ± 0.33 mg/dl, p=0.001) and blood urea (40.0 ± 30.38 versus 11.0 ± 4.32 mg/dl, p=0.001) in neonates with renal dysfunction. These results align with Rajesh et al. (2022) [18], who reported mean serum creatinine of 0.9 ± 0.44 mg/dl in HIE grade I, 2.2 ± 0.93 mg/dl in grade II, and 2.9 ± 0.38 mg/dl in grade III, with significant increases across grades (p<0.05). Roy et al. (2020) [21] similarly found significantly higher creatinine in asphyxiated cases (1.64 ± 0.33 versus 0.41 ± 0.09 mg/dl, p<0.001), with the highest values in HIE stage III. Sood et al. (2022) [17] confirmed that serum creatinine levels correlate with HIE severity. However, our creatinine values in the dysfunction group are notably lower than those reported in severe cases by other investigators, suggesting our population may have had relatively milder renal impairment overall.

Mean urine output in our study was 2.35 ± 3.4 ml/kg/hour overall, with lower output in those with renal dysfunction (1.06 ± 1.33 versus 5.09 ± 3.92 ml/kg/hr, p=0.077). Although not statistically significant, our values in the dysfunction group are comparable to Rajesh et al. (2022) [18], who reported 1.5 ± 0.38, 1.3 ± 0.53, and 1.1 ± 0.39 ml/kg/hour in HIE grades I, II, and III, respectively, with significant decreases as grades increased (p<0.05). Kumar et al. (2020) similarly found that urine output decreased as HIE stages progressed [22]. The lack of significance in our comparison may relate to the large standard deviation observed, reflecting substantial variability possibly due to collection difficulties. Zhang and Zeng (2020) [24] found that in preterm neonates with birth asphyxia, the incidence of urine output less than 1.0 ml/kg/h was 20.31%, comparable to the 21.7% oliguria rate we observed.

We documented hypocalcemia in 16.9% of asphyxiated neonates. This aligns closely with Yusuf et al. (2026) [20], who found 21.2% hypocalcemia in their cohort. The significant association between mechanical ventilation requirement and renal dysfunction (50% versus 25%, p=0.011) in our study aligns with findings from Gul et al. (2022) [16], who identified inotropic agent use as a significant risk factor for AKI, and Rumpel et al. (2022) [14], who found that neonates with AKI had more days of vasopressor use (median 2 versus 0 days, p=0.026). This association likely reflects overall illness severity rather than direct causality, as neonates requiring ventilation typically have more severe asphyxia and greater hemodynamic instability predisposing to kidney injury. Our overall mortality rate of 19.4% is consistent with international reports from similar settings. More importantly, we found a highly significant association between renal dysfunction and mortality, with 63.2% of deaths occurring in neonates with renal dysfunction versus 36.8% in those with normal function (p=0.004). This finding strongly aligns with Rumpel et al. (2022) [14], who reported mortality of 25% versus 2% in neonates with and without AKI (p=0.012), and with Gul et al. (2022), who found mortality rates of 16.5% versus 3.9% in affected versus unaffected neonates (p<0.001) [16]. These consistent findings across studies underscore the prognostic significance of renal involvement in asphyxiated neonates.

The pathophysiological basis for our findings is supported by experimental evidence from Lakat et al. (2024) [10], who demonstrated that perinatal asphyxia leads to acute kidney damage through increased expression of renal injury markers, proinflammatory cytokines, and fibrotic markers in a rat model. Their finding that perinatal asphyxia increases susceptibility to subsequent renal injury in adulthood highlights the potential long-term consequences beyond the neonatal period. Additionally, their machine learning model achieved 95.5% classification accuracy for identifying perinatal asphyxia based on renal biomarkers, suggesting potential for future diagnostic applications. Gallo et al. (2021) confirmed in their large cohort study that perinatal asphyxia was the most common etiology of AKI in term neonates (72.2%), with most cases diagnosed by serum creatinine criteria [25].

This study has several limitations, including incomplete urine output monitoring in some neonates, death of some subjects before repeat testing, absence of a non-asphyxiated control group, and lack of novel biomarker assessment. The sample size may have limited statistical power, particularly for detecting the association between HIE staging and renal dysfunction. However, our study provides important baseline data demonstrating that renal dysfunction is common in asphyxiated neonates and strongly associated with mortality, emphasizing the need for routine monitoring and early intervention.

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Conclusion

Perinatal asphyxia is a major cause of neonatal acute kidney injury, affecting approximately one-third of affected neonates in this study. Elevated serum creatinine and blood urea levels were significantly associated with renal dysfunction, while mechanical ventilation requirement emerged as a significant clinical predictor. Although a trend toward increasing renal dysfunction with HIE severity was observed, this did not reach statistical significance. Most importantly, renal dysfunction was strongly associated with mortality, highlighting the prognostic significance of kidney involvement. Early monitoring of renal function parameters is essential for timely identification and management of acute kidney injury in this vulnerable population.

Conflict of interest. Nil

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