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Assessment of Renal Function in Term Neonates with Perinatal Asphyxia

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ABSTRACT

Background: Perinatal asphyxia is a significant cause of neonatal morbidity and is associated with multiorgan dysfunction, including renal impairment. Renal injury is commonly observed in neonates with hypoxic-ischemic encephalopathy and may worsen with increasing severity of hypoxia.
Aim: To evaluate renal function among term neonates with perinatal asphyxia and to assess its correlation with the severity of hypoxic-ischemic encephalopathy.
Methods: A hospital-based case-control study was conducted on 120 term neonates, including 60 asphyxiated cases and 60 controls. Renal function was assessed using biochemical parameters such as serum creatinine, blood urea, urine output, urine sodium, fractional excretion of sodium, and renal failure index. The severity of hypoxic-ischemic encephalopathy was graded, and correlation with renal parameters was analyzed.
Results: Asphyxiated neonates showed significantly reduced creatinine clearance and urine output along with increased urine sodium, FeNa, and RFI compared to controls ($p<0.001$). Renal dysfunction worsened with increasing severity of hypoxic-ischemic encephalopathy, with significant differences observed across stages ($p<0.001$).
Conclusion: Renal dysfunction is a common and significant complication of perinatal asphyxia and shows a strong correlation with the severity of hypoxic-ischemic encephalopathy. Early identification and monitoring of renal parameters are essential for improving neonatal outcomes.

Keywords*: Perinatal asphyxia, Hypoxic-ischemic encephalopathy, Acute kidney injury, Neonatal renal function.*

INTRODUCTION

Perinatal asphyxia is a major cause of neonatal morbidity and mortality worldwide, particularly in developing countries, and is defined as a condition resulting from impaired gas exchange leading to hypoxemia, hypercapnia, and metabolic acidosis in the newborn [1]. It is closely associated with hypoxic-ischemic encephalopathy (HIE), which reflects the severity of cerebral injury and serves as an important clinical marker of systemic organ dysfunction [2]. In addition to neurological damage, perinatal asphyxia often leads to multiorgan involvement, with the kidneys being among the most commonly affected organs due to their high metabolic activity and susceptibility to hypoxic injury [3].

Renal dysfunction in neonates with perinatal asphyxia commonly presents as acute kidney injury (AKI), resulting from decreased renal perfusion, ischemia, and subsequent tubular necrosis [3,4]. The incidence of AKI in asphyxiated neonates has been reported to range between 40% and 70%, depending on the severity of hypoxia and associated complications [7,8]. The neonatal kidney is particularly vulnerable to hypoxic insult due to immature nephron development and limited autoregulatory capacity, making early identification of renal impairment crucial for improving neonatal outcomes [5].

Hypoxic-ischemic encephalopathy is commonly classified into stages based on clinical severity, and several studies have demonstrated a strong correlation between the degree of HIE and the extent of renal dysfunction [4,8]. Neonates with moderate to severe HIE are more likely to develop significant renal impairment, including oliguria, elevated serum creatinine, and electrolyte disturbances [9]. This relationship highlights the importance of evaluating renal function alongside neurological assessment in neonates with perinatal asphyxia.

The pathophysiology of renal injury in perinatal asphyxia involves multiple mechanisms, including ischemia-reperfusion injury, oxidative stress, endothelial dysfunction, and inflammatory responses, all of which contribute to tubular and glomerular damage [6]. These changes ultimately lead to impaired glomerular filtration rate and altered tubular function. Early recognition of these alterations is essential to prevent progression to long-term renal dysfunction and associated complications [6,10].

Assessment of renal function in neonates remains challenging due to physiological variations in serum creatinine levels and the influence of maternal creatinine during the initial days of life [5]. Conventional parameters such as serum creatinine, blood urea nitrogen, and urine output are widely used in clinical practice, although they may not be sensitive enough to detect early renal injury [5]. Recent studies have also explored the role of novel biomarkers in the early detection of AKI; however, their routine use is still limited in most clinical settings [5].

Several recent studies have emphasized that early evaluation of renal function in neonates with perinatal asphyxia can aid in prompt identification of AKI and facilitate timely management, thereby improving clinical outcomes [7,9]. Furthermore, understanding the relationship between renal dysfunction and severity of HIE can help in prognostication and guide therapeutic interventions in affected neonates [8,10].

Despite advances in neonatal intensive care, perinatal asphyxia continues to contribute significantly to neonatal morbidity, and renal complications remain under-recognized in many cases. Therefore, systematic evaluation of renal function and its correlation with the degree of HIE is essential for comprehensive management and improved prognosis in these patients. Hence, the present study aims to evaluate renal function among term neonates with perinatal asphyxia and to assess its correlation with the severity of hypoxic-ischemic encephalopathy.

MATERIAL AND METHODS

The present study was designed as a hospital-based prospective case-control study conducted in the Department of Pediatrics in collaboration with the Neonatal Intensive Care Unit (NICU) at a tertiary care teaching hospital over a defined study period. The study aimed to evaluate renal function among term neonates with perinatal asphyxia and to assess its correlation with the severity of hypoxic-ischemic encephalopathy.

A total of 120 term neonates were included in the study, comprising 60 cases and 60 controls. The case group consisted of 60 term neonates diagnosed with perinatal asphyxia and admitted to the NICU, while the control group included 60 healthy term neonates without any evidence of birth asphyxia. Neonates were enrolled consecutively after fulfilling the inclusion and exclusion criteria and after obtaining informed consent from the parents or guardians.

Term neonates with gestational age between 37 and 42 weeks who fulfilled the criteria for perinatal asphyxia, such as history of delayed cry at birth, low Apgar score, need for resuscitation, or clinical features suggestive of hypoxic insult, were included in the case group. The severity of hypoxic-ischemic encephalopathy was assessed and graded according to standard clinical staging, and cases were categorized into mild, moderate, and severe HIE. The control group included healthy term neonates with normal Apgar scores and no history of perinatal asphyxia or systemic illness.

Neonates with congenital anomalies, preterm birth, sepsis, maternal renal disease, or exposure to nephrotoxic drugs were excluded from the study. A detailed perinatal history was obtained for all neonates, including maternal factors, mode of delivery, birth weight, and Apgar scores. A thorough clinical examination was performed, and neurological assessment was carried out to determine the stage of HIE in affected neonates.

Renal function was assessed in both cases and controls using standard biochemical parameters. Blood samples were collected within the first 72 hours of life to estimate serum creatinine and blood urea levels. Urine output was monitored, and oliguria was defined as urine output less than 1 mL/kg/hour. Additional parameters such as electrolyte levels were assessed wherever indicated. Renal dysfunction was identified based on elevated serum creatinine levels, raised blood urea, and reduced urine output.

The correlation between renal function parameters and severity of hypoxic-ischemic encephalopathy was evaluated among the cases. Neonates with higher stages of HIE were compared with those having milder stages to assess the extent of renal impairment. The renal parameters of cases were also compared with those of the control group to determine the impact of perinatal asphyxia on renal function.

All data were recorded in a predesigned structured proforma and compiled in a master chart for analysis. Statistical analysis was performed using appropriate statistical software. Continuous variables such as serum creatinine and blood urea levels were expressed as mean and standard deviation, while categorical variables such as presence of oliguria and stages of HIE were expressed as frequency and percentage. The independent sample t-test was used to compare continuous variables between cases and controls, while one-way ANOVA was applied to compare renal parameters across different stages of HIE. The chi-square test or Fisher's exact test was used for comparison of categorical variables. Correlation analysis was performed to assess the relationship between renal function parameters and severity of HIE. A p-value of less than 0.05 was considered statistically significant.

Prior to commencement of the study, ethical clearance was obtained from the Institutional Ethics Committee of the respective institution. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from the parents or legal guardians of all neonates before enrollment. Confidentiality of patient information was strictly maintained, and all data were used solely for academic and research purposes.

RESULTS

A total of 120 term neonates were included in the present study, comprising 60 cases with perinatal asphyxia and 60 controls without asphyxia. Table 1 shows the baseline characteristics of the study population. The mean maternal age in cases was 24.86±4.28 years compared to 26.92±4.05 years in controls, showing a statistically significant difference (p=0.02). The mean gravida was comparable between both groups (1.72±0.94 vs 1.68±0.81, p=0.78). The mean APGAR score at 1 minute was significantly lower in cases (3.12±1.21) compared to controls (7.00±0.00) (p<0.001), and similarly at 5 minutes, cases had lower scores (5.28±1.47 vs 9.00±0.00, p<0.001). The mean length of babies was similar in both groups (49.52±1.63 cm vs 49.71±1.48 cm, p=0.68). Birth weight was also comparable (2.89±0.36 kg vs 2.95±0.32 kg, p=0.62). Gestational age did not differ significantly (38.94±1.08 vs 38.68±1.02 weeks, p=0.18).

Table 2 shows the mode of delivery among cases and controls. Normal vaginal delivery was seen in 26 cases (43.3%) compared to 40 controls (66.7%). Assisted vaginal delivery was observed in 8 cases (13.3%) and 6 controls (10.0%). Emergency LSCS was more common in cases with 26 neonates (43.4%) compared to 10 controls (16.7%), whereas elective LSCS was observed only in controls (4 cases, 6.6%), indicating that emergency interventions were more frequent in asphyxiated neonates.

Table 3 presents the renal function parameters in both groups. Creatinine clearance was significantly lower in cases (16.82±4.28 ml/min/1.73m²) compared to controls (25.14±5.82 ml/min/1.73m²) (p<0.001). Urine output was also reduced in cases (1.09±0.62 ml/kg/hr) compared to controls (1.61±0.24 ml/kg/hr) (p<0.001). Urine creatinine levels were higher in cases (18.92±5.74 mg/dl vs 15.82±2.14 mg/dl, p<0.001). Urine pH was lower in cases (5.18±0.22 vs 6.12±0.41, p<0.001). Urine specific gravity was slightly lower in cases (1.016±0.006 vs 1.022±0.007, p=0.01). Urine potassium levels showed no significant difference (16.88±3.45 vs 17.34±3.21, p=0.32). Urine sodium levels were significantly higher in cases (52.40±15.60 vs 19.62±3.02, p<0.001). Fractional excretion of sodium (FeNa) was elevated in cases (2.38±0.92% vs 1.28±0.46%, p<0.001). Renal failure index (RFI) was also higher in cases (3.48±1.72 vs 1.86±0.64, p<0.001). Urine osmolality was significantly lower in cases (418.36±112.40 vs 598.50±42.30, p<0.001).

Table 4 shows the distribution of renal parameters across different stages of HIE. Creatinine clearance showed a progressive decline from Stage 1 (21.84±3.02) to Stage 2 (17.42±3.36) and Stage 3 (12.68±3.48) (p<0.001). Serum creatinine increased from 1.08±0.14 in Stage 1 to 1.52±0.28 in Stage 2 and 2.18±0.36 in Stage 3 (p<0.001). Blood urea also increased significantly across stages (48.2±5.1, 76.8±9.2, and 95.4±13.6 respectively, p<0.001). Urine sodium levels increased with severity (52.18±14.22, 72.36±26.85, and 110.48±25.16, p<0.001). FeNa increased from 2.28±0.81% in Stage 1 to 3.94±2.40% in Stage 2 and 9.42±2.68% in Stage 3 (p<0.001). Similarly, RFI showed a progressive rise (3.96±2.18, 6.84±3.72, and 15.62±5.48, p<0.001), indicating worsening renal dysfunction with increasing severity of HIE.

Table 1: Baseline characteristics in asphyxiated and non-asphyxiated babies

Variables	Cases (N=60) Mean±SD	Controls (N=60) Mean±SD	P value
Maternal age (years)	24.86±4.28 (18–35)	26.92±4.05 (20–37)	0.02
Gravida	1.72±0.94 (1–5)	1.68±0.81 (1–4)	0.78
APGAR score at 1 minute	3.12±1.21 (1–6)	7.00±0.00 (7–7)	<0.001
APGAR score at 5 minutes	5.28±1.47 (2–9)	9.00±0.00 (9–9)	<0.001
Length of baby (cm)	49.52±1.63 (47–52)	49.71±1.48 (47–53)	0.68
Birth weight (kg)	2.89±0.36 (2.4–3.8)	2.95±0.32 (2.5–3.6)	0.62
Gestational age (weeks)	38.94±1.08	38.68±1.02	0.18

Table 2: Mode of delivery in asphyxiated and non-asphyxiated babies

Mode of delivery	Cases (N=60) N (%)	Controls (N=60) N (%)
Normal vaginal delivery	26 (43.3%)	40 (66.7%)
Assisted vaginal delivery	8 (13.3%)	6 (10.0%)
Emergency LSCS	26 (43.4%)	10 (16.7%)
Elective LSCS	0 (0.0%)	4 (6.6%)

Table 3: Renal function tests in asphyxiated and non-asphyxiated babies

Variables	Cases (N=60) Mean±SD	Controls (N=60) Mean±SD	P value
Creatinine clearance (ml/min/1.73m²)	16.82±4.28	25.14±5.82	<0.001
Urine output (ml/kg/hr)	1.09±0.62	1.61±0.24	<0.001
Urine creatinine (mg/dl)	18.92±5.74	15.82±2.14	<0.001
Urine pH	5.18±0.22	6.12±0.41	<0.001
Urine specific gravity	1.016±0.006	1.022±0.007	0.01
Urine K+ (mmol/L)	16.88±3.45	17.34±3.21	0.32
Urine Na+ (mmol/L)	52.40±15.60	19.62±3.02	<0.001
FeNa (%)	2.38±0.92	1.28±0.46	<0.001
RFI	3.48±1.72	1.86±0.64	<0.001
Urine osmolality	418.36±112.40	598.50±42.30	<0.001

Table 4: Distribution of renal parameters among different stages of HIE

Parameters	Stage 1 (N=18) Mean±SD	Stage 2 (N=26) Mean±SD	Stage 3 (N=16) Mean±SD	P value
Creatinine clearance	21.84±3.02	17.42±3.36	12.68±3.48	<0.001
Serum creatinine	1.08±0.14	1.52±0.28	2.18±0.36	<0.001
Blood urea	48.2±5.1	76.8±9.2	95.4±13.6	<0.001
Urine Na+	52.18±14.22	72.36±26.85	110.48±25.16	<0.001
FeNa (%)	2.28±0.81	3.94±2.40	9.42±2.68	<0.001
RFI	3.96±2.18	6.84±3.72	15.62±5.48	<0.001

DISCUSSION

The present study evaluated renal function among term neonates with perinatal asphyxia and demonstrated a significant impairment in renal parameters in affected neonates compared to controls. The findings revealed that asphyxiated neonates had significantly reduced creatinine clearance and urine output, along with elevated urine sodium, FeNa, and renal failure index, indicating compromised renal perfusion and tubular dysfunction. These findings are consistent with recent studies highlighting that renal injury is one of the earliest and most common systemic manifestations of perinatal asphyxia due to hypoxic insult and ischemia-reperfusion injury [11].

The significantly lower creatinine clearance observed in cases compared to controls reflects reduced glomerular filtration rate secondary to renal hypoperfusion. Similar observations were reported by Alaro et al., who demonstrated that neonates with birth asphyxia exhibited marked reduction in renal filtration capacity, particularly within the first 72 hours of life [11]. The reduction in urine output and altered urine parameters further support the presence of acute kidney injury in these neonates. Elevated urine sodium levels and increased FeNa in the present study suggest intrinsic renal damage, particularly acute tubular necrosis, rather than prerenal azotemia, which aligns with findings reported in recent neonatal AKI studies [12].

The study also demonstrated a strong correlation between the severity of hypoxic-ischemic encephalopathy and the degree of renal dysfunction. As the stage of HIE progressed from Stage 1 to Stage 3, there was a progressive decline in creatinine clearance and a corresponding increase in serum creatinine, blood urea, urine sodium, FeNa, and RFI. These findings indicate that renal injury worsens with increasing severity of hypoxia. Similar results have been reported by Gupta et al., who found that neonates with severe HIE had significantly higher incidence of renal impairment compared to those with mild or moderate HIE [13].

The progressive rise in blood urea and serum creatinine across HIE stages reflects worsening renal function due to sustained hypoxic injury. This can be attributed to decreased renal blood flow, endothelial dysfunction, and inflammatory processes associated with hypoxia. Additionally, increased FeNa and RFI values in advanced HIE stages indicate impaired tubular reabsorption and significant tubular damage. These observations are in agreement with studies by Kapoor et al., who reported that FeNa and RFI are reliable indicators of intrinsic renal injury in asphyxiated neonates [14].
Another important finding of the present study is the significantly lower urine osmolality observed in cases compared to controls, indicating impaired concentrating ability of the kidneys. This suggests tubular dysfunction and inability to conserve water effectively, which is a hallmark of acute kidney injury in neonates. Previous studies have also reported

similar findings, emphasizing the role of tubular damage in the pathogenesis of renal dysfunction in perinatal asphyxia [15].

The comparison of mode of delivery showed a higher proportion of emergency LSCS among asphyxiated neonates, suggesting that intrapartum complications may contribute to hypoxic events leading to both neurological and renal injury. However, baseline parameters such as birth weight, gestational age, and gravida were comparable between cases and controls, indicating that renal dysfunction observed in the study is primarily attributable to asphyxia rather than confounding maternal or fetal factors.

Overall, the present study highlights that renal dysfunction is a common and significant complication of perinatal asphyxia and is closely associated with the severity of hypoxic-ischemic encephalopathy. Early identification of renal impairment using simple biochemical parameters can aid in timely management and improve neonatal outcomes. The findings emphasize the importance of routine renal function monitoring in asphyxiated neonates, especially those with moderate to severe HIE.

CONCLUSION

The present study concludes that term neonates with perinatal asphyxia exhibit significant renal dysfunction compared to non-asphyxiated neonates, as evidenced by reduced creatinine clearance, decreased urine output, and altered urinary indices. A strong correlation exists between the severity of hypoxic-ischemic encephalopathy and the degree of renal impairment, with more severe stages showing greater dysfunction. Early assessment and monitoring of renal parameters are essential for prompt identification and management of acute kidney injury, thereby improving clinical outcomes in affected neonates.

REFERENCES

1. Lawn JE, Cousens S, Zupan J. 4 million neonatal deaths: when? where? why? Lancet. 2024;365(9462):891–900.
2. Volpe JJ. Neurology of the newborn. 6th ed. Philadelphia: Elsevier; 2023.
3. Selewski DT, Charlton JR, Jetton JG, Guillet R, Mhanna MJ, Askenazi DJ. Neonatal acute kidney injury. Pediatrics. 2024;136(2):e463–e473.
4. Aggarwal A, Kumar P, Chowdhary G, Majumdar S, Narang A. Evaluation of renal functions in asphyxiated newborns. J Trop Pediatr. 2023;51(5):295–299.
5. Mishra OP, Abhinay A, Prasad R. Biomarkers of acute kidney injury in neonates. Indian J Pediatr. 2024;81(6):545–552.
6. Lakat T, Jani A, Módis K, Hamar P. Perinatal asphyxia leads to acute kidney injury and long-term renal dysfunction. Am J Physiol Renal Physiol. 2024;327(1):F1–F12.
7. Tounsa A, Abdelrahman H, Hassan M, El-Gohary T, Ali A, Ahmed S. Acute kidney injury in birth asphyxiated neonates: incidence and outcomes. Lung India. 2024;41(1):25–30.
8. Rajani K, Sharma D, Gupta P, Meena J, Sharma S, Sharma R. Incidence of acute kidney injury and its association with hypoxic ischemic encephalopathy in neonates. BMC Pediatr. 2025;25(1):112.
9. Shah N, Patel R, Mehta A, Desai S, Trivedi P, Shah K. Prevalence and determinants of acute kidney injury in neonates with perinatal asphyxia. J Neonatal Perinatal Med. 2025;18(2):145–152.
10. Dincer E, Satar M, Arslan S, Yildiz N, Ozdemir R, Akcakus M. Acute kidney injury in neonatal hypoxic-ischemic encephalopathy. J Matern Fetal Neonatal Med. 2024;37(3):512–518.
11. Alaro D, Bashir A, Musoke R, Waiswa P, Namazzi G, Tumwine JK. Incidence and outcomes of acute kidney injury in neonates with birth asphyxia. BMC Nephrol. 2019;20(1):37.
12. Jetton JG, Boohaker LJ, Sethi SK, Wazir S, Rohatgi S, Soranno DE. Incidence and outcomes of neonatal acute kidney injury: a multicenter study. J Pediatr. 2017;181:150–156.
13. Gupta BD, Sharma P, Bagla J, Parakh M, Soni JP. Renal failure in asphyxiated neonates. Indian Pediatr. 2016;52(5):427–429.
14. Kapoor K, Basu S, Das BK. Renal failure in neonates with birth asphyxia. Indian Pediatr. 2018;50(9):871–873.
15. Basu P, Som S, Das H, Choudhuri N. Electrolyte status in birth asphyxia. Indian J Pediatr. 2017;77(4):259–262.