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Original Research Article

To Assess Cord Blood Electrolyte and Glucose Level in Birth Asphyxia and their Correlation with Severity of Asphyxia

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Abstract:
Background: Birth asphyxia, a leading cause of neonatal morbidity and mortality, disrupts oxygenation and causes metabolic derangements. Electrolyte imbalances—sodium, potassium, calcium and hypoglycemia correlate with severity, impacting organ function and neurological outcomes. Cord blood analysis offers early detection, aiding timely intervention and improved management of affected neonates.
Aim: to assess cord blood electrolyte and glucose level in birth asphyxia and their correlation with severity of asphyxia
Methods: Neonates meeting inclusion criteria were enrolled after parental consent. Cord blood (2 ml) was collected within 1 minute of birth for glucose and electrolyte analysis. Clinical data, birth history, and Apgar scores were recorded. Asphyxiated neonates were staged for HIE severity using Sarnat and Sarnat criteria, and biochemical and clinical outcomes were documented.
Results: Among 75 cases and 75 controls, males predominated (68% vs. 64%). Cases showed lower sodium (134.6 ± 4.27 vs. 137.73 ± 3.89 mEq/L), calcium (8.01 ± 0.63 vs. 9.02 ± 0.49 mg/dL), glucose (65.49 ± 9.45 vs. 81.82 ± 10.39 mg/dL) and higher potassium (5.15 ± 0.76 vs. 4.27 ± 0.35 mEq/L), all $p < 0.0001$. Biochemical disturbances correlated with HIE severity: sodium ($r = -0.51$), potassium ($r = 0.39$), calcium ($r = -0.78$), glucose ($r = -0.56$).
Conclusion: Birth asphyxia causes significant disturbances in cord blood electrolytes and glucose, correlating with hypoxic-ischemic encephalopathy severity. Sodium, calcium, and glucose decrease, while potassium rises with worsening asphyxia. Monitoring these parameters enables early detection, guides timely intervention, and supports effective management of affected neonates, potentially reducing complications and improving short- and long-term outcomes
Keywords: Cord Blood Electrolyte, Birth Asphyxia, Correlation With Severity, National Neonatology Forum.

Introduction

Birth asphyxia, defined as "the failure to establish breathing at birth," remains a leading cause of neonatal morbidity and mortality worldwide. Severity and outcomes are predicted by time to first breath, heart rate at 90 seconds, duration of resuscitation, and Apgar score at five minutes. [1] According to the National Neonatology Forum (NNF) of India, it is defined as "gasping and ineffective breathing or lack of breathing at one minute after birth." Sustained hypoxia leads to perinatal asphyxia causing hypoxic-ischemic injury to organs such as kidneys (50%), brain (28%), heart (25%), lungs (23%), liver, and intestines. Long-term complications include cerebral palsy, persistent pulmonary hypertension, renal cortical necrosis, hypotension, cardiogenic shock, or neonatal death. [2]

Globally, birth asphyxia accounts for ~4 million neonatal deaths and 3.2 million stillbirths annually, with perinatal hypoxia contributing to 23% and 29%, respectively. In India, it accounts for 28.8% of neonatal deaths and 45.5% of stillbirths, with 8% and 2% of newborns showing Apgar <7 at one and five minutes. The Apgar score, developed by Dr. Virginia Apgar in 1952, assesses skin color, heart rate, reflex irritability, muscle tone, and respiratory effort (0–2 each; total 10), guiding early risk assessment. [3]

Electrolytes—sodium, potassium, and calcium—are crucial for cellular homeostasis, nerve transmission, muscle contraction, and enzymatic activities, with calcium particularly important for myocardial function and clotting. [4] Neonates face challenges maintaining electrolyte balance due to immature renal function and sudden extrauterine transition. Birth asphyxia also causes hypoglycemia due to glycogen depletion and hyperinsulinemia, with anaerobic metabolism producing lactic acidosis. [5] Electrolyte disturbances include hyperkalemia, hyponatremia, and hypocalcemia.

Cord blood offers immediate, real-time assessment of these metabolic changes, providing a reliable indicator of neonatal status at birth. Early evaluation of glucose and electrolytes can guide timely interventions, prevent complications, and improve outcomes.

The aim of the study is to Assess Cord Blood Electrolyte and Glucose Level in Birth Asphyxia and their Correlation with Severity of Asphyxia

Materials and Methods

Study Design: Observational study.

Study Type: Hospital-based observational study.

Study Duration: From ethical committee approval until completion of required sample size.

Study Location: Department of Paediatric Medicine, Sir Padam Pat Institute of Neonatal and Paediatric Health (SPINPH), including affiliated hospitals: Zenana Hospital, Gangori Hospital, HB Kanwatiya Hospital, Mahila Chikitsalaya.

Sample Size: Calculated based on Gandhi KC et al. (2020) with 27% birth asphyxia prevalence, 95% confidence interval, 10% margin of error; final sample size = 75

- Formula: Sample Size = $Z^2 \times p(1-p) / E^2$
- Z = 1.96, Error (E) = 10%, Calculated size = 74.8 → rounded to 75

Inclusion Criteria: The inclusion criteria for this study comprised two groups. The case group included neonates with a gestational age of 37 to less than 42 weeks who either had an Apgar score of less than 7 at 5 minutes or required more than 1 minute of positive pressure ventilation. The control group consisted of healthy neonates without signs of asphyxia, with an Apgar score greater than 7 at 1 minute, normal fetal heart rate, and clear amniotic fluid.

Exclusion Criteria: Exclusion criteria included neonates with congenital anomalies, those born to mothers with metabolic disorders, alcohol or tobacco use, or taking antiepileptic drugs, as well as cases of maternal drug abuse and parents who did not provide consent for participation in the study.

Methodology: Neonates meeting inclusion criteria at SPINPH-affiliated hospitals were enrolled after obtaining parental consent. Data on birth weight, sex, maternal history, Apgar score, meconium staining, birth events, and resuscitation were recorded. Cord blood (2 ml) was collected for glucose and electrolyte analysis. Asphyxiated neonates were monitored for HIE using Sarnat staging, with clinical course, lab results, and outcomes documented.

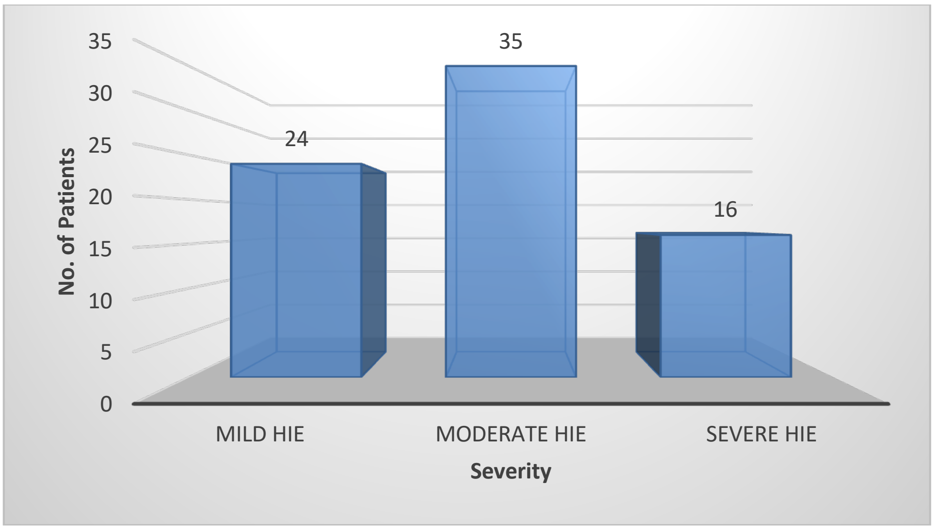
Results and observations

Among 75 cases and 75 controls, males predominated (68% vs. 64%), with no significant gender difference (p = 0.6). Birth weights were comparable: in cases, 4% <2500 g, 46.7% 2500–3000 g, 49.3% >3000 g; in controls, 6.7%, 40%, and 53.3%, respectively. Mean weights were 3003.7 ± 350.4 g (cases) and 2951.1 ± 297.4 g (controls), p = 0.32.

Table 1: Comparison of Biochemical Parameters Between Case and Control Groups.

Parameter	Case Group		Control Group		P-Value
	Mean	SD	Mean	SD	
Sodium (mEq/L)	134.6	4.27	137.73	3.89	<0.0001
Potassium (mEq/L)	5.15	0.76	4.27	0.35	<0.0001
Calcium (mg/dL)	8.009	0.63	9.02	0.49	<0.0001
Glucose (mg/dL)	65.49	9.45	81.82	10.39	<0.0001

Biochemical analysis showed significant differences between groups. Mean serum sodium was lower in cases (134.6 ± 4.27 mEq/L) versus controls (137.73 ± 3.89 mEq/L, p <0.0001). Potassium was higher in cases (5.15 ± 0.76 mEq/L) than controls (4.27 ± 0.35 mEq/L, p <0.0001). Calcium (8.009 ± 0.63 mg/dL vs 9.02 ± 0.49 mg/dL) and glucose (65.49 ± 9.45 mg/dL vs 81.82 ± 10.39 mg/dL) were lower in cases, all statistically significant, indicating marked metabolic disturbances.



Graph 1: Distribution of HIE Severity Among Cases

In the case group, 32% (24 patients) had mild HIE, 46.67% (35 patients) moderate, and 21.33% (16 patients) severe, indicating most cases were moderate, followed by mild and severe, reflecting a varied clinical spectrum of HIE severity.

Table 2: Severity-wise Distribution of Biochemical Parameters in HIE Cases.

Severity	Case Group							
	Sodium (mEq/L)		Potassium (mEq/L)		Calcium (mg/dL)		Glucose (mg/dL)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Mild HIE	137.7	2.53	4.83	0.5	8.61	0.5	70.95	6.62
Moderate HIE	133.75	4.23	5.13	0.8	7.94	0.26	66.25	9.04
Severe HIE	131.8	3.77	5.69	0.76	7.23	0.47	55.65	5.95
P-Value	0.0001		0.0015		0.0001		0.0001	

Biochemical parameters worsened with increasing HIE severity. Mild HIE: sodium 137.7 ± 2.53, potassium 4.83 ± 0.5, calcium 8.61 ± 0.5, glucose 70.95 ± 6.62. Moderate HIE: sodium 133.75 ± 4.23, potassium 5.13 ± 0.8, calcium 7.94 ± 0.26, glucose 66.25 ± 9.04. Severe HIE: sodium 131.8 ± 3.77, potassium 5.69 ± 0.76, calcium 7.23 ± 0.47, glucose 55.65 ± 5.95, all p <0.05.

Table 3: Pearson Correlation Between HIE Severity and Serum Sodium, Potassium, Calcium, and Glucose Levels.

Pearson Correlation	Severity	
Severity	**r-value**	**p-value**
Sodium (mEq/L)	-0.51	<0.0001
Potassium (mEq/L)	0.39	0.0005
Calcium (mg/dL)	-0.78	<0.0001
Glucose (mg/dL)	-0.56	<0.0001

The Pearson correlation analysis between serum electrolytes, glucose, and the severity of Hypoxic-Ischemic Encephalopathy (HIE) revealed significant associations. Serum sodium showed a moderate negative correlation with HIE severity ($r = -0.51$, $p < 0.0001$), while serum potassium had a moderate positive correlation ($r = 0.39$, $p = 0.0005$). Serum calcium demonstrated a strong negative correlation ($r = -0.78$, $p < 0.0001$), and serum glucose exhibited a moderate-to-strong negative correlation ($r = -0.56$, $p < 0.0001$), indicating that worsening HIE is linked to decreasing sodium, calcium, and glucose, but increasing potassium levels.

Discussion

Birth asphyxia is a critical perinatal condition contributing to neonatal morbidity and mortality. [6] It can cause hypoxic-ischemic brain injury and biochemical disturbances in glucose, calcium, sodium, and potassium. Cord blood analysis aids early detection, guiding electrolyte management to improve outcomes, prevent brain injury, and reduce long-term complications. [7]

The study observed lower sodium (134.6 ± 4.27 vs. 137.73 ± 3.89 mEq/L, $p < 0.0001$), calcium (8.009 ± 0.63 vs. 9.02 ± 0.49 mg/dL, $p < 0.0001$), and glucose (65.49 ± 9.45 vs. 81.82 ± 10.39 mg/dL, $p < 0.0001$) in cases, with higher potassium (5.15 ± 0.76 vs. 4.27 ± 0.35 mEq/L, $p < 0.0001$). Kavya M. Y et al [8] reported sodium 122.1 ± 6.0 and calcium 6.85 ± 0.95 mg/dL; Bahatkar K et al [9] found sodium 130.52 ± 5.27, calcium 8.96 ± 1.08 mg/dL, and glucose 43.68 ± 10.81 mg/dL.

Pearson correlation analysis showed serum sodium ($r = -0.51$, $p < 0.0001$) and calcium ($r = -0.78$, $p < 0.0001$) negatively correlated, and potassium ($r = 0.39$, $p = 0.0005$) positively correlated with HIE severity. Datta S et al [10] reported a significant negative correlation between sodium and HIE grades ($p < 0.05$), while Bahatkar K et al [9] found significant differences in sodium, calcium, and glucose levels ($p < 0.05$).

Biochemical disturbances in HIE correlate with severity. Bahatkar K et al [9] reported sodium decreasing from 136 ± 1.4 to 126.71 ± 6 mEq/L ($r = -0.591$, $p < 0.0001$) and calcium declining ($r = -0.484$, $p = 0.001$). Kavya M. Y et al [8] observed sodium drop from 137.38 ± 3.23 to 126.25 ± 2.95 mEq/L, potassium rise to 5.30 ± 0.54 mEq/L, and significant calcium and glucose decline ($p = 0.001$).

Pearson correlation analysis showed serum sodium negatively correlated with HIE severity ($r = -0.51$, $p < 0.0001$), calcium strongly negatively correlated ($r = -0.78$, $p < 0.0001$), potassium positively correlated ($r = 0.39$, $p = 0.0005$), and glucose negatively correlated ($r = -0.56$, $p < 0.0001$). Bahatkar K et al [9] reported significant differences in sodium, calcium, and glucose between cases and controls ($p < 0.05$), supporting these correlations. Datta S et al [10] also observed a significant negative correlation between sodium and HIE grades ($p < 0.05$).

Conclusion

Birth asphyxia causes significant disturbances in cord blood electrolytes and glucose, correlating with HIE severity. With increasing severity, sodium, calcium, and glucose decline, while potassium rises, reflecting metabolic derangements. Monitoring these parameters can serve as important indicators of asphyxia severity, enabling early recognition and timely intervention to improve neonatal outcomes.

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