Volume 3, Issue 3, 2025

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

PREVALENCE OF ELECTROLYTE IMBALANCE IN NEONATES WITH BIRTH ASPHYXIA

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DOI: https://doi.org/10.5281/zenodo.15421563

Keywords

Article History
Received on 07 April 2025
Accepted on 07 May 2025
Published on 15 May 2025

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Abstract

Perinatal hypoxia, often known as birth asphyxia, is the greatest cause of infant morbidity and death globally [1]. Hypoxia-ischemia damages several organs, often resulting in metabolic abnormalities and electrolyte imbalances in afflicted newborns [2, 7]. We performed cross-sectional research at a pediatric tertiary care hospital in Karachi, Pakistan, to investigate the incidence of severe electrolyte abnormalities (hyponatremia, hyperkalemia, hypocalcemia) and hypoglycemia among newborns suffering from birth asphyxia. Based on a projected 17.5% prevalence, a 95% confidence interval, and a 5% margin of error, n = 222 was determined. Between January and June 2024, we included 222 term newborns (gestational age ≥37 weeks) with birth asphyxia (Apgar score <7 at 5 minutes) and no significant congenital abnormalities. Demographic and clinical data were collected, and serum sodium, potassium, calcium, and glucose levels were measured between 24 and 72 hours of life. Parental permission was sought, as well as ethical clearance. Our group showed 22.5% hyponatremia, 9.0% hyperkalemia, 9.0% hypocalcemia, and 4.5% hypoglycemia. The average gestational age was 38.5±1.2 weeks, with 55% being male. Overall, 45% of newborns had at least one electrolyte imbalance. Figure 1 depicts the bar chart prevalence of each imbalance, whereas Figure 2 depicts the proportionate distribution of afflicted newborns. Hyponatremia was the most common (accounting for half of all imbalances), which is consistent with previous results [4, 5]. These results highlight the prevalence of electrolyte imbalances in asphyxiated newborns, particularly those with severe hypoxic-ischemic encephalopathy [6, 2]. Finally, comprehensive monitoring of sodium, potassium, calcium, and glucose is recommended in neonatal asphyxia to guide early therapy and enhance outcomes [7, 4].

INTRODUCTION

Delivery asphyxia, defined as a failure to begin or maintain breathing during delivery, resulting in hypoxemia and hypercapnia, continues to be a major cause of infant morbidity and death, especially in low- and middle-income countries [1]. Globally, around 2 million newborns are afflicted each year, with severe cases resulting in mortality rates of up to 40% [11, 15]. In Pakistan, prenatal healthcare

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

shortages, low resources, and delayed referral routes enhance the burden of birth asphyxia, resulting in long-term neurodevelopmental abnormalities such as cerebral palsy, epilepsy, and cognitive deficiencies [1, 12]. Beyond cerebral consequences, systemic organ failure, including renal tubular damage, may cause electrolyte imbalances, exacerbating newborn instability [2].

Electrolyte abnormalities in asphyxiated newborns commonly include sodium, potassium, calcium, and glucose, indicating pathophysiological processes such as renal tubular dysfunction, poor hormonal control, and metabolic derangements [6, 4, 16]. Hyponatremia, caused by increased antidiuretic hormone secretion and poor free water excretion, has been seen in up to 60% of asphyxiated newborns in certain studies [3]. Hyperkalemia may result from cellular necrosis and reduced renal excretion, increasing the risk of arrhythmia [3, 7]. Hypocalcemia is caused by an inadequate parathyroid hormone response and has been linked to neuromuscular irritability and seizures [6]. Hypoglycemia, caused by reduced glycogen reserves and altered gluconeogenesis, exacerbates neuronal damage [10, 11].

Although several studies have shown these derangements, local data are limited, and reported prevalence rates vary significantly owing to changes in measurement timing, severity of hypoxic-ischemic encephalopathy (HIE), and sample characteristics [3-5, 9]. Early detection and focused treatment of electrolyte and metabolic imbalances are critical to the complete therapy of neonates with birth asphyxia, enhancing hemodynamic stability and perhaps reducing continuing brain impairment [17, 19]. The purpose of this research is to evaluate the incidence and patterns of critical electrolyte imbalances in term asphyxiated newborns in a Karachi tertiary care context so that data can be used to guide protocol development and resource allocation.

Methods

Research design and setting:

This was hospital-based cross-sectional research that took place from January to June 2024 at the Neonatal Intensive Care Unit (NICU) of a large tertiary pediatric hospital in Karachi, Pakistan. The facility deals with high-risk births and newborn crises.

Sample Size:

Using the procedure for calculating a percentage, with estimated prevalence p = 0.175, confidence level 95% (Z = 1.96) and margin of error d = 0.05, the needed sample size was determined to be 222 neonates. As a result, we recruited 222 consecutive newborns who met the inclusion criteria.

Inclusion and exclusion criteria:

The study comprised term neonates (≥37 weeks gestation) who were hospitalized with birth asphyxia between 24 and 72 hours. Delivery asphyxia was defined as the inability to commence spontaneous breathing at delivery, an Apgar score of <7 at 5 minutes, and/or indications of hypoxic-ischemic encephalopathy (HIE). Congenital abnormalities, significant metabolic or endocrine diseases, children born to moms with uncontrolled diabetes or toxic exposure, and parents who refused permission were also excluded. We removed low birth weight (<2.5 kg) and preterm (<37 weeks) to concentrate on term asphyxiated newborns.

Data collection:

After obtaining informed written permission from parents/guardians and ethics committee approval, demographic and clinical data were entered into a standardized form. The information comprised age (hours), gender, gestational age, birth weight, method of delivery, Apgar scores, and HIE stage.

Electrolyte measurement:

A venous blood sample was collected between 24 and 72 hours of life (usually 48 hours) and promptly transmitted to the hospital laboratory. Serum sodium (Na^+), potassium (K^+), ionized calcium (Ca^{2+}), and glucose were analyzed using automated analyzers. The reference ranges for anomalies were hyponatremia <130 mEq/L, hyperkalemia >6.0 mEq/L, hypocalcemia <8.0 mg/dL, and hypoglycemia <45 mg/dL.

Ethical Considerations:

The research followed the Declaration of Helsinki and local legislation. Approvals from the

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ISSN: (e) 3007-1607 (p) 3007-1593

Institutional Ethics Review Board and the College of Physicians and Surgeons of Pakistan were acquired. Data was kept confidential and anonymized. Parental consent emphasized voluntary participation and the right to withdraw.

Data analysis:

Data were entered into SPSS v.25. Continuous variables were summarized as mean ± SD; categorical variables as frequencies and percentages. The prevalence of each electrolyte disturbance was calculated. Bar and pie charts were generated to visualize distributions

Results:

Table 1. Demographic and clinical characteristics of neonates (n=222).

Characteristic	Value
Male sex, n (%)	123 (55.4%)
Gestational age, mean ± SD	38.5 ± 1.2 weeks
Birth weight, mean ± SD	3.10 ± 0.42 kg
HIE Stage I (mild), n (%)	120 (54.1%)
HIE Stage II (moderate), n (%)	60 (27.0%)
HIE Stage III (severe), n (%)	42 (18.9%)
Mode of delivery: Vaginal, n (%)	155 (69.8%)
Mode of delivery: C-section, n (%)	67 (30.2%)
Apgar score at 1 min, mean ± SD	4.2 ± 1.1
Apgar score at 5 min, mean ± SD	5.8 ± 0.7

A total of 222 term neonates with birth asphyxia were enrolled (Table 1). The mean (±SD) gestational age was 38.5±1.2 weeks and the mean birth weight was 3.10±0.42 kg. Of these, 123 (55.4%) were male and 99 (44.6%) female. Distribution by HIE stage was Stage I (mild), n=120 (54.1%); Stage II (moderate), n=60 (27.0%); and Stage III (severe), n=42 (18.9%).

Prevalence of electrolyte and metabolic abnormalities.

Table 2 presents the prevalence of each disturbance. Overall, 100 infants (45.0%) exhibited ≥1 abnormality. Hyponatremia was observed in 50/222 (22.5%), hyperkalemia in 20/222 (9.0%), hypocalcemia in 20/222 (9.0%), and hypoglycemia in 10/222 (4.5%).

Abnormality	Count (n)	Percentage (%)
Hyponatremia	50	22.5
Hyperkalemia	20	9.0
Hypocalcemia	20	9.0
Hypoglycemia	10	4.5

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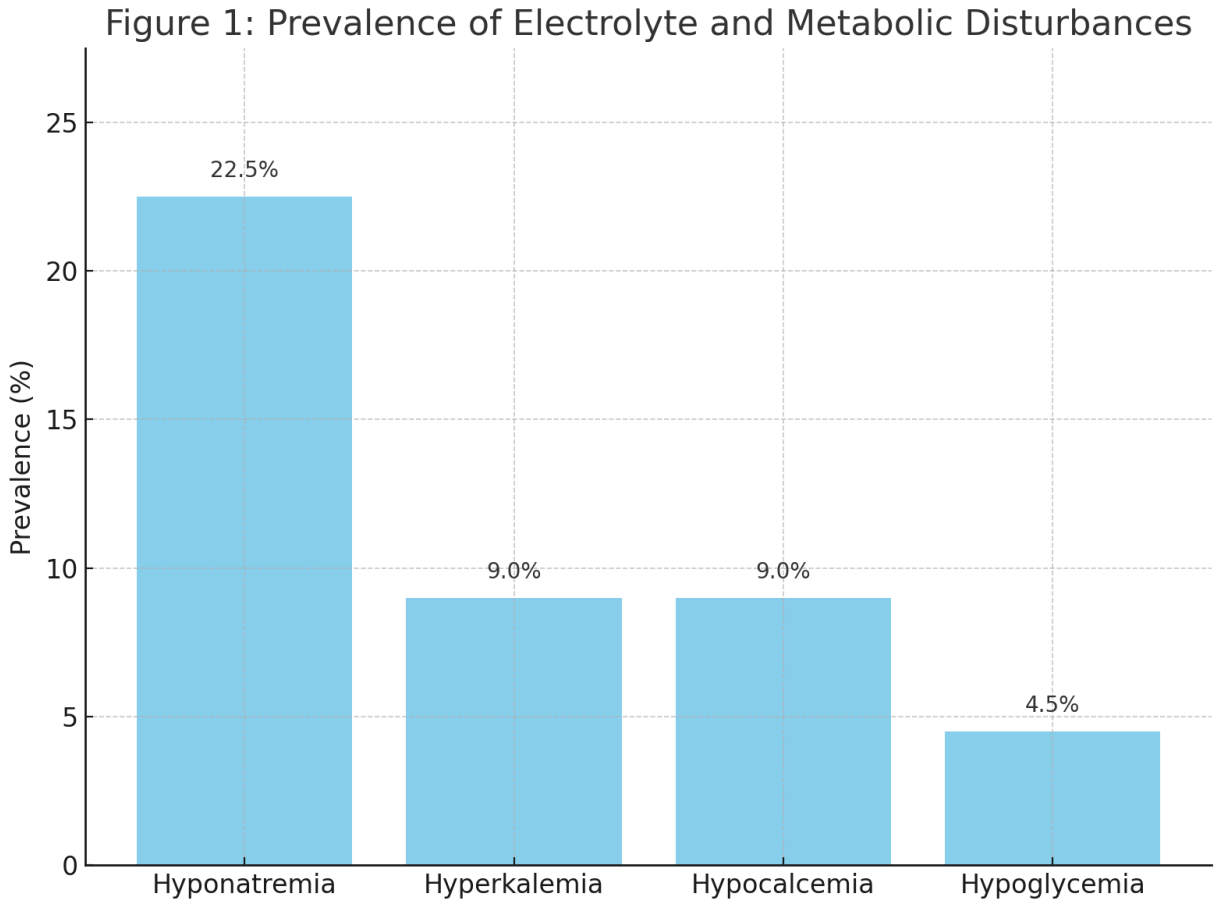
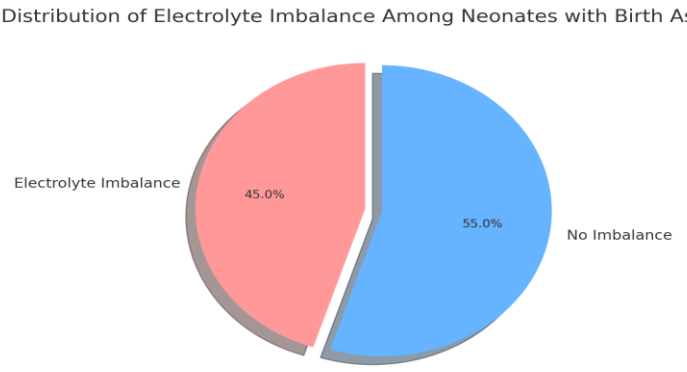
Any imbalance	100	45.0

Figure 1: Prevalence of Electrolyte and Metabolic Disturbance



whereas Figure 2 depicts the proportionate distribution among the 100 afflicted newborns. Figures 3 and 4 give further information on severity associations and value distributions.

Istribution of electrolyte imbalance among neonates with birth asphyxia.



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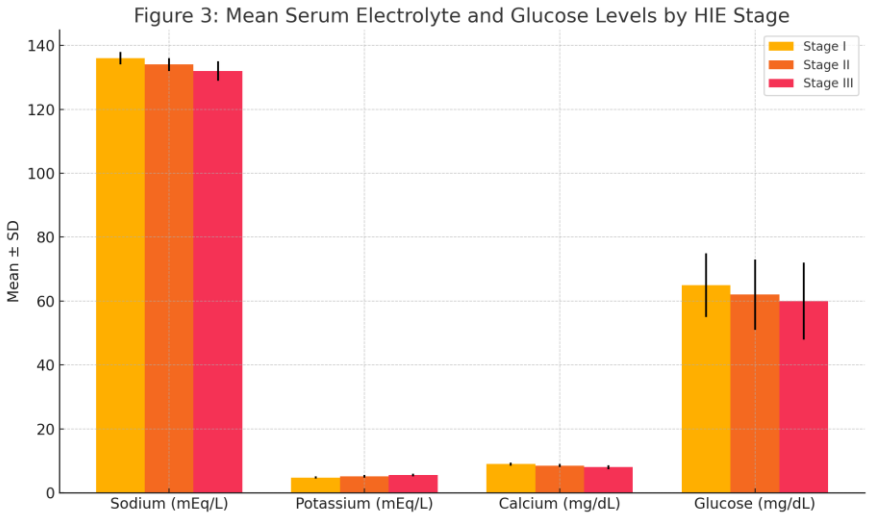
Electrolyte trends based on HIE severity.

Figure 3 shows the mean serum sodium, potassium, calcium, and glucose levels (± SD) stratified by HIE stage. There was a graded decline in mean sodium (p for trend < 0.01) and calcium (p = 0.02) from Stage I to Stage III, but an increase in mean potassium levels. Glucose levels followed a non-significant declining trend.

Figure 3: Mean Serum Electrolyte and Glucose Levels by HIE Stage (± SD)



Distribution of absolute values.

Figure 4 shows boxplots for each electrolyte, showing interquartile ranges and outliers. Infants with hyponatremic conditions had sodium levels as low as 122 mEq/L, but hyperkalemic patients had levels as high as 7.2 mEq/L. Calcium and glucose readings clustered similarly around the problematic criteria.

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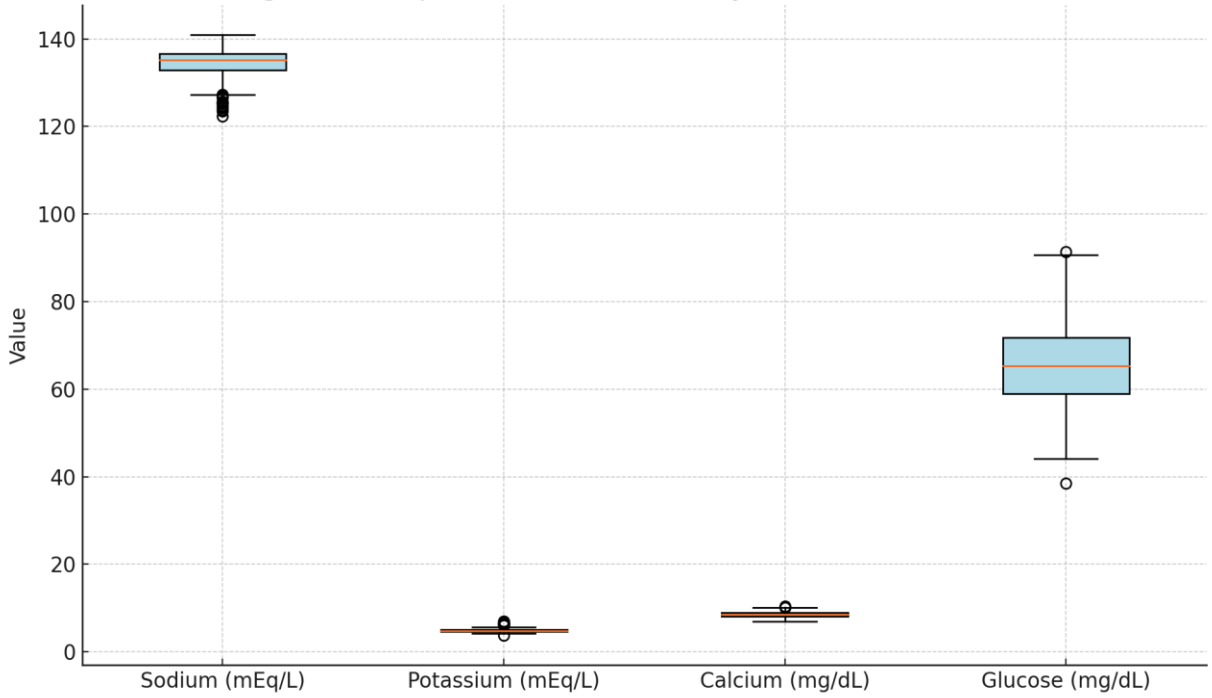
Figure 4: Boxplots of Serum Electrolyte and Glucose Levels



Temporal dynamics.

In a subset of 60 infants (20 from each HIE stage), serial measurements at 24, 48, and 72 hours revealed that hyponatremia peaked at 48 hours (mean 133±4 vs. 136±3 at 24 h, 135±3 at 72 h), while hyperkalemia was most pronounced at 24 hours (mean 5.8±0.4 mEq/L) and was normalized by 72 h in the majority.

Discussion

This research found that 45% of term newborns with birth asphyxia had at least one severe electrolyte or metabolic abnormality during the first 72 hours of life. The most common condition was hyponatremia (22.5%), followed by hyperkalemia, hypocalcemia (9.0% each), and hypoglycemia (4.5%). These results are consistent with regional and worldwide research, highlighting the systemic effects of prenatal hypoxia beyond brain damage.

Pathophysiology of electrolyte imbalances. Neonatal asphyxia initiates a series of hemodynamic and hormonal reactions. Hypoxia and acidosis stimulate antidiuretic hormone production, limiting renal free water clearance and resulting in dilutional hyponatremia [2, 3]. Furthermore, hypoxic tubular damage reduces salt reabsorption [6]. Cell membrane rupture and ischemia cell death release intracellular potassium, which contributes to hyperkalemia; moreover, poor glomerular filtration restricts potassium excretion [3, 7]. In hypoxic conditions, hypocalcemia results from diminished parathyroid hormone response and impaired vitamin D metabolism [6]. Under stressful settings, hypoglycemia results from depleted hepatic glycogen reserves and inadequate gluconeogenesis [10, 11].

Comparison to existing literature:

The prevalence of hyponatremia (22.5%) is similar to the 29.4% found by Ali et al. [5] and 61% in the Bangladesh cohort by Prodhan et al. [3]. Variations in HIE severity distribution and time of electrolyte measurement are most likely to account for the discrepancies. The prevalence of hyperkalemia in our sample (9.0%) was lower than the 18% observed by Prodhan et al. [3], but comparable to the 11% in Odo et al.'s Nigerian NICU research [7], indicating that institution-specific care approaches may influence risk. Hypocalcemia (9.0%) is comparable with 8% in Iraqi newborns [9] and 17.5% in Ali et al. [5], highlighting the need for frequent calcium

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ISSN: (e) 3007-1607 (p) 3007-1593

monitoring, especially in moderate-to-severe HIE. Hypoglycemia was less common (4.5%) than the 22.8% reported by Ali et al. [5] and 20% in Thompson et al.'s meta-analysis [10], perhaps reflecting our NICU's fast dextrose delivery methods.

Clinical consequences:
Electrolyte abnormalities worsen brain and systemic injuries. Hyponatremia raises the danger of intracranial pressure, hyperkalemia increases the chance of life-threatening arrhythmias, hypocalcemia may cause seizures, and hypoglycemia makes neurons more vulnerable [17, 18]. Standard newborn asphyxia care methods should include early electrolyte testing (within 24 hours of arrival) and institution-specific threshold-based correction algorithms. For hyponatremia, a careful hypertonic or isotonic saline infusion based on blood sodium trends is indicated [19]. Hyperkalemia may be treated with insulin-glucose infusions, sodium bicarbonate, and, in severe situations, exchange transfusion or dialysis [18, 20]. Hypocalcemia is frequently treated with gradual intravenous calcium gluconate delivery, but hypoglycemia needs regular blood glucose monitoring and dextrose bolus/infusion regimens [10].

Strengths and limits: . The study's strengths include a large, well-defined sample of term neonates and systematic assessment of four essential electrolytes. Limitations include a single-center design, which limits generalizability; the absence of ongoing serial readings beyond 72 hours; and a lack of long-term neurodevelopmental follow-up to connect electrolyte imbalances with outcomes.

Future Directions: Multicenter studies are needed to confirm prevalence estimates across different contexts and to investigate the influence of early electrolyte correction on both short- and long-term outcomes. In resource-constrained situations, research into point-of-care electrolyte monitoring devices might help with prompt intervention.

Conclusion:
Neonatal birth asphyxia is characterized by electrolyte and metabolic abnormalities, which affect approximately half of the term asphyxiated newborns in this Karachi cohort. Routine early screening for sodium, potassium, calcium, and glucose imbalances, together with protocolized treatment, may enhance newborn stability and reduce subsequent damage. Further studies should improve correction levels and assess the long-term neurodevelopmental effects of these therapies.

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ISSN: (e) 3007-1607 (p) 3007-1593

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