Section: Pediatrics

Original Research Article

CORRELATION OF SERUM IONIZED CALCIUM , SERUM SODIUM AND GLUCOSE ABNORMALITIES WITH SEVERITY OF HYPOXIC -ISCHEMIC ENCEPHALOPATHY IN NEONATES

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ABSTRACT

Background: Birth asphyxia is a major cause of neonatal morbidity and mortality, especially in developing countries like India. It frequently leads to hypoxic-ischemic encephalopathy (HIE), which is associated with significant neurological sequelae. Birth asphyxia is also known to cause metabolic disturbances such as hypocalcemia, hyponatremia, and hypoglycemia, which can worsen neonatal outcomes. This study was undertaken to evaluate the effect of birth asphyxia on serum ionized calcium, serum sodium, and serum glucose levels and to assess their association with the severity of birth asphyxia and stages of HIE. The aim is to study the effect of Birth asphyxia on serum ionized calcium, serum sodium and glucose levels. **Materials and Methods:** This hospital-based observational study was conducted on 60 term neonates diagnosed with birth asphyxia. Detailed maternal and neonatal data including gestational age, birth weight, gender, mode of delivery, Apgar scores, need for resuscitation, and clinical grading of asphyxia were recorded. HIE staging was done using the Sarnat-Sarnat classification. Serum ionized calcium, sodium, and glucose levels were measured within 24 hours of birth. Data were analyzed using SPSS software, and appropriate statistical tests were applied. A p-value <0.05 was considered statistically significant. **Result:** Among the studied neonates, 46.7% had HIE stage I, 36.7% had HIE stage II, and 16.6% had HIE stage III. Serum calcium, sodium, and glucose levels showed a significant decline with increasing severity of birth asphyxia and higher stages of HIE (p <0.001). Neonates with severe asphyxia and HIE stage III had the lowest mean biochemical values. No significant association was found between HIE severity and factors such as gender, mode of delivery, or booking status. **Conclusion:** Birth asphyxia is significantly associated with alterations in serum calcium, sodium, and glucose levels, and these abnormalities worsen with increasing severity of asphyxia and HIE stages. Early identification and prompt correction of these metabolic derangements may help improve neonatal outcomes and reduce complications.

INTRODUCTION

Neonatal morbidity and mortality remain major global health challenges, particularly in developing countries. Despite advances in obstetrics and perinatal care, the burden of neonatal deaths continues to be substantial, with India contributing significantly to global figures. Neonatal mortality constitutes the largest proportion of infant mortality and is widely regarded as a key indicator of a nation's overall health status.[1]

Perinatal asphyxia is a leading cause of neonatal morbidity and mortality. It is defined as a condition resulting from impaired gas exchange during labor and delivery, leading to fetal hypoxemia, hypercarbia, and metabolic acidosis.[2] WHO defines birth asphyxia as failure to initiate and sustain breathing , NNF defines birth asphyxia as Apgar scores of less than 7 at one minute of age wherein an Apgar 4-6 is considered moderate birth asphyxia and a score of 3 or lesser is considered severe birth asphyxia.[3] Severe cases may progress to hypoxic-ischemic encephalopathy (HIE), a serious neurological sequela associated with long-term

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neurodevelopmental impairment and significant socioeconomic burden.[4]

Birth asphyxia is frequently associated with multiple metabolic derangements, including hypoglycemia, hypocalcemia, hyponatremia, hyperphosphatemia, and metabolic acidosis.[5] Among these, glucose plays a critical role as the primary energy substrate for the brain. In neonates, serum glucose levels normally decline after birth and subsequently stabilize within a few hours.[6] However, in asphyxiated infants, hypoglycemia may occur due to glycogen depletion, increased metabolic demand, and altered hormonal regulation, including catecholamine surge and transient hyperinsulinemia.[7] These fluctuations can further exacerbate neurological injury if not promptly recognized and managed.[8] Calcium is another essential element involved in numerous physiological processes, including neuromuscular function and enzymatic activity. During fetal life, calcium is actively transported across the placenta, resulting in higher fetal calcium levels compared to maternal levels.[9] After birth, the sudden cessation of placental calcium transfer leads to a transient decline in serum calcium levels.[10] In neonates with perinatal asphyxia, this physiological adaptation may be disrupted, increasing the risk of hypocalcemia, which can contribute to neuromuscular irritability and seizures.[11]

Similarly, sodium balance is affected in asphyxiated neonates. While mild hypernatremia is expected in normal neonates due to postnatal fluid shifts, perinatal asphyxia is often associated with hyponatremia.[12] This is primarily due to increased secretion of antidiuretic hormone (ADH) in response to hypoxic stress, leading to water retention and dilutional hyponatremia.[13] Additional contributing factors include immature renal tubular function, limited sodium reabsorption capacity, and partial resistance to aldosterone.[14]

Given the high incidence of perinatal asphyxia and its associated metabolic complications, early identification and management of electrolyte and biochemical abnormalities are crucial in improving neonatal outcomes. Understanding the pattern and extent of these metabolic changes can aid in timely intervention and reduce the risk of both immediate and long-term complications.

MATERIALS AND METHODS

A cross-sectional observational study was conducted at T.S.Misra University in Lucknow to investigate the correlation between Serum Ionized calcium ,Serum sodium and glucose disturbances in all asphyxiated neonates born during period of 18 months, from 1st July 2024 to 31st December 2025. Before enrolling the patient, institutional ethical committee approval was taken. Informed consent was obtained from parents.

A minimum sample size of 60 neonates was achieved during the study period. Newborns with gross congenital anomalies, Pre term and IUGR babies, Suspected metabolic diseases, Babies born to mothers with Diabetes mellitus, mothers on anti epileptics, mothers with suspected or confirmed electrolyte abnormalities, Babies born to mothers with diuretics, general anesthesia, phenobarbitone, pethidine, magnesium sulphate, antihypertensive and drugs likely to cause depression and electrolyte disturbance in newborn were excluded.

Data collection was done within 24 hours of birth to ensure accuracy. A detailed history and examination of the patient was done and blood sample were collected and processed in the Department of Biochemistry.

Electrolyte estimation: Serum electrolytes (sodium, ionized calcium) were analysed using ion selective electrode by automated machine.

Serum Glucose was estimated by Glucose Oxidase Peroxidase method.

HIE Staging: Patients were classified according to SARNAT-SARNAT staging to grade the severity of HIE.

No cases were treated with therapeutic hypothermia due to the unavailability of such facility at our institute and cases were managed according to the protocol of the institute. Normal level of serum sodium, ionized calcium was taken as 135-145 meq/L, and 1–1.5 mmol/L respectively

Statistical Analysis: All Data were compiled and analyzed using SPSS version 29.0 (IBM Corp., Armonk, NY). Categorical variables, such as HIE stages and the proportion of newborns with electrolyte disturbances, were expressed as frequencies and percentages. Continuous variables, including serum sodium, ionized calcium, and glucose levels, were summarized as means ± standard deviations (SD).

The Chi-square test was used to assess associations between categorical variables (e.g., HIE stage and electrolyte abnormalities). A p-value <0.05 was considered statistically significant.

RESULTS

The study included a total of 60 newborns with birth asphyxia. The distribution of the studied cases based on gender revealed that 35 (58.3%) cases were males followed by 25 (41.7%) female cases.

Distribution of the studied patients based on need of resuscitation

The majority 44 (73.3%) patients required positive pressure ventilation (PPV). Intubation was performed in 13 (21.7%) patients. Only 3 patients (5.0%) needed initial steps alone without further ventilatory support.

APGAR Scores of babies: The study evaluated APGAR scores in the 60 asphyxiated newborns at 1 minute and 5 minutes after birth. At 1 minute, the majority of patients had low scores: 29 patients (48.3%) scored 3, 16 patients (26.7%) scored 4, 7

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patients (11.7%) scored 5, and 8 patients (13.3%) scored 6, with an overall mean APGAR score of 3.54 ± 0.98. At 5 minutes, the scores showed improvement: 3 patients (5.0%) scored 3, 14 patients (23.3%) scored 5, 18 patients (30.0%) scored 6, 17 patients (28.3%) scored 7, and 8 patients (13.3%) scored 8, resulting in a mean APGAR score of 5.60 ± 1.05.

Table 1: Distribution of the studied patients based on need of resuscitation

Need for resuscitation	Number of patients (n=60)	Percentage
PPV	44	73.3
Intubation	13	21.7
Initial steps	3	5.0

Table 2: APGAR Scores of babies

Score	Number of patients	Percentage
3	29	48.3
4	16	26.7
5	7	11.7
6	8	13.3
Mean: 3.54 ± 0.98		
3	3	5.0
5	14	23.3
6	18	30.0
7	17	28.3
8	8	13.3
Mean: 5.60 ± 1.05		

Distribution of the studied patients based on grading of HIE: In our study, 60 asphyxiated newborns were classified according to the grading of hypoxic-ischemic encephalopathy (HIE). Mild HIE (stage 1) was observed in 28 patients (46.7%), moderate HIE (stage 2) in 22 patients (36.7%), and severe HIE (stage 3) in 10 patients (16.7%).

Table 3: Distribution of the studied patients based on grading of HIE

Grading of HIE	Number of patients (n=60)	Percentage
HIE1	28	46.7
HIE2	22	36.7
HIE3	10	16.6

Association of stages of HIE with serum sodium, serum calcium and serum glucose

The mean serum sodium, calcium, and glucose levels showed a progressive decline with increasing severity of hypoxic-ischemic encephalopathy (HIE). In HIE stage 1 (n=28), the mean serum sodium was 136.8 ± 3.78 mEq/L, serum calcium was 8.85 ± 0.68 mg/dL, and serum glucose was 60.5 ± 3.64 mg/dL. In HIE stage 2 (n=22), these values decreased to 132.9 ± 4.29 mEq/L, 8.06 ± 0.48 mg/dL, and 53.7 ± 4.51 mg/dL, respectively. A further decline was observed in HIE stage 3 (n=10), with mean serum sodium of 127.1 ± 4.99 mEq/L, serum calcium of 7.58 ± 1.02 mg/dL, and serum glucose of 45.4 ± 3.94 mg/dL

There was a statistically significant association between the stages of mhypoxic-ischemic encephalopathy (HIE) and the levels of serum sodium, serum calcium, and serum glucose in the newborn participants ($p<0.05$).

Table 4: Association of stages of HIE with serum sodium, serum calcium and serum glucose

Stages of HIE	Serum sodium (mEq/L)	Serum calcium (mg/dL)	Serum glucose (mg/dL)
HIE 1 (n=28)	136.8 ± 3.78	8.85 ± 0.68	60.5 ± 3.64
HIE 2 (n=22)	132.9 ± 4.29	8.06 ± 0.48	53.7 ± 4.51
HIE 3 (n=10)	127.1 ± 4.99	7.58 ± 1.02	45.4 ± 3.94
p-value	<0.001	<0.001	<0.001

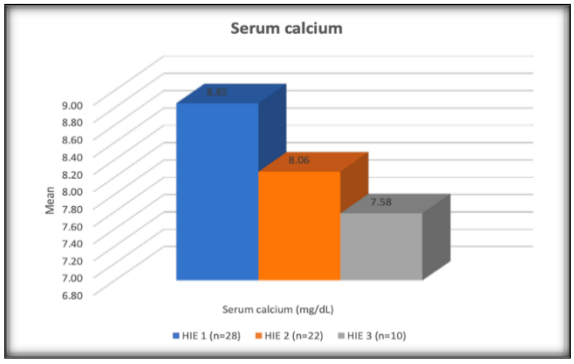

Figure 1: Serum sodium levels of stages of HIE

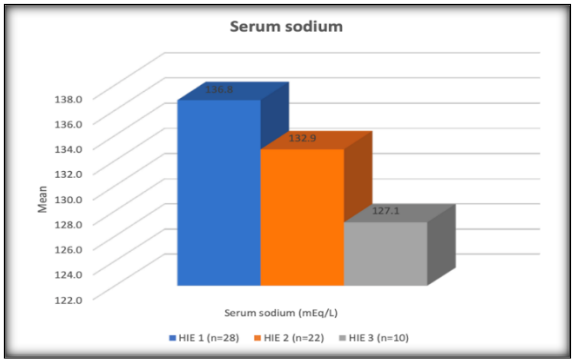

Figure 2: Serum Ionized Calcium levels of stages of HIE

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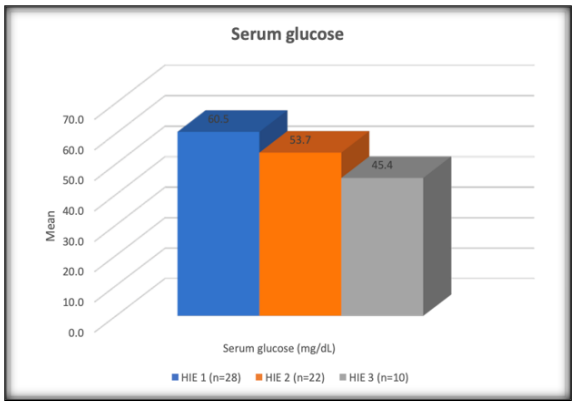

Figure 3: Serum Glucose levels of stages of HIE

DISCUSSION

In the present study, the mean maternal age was 26.06 years with a standard deviation of 5.47 years, while the mean gestational age was 37.91 weeks with a standard deviation of 2.19 weeks. The mean birth weight was 3.03 kilograms, with a standard deviation of 0.38 kilograms. 35 (58.3%) cases were males, followed by 25 (41.7%) female cases. Our findings were comparable to the findings of Lahari DS et al,[15] who reported that there was a male preponderance was observed, with 45 (60%) and 24 (40%) being female neonates. The mean birth weight was 2676±420 grams and the mean gestational period was 39.21±4.34 weeks.

In the present study, the majority 44 (73.3%) patients required positive pressure ventilation (PPV). Intubation was performed in 13 (21.7%) patients. Only 3 patients (5.0%) needed initial steps alone without further ventilatory support. Previous studies have reported similar trends. Rai S et al,[16] observed that nearly 70% of asphyxiated neonates required PPV, with intubation needed in approximately 20%, while only a minority responded to initial steps.

In the present study, at 1 minute, the majority of patients had low scores: 29 patients (48.3%) scored 3, 16 patients (26.7%) scored 4, 7 patients (11.7%) scored 5, and 8 patients (13.3%) scored 6, with an overall mean APGAR score of 3.54 ± 0.98. At 5 minutes, the scores showed improvement: 3 patients (5.0%) scored 3, 14 patients (23.3%) scored 5, 18 patients (30.0%)

scored 6, 17 patients (28.3%) scored 7, and 8 patients (13.3%) scored 8, resulting in a mean APGAR score of 5.60 ± 1.05. Our findings were consistent with the findings of Injeti G et al who reported that out of 85 neonates with birth asphyxia, at 1 minute of life, 8(9.41%) had an Apgar score <4, and 77 (90.59%) had an Apgar score 4-6. Out of 68 inborn neonates, 7 (10.29%) had an Apgar score <4, 61 (89.70%) were having 4-6 at 1 minute of life. Out of 17 outborn neonates, 1 (5.88%) had an Apgar score <4,16 (94.12%) had an Apgar score of 4-6 at 1 minute of life.

In the present study, more than half of the mothers were primigravida, with 32 cases (53.3%), while 28 (46.7%) were multigravida. The amniotic fluid was observed in 54 patients (90.0%), whereas it was stained in only 6 patients (10.0%). Regarding the mode of delivery, vaginal delivery occurred in 33 cases (55.0%), and cesarean section was performed in 27 cases (45.0%). In terms of antenatal care, 37 patients (61.7%) were from booked pregnancies, while 23 (38.3%) were unbooked. Our findings were in concordance with the findings of Injeti G et al,[17] who reported that 50.0% cases went through vaginal delivery.

In the present study, according to the clinical grades of asphyxia, mild asphyxia was present in 25 patients (41.7%), followed by moderate asphyxia in 23 patients (38.3%), and severe asphyxia in 12 patients (20.0%). Similar to the present study, Rai S et al,[16] reported that the study group was comprised of 53 cases of mild birth asphyxia, 26 cases of moderate birth asphyxia and 21 cases of severe birth asphyxia.

In the present study When assessed by stages of hypoxic-ischemic encephalopathy (HIE), newborns with HIE stage 1 had the highest mean serum calcium at 8.85±0.68 mg/dL, followed by 8.06±0.48 mg /dL in HIE stage 2, and 7.58±1.02 mg/dL in HIE stage 3, again demonstrating a highly significant association ($p < 0.001$).

In the present study When evaluated by stages of hypoxic-ischemic encephalopathy (HIE), newborns with HIE stage 1 exhibited the highest mean serum sodium at 136.8±3.78 mEq/L, followed by 132.9±4.29 mEq/L in HIE stage 2, and 127.1±4.99 mEq/L in HIE stage 3, again showing a statistically significant association ($p < 0.001$).

In the present study When assessed by stages of hypoxic-ischemic encephalopathy (HIE), newborns with HIE stage 1 had the highest mean serum glucose at 60.5 ±3.64 mg/dL, followed by 53.7±4.51 mg/dL in HIE stage 2, and 45.4±3.94 mg/dL in HIE stage 3, again demonstrating a statistically significant association ($p < 0.001$).

Our findings were similar to the findings of Joag G et al,[18] who reported that the mean calcium level was significantly lower in mild asphyxia cases than severe asphyxia (0.56 vs 0.88). In their study serum calcium was lower among asphyxiated neonates as compared to non-asphyxiated neonates and difference between 2 groups was statistically significant. Lower calcium levels are seen among asphyxiated neonates and are directly proportional to severity of birth asphyxia and HIE stages. Kavya MY et al,[19] reported that among the asphyxiated neonates serum sodium, calcium and glucose levels in arterial cord blood was significantly lower and has a linear correlation with the severity of birth asphyxia and different stages of HIE.

Our results are in concordance with Masood N et al,[20] which concluded that asphyxiated neonates had lower serum calcium levels compared to controls. The results of the present study were in concordance with those of Basu P et al,[21] reported that serum sodium (122.1±6.0 meq/l) and calcium (6.85±0.95 mg/dl) levels were significantly lower in asphyxiated neonates, compared to non- asphyxiate neonates

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serum sodium (138.8±2.7 meq/l; p<0.001) and calcium (9.50±0.51 mg/dl; p<0.001) levels.

According to Rai S et al mean glucose levels of the study and control groups were 54.4 ± 10.91 and 76 ± 15.5 mg/dl. The mean serum glucose level in the study was lower as compared to control group, and the statistical difference was highly significant (P < 0.001)

Strength and limitation of the study: A key strength of this study is it specifically assessed ionized calcium, sodium, and glucose, which are clinically relevant and directly linked to neonatal outcomes in asphyxia.

Biochemical changes were analyzed in relation to HIE staging, providing a clear clinical context and prognostic relevance.

However, a limitation of the study is the study was conducted on a relatively modest cohort, which may limit the generalizability of findings to wider populations.

Being restricted to one tertiary care center, the results may reflect local practices and patient demographics rather than broader national or global trends.

CONCLUSION

Birth asphyxia is strongly associated with significant metabolic derangements, particularly hypocalcemia, hyponatremia, and hypoglycemia, which correlate with the severity of hypoxic-ischemic encephalopathy. Low birth weight emerges as an important predictor of severe disease and biochemical abnormalities. Early identification and prompt management of these metabolic disturbances are crucial to reduce mortality and improve neurological outcomes. Routine biochemical monitoring, especially in high-risk neonates, is recommended to guide timely interventions and enhance prognosis.

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