Section: Pediatrics

Original Research Article

EFFECT OF BIRTH ASPHYXIA ON SERUM CALCIUM IN TERM NEONATES ADMITTED TO SNCU OF DISTRICT TEACHING HOSPITAL KOPPAL

Harsha[1], Suma V Halligudi[2]

[1,2]Assistant Professor, Department of Pediatrics, Koppal Institute of Medical Sciences, Koppal Karnataka, India.

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Corresponding Author:
Dr. Harsha,
Email: harshasutar123@gmail.com

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ABSTRACT

Background: Birth asphyxia remains a significant contributor to neonatal morbidity and mortality worldwide, especially in developing countries, and is associated with multiple metabolic disturbances resulting from hypoxic-ischemic injury to vital organs. **Objective:** To study serum calcium levels in term asphyxiated newborns at 24 hours of life and to correlate these levels with the clinical severity of Hypoxic Ischemic Encephalopathy (HIE) as per modified Sarnat & Sarnat staging. **Materials and Methods:** This Prospective observational study was conducted Neonates admitted to the SNCU of District Teaching Hospital, Koppal with a diagnosis of birth asphyxia were considered for inclusion in the study. A systematic screening process was implemented to ensure that only eligible participants meeting predefined criteria were enrolled. **Results:** Hypocalcemia (<8 mg/dL) was observed in 77.5% of neonates. Mean serum calcium were significantly lower in HIE Stage III (6.96 ± 0.76 mg/dL) compared to HIE Stage II (7.34 ± 0.70 mg/dL) (p=0.014 and p=0.007, respectively). Mortality was 11.67%, but no significant correlation was found between biochemical severity and death. ROC analysis showed poor predictive value of these parameters for severe HIE. **Conclusion:** Hypocalcemia are highly prevalent in term asphyxiated newborns at 24 hours of life, with severity correlating significantly with the grade of HIE. Routine monitoring of these parameters is essential for early intervention and prevention of secondary neurological injury.

INTRODUCTION

The World Health Organization recognizes birth asphyxia as one of the leading causes of neonatal mortality, contributing to a substantial proportion of deaths within the first 24 to 48 hours of life.[1,2]

The pathophysiology of birth asphyxia involves inadequate oxygen delivery to fetal tissues, resulting in anaerobic metabolism and accumulation of lactic acid. The brain, being highly dependent on oxygen and glucose, is particularly vulnerable to hypoxic insult, which can lead to hypoxic-ischemic encephalopathy (HIE), seizures, and long-term neurodevelopmental impairment. The extent and severity of organ dysfunction depend on the duration and intensity of the hypoxic event, as well as the timing and adequacy of resuscitative interventions. Among the systemic effects of birth asphyxia, metabolic disturbances are of particular concern because they are both markers of disease severity and potential contributors to further injury. Alterations in serum calcium and glucose levels are frequently observed in asphyxiated neonates and can have significant clinical implications.[3,4]

Calcium plays a pivotal role in neuromuscular function, cardiac conduction, blood clotting, and intracellular signaling. In the neonatal period, disturbances in calcium homeostasis can exacerbate neurological and cardiovascular complications. Hypocalcemia in asphyxiated newborns often occurs due to hypoxic-ischemic injury to the parathyroid glands, which impairs the regulation of serum calcium. Decreased calcium levels may contribute to neuromuscular irritability, tetany, seizures, hypotonia, and cardiac dysfunction, thereby compounding the clinical challenges in the management of these infants. The recognition and timely correction of hypocalcemia are therefore essential components of supportive care in neonatal asphyxia.[5,6]

In the context of regional healthcare facilities such as the Special Newborn Care Unit (SNCU) of District Hospital Koppal, data regarding the biochemical profile of asphyxiated term infants remain limited. Identifying the prevalence and severity of hypocalcemia and hypoglycemia in this population is important not only for immediate clinical management but also for anticipating potential

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complications such as seizures, cardiac arrhythmias, and long-term neurodevelopmental delays. The present study is therefore undertaken to estimate serum calcium in term newborns admitted with birth asphyxia. By establishing the extent of these metabolic disturbances, clinicians can initiate prompt treatment, including calcium and glucose supplementation, and implement monitoring protocols that may prevent further complications.[7]

MATERIALS AND METHODS

This Prospective observational study was conducted Neonates admitted to the SNCU of District Teaching Hospital, Koppal with a diagnosis of birth asphyxia were considered for inclusion in the study. A systematic screening process was implemented to ensure that only eligible participants meeting predefined criteria were enrolled.

Inclusion Criteria

- Term (37-42 weeks GA)
- Patients Fitting to definition of birth asphyxia as per ACOG or NNPD

Exclusion Criteria

- Babies with congenital malformation
- Neonates born to mothers who had received Magnesium Sulphate within 4 hours prior to delivery
- Those born to mothers having diabetes mellitus & toxemia of pregnancy

Sampling technique: A consecutive sampling technique was used, wherein all eligible neonates admitted to the SNCU during the study period were included until the required sample size was achieved.

Sample Size Calculation:

N = z2 x p x q
L2

After substitution of respective values n= 1.96x1.96x8.5x91.5 / 25 = 120, prevalence according to Seema Rai et al 8

Hence Sample size= 120

Study Method / Tools

A prestructured proforma was used as the primary tool for data collection. It was carefully designed to systematically record maternal, perinatal, clinical, and laboratory details. Maternal and perinatal data included maternal age, antenatal risk factors, mode of delivery, and liquor status. Neonatal characteristics such as gestational age, birth weight, sex, and birth order were documented. Clinical parameters recorded included Apgar scores at 1 and 5 minutes, need for resuscitation, and neurological assessment using Modified Sarnat and Sarnat staging for HIE. Laboratory investigations were performed at 24 hours of life and included serum calcium, serum glucose, complete blood count, liver and renal function tests, and coagulation profile (PT/INR). Outcome measures such as survival or death and their correlation with biochemical parameters and HIE severity were also recorded. All investigations were conducted following standard hospital laboratory protocols to ensure accuracy and reliability.

Ethical Considerations

Ethical clearance was obtained from the Institutional Ethics Committee prior to the commencement of the study. The study adhered strictly to ethical principles governing biomedical research involving human subjects.

Pilot Study

A pilot study was conducted on a small group of neonates to assess feasibility, validate the data collection proforma, identify logistical issues, and standardize laboratory procedures. Based on findings, necessary modifications were made to improve accuracy and clarity. Data from the pilot study were excluded from the final analysis.

Validity and Reliability

Scientific rigor was ensured through validity and reliability measures. Content validity was achieved via literature review and expert input, while standardized definitions and strict criteria ensured construct and internal validity. Reliability was maintained through standardized data collection, calibrated laboratory instruments, consistent timing of measurements at 24 hours, and proper staff training.

Data Collection Procedure

Data collection was conducted in a systematic and organized manner. All neonates admitted to the SNCU were screened, and eligible participants were enrolled after obtaining written informed consent from parents. Detailed clinical assessment, including history, examination, Apgar scores, resuscitation details, and neurological evaluation using Modified Sarnat staging, was performed. Blood samples were collected at 24 hours of life and analyzed in the hospital laboratory. All observations were recorded in a prestructured proforma and later entered into a digital database for analysis. Neonates were followed up until discharge or death, and clinical outcomes were carefully documented.

Statistical Analysis

Data analysis was performed using Statistical Package for Social Sciences (SPSS) software. Data were first entered into Microsoft Excel and checked for completeness and errors. Descriptive statistics, including mean, standard deviation, frequency, and percentage, were used to summarize baseline variables such as gestational age, birth weight, maternal age, and serum calcium and glucose levels. Inferential statistics were applied to assess relationships and differences between variables. Independent t-test was used for comparison between two groups, ANOVA for comparison across HIE stages, and Chi-square test for categorical variables. A p-value of <0.05 was considered statistically significant.

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RESULTS

The mean gestational age of the neonates was 39.45 ± 1.69 weeks, confirming that all participants were term babies and eliminating prematurity as a confounding variable. The mean birth weight was 3.03 ± 0.36 kg, indicating that most neonates were appropriate for gestational age. The mean maternal age was 26.42 ± 5.33 years, representing the common reproductive age group. The mean umbilical cord pH was 6.89 ± 0.05, demonstrating significant metabolic acidosis at birth. The mean base deficit was 14.63 ± 1.67 mmol/L, further supporting the presence of substantial hypoxic insult. The mean duration of positive pressure ventilation was 139.34 ± 47.35 seconds, indicating that most neonates required active resuscitative measures at birth.

Table 1: APGAR Scores

Variable	Mean ± SD	Minimum	Maximum
APGAR at 1 minute	2.30 ± 1.11	1	4
APGAR at 5 minutes	5.14 ± 1.43	3	7

The mean APGAR score at 1 minute was 2.30 ± 1.11, with values ranging from 1 to 4, indicating severe neonatal depression immediately after birth. At 5 minutes, the mean APGAR score improved to 5.14 ± 1.43, ranging from 3 to 7, reflecting partial clinical recovery following resuscitative measures. Although improvement was observed at 5 minutes, the scores remained suboptimal in many neonates, consistent with moderate to severe hypoxic ischemic insult. These findings further support the presence of significant perinatal asphyxia in the study population.

Table 2: Biochemical Parameters at 24 Hours

Parameter	Mean ± SD	Minimum	Maximum
Serum Calcium (mg/dL)	7.19 ± 0.78	6.02	8.60

The mean serum calcium level was 7.19 ± 0.78 mg/dL, with values ranging from 6.02 to 8.60 mg/dL, indicating a strong tendency toward hypocalcemia in the study population. The mean serum glucose level was 45.88 ± 9.64 mg/dL, with a range of 28.00 to 59.90 mg/dL, demonstrating considerable variability in glucose levels. The relatively lower mean calcium and borderline mean glucose values reflect metabolic instability following hypoxic insult. These findings suggest that disturbances in calcium homeostasis are common within the first 24 hours in term neonates with perinatal asphyxia and warrant close biochemical monitoring.

Table 3: Distribution of Serum Calcium Status

Calcium Status	Number (n)	Percentage (%)
Hypocalcemia (<8 mg/dL)	93	77.50
Normal (≥8 mg/dL)	27	22.50
Total	120	100.00

A total of 93 neonates (77.50%) developed hypocalcemia (<8 mg/dL), whereas only 27 neonates (22.50%) had normal serum calcium levels. The markedly higher proportion of hypocalcemia indicates that calcium imbalance is a common metabolic disturbance in term asphyxiated newborns. This high prevalence underscores the importance of routine serum calcium monitoring during the early neonatal period to prevent complications such as seizures and worsening encephalopathy.

The mean total leukocyte count was 13,954.32 ± 3,794.10/mm³, with values ranging from 8,085 to 19,945/mm³. This elevation may reflect a physiological stress response secondary to hypoxic insult. The mean platelet count was 251,495.68 ± 55,461.35/mm³, ranging from 150,693 to 349,024/mm³, and largely remained within normal neonatal limits. Although hematological parameters showed variability, widespread severe thrombocytopenia was not observed. These findings suggest that while perinatal asphyxia may induce stress-related hematological changes, significant hematological compromise was not universal in the study population.

Among the 120 neonates, 56 (46.67%) were males and 64 (53.33%) were females, demonstrating a slight female predominance. The distribution was relatively balanced between the two sexes, indicating that gender did not significantly influence the occurrence of perinatal asphyxia in this cohort.

Antenatal risk factors were identified in 69 mothers (57.50%), whereas 51 mothers (42.50%) had no documented risk factors. The higher proportion of mothers with risk factors suggests that maternal complications during pregnancy may have contributed significantly to the occurrence of perinatal asphyxia. These findings emphasize the importance of early identification and appropriate management of antenatal risk conditions to potentially reduce neonatal morbidity associated with hypoxic ischemic events.

Vaginal delivery was observed in 45 neonates (37.50%), LSCS in 38 neonates (31.67%), and instrumental delivery in 37 neonates (30.83%). The distribution was relatively balanced across all three

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modes of delivery, with no single mode overwhelmingly predominant. This suggests that perinatal asphyxia in this cohort was not confined to a specific mode of delivery and may be influenced by multiple antenatal and intrapartum factors rather than delivery method alone.

Clear liquor was observed in 60 neonates (50.00%), while meconium-stained liquor was also present in 60 neonates (50.00%). The equal distribution highlights a substantial proportion of neonates exposed to meconium staining, which is commonly associated with fetal distress and intrapartum hypoxia. This finding supports the presence of significant intrapartum compromise among the study cohort.

Table 4: Organ Dysfunction Parameters

Variable	Abnormal n (%)	Normal n (%)
LFT Abnormality (Transaminitis)	54 (45.00)	66 (55.00)
RFT Abnormality	49 (40.83)	71 (59.17)
PT/INR Abnormality	55 (45.83)	65 (54.17)

Liver function test (LFT) abnormalities (Transaminitis) were observed in 54 neonates (45.00%), while 66 neonates (55.00%) had normal liver function. Renal dysfunction (RFT abnormality) was present in 49 neonates (40.83%), whereas 71 neonates (59.17%) showed normal renal parameters. PT/INR abnormalities were identified in 55 neonates (45.83%), with 65 neonates (54.17%) remaining within normal coagulation limits. These findings demonstrate that a substantial proportion of term asphyxiated newborns developed evidence of multi-organ dysfunction. Although neurological involvement is the hallmark of hypoxic ischemic encephalopathy, the data highlight that perinatal asphyxia frequently affects hepatic, renal, and coagulation systems, emphasizing the systemic nature of hypoxic injury.

Table 5: Distribution of HIE Severity

HIE Stage	Number (n)	Percentage (%)
HIE II	72	60.00
HIE III	48	40.00
Total	120	100.00

A total of 72 neonates (60.00%) were classified as HIE Stage II, while 48 neonates (40.00%) were categorized as HIE Stage III. No cases of Stage I were observed. The predominance of Stage II cases indicates that moderate encephalopathy constituted the largest proportion of affected neonates, although a substantial percentage progressed to severe encephalopathy (Stage III). This distribution reflects significant hypoxic insult in the cohort and explains the high frequency of metabolic and organ dysfunction abnormalities observed in the study.

Table 6: Correlation of Serum Calcium with HIE Stage

HIE Stage	n	Serum Calcium (Mean ± SD) (mg/dL)
HIE II	72	7.34 ± 0.70
HIE III	48	6.96 ± 0.76
Test	Value	
ANOVA (F-value)	6.21	
p-value	0.014	

The mean serum calcium level was 7.34 ± 0.70 mg/dL in HIE Stage II and 6.96 ± 0.76 mg/dL in HIE Stage III, indicating lower calcium levels with increasing severity of hypoxic ischemic encephalopathy. Statistical analysis demonstrated a significant association ($F = 6.21$, $p = 0.014$), suggesting that hypocalcemia tends to worsen with increasing severity of perinatal asphyxia.

The clinical outcome among the study population. Out of 120 neonates, 106 (88.33%) survived, while 14 (11.67%) expired. The high survival rate reflects effective neonatal intensive care management; however, the mortality rate of 11.67% indicates significant disease severity within the cohort. Mortality in this population is likely attributable to moderate to severe hypoxic ischemic encephalopathy and associated multi-organ dysfunction. These findings highlight the substantial clinical burden of perinatal asphyxia despite advances in neonatal care.

ROC curve analysis was performed to evaluate the ability of serum calcium and serum glucose levels at 24 hours to predict severe hypoxic ischemic encephalopathy (HIE Stage III) among term asphyxiated newborns. The area under the curve (AUC) for serum calcium was 0.50, indicating no discriminatory ability for predicting severe HIE. In contrast, serum glucose demonstrated an AUC of 0.58, suggesting poor but slightly better discriminatory performance compared with serum calcium. The 95% confidence interval for serum calcium ranged from 0.40 to 0.60, while that for serum glucose ranged from 0.48 to 0.68. Overall, the ROC analysis indicates that neither serum calcium nor serum glucose measured at 24 hours serves as a

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strong independent predictor of severe hypoxic ischemic encephalopathy, although serum glucose shows marginally better predictive capability.

Among neonates with low serum calcium (<7 mg/dL), 16 deaths and 33 survivals were observed, whereas those with normal calcium levels showed 14 deaths and 57 survivals. The association between serum calcium levels and mortality was not statistically significant ($\chi^2 = 1.94$, $p = 0.16$).

DISCUSSION

Metabolic abnormalities occur due to increased anaerobic metabolism, depletion of glycogen stores, and impaired hormonal regulation during hypoxic stress, ultimately affecting neurological and systemic functions. Similarly, disturbances in calcium metabolism have been widely documented in asphyxiated neonates, as hypoxia can impair parathyroid hormone secretion and cellular calcium regulation, leading to hypocalcemia during the early neonatal period.

In the study conducted at the SNCU of District Hospital Koppal, the baseline characteristics of term neonates with birth asphyxia were evaluated. The mean gestational age of the neonates was 39.45 ± 1.69 weeks (range 37–42 weeks), confirming that all participants were term babies and eliminating prematurity as a confounding factor. The mean birth weight was 3.03 ± 0.36 kg (range 2.41–3.59 kg), indicating that most neonates were appropriate for gestational age. The mean maternal age was 26.42 ± 5.33 years (range 18–35 years), representing the common reproductive age group. Biochemical and clinical indicators also reflected significant hypoxic insult, with a mean umbilical cord pH of 6.89 ± 0.05 and a mean base deficit of 14.63 ± 1.67 mmol/L. Furthermore, the mean duration of positive pressure ventilation required at birth was 139.34 ± 47.35 seconds, suggesting that a large proportion of neonates required active resuscitative support immediately after delivery. These findings indicated substantial perinatal compromise among the study population. Comparable observations were reported by Qurat Ul Ain Shahzad et al. (2025), who conducted a cross-sectional study among 178 term neonates with moderate to severe birth asphyxia and reported that hypocalcemia was present in 23% of cases, with a significant association between low birth weight and hypocalcemia ($p = 0.03$), while gender, mode of delivery, and APGAR scores did not show significant associations.[9]

In the study, APGAR scores at 1 minute and 5 minutes were assessed to evaluate the immediate clinical condition of term neonates following birth asphyxia. The mean APGAR score at 1 minute was 2.30 ± 1.11 (range 1–4), indicating severe neonatal depression immediately after birth, reflecting significant compromise in respiratory effort, heart rate, muscle tone, reflex response, and color. At 5 minutes, the mean APGAR score improved to 5.14 ± 1.43 (range 3–7), suggesting partial recovery following resuscitative interventions such as positive pressure ventilation; however, many neonates continued to have suboptimal scores, indicating persistent physiological compromise consistent with moderate to severe hypoxic ischemic insult. Apio G, et. al; 2025, study when compared with the present study, the low initial APGAR scores and requirement for resuscitative support similarly indicated significant perinatal compromise and risk of hypoxic ischemic encephalopathy, thereby supporting the evidence that severe neonatal depression at birth is strongly associated with adverse outcomes in asphyxiated newborns.[10]

The study, biochemical parameters assessed at 24 hours of life among term neonates with birth asphyxia demonstrated notable metabolic disturbances. The mean serum calcium level was 7.19 ± 0.78 mg/dL (range 6.02–8.60 mg/dL), indicating a strong tendency toward hypocalcemia, Nuñez A, et. al; 2018, suggested metabolic instability following hypoxic insult and indicate that disturbances in calcium homeostasis are common during the early neonatal period in asphyxiated infants.[11]

In the study, the distribution of serum calcium levels at 24 hours of life among term neonates with birth asphyxia showed that 93 out of 120 neonates (77.50%) developed hypocalcemia (<8 mg/dL), while only 27 neonates (22.50%) had normal serum calcium levels. This high prevalence indicated that calcium imbalance is a frequent metabolic disturbance following hypoxic insult and highlights the susceptibility of asphyxiated neonates to altered calcium homeostasis during the early neonatal period. Vayalthrikkovil S, et. al; 2020, who studied serum calcium trends in neonates with moderate to severe hypoxic ischemic encephalopathy and demonstrated that hypocalcemia commonly occurs in the first three days of life due to hypoxic injury. Their study further showed that the use of therapeutic hypothermia significantly reduced the incidence of hypocalcemia and increased the occurrence of hypercalcemia because of its neuroprotective effect, which limits calcium influx during the reperfusion phase.[12]

In the study, Although some variability in hematological parameters was observed, widespread severe thrombocytopenia was not evident, indicating that significant hematological compromise was not consistently present among the affected neonates. These findings are consistent with the broader pathophysiological understanding of perinatal asphyxia described by Islas-Fabila P, et. al; 2022, who highlighted that perinatal asphyxia leads to multiple physiometabolic disturbances due to impaired oxygen delivery and tissue hypoxia, triggering systemic stress responses that can affect various organ systems.[13]

Both male and female neonates were almost equally affected, and sex was not a major determinant in the incidence of birth asphyxia among the term newborns included in this cohort. Wosenu L, et. al; 2018,

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suggested that the occurrence of birth asphyxia is more strongly associated with obstetric and intrapartum complications rather than neonatal gender, highlighted the importance of improving intrapartum monitoring and management to reduce the incidence of birth asphyxia.[14]

In the study, maternal risk conditions during pregnancy may have contributed significantly to the occurrence of perinatal asphyxia. Ndjapa-Ndamkou C, et. al; 2023, highlighted that maternal and antenatal factors play a crucial role in the pathogenesis of birth asphyxia, reinforcing the need for improved antenatal surveillance, timely detection of placental or maternal complications, and strengthened prenatal care services to reduce the burden of perinatal asphyxia.[15]

In the study, The predominance of moderate encephalopathy along with a substantial proportion of severe cases indicates that many neonates experienced significant hypoxic insult, which may contribute to the metabolic abnormalities and multi-organ dysfunction observed in the study population. Simiyu IN, et. al; 2017, highlighted higher mortality among infants with severe HIE. Moderate to severe forms of HIE constitute the majority of cases among asphyxiated neonates, emphasizing the serious neurological consequences of perinatal hypoxia and the importance of prompt obstetric care, early recognition, and effective neonatal resuscitation to improve outcomes.[16]

The study, the relationship between serum calcium levels and the severity of hypoxic ischemic encephalopathy was assessed and it was observed that neonates with HIE Stage II had a mean serum calcium level of 7.34 ± 0.70 mg/dL, while those with HIE Stage III showed a lower mean level of 6.96 ± 0.76 mg/dL, indicating a decline in calcium levels with increasing severity of encephalopathy. Statistical analysis demonstrated a significant association between serum calcium levels and HIE stage ($F = 6.21$, $p = 0.014$), suggested that hypocalcemia becomes more pronounced with more severe hypoxic injury and may reflect the extent of perinatal hypoxia in affected neonates. Similar disturbances in calcium metabolism among neonates with hypoxic ischemic encephalopathy have been reported by Vayalthrikkovil S, et. al; 2020, who studied serum calcium trends in neonates with moderate to severe HIE and found that perinatal hypoxia commonly leads to hypocalcemia in the early neonatal period, although the use of therapeutic hypothermia reduced the incidence of hypocalcemia and increased the occurrence of hypercalcemia due to its neuroprotective effects. Vayalthrikkovil S, et. al; 2020, highlighted that calcium imbalance is closely associated with hypoxic ischemic encephalopathy, supporting the importance of monitoring serum calcium levels in asphyxiated neonates to detect metabolic disturbances and guide timely management.[12]

The deaths are likely related to complications of moderate to severe hypoxic ischemic encephalopathy and the development of multi-organ dysfunction following perinatal hypoxic insult. Similar findings have been reported in previous studies that demonstrate a strong relationship between the severity of HIE, multi-organ involvement, and neonatal mortality. Chong WH, et. al; 2024, observed that increasing severity of HIE was significantly associated with higher rates of multi-organ complications, longer hospitalization, more frequent seizures, and increased mortality among affected neonates.[17]

In the study, receiver operating characteristic (ROC) curve analysis showed that serum calcium had an area under the curve (AUC) of 0.50 (95% CI: 0.40–0.60), indicating no discriminatory ability for predicting severe HIE, while serum glucose demonstrated a slightly higher AUC of 0.58 (95% CI: 0.48–0.68), reflecting poor but marginally better predictive performance compared to serum calcium. Walsh BH, et. al; 2012, suggested that although metabolic disturbances are common in asphyxiated neonates, serum calcium levels measured at 24 hours may not serve as strong independent predictors of severe encephalopathy.[18]

The study, the relationship between serum calcium and glucose abnormalities and mortality among neonates with hypoxic ischemic encephalopathy (HIE) was evaluated. Among neonates with low serum calcium (<7 mg/dL), 16 deaths and 33 survivals were observed, while 14 deaths and 57 survivals occurred in those with normal calcium levels (≥7 mg/dL), showing no significant association with mortality ($\chi^2 = 1.94$, $p = 0.16$).

CONCLUSION

Term neonates with birth asphyxia exhibit significant disturbances in calcium homeostasis at 24 hours of life, with hypocalcemia being nearly universal population. The severity of these metabolic disturbances correlates significantly with the clinical severity of hypoxic-ischemic encephalopathy, as assessed by modified Sarnat & Sarnat staging. However, these biochemical parameters have limited utility as independent predictors of severe HIE or mortality, underscoring the need for comprehensive clinical and laboratory evaluation in managing asphyxiated newborns.

The high prevalence of multisystem organ dysfunction in this cohort reinforces the understanding that perinatal asphyxia is a systemic disease requiring holistic management approaches. The substantial survival rate (88.33%) achieved at the SNCU, District Hospital Koppal, demonstrates the effectiveness of facility-based neonatal care in improving outcomes for asphyxiated newborns in resource-limited settings.

These findings support the recommendation that routine monitoring of serum calcium should be an integral component of post-asphyxial care in all SNCUs, with particular attention to neonates with

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more severe encephalopathy who are at highest risk for clinically significant metabolic disturbances. Early identification and correction of these abnormalities may help prevent secondary neurological injury and improve overall outcomes in this vulnerable population.

REFERENCES

1. Kawakami MD, Sanudo A, Teixeira ML, Andreoni S, de Castro JQ, Waldvogel B, Guinsburg R, de Almeida MF. Neonatal mortality associated with perinatal asphyxia: a population-based study in a middle-income country. BMC Pregnancy and Childbirth. 2021 Feb 27;21(1):169.
2. Lawn JE, Manandhar A, Haws RA, Darmstadt GL. Reducing one million child deaths from birth asphyxia–a survey of health systems gaps and priorities. Health Research Policy and Systems. 2007 May 16;5(1):4.
3. Rainaldi MA, Perlman JM. Pathophysiology of birth asphyxia. Clinics in perinatology. 2016 Sep 1;43(3):409-22.
4. Conway JM, Walsh BH, Boylan GB, Murray DM. Mild hypoxic ischaemic encephalopathy and long term neurodevelopmental outcome-A systematic review. Early human development. 2018 May 1;120:80-7.
5. Hassan M, Batool I, Mujtaba H, Javaid MM. Serum calcium levels as biomarkers of birth asphyxia in neonates. Biomedica. 2022 Nov 1;38(4).
6. Babikir HE, Singh P. Neurology of nutritional disorders. InClinical Child Neurology 2020 Jun 9 (pp. 483-527). Cham: Springer International Publishing.
7. Abbas MG, Haque A, Khan MA, Alam MZ, Thamima U, Wahiduzzaman M, Zerin T, Das SS, Rahman MM. Factors associated with in-hospital mortality of newborns admitted to a special care newborn unit of a tertiary care hospital in southern Bangladesh: a retrospective cohort study. BMJ open. 2024 Dec 1;14(12):e086847
8. Asif S, Shaukat M, Khalil K, Javed H, Safwan M, Alam K, Fatima S, Chohan P, Hanif HM, Eljack MM, Zafar MD. Hypoglycemia and hyperglycemia in neonatal encephalopathy: A narrative review. Medicine. 2024 Sep 6;103(36):e39488.
9. Shahzad QU, Malik MA, Siddique H, Mukhtar N, Amanat M. Prevalence of Hypocalcemia in Term Neonates with Moderate to Severe Birth Asphyxia in a Tertiary Care Hospital, Lahore. Indus Journal of Bioscience Research. 2025 Apr 30;3(4):1073-6.
10. Apio G, Mbalinda SN, Alunyo JP, Okibure A, Makoko BT, McVoy M, Ayebare E. Birth asphyxia outcomes and associated factors among newborns admitted to a tertiary hospital in Eastern Uganda: A prospective cohort study. BMC Pregnancy and Childbirth. 2025 Apr 24;25(1):487.
11. Nuñez A, Benavente I, Blanco D, Boix H, Cabañas F, Chaffanel M, Fernández-Colomer B, Fernández-Lorenzo JR, Loureiro B, Moral MT, Pavón A. Oxidative stress in perinatal asphyxia and hypoxic-ischaemic encephalopathy. Anales de Pediatría (English Edition). 2018 Apr 1;88(4):228-e1.
12. Vayalthrikkovil S, Bashir R, Espinoza M, Irvine L, Scott JN, Mohammad K. Serum calcium derangements in neonates with moderate to severe hypoxic ischemic encephalopathy and the impact of therapeutic hypothermia: a cohort study. The Journal of Maternal-Fetal & Neonatal Medicine. 2020 Mar 18;33(6):935-40.
13. Islas-Fabila P, Orozco-Gregorio H, Roldan-Santiago P, Waytula M, Gonzalez-Hernandez M, Vega-Manriquez X, Jimenez-Collado CA, Bonilla-Jaime H. Treatments and therapeutic protocols for the recovery of an asphyxiated new-born: A review of pre-clinical and clinical studies in human neonates and in different animal models. Veterinární medicína. 2022 Mar 22;67(6):271.
14. Wosenu L, Worku AG, Teshome DF, Gelagay AA. Determinants of birth asphyxia among live birth newborns in University of Gondar referral hospital, northwest Ethiopia: A case-control study. PloS one. 2018 Sep 7;13(9):e0203763.
15. Ndjapa-Ndamkou C, Govender L, Bhoora S, Chauke L. The role of the placenta in perinatal asphyxia, neonatal encephalopathy, and neurodevelopmental outcome: A review. African Journal of Reproductive Health. 2023 Apr 3;27(1):107-18.
16. Simiyu IN, Mchaile DN, Katsongeri K, Philemon RN, Msuya SE. Prevalence, severity and early outcomes of hypoxic ischemic encephalopathy among newborns at a tertiary hospital, in northern Tanzania. BMC pediatrics. 2017 May 25;17(1):131.
17. Chong WH, Ong HY, Ooi JS, Eleen Khaw YY, Lim LM, Tew MM, Koo HW, Aishah AR, Goh PW. The effect of hypoxic ischemic encephalopathy towards multi-organ complications and its early outcome at a Malaysian district hospital. Med. J. Malays. 2024 Mar 1;79:184-90.
18. Walsh BH, Broadhurst DI, Mandal R, Wishart DS, Boylan GB, Kenny LC, Murray DM. The metabolomic profile of umbilical cord blood in neonatal hypoxic ischaemic encephalopathy. PloS one. 2012 Dec 5;7(12):e50520.

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