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ORIGINAL RESEARCH

Assessment of the Correlation between Birth Asphyxia and Serum LDH Levels in Term Neonates

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ABSTRACT

Background: Birth asphyxia remains a major cause of neonatal morbidity and mortality, particularly in developing countries. Early identification of the extent of hypoxic injury is crucial for timely intervention. Serum Lactate Dehydrogenase (LDH), a marker of tissue damage, has been proposed as a potential indicator of perinatal asphyxia severity. **Aim:** To assess serum LDH levels in term neonates with birth asphyxia and evaluate its correlation with the severity of hypoxic-ischemic encephalopathy (HIE). **Material and Methods:** This prospective observational study was conducted over 12 months in the Department of Paediatrics at a tertiary care teaching hospital. A total of 110 term neonates were enrolled and divided into two groups: Group A (55 neonates with birth asphyxia) and Group B (55 healthy term neonates as controls). Serum LDH levels were measured within 6 hours of birth using an enzymatic UV-kinetic method. Clinical data including mode of delivery, Apgar scores, and HIE staging were recorded. Statistical analysis was performed using SPSS version 21.0, with significance set at $p < 0.05$. **Results:** Caesarean section was significantly more common in the asphyxiated group (60% vs. 38.18%, $p = 0.049$). Risk factors such as prolonged labour (32.73% vs. 5.45%), meconium-stained liquor (38.18% vs. 3.64%), and foetal distress (30.91% vs. 1.82%) were significantly associated with birth asphyxia ($p < 0.001$). Apgar scores were significantly lower in Group A at both 1 and 5 minutes ($p < 0.001$). Mean serum LDH levels were significantly higher in asphyxiated neonates (1126.35 ± 240.27 U/L) compared to controls (612.42 ± 156.54 U/L, $p < 0.001$). A progressive increase in LDH levels was noted with advancing HIE severity: Stage I (980.22 ± 115.43 U/L), Stage II (1132.85 ± 172.68 U/L), and Stage III (1346.73 ± 198.35 U/L, $p < 0.001$).

Conclusion: Serum LDH levels are significantly elevated in neonates with birth asphyxia and show a strong correlation with HIE severity. LDH can serve as a valuable biochemical marker for early diagnosis and prognostication of perinatal asphyxia when used alongside clinical indicators.

Keywords: Birth asphyxia, Neonates, Serum LDH, Hypoxic-ischemic encephalopathy, Apgar score

INTRODUCTION

Perinatal asphyxia continues to be one of the most pressing public health challenges in neonatal care, especially in low- and middle-income countries where perinatal mortality rates remain disproportionately high. Defined as a condition resulting from impaired gas exchange before, during, or immediately after birth, perinatal asphyxia leads to significant morbidity and mortality among neonates. The failure to

establish spontaneous respiration immediately after birth can lead to multiorgan dysfunction, particularly affecting the brain, thereby posing a serious threat to long-term neurodevelopmental outcomes.[1]

The burden of neonatal mortality remains unacceptably high in many developing nations, with perinatal asphyxia accounting for a significant proportion of early neonatal deaths. Studies from South Asia highlight the role of intrapartum-related complications as major contributors to neonatal mortality. In urban and rural healthcare settings alike, the lack of timely obstetric interventions, inadequate resuscitation practices, and insufficient neonatal intensive care infrastructure all contribute to the persistence of poor perinatal outcomes. Despite improvements in antenatal care and institutional deliveries, early identification and effective management of birth asphyxia continue to lag behind, especially in resource-constrained environments.[2]

The pathophysiology of birth asphyxia involves a complex cascade of events including hypoxia, hypercapnia, and acidosis, which lead to cellular energy failure, inflammation, and ultimately cell death. The brain is the most vulnerable organ in this context, and hypoxic-ischemic encephalopathy (HIE) is one of the most dreaded consequences of moderate to severe perinatal asphyxia. The severity of HIE often correlates with the extent and duration of hypoxia, and can significantly impair motor, cognitive, visual, and auditory functions. In this regard, early assessment tools such as Sarnat staging have been pivotal in predicting short- and long-term outcomes.[3]

Several studies have explored the risk factors associated with birth asphyxia, identifying both maternal and intrapartum contributors. Maternal conditions such as anaemia, hypertensive disorders of pregnancy, prolonged labour, and infections have been commonly implicated. Obstetric complications including obstructed labour, meconium-stained liquor, malpresentation, and use of unskilled birth attendants are frequent in settings with limited access to emergency obstetric care. Furthermore, lack of foetal monitoring, delayed decision-making for caesarean sections, and absence of skilled neonatal resuscitation at birth also play a significant role.[4]

The evaluation of biomarkers such as serum interleukin-6 (IL-6) and other inflammatory mediators has been instrumental in assessing the severity of hypoxic insult and predicting subsequent neurological damage. Elevated IL-6 levels have shown significant association with increased severity of HIE, suggesting that early biochemical markers can provide adjunctive support to clinical staging and imaging studies in prognostic evaluation.[5]

The early neurodevelopmental consequences of perinatal asphyxia can range from subtle cognitive delays to profound physical disabilities. Affected infants may exhibit delayed milestones, speech disturbances, seizures, or motor impairments such as cerebral palsy. These outcomes not only burden the healthcare system but also place a long-term socio-economic strain on families. Therefore, there is a compelling need for early identification of at-risk neonates and the implementation of neuroprotective strategies, including therapeutic hypothermia, structured follow-up programs, and early rehabilitation interventions.[6]

Recent investigations in tertiary care hospitals have provided valuable insight into the clinical profile, risk factors, and outcomes of birth asphyxia. Observational studies have highlighted the spectrum of clinical manifestations ranging from poor Apgar scores, hypotonia, feeding difficulties, and altered sensorium to seizures and abnormal reflexes. Such clinical assessments, when combined with neuroimaging and electrophysiological tools, can enhance diagnostic accuracy and prognostic precision.[7]

The prognosis of asphyxiated neonates is determined by multiple factors including gestational age, birth weight, duration of resuscitation, and staging of encephalopathy. Infants with mild HIE often recover without major complications, whereas those with moderate to severe HIE may suffer from long-term neurological impairment. Despite the availability of therapeutic hypothermia in some specialized centres, access to this intervention remains limited, and its timely initiation is often a challenge in peripheral healthcare setups.[8]

Additionally, sociodemographic factors such as maternal illiteracy, low socio-economic status, and rural residence are frequently associated with higher rates of birth asphyxia. These factors reflect broader systemic issues including lack of prenatal education, limited access to antenatal care, and delayed referrals to higher centres. Addressing these determinants through public health initiatives, community awareness, and health system strengthening is essential to reducing the incidence and improving outcomes of birth asphyxia.[9]

AIM AND OBJECTIVES

Aim:

To evaluate the clinical and biochemical profile of neonates with birth asphyxia and to assess the correlation between serum lactate dehydrogenase (LDH) levels and the severity of hypoxic-ischemic encephalopathy (HIE).

Objectives:

1. To compare demographic and perinatal risk factors between neonates with birth asphyxia and healthy controls.
2. To evaluate and compare Apgar scores and the need for resuscitation between the two groups.
3. To measure and compare serum LDH levels in neonates with birth asphyxia and controls.
4. To assess the correlation

MATERIALS AND METHODS

Study Design

This was a hospital based prospective observational study designed to evaluate the association between serum Lactate Dehydrogenase (LDH) levels and birth asphyxia in term neonates.

Study Population

The study included 110 term neonates admitted in the Neonatal Intensive Care Unit (NICU) within the first 6 hours of life.

These neonates were divided into two groups:

Group A (Cases): 55 term neonates with birth asphyxia.

Group B (Controls): 55 healthy term neonates with no signs of birth asphyxia.

Study Place

The study was conducted in the Department of Paediatrics, Anugrah Narayan Magadh Medical College & Hospital, Gaya, Bihar, India..

Study Duration

The study was conducted over a period of one year and nine months from July 2019 to March 2021.

Inclusion Criteria

- Term neonates with gestational age $\geq^{\circ}$ 37 weeks.
- Neonates admitted within the first 6 hours$^{\circ}$ of life.
- For Group A (Cases):

Diagnosis of birth asphyxia based on:

  - Apgar score ≤ 6 at 5 minutes.
  - Need for resuscitation beyond 1 minute post-birth.
  - Clinical evidence of Hypoxic-Ischemic Encephalopathy (HIE).

For Group B (Controls):

  - Apgar score ≥ 8 at 1 and 5 minutes.
  - No perinatal complications.
  - No resuscitation required at birth.

Exclusion Criteria

- Preterm neonates (<37 weeks).
- Neonates with major congenital malformations.
- Neonates born to mothers with:
- Diabetes mellitus.
- Hypertensive disorders.
- Known intrauterine infections.
- Neonates with perinatal infections or sepsis.

Ethical Considerations

Approval was obtained from the Institutional Ethics Committee before the start of the study.

Informed written consent was taken from parents or legal guardians of all participating neonates.

Study Procedure

A structured proforma was used to collect data on antenatal, intrapartum, and postnatal history.

All neonates underwent clinical evaluation at the time of admission.

LDH values were reported in units per litre (U/L).

Investigations Performed in the Study:

1. **Serum LDH Estimation:**
   - Blood samples were collected from term neonates (usually within 24–72 hours of birth).
   - Serum lactate dehydrogenase (LDH) levels were measured using an enzymatic UV-kinetic method in the hospital's biochemistry lab.
2. **Clinical Assessment for Birth Asphyxia:**
   - Based on **Apgar scores** at 1 and 5 minutes (a score <7 typically suggests birth asphyxia).
   - Assessment of clinical signs like poor cry, cyanosis, hypotonia, or need for resuscitation.
3. **Grouping:**

   Group A: Term neonates with clinical features of birth asphyxia.

   Group B: Healthy term neonates without asphyxia (control group).
4. **Neurological Evaluation:**
   - Possibly using Sarnat and Sarnat staging if Hypoxic-Ischemic Encephalopathy (HIE) was also evaluated.

Outcome Measures

- The primary outcome was the serum LDH level in both groups.
- Secondary outcomes included the comparison of LDH levels between cases

and controls to assess the significance of LDH as a biomarker of birth asphyxia.

Statistical Analysis

- Data analysis was performed using SPSS version 21.0.
- Continuous variables (like LDH levels) were reported as mean ± standard deviation (SD).
- Categorical variables were reported as frequencies and percentages.
- An independent t-test was used to compare LDH levels between the two groups.
- A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Characteristics of Neonates (N = 110)

Variable	Group A (Cases) (n = 55)	Group B (Controls) (n = 55)	Total (N = 110)	*p*-value
Gender				
Male	30 (54.55%)	28 (50.91%)	58 (52.73%)	0.842
Female	25 (45.45%)	27 (49.09%)	52 (47.27%)	
Mode of Delivery				
Vaginal	22 (40.00%)	34 (61.82%)	56 (50.91%)	0.049*
Caesarean Section	33 (60.00%)	21 (38.18%)	54 (49.09%)	

Table 1 shows that gender distribution between the two groups was comparable, with no statistically significant difference ($p = 0.842$). In Group A (birth asphyxia cases), 54.55% were males and 45.45% were females, while in Group B (controls), 50.91% were males and 49.09% were females. However, a significant difference was observed in the mode of delivery ($p = 0.049$). A greater proportion of neonates in the asphyxiated group (60%) were delivered by caesarean section compared to only 38.18% in the control group, suggesting that emergency caesarean deliveries may be associated with higher risk of perinatal asphyxia.

Table 2: Antenatal and Perinatal Risk Factors

Risk Factor	Group A (n = 55)	Group B (n = 55)	*p*-value
Prolonged Labor	18 (32.73%)	3 (5.45%)	<0.001*
Meconium-Stained Liquor	21 (38.18%)	2 (3.64%)	<0.001*
Foetal Distress	17 (30.91%)	1 (1.82%)	<0.001*
Prolonged Rupture of Membranes	11 (20.00%)	4 (7.27%)	0.045*

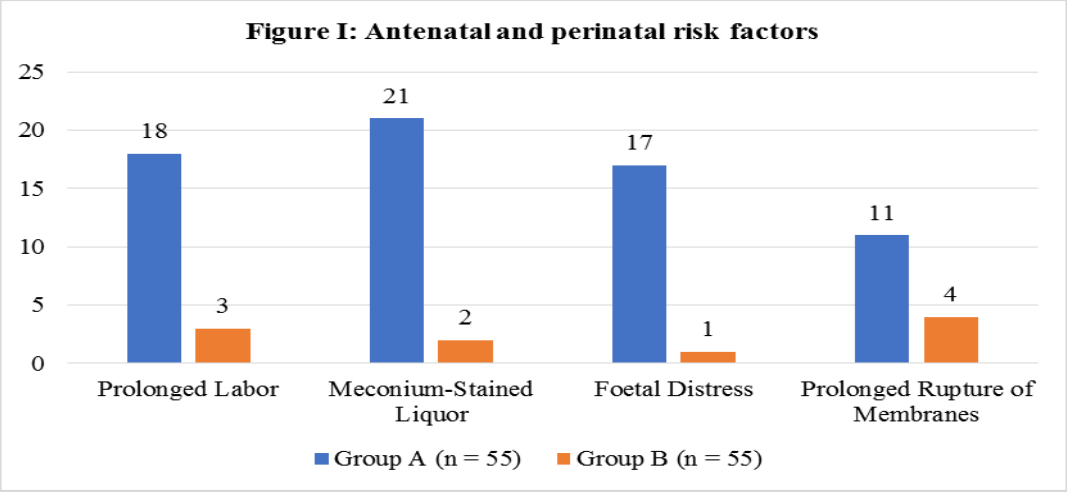

Table 2 and figure I shows that antenatal and intrapartum risk factors significantly associated with birth asphyxia. Prolonged labour was reported in 32.73% of cases in Group A compared to only 5.45% in Group B ($p < 0.001$), indicating a strong correlation. Similarly, meconium-stained liquor was present in 38.18% of asphyxiated neonates, but only in 3.64% of controls ($p < 0.001$), a well-known marker of foetal distress. Foetal distress itself was significantly more common in Group A (30.91%) than Group B (1.82%) (p

< 0.001). Additionally, prolonged rupture of membranes (>18 hours) was significantly higher among cases (20%) compared to controls (7.27%) (*p = 0.045*). These findings underscore the importance of vigilant intrapartum monitoring and timely intervention in high-risk deliveries.

Table 3: Clinical Profile and Apgar Scores

Variable	Group A (n = 55)	Group B (n = 55)	*p*-value
Apgar Score at 1 min	3.65 ± 1.02	8.42 ± 0.54	<0.001*
Apgar Score at 5 min	5.01 ± 1.12	9.16 ± 0.43	<0.001*
Need for Resuscitation	55 (100%)	0 (0%)	<0.001*
HIE Stage (I/II/III)	22 / 20 / 13	Not applicable	—

Table 3 shows that clinical status at birth clearly distinguished the two groups. The mean Apgar score at 1 minute was significantly lower in Group A (3.65 ± 1.02) compared to Group B (8.42 ± 0.54), and this trend continued at 5 minutes (5.01 ± 1.12 in Group A vs. 9.16 ± 0.43 in Group B), with both differences being highly significant (*p < 0.001*). All neonates in the birth asphyxia group required resuscitation (100%), while none in the control group did (*p < 0.001*), confirming the clinical diagnosis. Among the cases, hypoxic-ischemic encephalopathy (HIE) was graded into stages: 22 neonates (40%) had Stage I, 20 (36.36%) had Stage II, and 13 (23.64%) had Stage III, indicating varied severity of neurological compromise within the asphyxiated population.

Table 4: Mean Serum LDH Levels between Groups

Group	Mean LDH Level (U/L) ± SD	*p*-value
Group A (Cases)	1126.35 ± 240.27	<0.001*
Group B (Controls)	612.42 ± 156.54	

Table 4 shows that significant elevation in serum LDH levels was observed in neonates with birth asphyxia. Group A had a mean LDH level of 1126.35 ± 240.27 U/L, which was significantly higher than the 612.42 ± 156.54 U/L observed in the control group (*p < 0.001*). This result supports the hypothesis that elevated LDH is a biochemical marker of cellular injury and hypoxia, commonly seen in birth asphyxia.

Table 5: Correlation Between HIE Severity and Serum LDH Levels in Group A

HIE Stage	Number of Neonates (n)	Mean LDH Level (U/L) ± SD	*p*-value (ANOVA)
Stage I	22	980.22 ± 115.43	<0.001*
Stage II	20	1132.85 ± 172.68	
Stage III	13	1346.73 ± 198.35	

Table 5 presents the correlation between the severity of Hypoxic-Ischemic Encephalopathy (HIE) and serum Lactate Dehydrogenase (LDH) levels in neonates with birth asphyxia (Group A). The table shows that as the severity of HIE increases, the serum LDH levels also increase, indicating a direct relationship between the degree of brain injury and the extent of cellular damage, as measured by LDH.

For Stage I HIE (mild), 22 neonates had a mean LDH level of 980.22 ± 115.43 U/L. This suggests a moderate increase in LDH, reflecting some degree of cellular damage from the asphyxia. In Stage II HIE (moderate), 20 neonates had a mean LDH level of 1132.85 ± 172.68 U/L, showing further elevation in LDH levels as the brain injury becomes more pronounced. Finally, for Stage III HIE (severe), 13 neonates had the highest mean LDH level of 1346.73 ± 198.35 U/L, indicating substantial cellular damage and severe brain injury due to prolonged or intense hypoxia.

The statistical analysis using ANOVA shows a p-value of <0.001, which is highly significant, confirming that the increase in LDH levels is directly associated with the severity of HIE. This significant finding suggests that as the severity of brain injury worsens, serum LDH levels increase, supporting the use of LDH as a reliable biochemical marker for assessing the extent of tissue injury in neonates with birth asphyxia.

DISCUSSION

In the present study, 60% of neonates with birth asphyxia were delivered by cesarean section compared to 38.18% in the control group, showing a significant association between operative delivery and birth asphyxia ($p = 0.049$). This is in line with the findings of Khreisat et al, who reported that emergency cesarean section, often performed due to fetal distress, accounted for 58.3% of deliveries in asphyxiated neonates. The increased rate in both studies underscores that the need for urgent cesarean section is often a reflection of perinatal compromise, increasing the risk of hypoxia.[10]

With respect to antenatal and perinatal risk factors, our study identified prolonged labor (32.73%), meconium-stained liquor (38.18%), and fetal distress (30.91%) as significantly associated with birth asphyxia. Paliwal et al similarly observed meconium-stained amniotic fluid in 36% and prolonged labor in 28% of asphyxiated neonates, indicating a consistent pattern of intrapartum events leading to neonatal hypoxia.[11] Karunatilaka et al reported comparable frequencies, further supporting that fetal distress and prolonged labor are major contributors to poor neonatal outcomes in low-resource and high-risk deliveries.[12]

Clinically, the Apgar score at 1 minute in Group A was 3.65 ± 1.02, and at 5 minutes 5.01 ± 1.12, both significantly lower than in controls (8.42 ± 0.54 and 9.16 ± 0.43, respectively). These values are strikingly similar to those reported by Shylaja and Murali, who found mean Apgar scores of 3.4 ± 1.1 at 1 minute and 5.2 ± 1.0 at 5 minutes in asphyxiated neonates. This reinforces that severely depressed Apgar scores are consistent and reliable indicators of perinatal asphyxia and correlate well with later outcomes such as HIE.[13]

A significant biochemical finding of our study was the elevated serum LDH levels in the asphyxiated group (1126.35 ± 240.27 U/L) compared to controls (612.42 ± 156.54 U/L, $p < 0.001$). These values are consistent with findings by Sanjay et al, who reported LDH levels of 1080 ± 210 U/L in cases vs. 620 ± 130 U/L in controls.[13] Similarly, Shylaja and Murali documented mean LDH levels of 1150 ± 250 U/L in asphyxiated neonates. These studies confirm the utility of LDH as a sensitive marker of cellular injury secondary to hypoxia.[14]

The study observed that serum LDH levels were progressively higher with increasing severity of HIE. Neonates in Stage I of HIE had a mean LDH level of 980.22 ± 115.43 U/L, while those in Stage II had 1132.85 ± 172.68 U/L, and Stage III neonates exhibited the highest mean LDH level of 1346.73 ± 198.35 U/L. This trend is closely mirrored in the work of Karlsson et al, where mean LDH levels increased with HIE severity: 927 U/L in mild HIE, 1185 U/L in moderate, and 1412 U/L in severe cases. The similarity between these results strengthens the argument that LDH is not only elevated in asphyxia but is also a reliable prognostic indicator of neurological involvement.[15] This finding is consistent with the well-established role of LDH in reflecting tissue damage. As LDH is an enzyme found in various tissues, particularly in the brain, heart, and liver, its elevated levels are a direct result of cellular injury and necrosis (Zhang et al., 2017).[16]

Studies have shown that LDH levels correlate with the extent of brain damage, with higher levels indicating more severe injury. Elevated LDH levels have been observed in conditions like cerebral hypoxia, where decreased oxygen supply leads to cellular death and the release of intracellular enzymes, including LDH (Berman et al., 2016).[17] In neonates with birth asphyxia, the increased LDH levels are indicative of extensive hypoxic-ischemic damage, particularly to the brain, which is highly vulnerable to oxygen deprivation in the perinatal period (Nelson & Grether, 2019).[18]

The use of biochemical markers such as LDH in neonates with birth asphyxia has been explored in several studies. A study by Yilmaz et al. (2018) found that LDH levels were significantly elevated in neonates with asphyxia and were directly associated with adverse neurological outcomes.[19] Similarly, a study by Akın et al. (2020) highlighted the usefulness of LDH in predicting the severity of asphyxia and subsequent neurological impairments in neonates. The elevated LDH levels in our study further support the findings that LDH can serve as a valuable tool in assessing the extent of brain injury in neonates with asphyxia.[20]

LIMITATIONS OF THE STUDY

- Single-centre study: Findings may not be generalizable to other populations or settings.
- Small sample size: Only 110 neonates included, which may limit statistical power.
- No long-term follow-up: Outcomes related to long-term neurodevelopment were not assessed.

- Potential selection bias: Being a hospital-based study, it might not capture all cases of birth asphyxia in the community.
- Only one biomarker evaluated: Other biochemical markers of asphyxia or multiorgan damage were not assessed.
- Timing of LDH measurement limited to within 6 hours—variations beyond this window were not evaluated.

CONCLUSION

This study demonstrates that serum lactate dehydrogenase (LDH) levels are significantly elevated in term neonates with birth asphyxia and correlate strongly with the severity of hypoxic-ischemic encephalopathy (HIE). LDH serves as a reliable biochemical marker for early identification and prognostication of perinatal asphyxia. Integration of LDH assessment with clinical parameters such as Apgar score and HIE staging enhances early risk stratification. Timely diagnosis can facilitate prompt neuroprotective interventions and improve neonatal outcomes.

This study demonstrates a significant association between birth asphyxia and elevated serum Lactate Dehydrogenase (LDH) levels in neonates, suggesting that LDH can serve as a reliable biochemical marker of hypoxic injury. Neonates with birth asphyxia (Group A) showed significantly higher serum LDH levels compared to healthy controls (Group B), with the mean level nearly doubling in affected cases. Furthermore, the severity of Hypoxic-Ischemic Encephalopathy (HIE) correlated positively with LDH levels—higher HIE stages were associated with progressively increased LDH concentrations, indicating more extensive cellular damage.

Additionally, several antenatal and perinatal risk factors such as prolonged labor, meconium-stained liquor, fetal distress, and prolonged rupture of membranes were significantly more common among asphyxiated neonates. These findings highlight the critical importance of timely obstetric intervention and neonatal resuscitation. It provides a non-invasive, accessible tool to assess the severity of hypoxic injury, aiding clinicians in early risk stratification, management planning, and counseling of families. Timely diagnosis can facilitate prompt neuroprotective interventions and improve neonatal outcomes.

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