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Original Article

Umbilical Cord Arterial Blood Lactate Dehydrogenase Levels as an Early Predictor of Adverse Perinatal Outcomes in High-Risk Term Pregnancies: A Cross-Sectional Observational Study

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ABSTRACT

Background: Perinatal hypoxia remains a leading cause of neonatal morbidity and mortality. Umbilical cord blood biomarkers, particularly lactate dehydrogenase (LDH), offer objective assessment of fetal distress in high-risk pregnancies.

Aims and Objectives: This study aimed to evaluate the association between umbilical cord arterial blood LDH levels and adverse perinatal outcomes in high-risk term pregnancies and to determine its predictive efficacy for early risk stratification.

Materials and Methods: A cross-sectional observational study was conducted in the NICU of a tertiary care hospital over 12 months (March 2024–March 2025). Three hundred neonates born to women with high-risk term singleton pregnancies were enrolled consecutively. Umbilical cord arterial blood LDH levels were measured immediately after delivery. Maternal and neonatal clinical variables, Apgar scores, resuscitation needs, NICU admission, and complications were recorded. Statistical analysis included Mann-Whitney U tests, Spearman correlations, ROC curve analysis, and logistic regression ($p < 0.05$ significant).

Results: Median cord LDH was significantly higher in neonates with adverse outcomes, resuscitation requirement, NICU admission, and complications ($p < .001$). Strong inverse correlations existed with Apgar scores and positive correlations with oxygen therapy and hospital stay duration. ROC analysis showed AUC 0.884 (95% CI 0.842–0.926) at cutoff ≥985 U/L (sensitivity 84.4%, specificity 89.0%). Elevated LDH was the strongest independent predictor (aOR 34.62, $p < .001$).

Conclusion: Umbilical cord arterial LDH is a reliable early predictor of adverse perinatal outcomes in high-risk term pregnancies, supporting its use for timely intervention and optimized neonatal care.

Keywords: Lactate Dehydrogenase; Umbilical Cord; Asphyxia Neonatorum; Perinatal Care; High-Risk Pregnancy; Hypoxia, Brain.

INTRODUCTION

Globally, significant advancements in healthcare infrastructure and obstetric management have contributed to a downward trend in childhood mortality over recent decades, yet reducing neonatal mortality remains a critical global challenge [1]. Recognizing and managing disorders in newborns during the immediate postnatal period is exceptionally challenging because early clinical presentations are frequently diverse, subtle, and highly nonspecific [2].

Among the various pathophysiological insults, perinatal hypoxia and subsequent birth asphyxia stand out as leading causes of preventable organ injury. In conditions where tissue perfusion or oxygen concentration becomes severely deficient, cellular metabolism shifts dynamically from aerobic to anaerobic pathways [3]. Under these anaerobic conditions, glucose is metabolized less efficiently, causing an accumulation of pyruvate, which is subsequently deoxidized into lactate by lactate dehydrogenase. Lactate dehydrogenase (LDH) is a ubiquitous intracellular enzyme found across almost all major organ systems that responds directly to energy shortages and cellular distress [4].

To identify infants at risk before irreversible damage occurs, objective diagnostic strategies are vital. Umbilical cord blood analysis has emerged as a reliable approach for the early prediction of hypoxia-related damages and metabolic acidosis in the fetus [5]. Traditionally, clinical assessment tools like cord arterial blood pH and base deficit estimation have been utilized to evaluate the severity of intrapartum distress, alongside lactate dehydrogenase level estimation [6].

Hypoxia triggers widespread systemic organ damage, and the fetal liver is highly vulnerable to hypoxic insults, resulting in an early, abrupt, and transient increase in aminotransferases, alkaline phosphatase, and LDH levels. Under normal circumstances, fetal lactate generated during hypoxic episodes is cleared continuously across the placenta. When severe or sustained intrapartum hypoxia impairs this clearance mechanism, umbilical artery lactate levels rise significantly, making them an efficient and accurate technique for diagnosing fetal hypoxia [7].

LDH levels correlate well with disease severity, such as asphyxia, respiratory distress, necrotizing enterocolitis, and hypoxic-ischemic encephalopathy (HIE) [8]. Clinical studies emphasize the utility of cord blood biomarkers in high-risk pregnancies, where data suggest that high cord blood lactate values are significantly associated with adverse neonatal outcomes in high-risk term pregnancies. Investigating these biochemical fluctuations helps clinicians risk-stratify neonates immediately at birth, facilitating proactive management [9, 10].

This study sought to determine the association between cord blood LDH and perinatal outcomes in high-risk term pregnancies. The primary aim of this study is to determine the role of umbilical cord arterial blood LDH levels in the early prediction of adverse perinatal outcomes in term neonates of high-risk pregnancy, to reduce the overall burden of perinatal morbidity and mortality associated with birth asphyxia. The specific objectives are to demonstrate that umbilical cord arterial blood LDH can be used for early prediction and intervention, thereby optimizing neonatal intensive care resource utilization and achieving the secondary outcome of reducing overall neonatal morbidity and mortality.

MATERIALS AND METHODS

Study Setting: This study was conducted as a cross-sectional observational study. The designated study center was the Neonatal Intensive Care Unit (NICU) at the Government Kasturba Gandhi Hospital for Women and Children, located in Chepauk, Chennai. The investigation was carried out over a strict study duration of twelve months, spanning from March 2024 to March 2025.

Study Participants: The study population comprised neonates delivered to antenatal women categorized as having high-risk pregnancies. To be eligible for inclusion, neonates had to be born to antenatal women at a gestational age $\geq$ 37 weeks, representing term gestations, and must have been from singleton pregnancies. The pregnancies had to present with one or more established high-risk factors, which included maternal anemia, hypertensive disorders of pregnancy, a previous history of cesarean section, malpresentation, thyroid disorders, gestational diabetes mellitus, seizure disorders, Rh incompatibility, intrauterine growth restriction (IUGR), oligohydramnios, polyhydramnios, prelabour rupture of membranes (PROM), maternal infections, or intrahepatic cholestasis of pregnancy. Additionally, obtaining written informed consent from the parent or legal guardian was a strict prerequisite for enrollment.

The study excluded neonates born to antenatal women with preterm or post-term delivery (defined as 42 weeks). Neonates derived from low-risk or normal pregnancies, cases involving intrauterine fetal death, or infants diagnosed with a congenitally malformed fetus were also excluded. Furthermore, multifetal gestations and cases where the family refused to participate were excluded from the final sample.

Sample Size and Sampling Technique: The required sample size for this study was determined to be 300 neonates. This sample size was calculated using OpenEpi software, assuming a 95% confidence interval and an 80% statistical power. To recruit the required number of participants, a consecutive sampling technique was implemented, wherein every eligible neonate meeting the predefined criteria was enrolled sequentially until the required duration of the study was reached.

Study Tools: The primary study tools utilized for data collection, clinical evaluation, and biochemical assessment consisted of two primary components: the patient's comprehensive case records and the quantified cord blood LDH levels. The clinical case records comprised a structured data collection sheet and a final proforma utilized by the pediatrician to capture maternal demographics, obstetric variables, and direct neonatal outcomes. This documentation included baby details (such as name, age, sex, birth weight, weight classification like AGA/SGA/LGA, date and time of birth, Apgar scores, mode of delivery, indication for admission, and provisional diagnosis) and maternal profiles (including age, parity, LMP, EDD, gestational age, blood group, specific maternal risk factors, indications for LSCS, and maternal medications).

The second primary tool, the biochemical measurement of LDH levels, was derived directly from umbilical cord arterial blood collected in a 0.5 ml lithium heparin tube immediately post-delivery. Supplementary tools integrated within the clinical records included standard laboratory investigation charts (tracking parameters such as CBC, TC, RBC/Hb, PCV, DC, PLT, CRP, serum calcium, and USG imaging) and treatment monitoring tables designed to quantify the exact duration of clinical interventions, such as supplemental oxygen, antibiotics, inotropes, phototherapy, total duration of hospital stay, and final outcome of the baby.

Study Procedure: Upon identification of an eligible high-risk pregnancy, the clinician provided a written study information handout to the primary caregiver, verbally explained the details and implications of the procedure, and obtained formal written consent. Maternal socio-demographic features, including age, gestation, parity, high-risk factors, and the mode of delivery, were systematically recorded on the structured data collection sheet. Immediately following either a vaginal or a cesarean delivery, the umbilical cord was doubly clamped and cut at two places to isolate a segment of 10–12 cm.

Umbilical cord arterial blood samples were then carefully drawn from this isolated segment using a sterile syringe, and 0.5 ml of the collected blood sample was immediately transferred into a lithium heparin tube for specialized LDH biochemical analysis. Following delivery, the attending pediatrician recorded neonatal details on the final datasheet, including the gender of the baby, birth weight, Apgar scores determined at 1 minute and 5 minutes, the immediate need for resuscitation, requirement for NICU admission, development of early neonatal complications, and the overall neonatal outcome. Primary and secondary clinical outcomes were fully assessed at the conclusion of the study period.

Ethical Issues: Prior to commencing the study, permission was obtained from the institutional ethical committee. The study maintained participant confidentiality. There was no potential harm caused to the child during the course of this study. Any unforeseen adverse reactions or complications, had they occurred, were documented, treated, and the child would be excluded from this study.

Statistical Analysis: Data were analyzed using Jamovi 2.3.21 statistical analysis software for windows. Descriptive statistics was done for continuous variables like maternal age, birth weight, and cord blood LDH levels as mean ± standard deviation or median with interquartile range based on normality testing, while categorical variables will be computed using frequencies and percentages with a 95% confidence interval. Inferential analysis included Independent Samples t-tests or Mann-Whitney U tests to compare LDH levels across binary neonatal outcomes. Chi-square tests will assess categorical clinical associations. Spearman's correlation analyzed relationships between LDH levels, Apgar scores, and duration of hospital stay. Receiver Operating Characteristic (ROC) curve analysis determined the diagnostic efficacy, Area Under the Curve (AUC), and optimal predictive cut-off values of cord blood LDH for adverse perinatal outcomes. In all these tests, p-value of less than 0.05 was considered to be statistically significant.

RESULTS

A total of 300 neonates born to high-risk mothers were included in the analysis. Maternal age distribution did not differ significantly between outcome groups (p = .482). However, the mode of delivery demonstrated a significant association with adverse perinatal outcomes, with emergency lower segment cesarean section (LSCS) occurring more frequently among neonates with adverse outcomes than among those with normal outcomes (p = .015). Among maternal high-risk conditions, hypertensive disorders of pregnancy and severe intrauterine growth restriction (IUGR) were significantly associated with adverse neonatal outcomes (both p < .001) (Table 1).

Table 1: ***Baseline Maternal and Neonatal Categorical Characteristics and Their Association With Adverse Perinatal Outcomes (N = 300)***

Variable	Total Cohort, *n* (%)	Normal Outcome (*n* = 210)	Adverse Outcome (*n* = 90)	Test Statistic (*p*)	95% CI
Maternal age (years)					
< 25	84 (28.0)	62 (29.5)	22 (24.4)	.482	[23.0, 33.5]
25–35	186 (62.0)	131 (62.4)	55 (61.1)	Reference	[56.2, 67.5]

> 35	30 (10.0)	17 (8.1)	13 (14.5)	.124	[7.0, 14.0]
Mode of delivery					
Vaginal delivery	126 (42.0)	98 (46.7)	28 (31.1)	.015	[36.4, 47.8]
Emergency LSCS	174 (58.0)	112 (53.3)	62 (68.9)	.015	[52.2, 63.6]
Primary high-risk factors					
Hypertensive disorders	96 (32.0)	52 (24.8)	44 (48.9)	< .001	[26.8, 37.6]
Maternal anemia	72 (24.0)	45 (21.4)	27 (30.0)	.112	[19.3, 29.3]
Severe IUGR	42 (14.0)	18 (8.6)	24 (26.7)	< .001	[10.4, 18.5]
Oligohydramnios/Polyhydramnios	48 (16.0)	38 (18.1)	10 (11.1)	.134	[12.1, 20.7]
Other conditions (PROM, GDM, etc.)	42 (14.0)	57 (27.1)	29 (32.2)	.378	[10.4, 18.5]
Neonatal sex					
Male	162 (54.0)	114 (54.3)	48 (53.3)	.881	[48.2, 59.7]
Female	138 (46.0)	96 (45.7)	42 (46.7)	.881	[40.3, 51.8]

Note. LSCS = lower segment cesarean section; IUGR = intrauterine growth restriction; PROM = prelabor rupture of membranes; GDM = gestational diabetes mellitus.

Median LDH concentrations were significantly higher among neonates requiring immediate resuscitation compared with those who did not require resuscitation (U = 2,145.5, p < .001). Similarly, neonates admitted to the neonatal intensive care unit (NICU) demonstrated markedly elevated LDH concentrations relative to those not requiring admission (U = 2,412.0, p < .001) (Table 2).

Table 2: ***Comparison of Umbilical Cord Arterial Blood LDH Levels Across Neonatal Clinical Outcomes (N = 300)***

Neonatal Clinical Parameter	***n***	**Median LDH (U/L)**	**IQR (U/L)**	**Mann–Whitney *U***	***p***
Immediate need for resuscitation					
Yes	78	1,185.5	895.0–1,462.0	2,145.5	< .001
No	222	684.0	542.5–846.0	—	—
NICU admission					
Admitted	90	1,142.0	884.5–1,410.5	2,412.0	< .001
Not admitted	210	672.0	536.0–814.5	—	—
Early neonatal complications					
Present (Asphyxia, HIE, RDS)	66	1,248.0	954.0–1,518.0	1,384.0	< .001
Absent	234	702.5	554.0–864.0	—	—

Note. LDH = lactate dehydrogenase; IQR = interquartile range; NICU = neonatal intensive care unit; HIE = hypoxic-ischemic encephalopathy; RDS = respiratory distress syndrome.

Cord blood LDH levels demonstrated strong inverse correlations with both the 1-minute Apgar score (r = −.684, p < .001) and the 5-minute Apgar score (r = −.712, p < .001), indicating that higher LDH concentrations were associated with poorer neonatal condition immediately after birth. Positive correlations were observed between LDH levels and duration of oxygen therapy (r = .586, p < .001) as well as duration of hospital stay (r = .624, p < .001) (Table 3).

Table 3: ***Spearman Rank Correlation Matrix Between Umbilical Cord Blood LDH Levels and Continuous Clinical Variables***

Variable	**1**	**2**	**3**	**4**	**5**	**6**
1. Cord blood LDH (U/L)	1.000					
2. Apgar score (1 min)	−.684	1.000				
3. Apgar score (5 min)	−.712	.842	1.000			
4. Duration of oxygen therapy (days)	.586	−.514	−.562	1.000		
5. Duration of hospital stay (days)	.624	−.498	−.548	.742	1.000	
6. Birth weight (kg)	−.055	.082	.094	−.042	−.061	1.000

Note. Correlations were calculated using Spearman's rank-order correlation coefficient

Receiver operating characteristic (ROC) curve analysis demonstrated excellent discriminative ability, with an area under the curve (AUC) of 0.884 (95% CI [0.842, 0.926], $p < .001$). The optimal diagnostic threshold, determined using Youden's Index, was ≥ 985 U/L. At this cut-off, cord blood LDH demonstrated a sensitivity of 84.4% and a specificity of 89.0%. The positive predictive value (PPV) and negative predictive value (NPV) were 76.8% and 93.1%, respectively (Table 4).

Table 4: ***Receiver Operating Characteristic (ROC) Analysis of Umbilical Cord Blood LDH for Predicting Adverse Perinatal Outcomes***

Diagnostic Parameter	Value	95% CI	*p*
Area under the curve (AUC)	0.884	0.842–0.926	< .001
Optimal cut-off value	≥ 985 U/L	—	
Sensitivity (%)	84.4	75.3–91.2	
Specificity (%)	89.0	84.1–92.9	
Positive predictive value (PPV, %)	76.8	67.2–84.7	
Negative predictive value (NPV, %)	93.1	88.8–96.1	
Youden's Index (*J*)	0.734	—	

Note. ROC = receiver operating characteristic; AUC = area under the curve; LDH = lactate dehydrogenase; CI = confidence interval; PPV = positive predictive value; NPV = negative predictive value. The optimal cut-off value was determined using Youden's Index.

In the univariate analysis, neonates with cord blood LDH levels ≥ 985 U/L had 41.25 times greater odds of developing adverse perinatal outcomes than those with lower LDH concentrations (95% CI [21.42, 79.44], $p < .001$). After adjustment for maternal age, gestational age, hypertensive disorders of pregnancy, emergency lower segment cesarean section (LSCS), and severe intrauterine growth restriction (IUGR), elevated cord blood LDH remained the strongest independent predictor of adverse outcomes (adjusted OR [aOR] = 34.62, 95% CI [16.84, 71.18], $p < .001$). Hypertensive disorders of pregnancy (aOR = 2.14, $p = .021$) and severe IUGR (aOR = 2.84, $p = .011$) also remained significant independent predictors. In contrast, maternal anemia, emergency LSCS, and gestational age were not independently associated with adverse outcomes after multivariable adjustment (Table 5).

Table 5: ***Univariate and Multivariable Logistic Regression Analysis of Factors Associated With Adverse Perinatal Outcomes***

Predictor	Crude OR	95% CI	*P*	Adjusted OR	95% CI	*P*
Elevated LDH (≥ 985 U/L)	41.25	21.42–79.44	< .001	34.62	16.84–71.18	< .001
Hypertensive disorders	2.91	1.74–4.86	< .001	2.14	1.12–4.08	.021
Severe maternal anemia	1.57	0.89–2.76	.114	1.22	0.61–2.44	.574
Severe IUGR	3.88	1.98–7.61	< .001	2.84	1.28–6.32	.011
Emergency LSCS	1.94	1.14–3.29	.016	1.48	0.78–2.82	.228
Gestational age (per week)	0.84	0.62–1.14	.264	0.91	0.64–1.30	.608

Note. OR = odds ratio; aOR = adjusted odds ratio; CI = confidence interval; LDH = lactate dehydrogenase; IUGR = intrauterine growth restriction; LSCS = lower segment cesarean section.

DISCUSSION

The present study demonstrates a robust association between elevated umbilical cord arterial blood lactate dehydrogenase (LDH) levels and adverse perinatal outcomes in neonates born to high-risk term pregnancies. In a cohort of 300 neonates, cord blood LDH concentrations were significantly higher in those requiring resuscitation, NICU admission, or experiencing early complications such as birth asphyxia, hypoxic-ischemic encephalopathy (HIE), and respiratory distress syndrome (RDS). The median LDH was markedly elevated in affected groups (e.g., 1,185.5 U/L vs. 684.0 U/L for resuscitation need; $p < .001$), highlighting LDH's utility as an early biochemical marker of hypoxic stress.

These findings align with the established pathophysiology of perinatal hypoxia. During intrapartum oxygen deprivation, fetal tissues shift to anaerobic glycolysis, leading to pyruvate accumulation and its conversion to lactate via LDH. This enzyme, ubiquitous in tissues including liver, heart, and brain, is released upon cellular injury, reflecting both metabolic acidosis and organ-level damage [11].

In high-risk pregnancies—particularly those complicated by hypertensive disorders or severe intrauterine growth restriction (IUGR)—placental insufficiency exacerbates fetal hypoxia, impairing lactate clearance and elevating cord arterial LDH [12]. The study's observation of strong inverse correlations between LDH and Apgar scores ($r = -0.684$ at 1 minute and $r = -0.712$ at 5 minutes; both $p < .001$) further supports this, as lower Apgar scores indicate compromised cardiorespiratory adaptation directly linked to hypoxic insult. Positive correlations with duration of oxygen therapy ($r = 0.586$) and hospital stay ($r = 0.624$) highlight LDH's prognostic value for resource utilization and recovery trajectory [13]. Receiver Operating Characteristic (ROC) analysis revealed excellent discriminative performance, with an area under the curve (AUC) of 0.884 (95% CI 0.842–0.926), confirming LDH's strong predictive accuracy for adverse outcomes. The optimal cutoff of ≥985 U/L yielded 84.4% sensitivity and 89.0% specificity, with a negative predictive value of 93.1%.

The slightly higher cutoff in the present study may reflect population-specific factors, such as the prevalence of hypertensive disorders (48.9% in adverse outcomes) or methodological differences in LDH assay timing and sample handling [14].

Logistic regression reinforced LDH's independent predictive strength. Neonates with LDH ≥985 U/L had over 41-fold increased crude odds of adverse outcomes, remaining highly significant after multivariable adjustment (aOR 34.62, 95% CI 16.84–71.18; $p < .001$). Hypertensive disorders and severe IUGR retained independent associations, consistent with their roles in placental dysfunction and chronic fetal stress. These results echo broader literature linking elevated LDH to severity in preeclampsia and related conditions, where it signals endothelial damage, hemolysis, and multi-organ involvement extending to the fetus [15].

Clinically, the integration of cord blood LDH offers several advantages. Unlike Apgar scoring, which is subjective and influenced by non-hypoxic factors (e.g., maternal medications or resuscitation efforts), LDH provides an objective, quantifiable measure available immediately post-delivery [16]. Routine measurement in high-risk term pregnancies could facilitate rapid risk stratification, enabling timely NICU triage, neuroprotective interventions (such as therapeutic hypothermia for HIE), and optimized resource allocation. This is particularly relevant in resource-constrained settings like tertiary care centers in developing regions, where the study was conducted. Early identification may reduce long-term neurodevelopmental sequelae, a major concern given HIE's contribution to childhood disability [17, 18].

The study's strengths include its prospective consecutive sampling, adequate power (n=300), and comprehensive adjustment for confounders in multivariable analysis. Use of standardized protocols for cord blood collection (immediate arterial sampling from clamped segment) minimizes pre-analytical variability. Exclusion of preterm, malformed, and low-risk cases enhances internal validity for the target high-risk term population. Statistical rigor, employing non-parametric tests for non-normal LDH distribution, Spearman correlations, and ROC/Youden's Index, bolsters reliability.

Limitations must be acknowledged. As a single-center observational study, generalizability may be limited by local demographics, referral patterns, and management protocols at Government Kasturba Gandhi Hospital. Consecutive sampling, while practical, risks selection bias toward more severe cases in a high-volume tertiary NICU. LDH elevation, though highly suggestive of hypoxia, is not entirely specific; other conditions (e.g., hemolysis, infection, or maternal influences) could contribute.

Biomarkers like cord LDH bridge the gap between subtle intrapartum signs and overt neonatal decompensation, aligning with WHO and national guidelines emphasizing early detection in high-risk deliveries. By enabling precise intervention, such strategies could meaningfully lower the burden of perinatal asphyxia-related morbidity.

CONCLUSION

In high-risk term pregnancies, umbilical cord arterial blood LDH levels serve as a reliable, independent predictor of adverse perinatal outcomes, with a cutoff of ≥985 U/L demonstrating excellent diagnostic accuracy. Routine incorporation of this biomarker facilitates early risk stratification, timely interventions, and optimized NICU utilization, ultimately contributing to reduced neonatal morbidity and mortality.

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