The Egyptian Journal of Hospital Medicine (July 2025) Vol. 100, Page 3217-3224

Role of Lactate Dehydrogenase Testing in the Prediction of Severe Conditions in Newborn

Elseaid Rashad Fouda[1], Shaimaa Karam Abdel Alim[1], Hadeel Mohamed Shalaby[2], Amany Ahmed El-Banna[1]
Departments of [1]Pediatrics and [2]Clinical Pathology, Faculty of Medicine, Menoufia University, Egypt
***Corresponding author:** Shaimaa Karam Abdel Alim, **Mobile:** (+20) 010 00377091, **Email:** badawymohamed039@gmail.com

ABSTRACT

Background: The neonatal period, particularly the first week of life, presents significant challenges due to high morbidity and mortality rates. Lactate Dehydrogenase (LDH) has emerged as a promising biomarker for early detection of severe complications in neonates, as it rises during cellular hypoxia and tissue injury.

Objective: This study aimed to investigate the relationship between LDH levels at Neonatal Intensive Care Unit (NICU) admission and the occurrence of severe complications in neonates, evaluating its potential as a predictive biomarker for neonatal outcomes. **Patients and methods:** A cross-sectional study was conducted at the NICU of Menoufia University Hospital. The study included 53 neonates admitted with serious illness symptoms. Comprehensive data collection included demographic information, clinical examinations, and laboratory investigations, including LDH levels.

Results: Among the neonates studied, LDH levels showed significant negative correlations with Apgar scores at 1 and 5 minutes ($r = -0.581$ and $r = -0.530$, respectively, $p \leq 0.001$), pH ($r = -0.488$, $p \leq 0.001$), and oxygen saturation ($r = -0.523$, $p \leq 0.001$). Neonates requiring mechanical ventilation (MV) showed significantly higher LDH levels (1121.6 ± 182.87 U/L, $p \leq 0.001$). ROC curve analysis revealed that LDH had moderate predictive value for mortality (AUC = 0.81, 95% CI: 0.69-0.93), with a cutoff value of 895.50 U/L yielding 79.2% sensitivity and 55.2% specificity.

Conclusion: LDH demonstrated potential as a valuable biomarker for predicting adverse outcomes in neonates, particularly mortality risk. Its significant associations with poor Apgar scores, respiratory support requirements, and various clinical parameters suggest its utility in early risk assessment. However, larger studies are needed to validate these findings and establish definitive clinical guidelines for LDH use in neonatal care.

Keywords: Lactate dehydrogenase, Neonatal mortality, Biomarker, Respiratory distress, NICU, MV.

INTRODUCTION

Critically ill neonates are infants with a broad spectrum of diseases and disorders that emerge pre-, peri- or post-natal, and who need life support treatment in the NICU. The neonatal period, especially the first week of life, is critical due to high rates of morbidity and mortality. Early diagnosis and intervention are essential for improving outcomes in newborns with severe illnesses. However, recognizing these conditions promptly is challenging as neonates often exhibit nonspecific symptoms that are difficult to interpret [(1)]. Clinical signs such as respiratory distress, poor feeding, or general lethargy may indicate serious underlying conditions, but these symptoms alone lack specificity, necessitating reliable biomarkers for early detection and intervention in NICU settings [(2)].

Lactate dehydrogenase (LDH) has emerged as a promising biomarker in this context. An intracellular enzyme involved in anaerobic metabolism. LDH levels rise in response to cellular hypoxia and energy deficit making it an indicator of tissue oxygenation and cellular integrity [(3)]. During hypoxia or tissue injury, cell membranes become compromised, releasing LDH into the plasma. This release occurs in a range of neonatal conditions, from hypoxic-ischemic encephalopathy (HIE) and respiratory distress to sepsis, where LDH levels can reflect the extent of cellular damage and the severity of illness [(4)].

Numerous studies underscore the association between elevated LDH levels and various neonatal conditions. For example, in infants experiencing fetal distress during birth, elevated LDH has shown promise in predicting HIE when a threshold of 1050 U/L is exceeded [(5)]. Similarly, LDH correlates with oxygen requirements in respiratory distress syndrome (RDS), linking LDH to the duration and severity of hypoxic stress in neonates. These findings suggest LDH may offer an accessible, rapid indicator to support early diagnosis and guide therapeutic decisions for neonates in critical care [(6)].

In developing countries, where NICU resources are often limited, the ability to triage neonates effectively is paramount. Identifying reliable biomarkers like LDH could enable quicker assessment, facilitating timely transfer and appropriate care, potentially reducing neonatal mortality and long-term complications. Our study aimed to investigate the relationship between LDH levels at NICU admission and the occurrence of severe complications in neonates, providing further insights into LDH's predictive value in neonatal care.

PATIENTS AND METHODS

Across-sectional study was carried out on 53 newborns at the NICU of Menoufia University Hospital, over a period of one year from January 2022 to January 2023.

Inclusion criteria: Neonates admitted to the NICU during the early neonatal period, who were born at a gestational age of either less than or greater than 37 weeks and exhibited signs of serious illness, including respiratory distress, neonatal sepsis, asphyxia and poor feeding.

Exclusion criteria: Stable neonates who did not require respiratory or cardiac support, such as those admitted solely for jaundice management.

Received: 10/03/2025
Accepted: 08/05/2025

All patients underwent a comprehensive clinical evaluation, which involved full history taking and thorough clinical examination and laboratory investigations.

Sample collection: 5 milliliters of blood were collected from each patient under complete aseptic conditions. The blood was divided into: 1 milliliter in EDTA anticoagulant for complete blood count (CBC) (Sysmex XN1000, Kobe, Japan), 1 milliliter in heparin anticoagulant for ABG using (Gem 3000) and 3 milliliters in a plain vacutainer, left for 10 minutes to clot and the serum was separated by centrifugation. Assay of creatinine, ALT, AST was done using AU680 Beckmen autoanalyser (Bechman, Instrument Inc., USA). CRP was determined by nephelometric method using Agappe Mispa i3 CRP Diagnostic Test Kit (supplied by AGAPPE Diagnostics Switzerland GmbH, Cham, Switzerland) according to manufacturer's instructions. The rest of sera were used for assay of LDH. Detection of LDH level was done using a Human LDH ELISA Kit (1st Industrial area, Obour City, Egypt). Catalogue No:. 1206 101, along with the manufacturer's instructions.

Outcome measures: The primary outcome was to investigate the relationship between LDH levels at NICU admission and the occurrence of severe complications in neonates. LDH was assessed as a potential biomarker for early detection of serious neonatal illness, and its levels were correlated with clinical severity, including the requirement for extended oxygen support, MV, or other critical interventions.

Ethical approval: The Research Ethics Committee of Medical Research, Faculty of Medicine, Menoufia University approved this study with a number and date of 1/2022 PEDI 43. Informed written consents were obtained from all parents. The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis

Data were analyzed using the SPSS version 26. The normality of data was tested using the one-sample Kolmogorov-Smirnov test. Qualitative data were described using numbers and percentages, with associations between categorical variables evaluated by X^2-test or Fisher's exact test when expected cell counts were less than 5. Continuous variables were presented as mean ± SD for normally distributed data and as median (Min-Max) for non-normally distributed data. Independent t-tests (parametric) and Mann-Whitney tests (non-parametric) were used to compare two groups. For comparisons among more than two groups, ANOVA was used. Correlations between continuous variables were assessed using Pearson correlation (for parametric data) and Spearman correlation (for non-parametric data). Sensitivity and specificity at various cutoff points were analyzed through ROC curve analysis. The significance threshold was set at 5% ($p \leq 0.05$). Smaller p-values were considered to indicate greater significance.

RESULTS

The mean age of the neonates was 9.32 ± 2.10 hours, ranging from 6 to 12 hours. Among them, 32 (60.4%) were males, and 21 (39.6%) were females. The mean gestational age was 36.07 ± 2.36 weeks, with a range from 27 to 38 weeks. Consanguinity was positive in 4 cases (7.5%) and negative in 49 cases (92.5%). Family history of similar conditions was reported in 9 neonates (17.0%), while 44 (83.0%) had no relevant family history. Maternal co-morbidities were noted in 12 cases (22.6%), while 41 (77.4%) of the mothers had no co-morbid conditions. The mode of delivery was predominantly Cesarean section (CS) accounting for 50 cases (94.3%), with only 3 neonates (5.7%) delivered via normal vaginal delivery (Table 1).

Table (1): Demographic and clinical characteristics of the studied neonates

Demographic data	The studied group (no=53)
Age (Hours)	
Mean ± SD	9.32±2.10
Min-Max	6-12
Sex	
Male	32 (60.4%)
Female	21 (39.6%)
Gestational age (weeks)	
Mean ± SD	36.07±2.36
Min-Max	27-38
Consanguinity	
Positive	4 (7.5%)
Negative	49 (92.5%)
Family history	
Positive	9 (17.0%)
Negative	44 (83.0%)
Maternal co-morbidities	
Positive	12 (22.6 %)
Negative	41 (77.4 %)
Mode of delivery	
CS	50 (94.3%)
NVD	3 (5.7%)

Among the diagnoses in the studied neonates, 30 cases (56.6%) were full-term with respiratory distress (RD), while 18 cases (34.0%) were preterm with RDS. Neonatal sepsis was diagnosed in 14 cases (26.4%), HIE in 8 cases (15.1%), and congenital heart disease (CHD) in 3 cases (5.7%) (Table 2).

Table (2): Diagnosis among the studied neonates

Diagnosis	The studied group (no=53)
FT with RD	30 (56.6%)
PT with RDS	18 (34.0%)
Neonatal sepsis	14 (26.4%)
HIE	8 (15.1%)
CHD	3 (5.7%)

Association between outcome and neonatal characteristics: In the analysis of neonatal characteristics associated with outcomes, a total of 24 neonates died and 29 were discharged. The mean birth weight was significantly lower in the deceased group (2.32 ± 0.56 kg) compared to the discharged group (2.76 ± 0.49 kg), with a p-value of 0.003. A significant difference was also observed in the Apgar scores, with the deceased group scoring lower at both 1 minute (4.91 ± 1.17 vs. 7.00 ± 0.80, p≤ 0.001) and 5 minutes (6.79 ± 0.77 vs. 8.45 ± 0.73, p≤0.001). Blood gas analysis revealed that the deceased group had a lower pH (7.25±0.07 vs. 7.35±0.07, p≤0.001) and bicarbonate levels (13.64±2.57 vs. 17.90±3.84, p≤0.001). Additionally, the mean oxygen saturation was significantly lower in the deceased neonates (92.33±1.57%) compared to those discharged (95.48±1.78%, p≤0.001). There were no significant differences in the use of antenatal steroids, respiratory rate, heart rate, temperature, or mean blood pressure between the two groups (Table 3).

Table (3): Association between outcome and neonatal characteristics

Neonatal characteristics	Died (no=24)	Discharged (no=29)	Test of significance	p value
Birth weight (KG)	2.32±0.56	2.76±0.49	t=3.07	**0.003***
Use of antenatal steroids Yes No	 6 (25.0%) 18 (75.0%)	 11 (37.9%) 18 (62.1%)	χ^2=1.01	0.315
Apgar score at 1st min	4.91±1.17	7.00±0.80	t=7.63	**≤0.001***
Apgar score at 5 min	6.79±0.77	8.45±0.73	t=7.94	**≤0.001***
PH	7.25±0.07	7.35±0.07	t=4.821	**≤0.001***
Paco2 (mmHg)	31.85±9.18	33.14±9.56	t=0.495	0.623
HCO3 mmol/L	13.64±2.57	17.90±3.84	t=4.64	**≤0.001***
Spo2%	92.33±1.57	95.48±1.78	t=6.73	**≤0.001***
R.R (C/M)	69.37±4.31	67.75±3.69	t=1.46	0.148
HR(B/M)	134.83±16.36	135.41±12.45	t=0.147	0.884
Temperature	36.94±0.29	36.85±0.32	t=1.07	0.289
MBP	44.50±7.30	44.07±5.89	t=0.238	0.813

Association between outcome, disorders, and respiratory support: Among neonates who died, 7 (29.2%) received survanta, compared to 4 (13.8%) in the discharged group, though this difference was not significant (p = 0.170). Median hospital stays were similar between the deceased (8.5 days) and discharged (8 days) groups (p = 0.661). MV was significantly more common in the deceased group (70.8%) than in the discharged group (6.9%, p ≤ 0.001). HIE was present in 8 (33.3%) of the deceased neonates but in none of the discharged (p = 0.001), while other diagnoses, such as full-term with respiratory distress (FT with RD), preterm with RDS (PT with RDS), and CHD, showed no significant differences between groups (Table 4).

Table (4): Association between outcome and newborn infant disorders& respiratory support

	Died (no=24)	Discharged (no=29)	Test of significance	p value
Receiving survanta Yes No	 7 (29.2%) 17 (70.8%)	 4 (13.8%) 25 (86.2%)	χ^2=1.88	0.170
Hospital stay (days)	8.5 (3-34)	8 (3-48)	Z=0.439	0.661
Respiratory support MV CPAP Nasal Canula	 17 (70.8%) 7 (29.2%) 0 (0%)	 2 (6.9%) 13 (44.8%) 14 (48.3%)	χ^2=27.41	**≤0.001***
FT with RDS	13 (54.2%)	17 (58.6%)	χ^2=0.106	0.745
PT with RDS	7 (29.2%)	11 (37.9%)	χ^2=0.450	0.502
Neonatal sepsis	7 (29.2%)	7 (24.1%)	χ^2=0.171	0.679
HIE	8 (33.3%)	0 (0%)	χ^2=11.38	**0.001***
CHD	2 (8.3%)	1 (3.4%)	FET	0.584

LDH levels showed significant correlations with several neonatal variables. A positive correlation was observed between LDH and gestational age ($r = 0.324$, $p = 0.018$) and mean blood pressure ($r = 0.294$, $p = 0.033$). Conversely, LDH negatively correlated with Apgar scores at both 1 and 5 minutes ($r = -0.581$ and $r = -0.530$, respectively, $p \leq 0.001$ for both), pH ($r = -0.488$, $p \leq 0.001$), bicarbonate (HCO_3^-) levels ($r = -0.430$, $p = 0.001$), and oxygen saturation (SpO_2) ($r = -0.523$, $p \leq 0.001$). Additionally, a positive correlation between LDH and aspartate transaminase (AST) levels was noted ($r = 0.303$, $p = 0.027$). No significant correlations were found between LDH and age, birth weight, respiratory rate, heart rate, temperature, hemoglobin (HG), platelets, total leukocyte count (TLC), liver enzymes (ALT), or electrolytes ($p > 0.05$) (Table 5).

Table (5): Correlation between LDH and neonatal data

Neonatal data	LDH	
	r	p
Age (Hours)	0.010	0.944
Gestational age (weeks)	0.324	**0.018***
Birth weight (KG)	0.100	0.477
Apgar score at 1st min	-0.581	**≤0.001***
Apgar score at 5 min	-0.530	**≤0.001***
PH	-0.488	**≤0.001***
$PaCO_2$ (mmHg)	-0.023	0.868
HCO_3^- (mmol/L)	-0.430	**0.001***
SpO_2% (mmHg)	-0.523	**≤0.001***
R.R (C/M)	0.264	0.057
HR (B/M)	-0.026	0.853
Temperature	0.105	0.454
MBP	0.294	**0.033***
HG G/dl	-0.026	0.852
platelets	-0.238	0.086
TLCs	-0.021	0.879
ALT (U/L)	-0.004	0.978
AST (U/L)	0.303	**0.027***
Na (mmol/L)	0.002	0.988
K (mmol/L)	-0.101	0.471
Ca (mg/dl)	-0.058	0.681
Creatinine (mg/dl)	0.176	0.207
RBS (mg/dl)	-0.027	0850
Hospital stay	0.182	0.191

No significant difference in LDH levels was found with consanguinity, family history, receiving survanta, or maternal medical history ($p > 0.05$). However, neonates who did not receive antenatal steroids had significantly higher LDH levels (992.28 ± 189.57 U/L) compared to those who did (878.53 ± 167.22 U/L, $p = 0.039$). LDH levels also varied significantly with respiratory support, showing the highest levels in neonates on MV (1121.6 ± 182.87 U/L, $p \leq 0.001$), and were elevated in neonates with full-term respiratory distress (FT with RD, $p = 0.011$) and HIE ($p \leq 0.001$) (Table 6).

Table (6): Association between history, diagnosis and LDH level

	LDH Mean ± SD	Test of significance	P value
Consanguinity Positive (n=4) Negative (n=49)	 993.50±258.00 952.71±185.33	t=0.412	0.682
Family history Positive (n=9) Negative (n=44)	 953.67±203.43 956.23±188.22	t=0.037	0.971
Use of antenatal steroids Yes (n=17) No (n=36)	 878.53±167.22 992.28±189.57	t=2.11	**0.039***
Receiving survanta Yes (n=11) No (n=42)	 882.73±70.61 974.93±205.45	t=1.45	0.151
Maternal medical history Positive (n=12) Negative (n=41)	 988.58±184.85 946.20±191.18	t=0.68	0.499
Respiratory support MV (n=19) CPAP (n=20) Nasal Cannula (n=14)	 1121.6±182.87 899.7±115.67 810.93±95.841	F=22.52	**≤0.001***
FT with RDS Yes (n=30) No (n=23)	 1012.7±191.24 881.57±160.97	t=2.46	**0.011***
PT with RDS Yes (n=18) No (n=35)	 830.22±114.01 1020.4±188.17	t=3.92	**≤0.001***
Neonatal sepsis Yes(n=14) No(n=39)	 983.43±159.93 945.87±199.18	t=0.635	0.529
HIE Yes (n=8) No (n=45)	 1237.1±142.9 905.78±148.16	t=5.85	**≤0.001***
CHD Yes (n=3) No (n=50)	 1075.0±317.05 948.64±181.17	t=1.13	0.264

Furthermore, logistic regression analysis was constructed to evaluate predictors of mortality, LDH was found to be significant factor influencing the death rate among the patients under study according to logistic regression analysis as each one unit change of serum LDH will increase the odds ratio in favor of mortality than survival by 1.01, which was statistically significant (p value 0.001) (Table 7).

Table (7): Logistic regression analysis for independent predictors of mortality

	β	SE	p value	Odds ratio	95% Confidence interval
Positive family history	2.639	1.106	0.017	14.00	1.60-122.33
Birth weight (KG)	-1.598	0.596	0.007	0.202	0.063-0.651
Apgar score at 1st min	-2.449	0.701	≤0.001	0.086	0.022-0.341
Apgar score at 5 min	-2.756	0.713	≤0.001	0.064	0.016-0.257
HCO_3^- (mmol/L)	-0.413	0.125	0.001	0.662	0.518-0.846
Spo2%	-0.993	0.256	≤0.001	0.371	0.224-0.613
LDH	0.009	0.003	0.001	1.01	1.003-1.014
Respiratory support MV	2.759	0.882	0.002	15.78	2.80-88.99

Receiver Operating Characteristic (ROC) curve analysis was used to assess the predictive value of LDH levels for neonatal mortality. The area under the curve (AUC) was 0.81 (95% CI: 0.69-0.93), with LDH cutoff of 895.50 U/L yielding a sensitivity of 79.2% and specificity of 55.2%. The positive predictive value (PPV) was 59.4%, negative predictive value (NPV) was 76.2%, and overall accuracy was 66% (Table 8).

Table (8): Receiver operating characteristics curve for prediction of mortality by LDH

AUC	95% CI	Cutoff	Sensitivity	Specificity	PPV	NPV	Accuracy
0.81	0.69-0.93	895.50	79.2%	55.2%	59.4	76.2	66%

AUC: Area under the curve, CI: Confidence interval, PPV: positive predictive value, NPV: negative predictive value

---

DISCUSSION

LDH is increasingly recognized as a potential biomarker for neonatal morbidity and mortality due to its association with cellular injury and metabolic stress. Elevated LDH levels may reflect adverse neonatal outcomes, particularly in high-risk infants requiring intensive care [7]. In our study, we aimed to assess the correlation between LDH levels and various clinical and biochemical parameters in neonates, with a focus on identifying predictors of mortality.

Our study identified significant associations between LDH levels and several neonatal factors. Elevated LDH levels were negatively correlated with gestational age, Apgar scores at 1 and 5 minutes, pH, and bicarbonate levels, suggesting that high LDH may reflect neonatal stress or hypoxia. Neonates requiring MV exhibited the highest LDH levels, while those on less intensive respiratory support, such as CPAP or nasal cannula, had lower LDH levels. Our ROC analysis demonstrated that LDH had moderate predictive value for mortality, with an AUC of 0.81, suggesting its usefulness as a prognostic tool.

In our study, we analyzed the diagnoses and outcomes of 53 neonates, revealing significant associations between neonatal characteristics and mortality. The predominant diagnoses included full-term (FT) neonates with respiratory distress (RD) (56.6%) and preterm (PT) neonates with RDS (34.0%). Our findings align with **Baseer *et al.*** [8], which often highlights respiratory complications as a leading cause of neonatal morbidity and mortality. Notably, we observed a significant difference in birth weight between neonates who died (2.32±0.56 kg) and those who discharged (2.76±0.49 kg), which is corroborating with **Pusdekar *et al.*** [9] that emphasize low birth weight as a critical risk factor for adverse outcomes.

Apgar scores at both 1 and 5 minutes were significantly lower in the deceased group, which is consistent with research indicating that low Apgar scores are predictive of neonatal mortality [10]. Furthermore, blood gas analyses revealed significantly lower pH and bicarbonate levels in the deceased group, which aligns with findings from a study by **Paul *et al.*** [11] that linked metabolic acidosis to higher mortality rates in neonates.

In terms of respiratory support, our results showed that a significant proportion of neonates who died were on MV (70.8%), contrasting sharply with the discharged group, where only 6.9% required MV. This finding is supported by earlier studies indicating that the need for advanced respiratory support is associated with increased mortality in neonates [12]. Additionally, we noted that the presence of HIE was significantly associated with mortality, which is aligning with literature that highlights HIE as a critical determinant of poor neurological outcomes and mortality in neonates [13].

However, our study did not find significant differences in the use of antenatal steroids, which contrasts with other studies suggesting a protective effect of antenatal steroids in reducing neonatal mortality and morbidity [14]. This discrepancy may be attributed to variations in sample size, demographics, or clinical protocols across studies.

In our investigation of the correlation between LDH levels and neonatal data, we found significant

associations that align with and expand upon existing literature. Notably, various Apgar scores were negatively correlated with LDH levels, which is reinforcing findings from previous studies that highlight the role of LDH as a biomarker for neonatal stress and as a predictor of adverse outcomes [15]. Specifically, lower Apgar scores at 1 and 5 minutes were significantly associated with elevated LDH levels, suggesting that LDH may reflect the extent of neonatal asphyxia or hypoxia, as reported by **Lee *et al.*** [6].

Furthermore, our study demonstrated a significant negative correlation between pH and bicarbonate levels with LDH, which corroborates with earlier research that associates metabolic acidosis with increased LDH, indicating poor neonatal outcomes [16]. Interestingly, while we found a significant relationship between the use of antenatal steroids and LDH levels, previous studies have shown mixed results regarding the impact of antenatal steroids on neonatal outcomes, often highlighting their protective effects against respiratory distress [17]. Our findings suggest that the use of antenatal steroids may indeed influence metabolic markers like LDH, indicating a potential area for further exploration. The association between respiratory support and LDH levels was particularly striking, with neonates requiring MV exhibiting the highest LDH levels. This finding is consistent with the work of **Ergenc *et al.*** [18] who reported that elevated LDH levels correlate with the need for advanced respiratory support, thus serving as a potential marker for clinical deterioration. However, while the relationship between respiratory support and LDH is evident, the literature is not entirely conclusive, as some studies indicate that LDH levels may not significantly differ among various forms of respiratory support [19].

Moreover, our ROC analysis revealed that LDH had moderate predictive value for mortality, with an area under the curve (AUC) of 0.81. This supports findings from **Algebaly *et al.*** [20] who similarly identified LDH as a useful prognostic indicator in neonates. However, the sensitivity and specificity in our study suggest that while LDH is a valuable marker, it should be utilized in conjunction with other clinical parameters for optimal predictive accuracy.

One strength of this study is the inclusion of multiple clinical and biochemical parameters, which allowed for a comprehensive analysis of LDH's role as a biomarker in neonates. Additionally, our study's inclusion of neonates requiring various levels of respiratory support provided insights into how LDH levels may differ across clinical scenarios.

LIMITATIONS

Being relatively small sample size, which may limit the generalizability of our findings, and its observational nature, which restricts our ability to establish causality. Furthermore, other confounding factors affecting LDH levels, such as concurrent infections or metabolic disorders, were not fully accounted for.

CONCLUSION

Our study underscored LDH's potential as a useful biomarker for predicting adverse outcomes in neonates, particularly for identifying those at higher risk of mortality. The significant correlations between elevated LDH and poor Apgar scores and need for intensive respiratory support suggest that LDH may serve as a valuable clinical marker for early intervention. However, further studies with larger sample sizes are needed to validate these results and explore LDH's role in neonatal care more comprehensively.

Conflict of interest: None.
Financial disclosures: None.

REFERENCES

1. **Dol J, Hughes B, Bonet M *et al.* (2023):** Timing of neonatal mortality and severe morbidity during the postnatal period: a systematic review. JBI Evidence Synthesis, 21 (1): 98–199.
2. **Yadav S, Lee B, Kamity R (2023):** Neonatal Respiratory Distress Syndrome. Treasure Island (FL): StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK560779/
3. **Farhana A, Lappin S (2023):** Biochemistry, Lactate Dehydrogenase. Treasure Island (FL): StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK557536/
4. **Ranjan A, Gulati A (2023):** Advances in therapies to treat neonatal hypoxic-ischemic encephalopathy. Journal of Clinical Medicine, 12 (20): 6653. https://doi.org/10.3390/jcm12206653
5. **Moustafa A, Mohamed Ahmed M, El-Mazary A *et al.* (2024):** Can Serum Lactate Dehydrogenase Differentiate between Respiratory Distress Syndrome (RDS) and Transient Tachypnea of Newborn (TTN) in Preterm Neonates?. Annals of Neonatology, 6 (2): 92-101.
6. **Lee M, Lee N, Bae M *et al.* (2021):** Using lactate dehydrogenase to predict the severity of respiratory distress in term newborn infants with no perinatal asphyxia. The Turkish Journal of Pediatrics, 63 (3): 393–403.
7. **Anh T, Hao T, Hoang H *et al.* (2020):** The role of plasma lactate dehydrogenase testing in the prediction of severe conditions in newborn infants: A prospective study. Research and Reports in Neonatology, 10: 31-35.
8. **Baseer K, Mohamed M, Abd-Elmawgood E (2020):** Risk factors of respiratory diseases among neonates in neonatal intensive care unit of Qena University Hospital, Egypt. Annals of Global Health, 86 (1): 22. doi:10.5334/aogh.2739.
9. **Pusdekar Y, Patel A, Kurhe K *et al.* (2020):** Rates and risk factors for preterm birth and low birthweight in the global network sites in six low-and low middle-income countries. Reproductive Health, 17 (3): 187. doi:10.1186/s12978-020-01029-z.
10. **Cnattingius S, Johansson S, Razaz N (2020):** Apgar score and risk of neonatal death among preterm infants. New England Journal of Medicine, 383 (1): 49-57.

11. **Paul M, Partridge J, Barrett-Reis B *et al.* (2020):** Metabolic acidosis in preterm infants is associated with a longer length of stay in the neonatal intensive care unit. PharmacoEconomics-Open, 4 (3): 541-547.
12. **Wyckoff M, Wyllie J, Aziz K *et al.* (2020):** Neonatal life support: 2020 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Circulation, 142 (16): 185-221.
13. **Debillon T, Sentilhes L, Kayem G *et al.* (2023):** Risk factors for unfavorable outcome at discharge of newborns with hypoxic-ischemic encephalopathy in the era of hypothermia. Pediatric Research, 93 (7): 1975-1982.
14. **McDougall A, Aboud L, Lavin T *et al.* (2023):** Effect of antenatal corticosteroid administration-to-birth interval on maternal and newborn outcomes: a systematic review. EClinicalMedicine, 58: 101916. doi: 10.1016/j.eclinm.2023.101916.
15. **Lee I, Wong S, Wang X *et al.* (2021):** Identifying early diagnostic biomarkers associated with neonatal hypoxic-ischemic encephalopathy. Diagnostics, 11 (5): 897. https://doi.org/10.3390/ diagnostics11050897.
16. **Naseh A, Ashrafzadeh S, Erfanian-Taghvaie M (2021):** Is Serum Lactate Dehydrogenase Level a Good Indicator for Diagnosis of Neonatal Birth Asphyxia? Diabetes, 2 (4): 1088. https://doi.org/10.24911/AP.106-1088
17. **Jobe A, Goldenberg R, Kemp M (2024):** Antenatal corticosteroids: an updated assessment of anticipated benefits and potential risks. American Journal of Obstetrics and Gynecology, 230 (3): 330-339.
18. **Ergenc I, Capar E, Erturk S *et al.* (2023):** Diagnostic performance of lactate dehydrogenase (LDH) isoenzymes levels for the severity of COVID-19. Journal of Medical Biochemistry, 42 (1): 16-26.
19. **Doandes F, Manea A, Lungu N *et al.* (2021):** Clinical, biological and electroencephalographic monitoring of newborns with neurological risk in the Neonatal Intensive Care Unit. Experimental and Therapeutic Medicine, 22 (1): 760. doi:10.3892/etm.2021.10192.
20. **Algebaly H, Abd-Elal A, El Kaffas R *et al.* (2021):** Predictive value of serum lactate dehydrogenase in diagnosis of septic shock in critical pediatric patients: A cross-sectional study. Journal of Acute Disease, 10 (3): 107-111.