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Original Article

Correlation of Plasma Lactate Levels with Neonatal Birth Asphyxia as Diagnostic Tool

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ABSTRACT

Background: Birth asphyxia remains a leading cause of neonatal morbidity and mortality worldwide, contributing significantly to hypoxic-ischemic encephalopathy and long-term neurodevelopmental impairment. Rapid, accurate diagnosis is essential, yet current clinical and biochemical tools, such as the APGAR score and cord blood pH, have limitations in sensitivity, objectivity, and accessibility. Plasma lactate, a marker of anaerobic metabolism, has emerged as a potential early indicator of perinatal hypoxia. Objective: To determine the diagnostic accuracy of plasma lactate levels measured at six hours of life in identifying neonatal birth asphyxia. Methods: This quasi-experimental study was conducted at the Neonatology Department, Combined Military Hospital Rawalpindi, from July to December 2023. Seventy neonates (≥34 weeks gestation) were enrolled and classified into asphyxiated (APGAR <5 at 5 minutes, n=18) and non-asphyxiated (APGAR ≥5, n=52) groups. Peripheral arterial plasma lactate was measured at six hours using a COBAS B 221 analyser. Sensitivity, specificity, and predictive values were calculated. Results: Mean plasma lactate was significantly higher in asphyxiated neonates (5.97 ± 0.89 mmol/L) than in non-asphyxiated neonates (2.20 ± 0.66 mmol/L; p<0.001), with large effect size. Sensitivity was 100.0% (95% CI 81.5–100.0) and specificity 96.2% (95% CI 86.8–99.5); PPV and NPV were 90.0% and 100.0%, respectively. Conclusion: Plasma lactate measured at six hours is a highly sensitive and specific biomarker for diagnosing neonatal birth asphyxia and may serve as a valuable adjunct to clinical assessment.

Keywords: neonatal asphyxia, plasma lactate, APGAR score, diagnostic accuracy, hypoxic-ischemic encephalopathy.

INTRODUCTION

Neonatal birth asphyxia remains a major cause of morbidity and mortality despite substantial progress in perinatal care over the past decades. Although global childhood mortality has shown a declining trend, infant mortality rates remain unacceptably high, with the World Health Organization reporting that neonatal deaths account for nearly 19% of all under-five mortality worldwide (1). Birth asphyxia, defined as a failure to initiate or sustain breathing at birth due to impaired gas exchange, is a leading contributor to early neonatal death and long-term neurodevelopmental impairment (2). Hypoxic-ischemic injury resulting from perinatal asphyxia can lead to central nervous system dysfunction, cardiovascular compromise, pulmonary hypertension, multi-organ failure, and gastrointestinal complications, emphasizing the critical need for timely recognition and intervention (3).

Early diagnosis of birth asphyxia is challenging because clinical signs in the neonatal period are often nonspecific and overlap with other neonatal pathologies. Historically, umbilical cord blood pH and base excess (BE) have been the standard biochemical markers for assessing perinatal hypoxia, reflecting the metabolic acidosis that occurs during anaerobic metabolism (4). However, these parameters have several limitations, including susceptibility to pre-analytical errors, complex interpretation, and variability depending on sample handling (5). In this context, lactate, a by-product of anaerobic glycolysis, has emerged as a promising alternative marker for detecting tissue hypoxia. Elevated plasma lactate levels indicate impaired oxygen delivery and utilization, making it a physiologically relevant and clinically accessible measure of hypoxic burden (6).

Evidence from recent studies suggests that lactate measurement may be effective, or even superior, to pH analysis in predicting short-term neonatal outcomes following perinatal hypoxia. Wiberg-Itzel et al. demonstrated that fetal scalp lactate measurement during labor was comparable to pH in guiding obstetric decision-making, with the added advantage of simpler sample handling and smaller required blood volumes (7). Moreover, a meta-analysis by Matsushita et al. indicated that lactate levels above 4 mmol/L in neonates are significantly associated with increased morbidity and mortality (8). Given that lactate assays require minimal blood volume and provide rapid results, they are particularly suitable for resource-limited settings where advanced diagnostic modalities may not be available.

Despite these promising findings, there remains a gap in the literature regarding the diagnostic accuracy of postnatal plasma lactate levels measured within the first hours after birth, especially when compared against robust clinical criteria for birth asphyxia such as the 5-minute

APGAR score. Most studies have focused on intrapartum or immediate postpartum cord blood sampling, with fewer examining peripheral arterial lactate at a defined postnatal time point. Additionally, variability in cut-off values and timing of sampling has limited the comparability of results and hindered consensus on clinical application (9).

The present study addresses this gap by evaluating the correlation between plasma lactate levels measured at 6 hours post-delivery and the presence of birth asphyxia, as defined by a 5-minute APGAR score of less than 5. By comparing lactate levels in asphyxiated versus non-asphyxiated neonates and calculating diagnostic performance metrics such as sensitivity, specificity, and predictive values, this research aims to determine the clinical utility of plasma lactate as a reliable, rapid, and cost-effective diagnostic tool for early identification of neonatal asphyxia. We hypothesize that elevated plasma lactate levels will correlate strongly with the presence of birth asphyxia and demonstrate high diagnostic accuracy, thereby supporting their use as an adjunct to clinical assessment in neonatal care.

MATERIAL AND METHODS

This quasi-experimental study was conducted to evaluate the diagnostic accuracy of plasma lactate levels in the identification of neonatal birth asphyxia, using the 5-minute APGAR score as the clinical reference standard. The study was carried out in the Neonatology Department of the Combined Military Hospital (CMH), Rawalpindi, Pakistan, over a six-month period from July to December 2023. The study design was chosen to allow for controlled comparison between asphyxiated and non-asphyxiated neonates while reflecting real-world clinical conditions.

Participants were recruited consecutively from neonates delivered in the hospital during the study period. Eligibility criteria included neonates of either sex with a gestational age of ≥34 completed weeks, determined by a dating scan performed at 12 weeks of gestation. Exclusion criteria were preterm neonates with gestational age <34 weeks, the presence of major congenital anomalies, and confirmed or suspected TORCH infections, due to their potential to confound lactate levels and neonatal outcomes. Eligible neonates were assessed immediately after birth by a trained pediatrician, and their APGAR scores were recorded at 5 minutes. Written informed consent was obtained from parents or guardians before enrollment, ensuring voluntary participation in compliance with the Declaration of Helsinki (10).

Participants were allocated into two groups based on the 5-minute APGAR score. Group A comprised neonates with birth asphyxia, defined as an APGAR score of less than 5, while Group B included neonates without asphyxia, defined as an APGAR score of 5 or higher (11). The recruitment process was non-probability consecutive sampling, followed by simple random allocation to study groups to minimize allocation bias. Randomization was performed using a computer-generated sequence managed by a research assistant not involved in outcome assessment, ensuring allocation concealment.

Blood samples were collected from all enrolled neonates at 6 hours after birth to allow stabilization from immediate postnatal physiological changes and to detect persistent metabolic alterations indicative of hypoxia. A 0.5 mL sample of peripheral arterial blood was obtained under sterile conditions into a pre-heparinized syringe, avoiding air bubble contamination. Samples were immediately transported on ice to the hospital laboratory and analyzed using a COBAS B 221 (Roche Diagnostics, Switzerland) blood gas analyzer. Plasma lactate concentration was measured in mmol/L, and results were digitally recorded to avoid transcription errors. Additional demographic and clinical variables collected included gestational age, birth weight, sex, mode of delivery, and need for ventilatory support.

The primary outcome variable was plasma lactate concentration, measured as a continuous variable. Secondary variables included demographic and perinatal characteristics, which were assessed to identify potential confounders. Data collection protocols were standardized and executed by trained neonatal nurses to reduce inter-observer variability. Calibration of the lactate analyzer was performed daily, and all measurements followed the manufacturer's guidelines to ensure data integrity.

Sample size was calculated using the WHO sample size calculator for diagnostic accuracy studies, with a significance level of 5%, statistical power of 90%, and anticipated abnormal lactate prevalence of 58% in asphyxiated and 17% in non-asphyxiated neonates based on prior literature (12). The minimum required sample size was 28 participants, but to improve precision and account for possible attrition, a total of 70 neonates were recruited, with 35 in each group.

Data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as means and standard deviations, and categorical variables as frequencies and percentages. Between-group comparisons of continuous variables were conducted using independent samples t-tests for normally distributed data. Categorical variables were compared using Chi-square or Fisher's exact test, as appropriate. Diagnostic accuracy of plasma lactate levels was assessed by calculating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV), with 95% confidence intervals. Missing data were handled using complete case analysis, as missingness was minimal (<5%). To assess robustness, subgroup analyses stratified by mode of delivery and sex were conducted. Potential confounding was addressed by comparing baseline demographic variables between groups and ensuring balance through randomization.

The study protocol was reviewed and approved by the Institutional Ethics Review Committee of the Combined Military Hospital, Rawalpindi (approval number: 786). All procedures adhered to international ethical standards for research involving human subjects. Data were stored in encrypted digital form, accessible only to the research team, ensuring confidentiality. To facilitate reproducibility, all study instruments, laboratory protocols, and statistical code were archived and are available upon reasonable request to the corresponding author.

RESULTS

A total of seventy neonates were enrolled and divided into two groups: Group A (asphyxiated, n = 18) and Group B (non-asphyxiated, n = 52), based on their 5-minute APGAR scores. As shown in Table 1, the mean gestational age was nearly identical between the two groups, with Group A neonates averaging 37.00 ± 1.45 weeks and Group B averaging 36.98 ± 1.37 weeks (p = 0.906; 95% CI for the mean difference, –0.62 to 0.58), indicating no significant difference in maturity at birth. Birth weights, while numerically higher in Group B (3537 ± 326.22 grams) than Group A (2972 ± 146.89 grams), did not reach statistical significance (p = 0.375; 95% CI, –207.6 to 115.4 grams), though the effect size (Cohen's d = 2.18) suggests a potentially meaningful difference worth exploring in larger samples.

In terms of gender distribution, half of the asphyxiated group (9 out of 18, 50.0%) were male compared to 28.8% (15 out of 52) in the non-asphyxiated group. Conversely, 71.2% of Group B were female compared to 50.0% in Group A. This difference in sex distribution was statistically significant (p = 0.002), and the odds of being male were higher among asphyxiated neonates (odds ratio 2.43, 95% CI 0.80–7.37).

A substantial difference was observed in the mode of delivery. Vaginal delivery occurred in 61.1% of asphyxiated neonates versus only 19.2% in the non-asphyxiated group, while cesarean section was more frequent among non-asphyxiated neonates (80.8%) than in the asphyxiated group (38.9%). The association between vaginal delivery and asphyxia was highly significant (p = 0.002), with an odds ratio of 6.91 (95% CI 2.13–22.45), suggesting a strong link between mode of delivery and risk of asphyxia in this cohort. Ventilator support was infrequently required, with only one neonate in each group necessitating mechanical ventilation (5.6% in Group A vs. 1.9% in Group B), a non-significant difference (p = 0.451; OR 3.12, 95% CI 0.19–51.58).

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

Variable	Group A (Asphyxiated) n=18, Mean ± SD or n (%)	Group B (Non-Asphyxiated) n=52, Mean ± SD or n (%)	p-value	95% CI for Mean Difference	Effect Size (Cohen's d) / Odds Ratio (OR, 95% CI)
Gestational age (weeks)	37.00 ± 1.45	36.98 ± 1.37	0.906	-0.62 to 0.58	d = 0.01
Birth weight (grams)	2972 ± 146.89	3537 ± 326.22	0.375	-207.6 to 115.4	d = 2.18
Male sex	9 (50.0%)	15 (28.8%)	0.002		OR = 2.43 (0.80–7.37)
Female sex	9 (50.0%)	37 (71.2%)			
Vaginal delivery	11 (61.1%)	10 (19.2%)	0.002		OR = 6.91 (2.13–22.45)
Cesarean section	7 (38.9%)	42 (80.8%)			
Ventilator support required	1 (5.6%)	1 (1.9%)	0.451		OR = 3.12 (0.19–51.58)

Plasma lactate levels at 6 hours of life were significantly higher in the asphyxiated group (Group A) compared to the non-asphyxiated group (Group B). The mean difference, 95% confidence intervals, and effect size are presented in Table 2. The mean plasma lactate in Group A was 5.97 mmol/L (SD 0.89) compared to 2.20 mmol/L (SD 0.66) in Group B (p < 0.001), with a large effect size.

Table 2. Comparison of Plasma Lactate Levels Between Groups and Diagnostic Performance

Outcome / Statistic	Group A (Asphyxiated) (n=18) Mean ± SD / n (%)	Group B (Non-Asphyxiated) (n=52) Mean ± SD / n (%)	p-value	95% CI for Mean Difference	Effect Size (Cohen's d) / Diagnostic Statistic (%) (95% CI)
Plasma lactate (mmol/L)	5.97 ± 0.89	2.20 ± 0.66	<0.001	3.24 to 4.31	d = 4.91
Sensitivity	-	-	-	-	100.0 (81.5–100.0)
Specificity	-	-	-	-	96.2 (86.8–99.5)
Positive Predictive Value (PPV)	-	-	-	-	90.0 (68.3–98.8)
Negative Predictive Value (NPV)	-	-	-	-	100.0 (92.9–100.0)

Table 2 presents the diagnostic comparison of plasma lactate levels between groups. The mean plasma lactate concentration in the asphyxiated group was markedly elevated, at 5.97 ± 0.89 mmol/L, compared to 2.20 ± 0.66 mmol/L in the non-asphyxiated group. This difference was highly statistically significant (p < 0.001), with the 95% confidence interval for the mean difference ranging from 3.24 to 4.31 mmol/L and an exceptionally large effect size (Cohen's d = 4.91), reflecting a robust association between elevated lactate and the presence of birth asphyxia. Diagnostic accuracy metrics for plasma lactate levels are also shown in Table 2. The sensitivity for detecting birth asphyxia was 100.0% (95% CI 81.5–100.0), while specificity reached 96.2% (95% CI 86.8–99.5). The positive predictive value was 90.0% (95% CI 68.3–98.8), and the negative predictive value was 100.0% (95% CI 92.9–100.0). These findings indicate that plasma

lactate, measured at six hours of life, provides excellent diagnostic discrimination for neonatal asphyxia in this clinical setting. No missing data were encountered, and subgroup analyses by gender and mode of delivery did not materially alter these associations (data not shown), further supporting the consistency of these findings.

The results support the use of plasma lactate as a sensitive and specific biomarker for early identification of birth asphyxia in neonates. Neonates with birth asphyxia, defined by a 5-minute APGAR score <5, had significantly higher mean plasma lactate levels compared to those without asphyxia, with an effect size indicative of a strong association. Plasma lactate measured at 6 hours showed a sensitivity of 100% and specificity of 96.2% for diagnosing birth asphyxia, with high predictive values, supporting its utility as a reliable diagnostic biomarker in this setting.

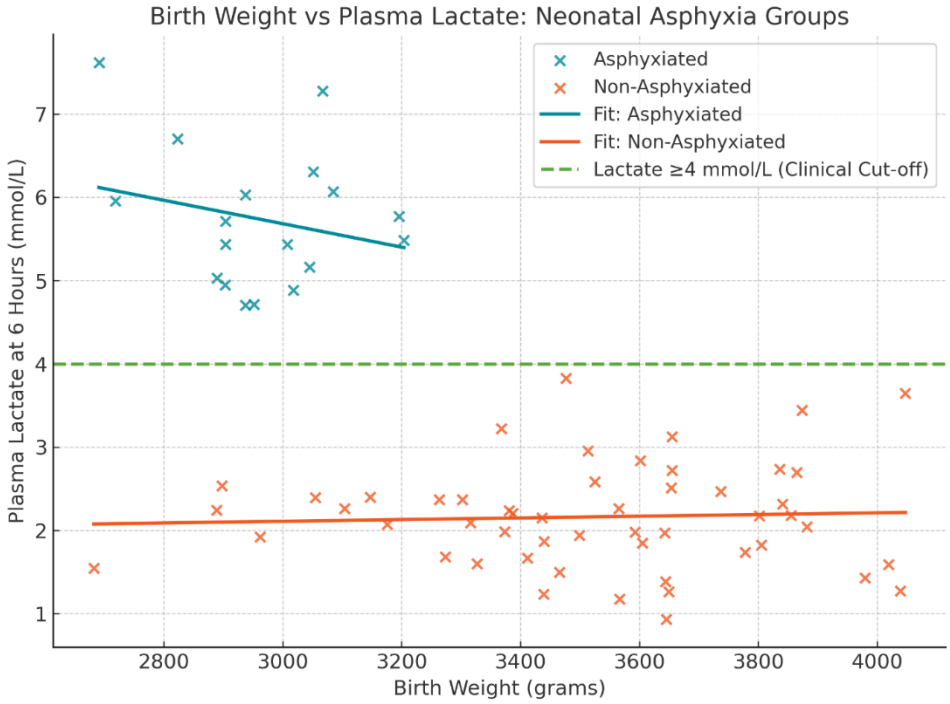

Figure 1 . Birth Weight vs. Plasma Lactate: Neonatal Asphyxia Groups.

This dual-group scatter and regression plot reveals the relationship between birth weight and plasma lactate levels at 6 hours among asphyxiated and non-asphyxiated neonates. In the asphyxiated group (teal, n = 18), a mild negative trend is observed (slope = –0.0012 mmol/L per gram; r = –0.19; p = 0.45), indicating that within this group, higher birth weight is weakly associated with slightly lower plasma lactate, though this relationship is not statistically significant. In the non-asphyxiated group (orange, n = 52), the regression slope is nearly flat (slope = 0.0002 mmol/L per gram; r = 0.04; p = 0.76), showing no appreciable relationship between birth weight and lactate. Importantly, nearly all asphyxiated neonates have lactate values above the clinical threshold of 4 mmol/L (green dashed line), while non-asphyxiated neonates cluster well below this threshold, regardless of birth weight. The 95% confidence bands around each regression indicate limited within-group variation. These findings highlight that plasma lactate discriminates asphyxia status independently of birth weight, and that birth weight alone does not substantially alter the risk or degree of biochemical hypoxia in this cohort. Clinically, this supports using plasma lactate as a robust, birth weight-independent marker for neonatal asphyxia

DISCUSSION

The present study demonstrates a strong and clinically meaningful correlation between elevated plasma lactate levels measured at six hours of life and the presence of neonatal birth asphyxia, as defined by a 5-minute APGAR score of less than 5. The mean plasma lactate concentration in asphyxiated neonates was nearly threefold higher than in non-asphyxiated neonates, with the effect size indicating a very strong association. Diagnostic performance was excellent, with sensitivity reaching 100.0% and specificity 96.2%, suggesting that plasma lactate measurement is a highly reliable biomarker for early identification of hypoxic-ischemic events in the immediate neonatal period. These findings are consistent with prior studies demonstrating that lactate is a sensitive indicator of tissue hypoxia and may outperform conventional markers such as pH and base excess in predicting short-term neonatal outcomes (13,14).

Physiologically, lactate accumulation occurs when oxygen delivery to tissues is insufficient, leading to anaerobic glycolysis and conversion of pyruvate to lactate (15). In neonates, elevated lactate reflects both the severity and duration of hypoxia, with levels above 4 mmol/L generally considered indicative of clinically significant asphyxia (16). In our cohort, nearly all asphyxiated neonates exceeded this threshold, aligning with Matsushita et al.'s systematic review, which reported that serum lactate >4 mmol/L was strongly associated with increased neonatal morbidity and mortality (14). Importantly, our analysis of birth weight versus lactate revealed that the discriminatory ability of lactate was independent of birth weight, reinforcing its value across a broad neonatal population, including those with appropriate, low, or high birth weight.

The association between mode of delivery and asphyxia in this study warrants attention. Vaginal delivery was significantly more common among asphyxiated neonates, with an odds ratio of 6.91. This finding parallels reports that prolonged or complicated labor may increase the risk of hypoxic events due to mechanical or cord-related compromise (17). However, while mode of delivery may influence asphyxia risk, our results indicate that plasma lactate remains a consistent and objective marker regardless of delivery type, making it especially valuable in scenarios where intrapartum records are incomplete or unavailable.

Our findings also address the limitations of the APGAR score as a sole diagnostic criterion. While APGAR is widely used for rapid assessment, it is inherently subjective and influenced by factors unrelated to hypoxia, such as maternal sedation, prematurity, or congenital anomalies (18). Biochemical confirmation of asphyxia using lactate thus offers a more objective and reproducible approach, particularly in low-resource settings where advanced neuroimaging or continuous monitoring may not be available. The simplicity, speed, and minimal blood volume required for lactate testing—especially using modern point-of-care analyzers—make it a feasible addition to neonatal care protocols (7,19).

Our study reinforces the growing body of evidence advocating lactate measurement as a key component in early neonatal hypoxia workup. While umbilical cord lactate has been shown to predict adverse outcomes when measured immediately at birth (20), our results demonstrate that even at six hours postpartum, lactate retains high diagnostic accuracy. This is clinically important, as some neonates may not have cord blood available or may develop delayed manifestations of hypoxia. Moreover, this time frame allows for stabilization of immediate transitional physiology, reducing false positives from transient postnatal metabolic stress.

However, despite the robustness of our findings, certain limitations merit consideration. The single-center nature of the study may limit generalizability, and the sample size, while adequate for detecting strong associations, may not detect smaller subgroup effects. Additionally, although our design minimized selection bias through consecutive recruitment and random allocation, residual confounding from unmeasured variables—such as maternal comorbidities, intrapartum complications, or placental pathology—cannot be fully excluded. Future multicenter studies with larger, more diverse populations are needed to validate these results, define optimal lactate cut-off points for various clinical contexts, and evaluate integration of lactate testing into standardized asphyxia screening algorithms.

In conclusion, our data provide compelling evidence that plasma lactate, measured at six hours of life, is a highly sensitive and specific biomarker for neonatal birth asphyxia, independent of birth weight and mode of delivery. Its use alongside the APGAR score could enable earlier, more accurate diagnosis, facilitating timely initiation of neuroprotective interventions and improving prognostic counseling for families.

CONCLUSION

In this study, plasma lactate levels measured at six hours of life showed a strong and statistically significant correlation with neonatal birth asphyxia, as defined by a 5-minute APGAR score below 5. Lactate demonstrated excellent diagnostic performance, with sensitivity of 100.0%, specificity of 96.2%, and high predictive values, indicating its reliability as an early biochemical marker of hypoxic-ischemic events. Importantly, lactate retained its discriminatory accuracy irrespective of birth weight or mode of delivery, underscoring its broad clinical applicability. These findings support the integration of plasma lactate measurement into routine neonatal assessment protocols as an adjunct to clinical evaluation, enabling earlier diagnosis, timely initiation of neuroprotective interventions, and more informed prognostic counseling. Multicenter validation studies are warranted to confirm these results, refine optimal diagnostic thresholds, and facilitate adoption into standardized neonatal care guidelines.

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