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Research Article

Accuracy Of Umbilical Cord Blood Lactate Dehydrogenase Levels Versus Umbilical Cord Blood Ph in Neonates with Birth Asphyxia: A Cross-Sectional Study

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ABSTRACT

Background: Birth asphyxia remains a major cause of neonatal morbidity and mortality worldwide, particularly in developing countries. Early identification of hypoxic injury is essential for timely management and prevention of long-term neurological sequelae. Umbilical cord blood pH is commonly used to assess fetal metabolic status at birth; however, biochemical markers such as lactate dehydrogenase (LDH), which reflect cellular injury, may serve as useful adjunct indicators of hypoxic insult.
Objective: To evaluate the diagnostic accuracy of umbilical cord blood lactate dehydrogenase (LDH) levels compared with umbilical cord blood pH in neonates with birth asphyxia.
Methods: This hospital-based cross-sectional study was conducted in a tertiary care hospital over a two-year period. A total of 75 neonates diagnosed with birth asphyxia were included in the study. Umbilical cord arterial blood samples were collected immediately after delivery for measurement of cord blood pH using a blood gas analyzer and LDH levels using an automated biochemical analyzer. Serum LDH levels were also estimated. Demographic and clinical variables were recorded. Statistical analysis was performed using appropriate tests including the Chi-square test, and a p-value <0.05 was considered statistically significant.
Results: Among the 75 neonates included in the study, 61.3% were males and 38.7% were females. The majority of neonates were term (58.7%), while 41.3% were preterm. Mild acidemia (pH >7.2) was observed in 57.3%, moderate acidemia (pH 7.01–7.19) in 32.0%, and severe acidemia (pH <7.0) in 10.7% of neonates. Elevated cord LDH levels (>612 IU/L) were significantly associated with decreasing cord blood pH values. A significant association was observed between cord blood pH and cord LDH levels (p = 0.004) as well as between cord blood pH and serum LDH levels (p = 0.001). Higher LDH levels were observed in neonates with severe acidemia, suggesting greater cellular injury due to hypoxia.
Conclusion: Umbilical cord blood LDH levels showed a significant association with cord blood pH and the severity of metabolic acidosis in neonates with birth asphyxia. Elevated LDH levels may serve as a useful adjunct biochemical marker for early detection and assessment of neonatal hypoxia, particularly in settings where cord blood gas analysis is not readily available.

Keywords*: Birth asphyxia; Neonate; Umbilical cord blood pH; Lactate dehydrogenase; Hypoxia; Neonatal outcome.*

INTRODUCTION

Birth asphyxia remains one of the leading causes of neonatal morbidity and mortality worldwide and continues to be a major contributor to neonatal deaths, particularly in developing countries. It is characterized by impaired gas exchange leading to hypoxemia, hypercapnia, and metabolic acidosis during the perinatal period. Globally, nearly one million neonatal deaths annually are attributed to birth asphyxia, and a substantial proportion of survivors experience long-term neurological complications such as cerebral palsy, developmental delay, and cognitive impairment [1]. Early recognition

of neonatal hypoxia is therefore crucial to initiate timely resuscitation and appropriate neonatal intensive care management.

Umbilical cord blood gas analysis has long been considered the most objective method for assessing fetal metabolic status immediately after birth. Among its parameters, umbilical arterial pH is widely used as an indicator of intrapartum hypoxia and metabolic acidosis. A cord arterial pH value below 7.20 indicates fetal acidemia, while values below 7.00 are often associated with severe acidemia and increased risk of adverse neonatal outcomes [2]. However, several studies have demonstrated that low cord pH alone does not always correlate with neurological injury or long-term morbidity, as some neonates with low pH remain clinically well without significant complications. Consequently, reliance solely on umbilical cord pH may not accurately reflect the degree of tissue hypoxia or cellular injury occurring during birth asphyxia [3].

In recent years, biochemical markers reflecting cellular damage have been investigated as potential indicators of perinatal hypoxia. Lactate dehydrogenase (LDH) is an intracellular enzyme involved in anaerobic glycolysis and is widely distributed in body tissues including the brain, liver, kidneys, and myocardium. During hypoxic injury, cellular membrane disruption results in the release of LDH into the circulation, leading to elevated serum levels. Increased LDH levels have therefore been proposed as a biochemical marker of tissue hypoxia and cellular injury in neonates with birth asphyxia [4]. Studies have demonstrated a significant association between elevated LDH levels and the severity of neonatal asphyxia, suggesting that LDH may serve as a useful indicator of hypoxic-ischemic injury in newborns [5].

Several investigators have attempted to compare biochemical markers with umbilical cord blood gas parameters in predicting neonatal outcomes. Research has shown that serum LDH levels correlate inversely with cord blood pH and directly with markers of acidosis such as $PaCO_2$, indicating that higher LDH concentrations are associated with more severe hypoxic insult [4]. Similarly, studies evaluating cord blood LDH and pH have demonstrated that both parameters are associated with adverse neonatal outcomes including need for resuscitation, NICU admission, and early neonatal mortality [6]. In some analyses, cord blood LDH has shown comparable or even superior predictive value compared with pH in identifying neonates at risk of complications.

Despite the availability of cord blood gas analysis, it is not always feasible in many resource-limited settings or during emergency referrals when cord blood samples are not collected immediately after birth. In such circumstances, alternative biochemical markers capable of reflecting perinatal hypoxia may provide valuable diagnostic information. Measurement of LDH levels from cord blood or early neonatal blood samples may therefore serve as a practical and accessible adjunct marker in identifying birth asphyxia.

Considering the need for reliable and easily measurable biomarkers for early detection of neonatal asphyxia, the present study was conducted to evaluate the diagnostic accuracy of umbilical cord blood lactate dehydrogenase (LDH) levels in comparison with umbilical cord blood pH among neonates with birth asphyxia. Understanding the relationship between these parameters may help determine whether LDH can serve as a useful adjunct or alternative biomarker for assessing perinatal hypoxia and predicting neonatal outcomes.

MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted in the Neonatal Intensive Care Unit (NICU), labour room, and obstetrics operation theatre of a tertiary care hospital over a period of 12 months from January 2025 to December 2025. The study population comprised neonates diagnosed with birth asphyxia who were delivered at the study institution during the study period. Prior approval for the study was obtained from the Institutional Ethics Committee, and written informed consent was obtained from the parents or legal guardians of all neonates before inclusion in the study.

The sample size for the present study was calculated based on previously published literature evaluating the diagnostic value of lactate dehydrogenase in birth asphyxia. Considering an anticipated sensitivity of approximately 94% reported in earlier studies and allowing for adequate statistical power and precision, the calculated sample size was increased and a total of 75 neonates fulfilling the eligibility criteria were included in the study. A convenience sampling technique was adopted, and all consecutive neonates meeting the inclusion criteria during the study period were enrolled until the required sample size was achieved.

Neonates diagnosed with birth asphyxia were included in the study. Birth asphyxia was defined according to the World Health Organization (WHO) definition as the failure to initiate or sustain breathing at birth. Clinically, neonates were categorized based on Apgar scores and respiratory status at birth. Neonates with Apgar scores between 4 and 6 at one minute of life were considered to have moderate birth asphyxia, while neonates with Apgar scores between 0 and 3 were categorized as severe birth asphyxia. Neonates with multiple congenital anomalies or those born from multifetal gestations were excluded from the study to avoid confounding factors that might influence biochemical parameters.

Immediately after delivery, irrespective of the mode of delivery (normal vaginal delivery or lower segment caesarean section), umbilical cord blood samples were collected under strict aseptic precautions. The umbilical cord was clamped at both the placental and fetal ends and then cut between the clamps. The placental end of the cord was cleaned with 70% isopropyl alcohol before sample collection. Using a sterile 22-gauge syringe, approximately 1 mL of umbilical cord

arterial blood was collected into a pre-heparinized syringe for arterial blood gas analysis. Umbilical cord blood pH was measured using an automated blood gas analyzer (ABL 80 FLEX Basic Gas Analyzer, Radiometer, 2015) at a temperature of 37°C.

In addition, approximately 2 mL of umbilical cord arterial blood was collected in a plain sterile bulb for the estimation of lactate dehydrogenase (LDH) levels. The collected blood samples were immediately transported to the central laboratory in the Department of Biochemistry for analysis. The samples were centrifuged for approximately 15 minutes to obtain serum, and LDH levels were measured using an automated biochemical analyzer (Dimension RX L Max Analyzer, Siemens DMNS1, 2012). The obtained LDH values were recorded and later compared with corresponding umbilical cord blood pH values to assess their diagnostic relationship in neonates with birth asphyxia.

For the purpose of analysis, umbilical cord blood pH values were categorized based on the degree of acidemia. A pH value greater than 7.20 was considered mild or normal acid-base status, values between 7.01 and 7.19 were categorized as moderate acidemia, and values less than 7.00 were classified as severe acidemia. Similarly, cord blood LDH levels were categorized based on the laboratory reference value, with levels below 612 IU/L considered within normal limits and values above 612 IU/L considered elevated.

Relevant maternal and neonatal demographic and clinical variables were recorded using a structured case record proforma. These included neonatal gender, gestational maturity (term or preterm), birth weight, mode of delivery, umbilical cord blood pH, cord blood LDH levels, and serum LDH levels. All collected data were compiled systematically and entered into Microsoft Excel spreadsheets for data management and preliminary analysis.

Statistical analysis was performed using appropriate statistical software. Quantitative variables were expressed as mean and standard deviation, while categorical variables were presented as frequency and percentage. Associations between categorical variables were assessed using the Chi-square test or Fisher's exact test as appropriate. Diagnostic performance parameters including sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall diagnostic accuracy were calculated to evaluate the ability of umbilical cord LDH levels in comparison with umbilical cord blood pH to identify birth asphyxia. Agreement between the two diagnostic parameters was evaluated using Cohen's kappa coefficient. A p-value of less than 0.05 was considered statistically significant.

All ethical principles related to biomedical research involving human participants were strictly adhered to during the conduct of the study. Confidentiality of patient information was maintained, and all collected data were used solely for research purposes.

RESULTS

A total of 75 neonates diagnosed with birth asphyxia were included in the study. Demographic characteristics, perinatal factors, and biochemical parameters including umbilical cord blood pH, cord blood lactate dehydrogenase (LDH), and serum LDH levels were analyzed to evaluate the diagnostic accuracy of LDH compared with cord blood pH.

Demographic and Perinatal Characteristics: Table 1 summarizes the demographic and perinatal characteristics of the study population. Among the 75 neonates, 46 (61.3%) were males and 29 (38.7%) were females, indicating a slight male predominance. Regarding gestational maturity, 44 neonates (58.7%) were term, while 31 neonates (41.3%) were preterm.

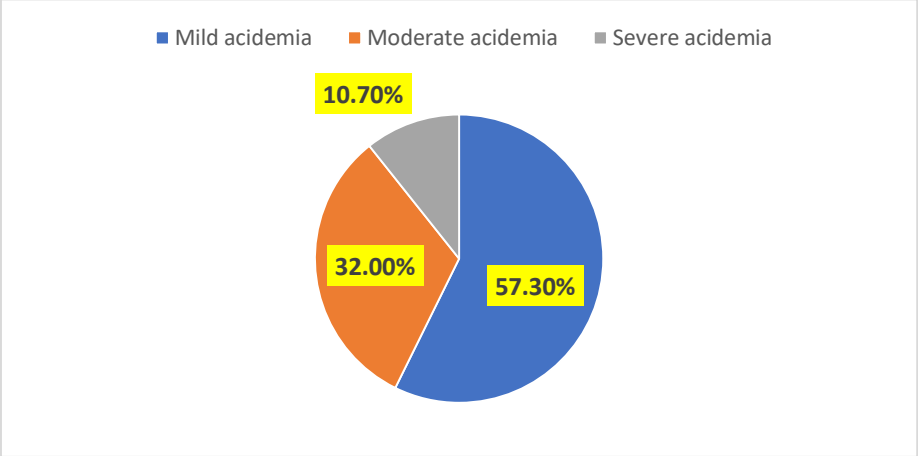
Birth weight analysis showed that the majority of neonates had birth weights between 1.5–2.5 kg (52%), followed by >2.5 kg (40%), whereas 8% had very low birth weight (<1.5 kg).With respect to mode of delivery, 40 neonates (53.3%) were delivered by lower segment cesarean section (LSCS) and 35 neonates (46.7%) were delivered by normal vaginal delivery (NVD).

Table 1: Demographic and perinatal characteristics of study subjects

Variable	Category	Frequency	Percentage
Gender	**Male**	46	61.30%
	Female	29	38.70%
Gestational maturity	**Term**	44	58.70%
	Preterm	31	41.30%
Birth weight	**>2.5 kg**	30	40.00%
	1.5–2.5 kg	39	52.00%
	<1.5 kg	6	8.00%
Mode of delivery	**NVD**	35	46.70%
	LSCS	40	53.30%

Distribution According to Umbilical Cord Blood pH: Umbilical cord blood pH values were categorized according to severity of acidosis. As shown in Figure 1, the majority of neonates 43 (57.3%) had mild acidemia (pH >7.2). Moderate acidemia (pH 7.01–7.19) was observed in 24 neonates (32.0%), while 8 neonates (10.7%) had severe acidemia (pH <7).

Figure 1: Distribution of study subjects according to cord blood pH



Cord LDH and Serum LDH Levels in Term and Preterm Neonates: Cord blood LDH and serum LDH levels were compared according to gestational maturity (Table 2). Among preterm neonates, 45.2% had elevated cord LDH levels (>612 IU/L), whereas 70.5% of term neonates showed elevated cord LDH levels.Similarly, elevated serum LDH levels were observed in 29.0% of preterm neonates and 61.4% of term neonates. However, the difference between term and preterm neonates in LDH elevation was not statistically significant (p = 0.08).

Table 2: Cord LDH and serum LDH levels according to gestational maturity

Gestational Age	Cord LDH		Serum LDH	
	>612 IU/L	<612 IU/L	>612 IU/L	<612 IU/L
Preterm (n=31)	14 (45.2%)	17 (54.8%)	9 (29.0%)	22 (71.0%)
Term (n=44)	31 (70.5%)	13 (29.5%)	27 (61.4%)	17 (38.6%)
p-value	**0.08**		**0.06**	

Association Between Cord Blood pH and Cord LDH: The relationship between cord blood pH and cord LDH levels is shown in Table 3. Elevated LDH levels were significantly associated with increasing severity of acidemia. Among neonates with severe acidemia (pH <7), 75% had elevated cord LDH levels, compared with 25.5% among neonates with pH >7.2.Statistical analysis revealed a significant association between cord blood pH and cord LDH levels (Chi-square test, p = 0.004).

Association Between Cord Blood pH and Serum LDH: Similarly, serum LDH levels demonstrated a significant relationship with cord blood pH values (Table 4). Elevated serum LDH was observed in 87.5% of neonates with severe acidemia, compared with 39.5% among neonates with mild acidemia.Statistical analysis showed a significant association between serum LDH levels and cord blood pH (p = 0.001).

Table 3: Association between cord blood pH and cord LDH levels

Cord pH	Total	Cord LDH	
		>612 IU/L	<612 IU/L
>7.2	43	11 (25.5%)	32 (74.5%)
7.01–7.19	24	16 (66.7%)	8 (33.3%)
<7.0	8	6 (75.0%)	2 (25.0%)
p-value	**0.004**		

Table 4: Association between cord blood pH and serum LDH levels

Cord pH	Total	Serum LDH	
		>612 IU/L	<612 IU/L
>7.2	43	17 (39.5%)	26 (60.5%)
7.01–7.19	24	19 (79.2%)	5 (20.8%)
<7.0	8	7 (87.5%)	1 (12.5%)
p-value	**0.001**		

DISCUSSION

Birth asphyxia remains one of the most important causes of neonatal morbidity and mortality, particularly in developing countries where access to immediate biochemical assessment may be limited. Early identification of neonates at risk of hypoxic injury is crucial for prompt intervention and prevention of long-term neurological complications. In the present study, the diagnostic value of umbilical cord blood lactate dehydrogenase (LDH) was evaluated in comparison with umbilical cord blood pH among neonates with birth asphyxia.

In the current study, male neonates constituted 61.3% of the study population, while females accounted for 38.7%, indicating a higher proportion of male neonates affected by birth asphyxia. Similar findings have been reported in several studies where male neonates were more frequently affected by perinatal asphyxia. A study by Shah et al. observed that approximately 60% of neonates with birth asphyxia were males, suggesting a possible increased susceptibility of male neonates to perinatal hypoxic stress [7]. Although the exact mechanism is not fully understood, several researchers have suggested that male neonates may have relatively reduced tolerance to hypoxic injury compared with females.

With respect to gestational maturity, 58.7% of neonates in the present study were term, while 41.3% were preterm. These findings are comparable with those reported by Kumar and Yadav, who found that the majority of neonates with birth asphyxia were delivered at term gestation [6]. Term neonates often constitute a larger proportion of asphyxia cases in hospital-based studies because high-risk term pregnancies frequently undergo close monitoring and hospital delivery. Nevertheless, preterm neonates remain particularly vulnerable to hypoxic injury due to immature organ systems and reduced physiological reserve.

Birth weight analysis in the present study demonstrated that 52% of neonates had birth weights between 1.5 and 2.5 kg, followed by 40% weighing more than 2.5 kg, whereas 8% had very low birth weight (<1.5 kg). Similar findings were reported by Kader et al., who observed that low birth weight was commonly associated with neonatal asphyxia and increased risk of adverse neonatal outcomes [8]. Low birth weight may reflect intrauterine growth restriction or prematurity, both of which are recognized risk factors for perinatal hypoxia.

In the present study, 53.3% of neonates were delivered by lower segment cesarean section (LSCS), while 46.7% were delivered by normal vaginal delivery. The higher rate of cesarean delivery among neonates with birth asphyxia may be explained by the presence of intrapartum fetal distress or obstetric complications necessitating emergency operative delivery. Similar trends have been reported in previous studies evaluating risk factors associated with birth asphyxia [9].

Analysis of umbilical cord blood pH levels in the present study showed that 57.3% of neonates had mild acidemia (pH >7.2), 32% had moderate acidemia (pH 7.01–7.19), and 10.7% had severe acidemia (pH <7). Umbilical cord blood pH remains one of the most widely accepted indicators of fetal metabolic status at birth. Previous studies have shown that severe acidemia is associated with increased risk of hypoxic ischemic encephalopathy and other adverse neonatal outcomes. Low et al. reported that neonates with cord pH less than 7.0 were at significantly higher risk of neurological complications and neonatal morbidity [3].

The present study also evaluated cord blood LDH levels among term and preterm neonates. Elevated cord LDH levels were observed in 70.5% of term neonates and 45.2% of preterm neonates. However, the difference between the two groups was not statistically significant. These findings suggest that LDH elevation may occur irrespective of gestational maturity and may primarily reflect the degree of hypoxic cellular injury. Similar observations were reported by Naseh et al., who demonstrated that serum LDH levels in neonates were significantly associated with biochemical markers of acidosis rather than with maternal or demographic characteristics [4].

One of the key findings of the present study was the significant association between cord blood pH and cord LDH levels. Elevated cord LDH levels were found in 75% of neonates with severe acidemia, compared with 25.5% among neonates with mild acidemia. This association was statistically significant ($p = 0.004$). These results indicate that cord LDH levels increase as the severity of metabolic acidosis increases, reflecting greater cellular damage resulting from hypoxic injury. Similar findings were reported by Kumar and Yadav, who demonstrated that elevated cord LDH levels were significantly associated with adverse neonatal outcomes including need for resuscitation, NICU admission, and neonatal complications [6].

In addition to cord LDH levels, serum LDH levels also showed a strong association with cord blood pH values. Elevated serum LDH was observed in 87.5% of neonates with severe acidemia, whereas only 39.5% of neonates with mild acidemia had elevated serum LDH levels. This relationship was statistically significant ($p = 0.001$). These findings support the hypothesis that LDH levels reflect the degree of tissue hypoxia and cellular injury occurring during birth asphyxia. Previous studies have also reported a negative correlation between serum LDH levels and cord blood pH, suggesting that increasing acidosis leads to increased release of intracellular enzymes into the circulation [4].

The elevation of LDH in neonates with birth asphyxia can be explained by the underlying pathophysiology of hypoxic injury. During hypoxia, anaerobic metabolism predominates, resulting in accumulation of lactate and cellular energy depletion. Cellular membrane integrity becomes compromised, leading to the release of intracellular enzymes such as LDH into the bloodstream. Because LDH is present in multiple tissues including the brain, liver, kidneys, and myocardium, elevated LDH levels may reflect multisystem cellular injury associated with severe hypoxia [10].

Another important observation from the present study is that LDH levels appear to increase progressively with increasing severity of acidemia, suggesting that LDH may serve as a useful biochemical marker for assessing the severity of birth asphyxia. In settings where cord blood gas analysis is unavailable or delayed, measurement of LDH levels may provide valuable information regarding the degree of hypoxic injury. This is particularly relevant in resource-limited settings where laboratory facilities for arterial blood gas analysis may not be readily available.[11-15]

Overall, the findings of the present study suggest that umbilical cord blood LDH and serum LDH levels have a strong association with cord blood pH and severity of metabolic acidosis in neonates with birth asphyxia. These results support the potential role of LDH as a useful adjunct biochemical marker for early detection of neonatal hypoxia and assessment of asphyxia severity.

CONCLUSION

The present study demonstrated that umbilical cord blood lactate dehydrogenase (LDH) levels showed a significant association with umbilical cord blood pH and the severity of metabolic acidosis in neonates with birth asphyxia. Elevated cord blood LDH and serum LDH levels were more frequently observed in neonates with lower cord blood pH values, indicating a strong relationship between biochemical markers of cellular injury and hypoxic insult during the perinatal period.

The findings of this study suggest that LDH levels increase progressively with increasing severity of acidosis, reflecting underlying tissue hypoxia and cellular damage associated with birth asphyxia. A significant association between elevated LDH levels and decreasing cord blood pH supports the potential role of LDH as a useful biochemical marker for assessing the severity of perinatal hypoxia.

Although umbilical cord blood pH remains the gold standard for evaluating fetal metabolic status at birth, measurement of LDH levels may serve as a valuable adjunct diagnostic tool, particularly in settings where cord blood gas analysis is not readily available. LDH estimation is relatively simple, cost-effective, and widely available, making it a practical alternative biomarker in resource-limited healthcare settings.

Therefore, umbilical cord blood LDH can be considered a useful supportive biochemical marker for early identification and assessment of birth asphyxia, which may help clinicians initiate timely management and potentially reduce neonatal morbidity and mortality. Further large-scale prospective studies are recommended to validate the diagnostic accuracy and prognostic significance of LDH in neonatal birth asphyxia.

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REFERENCES

1. Lawn JE, Cousens S, Zupan J. 4 million neonatal deaths: when? Where? Why? Lancet. 2005;365(9462):891–900.
2. American College of Obstetricians and Gynecologists, American Academy of Pediatrics. Neonatal encephalopathy and neurologic outcome. 2nd ed. Washington (DC): ACOG; 2014.
3. Low JA, Pickersgill H, Killen H, Derrick EJ. The prediction and prevention of intrapartum fetal asphyxia in term pregnancies. Am J Obstet Gynecol. 2001;184(4):724–730.
4. Naseh A, Ashrafzadeh S, Erfanian-Taghvaie M. Is serum lactate dehydrogenase level a good indicator for diagnosis of neonatal birth asphyxia? Ann Pediatr. 2021;4(2):1088.
5. Shah GS, Singh MK, Pandey TR, Kalakheti BK, Bhandari GP. Serum lactate dehydrogenase in neonatal asphyxia. J Nepal Paediatr Soc. 2004;23(2):73-76.
6. Kumar N, Yadav A. Umbilical cord arterial blood lactate dehydrogenase and pH as predictors of perinatal outcome in high-risk term pregnancies: a cohort study. J Mother Child. 2022;26(1).
7. Shah GS, Singh MK, Pandey TR, Kalakheti BK, Bhandari GP. Serum lactate dehydrogenase in neonatal asphyxia. J Nepal Paediatr Soc. 2004;23(2):73-76.

8. Kader FA, Paul S. Association between cord blood gas analysis and outcome in immediate newborn period. Dissertation, Tamil Nadu Dr MGR Medical University. 2017.
9. Nelson KB, Leviton A. How much of neonatal encephalopathy is due to birth asphyxia? Am J Dis Child. 1991;145(11):1325-1331.
10. Volpe JJ. Hypoxic-ischemic encephalopathy. In: Volpe's Neurology of the Newborn. 6th ed. Philadelphia: Elsevier; 2018.
11. Perlman JM. Intervention strategies for neonatal hypoxic-ischemic cerebral injury. Clin Ther. 2006;28(9):1353–1365.
12. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress: a clinical and electroencephalographic study. Arch Neurol. 1976;33(10):696–705.
13. Martin RJ, Fanaroff AA, Walsh MC. Fanaroff and Martin's Neonatal-Perinatal Medicine: Diseases of the Fetus and Infant. 11th ed. Philadelphia: Elsevier; 2020.
14. Buonocore G, Perrone S, Bracci R. Biochemical markers of neonatal brain injury. Clin Chem. 2002;48(11):2109–2116.
15. Thorngren-Jerneck K, Herbst A. Perinatal factors associated with cerebral palsy in children born in Sweden. Obstet Gynecol. 2006;108(6):1499–1505.