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Original Research Article

Serum lactate dehydrogenase as an early diagnostic biomarker to differentiate respiratory distress syndrome from transient tachypnea of the newborn

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ABSTRACT

Background: Early differentiation between respiratory distress syndrome (RDS) and transient tachypnea of the newborn (TTN) remains clinically challenging, particularly in settings where imaging and advanced diagnostics may be delayed. Lactate dehydrogenase (LDH) is a marker of cellular injury and hypoxic stress and may assist in early etiologic discrimination. Objectives of the study were to evaluate the diagnostic performance of serum LDH measured within the first 12 hours of life in differentiating RDS from TTN, and to examine whether LDH provides independent diagnostic value beyond gestational age and birthweight.
Methods: In this prospective diagnostic study, 151 neonates were enrolled: 50 normal controls, 63 with TTN, and 38 with RDS. Serum LDH levels were measured within 12 hours of birth. Group comparisons were performed using appropriate parametric or non-parametric tests. Diagnostic performance was assessed using receiver operating characteristic (ROC) analysis. Univariable and multivariable logistic regression models were constructed to evaluate the independent association of LDH with RDS.
Results: LDH levels differed significantly across groups, demonstrating a stepwise increase from normal neonates to TTN and RDS ($p<0.001$). In symptomatic infants (TTN versus RDS), LDH showed good discriminatory accuracy (AUC=0.84; 95% CI: 0.75–0.92). A cutoff of 657 U/l yielded 97.4% sensitivity and 69.8% specificity. In univariable analysis, LDH predicted RDS (OR per 100 U/l=1.25; 95% CI: 1.08–1.45; $p=0.003$). However, after adjustment for gestational age and birthweight, LDH was no longer independently associated with RDS.
Conclusions: Serum LDH demonstrates good early discriminatory performance for differentiating RDS from TTN and shows a biologically plausible gradient across disease severity. However, its association is largely mediated by prematurity and illness severity. LDH may serve as a useful adjunctive biomarker but should be interpreted in conjunction with perinatal and clinical parameters.

Keywords: RDS, TTN, Transient tachypnea of the newborn, LDH, Neonatal biomarkers

INTRODUCTION

Respiratory disorders in neonatal period are the most common and challenging clinical presentations that contributing significantly to neonatal morbidity and mortality. Respiratory distress syndrome (RDS) and transient tachypnea of the newborn (TTN) were the most common causes of these disorders. The overlapping clinical manifestations of these disorders make early differentiation difficult, especially at the borderline gestational ages. Precise distinction between the two disorders is crucial, as their underlying pathophysiology, progression, and management strategies differ significantly.[1-3]

RDS primarily caused by surfactant deficiency and structural lung immaturity, leading to progressive alveolar atelectasis, impaired gas exchange, ventilation perfusion mismatch, and progressive hypoxemia.[4] TTN, by contrast, is largely a benign and self-limited condition caused by delayed resorption of fetal lung fluid, typically occurring in term or late-preterm infants.[5] Although TTN often resolves with supportive care, RDS may require timely interventions such as continuous positive airway pressure (CPAP), surfactant therapy, or mechanical ventilation. Thus, early and accurate discrimination is essential to avoid both overtreatment of TTN and delayed life-saving therapy in infants with RDS.[6,7]

Biomarkers that reflect lung injury, hypoxia, cellular stress, or inflammation may enhance the diagnostic accuracy of early neonatal assessment. Lactate dehydrogenase (LDH), a cytosolic enzyme present in almost all tissues, is released into the circulation during cellular injury or metabolic stress and therefore represents a plausible early biochemical indicator of lung involvement. LDH isoenzymes show distinctive tissue distributions, and neonatal studies indicate that LDH-5, which has strong pulmonary expression, tends to peak within the first 24-48 hours of life, particularly in the presence of hypoxia or alveolar–capillary membrane injury.[8,9] Injured type I and type II pneumocytes in RDS release LDH in greater amounts compared with neonates experiencing TTN, whose pathology is primarily related to transient fluid retention rather than structural damage.[10]

Emerging data support LDH as a potential early discriminator between neonatal respiratory disorders. Animal model studies have shown elevated LDH levels in premature subjects with experimentally induced respiratory distress.[11] Point-of-care LDH measurement has also been explored as a rapid diagnostic tool in human neonates and demonstrated promise in differentiating forms of respiratory distress shortly after birth.[12] Further studies conclude that elevated LDH was associated with other conditions like bronchiolitis, indicating its sensitivity to lung injury and epithelial disruption.[9] Other studies that investigate the oxidative stress in RDS correlate elevated LDH as part of the biochemical cascade associated with alveolar injury and inflammatory process.[4] Moreover, novel diagnostic approaches such as lung fluid quantification and metabolomic profiling emphasize the growing interest in biomarkers for early prediction of respiratory disease severity, highlighting the need for simple, low-cost laboratory markers applicable at the bedside.[5, 10]

Distinguishing RDS from TTN at the earliest possible stage is clinically imperative. Misclassification may result in unnecessary administration of surfactant, inhaled nitric oxide, or mechanical ventilation in TTN, or conversely, delayed intervention and higher morbidity in true RDS cases. LDH being widely available, simple to measure, and rapidly obtainable may provide neonatologists with an adjunctive tool to support clinical judgment, radiologic evaluation, and other laboratory markers during the critical first hours of life.

Accordingly, the present study investigates the diagnostic value of serum LDH levels measured within the first 12 hours of life as an early biomarker and its potential role to distinguish RDS from TTN in neonates presenting with respiratory distress.

METHODS

Study design and setting

This was a prospective hospital-based observational diagnostic study conducted at the neonatal care unit (NCU) of the Regional Pediatrics Hospital, Najaf, Iraq, over a 6-month period from May 2024 to October 2024. The study was designed to evaluate the diagnostic performance of serum LDH measured within the first 12 hours of life in differentiating RDS from TTN.

A total of 151 neonates were enrolled, 63 neonates were admitted to the NCU and diagnosed with TTN and 38 neonates were admitted to the NCU and diagnosed with RDS and 50 healthy newborn infants were enrolled as a normal group from those delivered in the hospital.

Diagnostic criteria

The diagnosis of RDS and TTN was established based on a combination of clinical presentation and radiological findings in accordance with standard neonatal practice.

RDS was diagnosed in preterm neonates presenting with early-onset respiratory distress (tachypnea, grunting, chest retractions, and oxygen requirement) accompanied by characteristic chest radiograph findings, including reduced lung volumes, diffuse reticulogranular (ground-glass) appearance, and air bronchograms.

TTN was diagnosed in neonates with respiratory distress occurring shortly after birth, typically in term or late-preterm infants, with chest radiographic findings suggestive of retained fetal lung fluid (prominent central vascular markings, fluid in interlobar fissures, hyperinflation, and mild cardiomegaly) and a self-limited clinical course without evidence of surfactant deficiency.

All chest radiographs were interpreted by experienced neonatologists in conjunction with clinical findings.

Neonates with major congenital anomalies, structural malformations, meconium aspiration syndrome, perinatal asphyxia, sepsis, pneumonia, or those with incomplete clinical or laboratory records due to early discharge or missing data were excluded.

Data were collected prospectively using a structured questionnaire designed by the researcher, comprising both maternal and neonatal variables.

Neonatal parameters included the gestational age, sex and birth weight and the clinical features (Oxygen saturation, the use of nasal cannula, CPAP, or endotracheal tube, Apgar scores at 1 and 5 minutes, requirement for neonatal resuscitation at birth and the duration of respiratory support duration of admission. While maternal parameters included maternal age, mode of delivery (vaginal, caesarean).

A 1 ml venous blood sample was collected from each newborn infant within the first 12 hours after delivery using sterile technique. All samples were labeled, centrifuged to obtain serum, and stored at -20°C in the hospital laboratory biorepository. Serum samples were subsequently analyzed, and LDH levels were measured, recorded and integrated into each patient's data sheet.

The study proposal was approved by the ethical committee of pediatrics, Iraqi Board for Medical Specializations. Verbal informed consent was obtained from parents prior to sample collection. All patient information was treated confidentially. Administrative approval was granted by Al-Zahraa Teaching Hospital.

Statistical analysis

Data were analyzed using statistical package for the social sciences (SPSS), version 27 (IBM Corp., Armonk, NY, USA).

Continuous variables were assessed for normality using the Shapiro–Wilk test and visual inspection of histograms. As serum LDH and several clinical variables demonstrated non-normal distribution, continuous data were expressed as median and interquartile range (IQR). Comparisons between the three study groups (normal, TTN, and RDS) were performed using the Kruskal–Wallis test for continuous variables, followed by Bonferroni-adjusted pairwise comparisons when appropriate. For comparisons between two groups (TTN versus RDS), the Mann–Whitney U test was used.

Categorical variables were expressed as frequencies and percentages and compared using the Chi-square test or Fisher's exact test when expected cell counts were <5.

Correlation between serum LDH and clinical parameters was assessed using Spearman's rank correlation coefficient (ρ).

The diagnostic performance of LDH in differentiating RDS from TTN was evaluated using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC) with 95% confidence intervals (CI) was calculated. The optimal cutoff value was determined using Youden's index.

Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated.

To evaluate the association between LDH and RDS, univariable logistic regression analysis was first performed. Subsequently, a multivariable logistic regression model was constructed including gestational age and birthweight as a priori confounders to assess whether LDH independently predicted RDS. Odds ratios (ORs) with 95% confidence intervals were reported. All statistical tests were two-tailed, and a p value <0.05 was considered statistically significant.

RESULTS

A total of 151 neonates were included in the study: 50 normal newborns, 63 diagnosed with TTN, and 38 diagnosed with RDS. Baseline characteristics differed significantly among the three groups. Neonates with RDS had significantly lower gestational age and birthweight compared with both TTN and normal infants (p<0.001). Apgar scores at 1 and 5 minutes were also significantly lower in the RDS group (p<0.001). Duration of oxygen therapy and length of hospital stay were progressively longer from normal to TTN to RDS (p<0.001). There were no statistically significant differences in sex distribution or mode of delivery between groups (Table 1).

Table 1: Clinical characteristics and comparison of the study groups.

Variables	Normal (n=50)	TTN (n=63)	RDS (n=38)	P value
Gestational age (weeks)	37.25 (37–38)	37 (37–38)	32 (32–34)	<0.001
Birth-weight (g)	3200 (3100–3375)	3000 (2800–3200)	1900 (1625–2100)	<0.001
Apgar 1 min	8 (6–8)	6 (6–7)	5 (4–6)	<0.001
Apgar 5 min	9.5 (9–10)	8 (8–8)	7 (6–8)	<0.001
Oxygen duration (days)	0 (0–0)	2 (2–3)	5 (3–7)	<0.001
Admission duration (days)	0 (0–0)	2 (2–3)	5 (3–7.75)	<0.001
LDH (U/l)	391 (342–447)	601 (548–717)	868.5 (748–1060)	<0.001
Male sex, N (%)	22 (44)	38 (60.3)	19 (50)	0.214
Caesarean section, N (%)	39 (78)	49 (77.8)	32 (84.2)	0.705

Serum LDH concentrations demonstrated a significant difference across the three groups (p<0.001), with a clear stepwise increase corresponding to disease severity. Median (IQR) LDH values were: normal group: 391 U/l

(342–447), TTN group: 601 U/l (548–717), RDS group: 868 U/l (748–1060).

Post-hoc pairwise comparisons confirmed significant differences between all groups (normal versus TTN, normal versus RDS, and TTN versus RDS; all p<0.001) (Table 1).

In the symptomatic cohort (TTN and RDS combined), serum LDH showed significant associations with clinical parameters. LDH correlated negatively with: gestational age (ρ=−0.606, p<0.001), birthweight (ρ=−0.612, p<0.001) and positively with duration of oxygen therapy (ρ= 0.652, p<0.001) and length of hospital stay (ρ=0.639, p<0.001) (Table 2).

Receiver operating characteristic (ROC) analysis demonstrated that LDH has good discriminative accuracy for distinguishing RDS from TTN, with an area under the curve (AUC) of 0.84 (95% CI: 0.75–0.93), p<0.001. The optimal LDH cutoff identified by Youden's index was 657 U/l, with sensitivity=97.4%, specificity=69.8%, PPV=66.1% and NPV=97.8% (Table 3).

In univariable logistic regression, LDH was a significant predictor of RDS. For every 100-U/l increase in LDH, the odds of having RDS increased by 25% (OR: 1.25, 95% CI: 1.08–1.45, p=0.003). In multivariable model (adjusted for gestational age and birthweight), LDH was not independently significant, gestational age and birthweight were significant predictors. This indicates that the association between LDH and RDS is largely mediated by prematurity (Table 4).

Table 2: Spearman correlation between LDH and clinical variables.

Variables	ρ (Spearman)	P value
Gestational age	−0.606	<0.001
Birthweight	−0.612	<0.001
Apgar 1 min	−0.467	<0.001
Apgar 5 min	−0.526	<0.001
Oxygen duration	0.652	<0.001
Admission duration	0.639	<0.001

Table 3: LDH cutoffs, sensitivity and specificity.

LDH cutoff (U/l)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
600	97.4	49.2	53.6	96.9
650	97.4	66.7	63.8	97.7
657 (optimal)	97.4	69.8	66.1	97.8
700	86.8	73.0	66.0	90.2
800	60.5	81.0	65.7	77.3

Table 4: Univariable and multivariable logistic regression analysis for predictors of RDS (versus TTN).

Variables	Unit of increase	Univariable OR (95% CI)	P value	Adjusted OR* (95% CI)	P value
LDH	Per 100 U/l	1.25 (1.08–1.45)	0.003	1.04 (0.94–1.16)	0.41
Gestational age	Per 1 week	—	—	0.42 (0.31–0.57)	<0.001
Birthweight	Per 100 g	—	—	0.75 (0.68–0.83)	<0.001
Male sex	Reference: female	0.66 (0.30–1.47)	0.31	0.79 (0.32–1.96)	0.61
Cesarean delivery	Reference: vaginal	1.49 (0.50–4.42)	0.47	1.28 (0.37–4.45)	0.70

DISCUSSION

In this prospective hospital-based diagnostic study, we demonstrated that serum LDH measured within the first 12 hours of life exhibits a biologically coherent and statistically robust gradient across normal neonates, TTN, and RDS. LDH showed good discriminatory performance for differentiating RDS from TTN (AUC=0.84), with very high sensitivity at clinically relevant thresholds. Consistent with the established pathophysiological distinctions between these two conditions, neonates with RDS exhibited significantly lower gestational age, lower birth weight, poorer Apgar scores, and substantially prolonged hospital and oxygen therapy durations compared with infants diagnosed with TTN or healthy newborns, these findings align with existing literature.[1-4,13,14]

However, after adjustment for gestational age and birthweight, LDH was no longer independently associated with RDS. This finding should not be interpreted as a failure of LDH as a biomarker, but rather as evidence that LDH reflects the biological consequences of prematurity and pulmonary immaturity. In this sense, LDH functions as a marker of disease severity rather than an etiologically independent discriminator.

Our findings align with previous evidence linking LDH elevation to oxidative stress and epithelial disruption in neonatal respiratory pathology. Negi et al demonstrated associations between oxidative biomarkers and RDS severity, while more recent work has emphasized metabolic injury pathways in preterm lung disease.[4,15,16] Furthermore, metabolomic analyses of gastric fluid in very preterm infants have shown biochemical signatures predictive of surfactant requirement, underscoring the metabolic component of RDS pathophysiology.[10]

Recent studies have reported elevated LDH in neonates with RDS compared to TTN. Moustafa et al confirmed

higher LDH levels in preterm infants with RDS and suggested diagnostic utility in early differentiation.[15] Similar diagnostic patterns have been reported in earlier work by An et al, who found that LDH reliably differentiated RDS from TTN in Korean neonates, and by Lee et al, who demonstrated LDH's effectiveness to correlate with increasing severity of neonatal respiratory distress.[16,18] Moreover, Elfarargy et al identified LDH among several promising biochemical markers capable of distinguishing RDS from TTN in early neonatal life.[17,20]

However, many prior studies relied primarily on unadjusted comparisons or ROC analysis. Our study extends this literature by incorporating multivariable logistic regression adjusting for gestational age and birthweight—two dominant biological determinants of RDS. The attenuation of LDH's independent association after adjustment provides a more nuanced interpretation and avoids overestimating its standalone diagnostic value.

In parallel, recent advances in neonatal diagnostics increasingly emphasize multimodal approaches. Lung ultrasound scoring systems have shown strong diagnostic performance in differentiating TTN from RDS, particularly in preterm populations.[20] Studies have shown that combining biochemical markers such as LDH, CRP, and inflammatory cytokines enhances etiologic differentiation of neonatal respiratory distress.[21,22] Furthermore, De Bernardo et al demonstrated that incorporating umbilical cord blood gas analysis may promote early prediction of RDS risk in vulnerable newborns.[23] These studies collectively support a paradigm shift toward integrated risk stratification rather than reliance on a single marker.

Within this evolving framework, LDH retains practical relevance because of its universal availability, low cost, and rapid turnaround time—especially in low-resource or time-sensitive settings. LDH may assist in triaging newborns who are more likely to require aggressive interventions such as CPAP, surfactant administration, or mechanical ventilation interventions strongly associated with clinical outcomes in RDS.[2,24]

The high sensitivity (97.4%) and negative predictive value (97.8%) observed at the optimal LDH cutoff suggest that LDH may function effectively as a rule-out biomarker for RDS during the early postnatal period. In neonates presenting with respiratory distress and LDH values below approximately 650 U/l, the probability of RDS appears low, potentially reducing unnecessary early surfactant administration or invasive ventilation.

Early differentiation between TTN and RDS remains challenging, particularly in late-preterm infants where clinical and radiographic overlap is common. An adjunctive biochemical marker that correlates with illness burden may assist clinicians in early triage and monitoring decisions, especially when imaging access is delayed.

Moreover, the observed correlations between LDH and oxygen requirement and hospitalization duration suggest that LDH may also serve as an early indicator of respiratory severity and resource utilization.

To our knowledge, few studies have simultaneously evaluated intergroup biochemical gradients, ROC-based discriminatory performance, correlation with illness severity markers and multivariable adjustment for prematurity-related confounders within a single prospective neonatal cohort. This integrated analytical framework enhances interpretive robustness and reduces the risk of oversimplifying biomarker utility.

Limitations

Several limitations warrant consideration. First, the significant gestational age difference between RDS and TTN groups may introduce residual confounding despite statistical adjustment. Second, LDH is a nonspecific marker of cellular injury and may be elevated in other neonatal conditions such as sepsis or perinatal hypoxia. Third, this was a single-center study, potentially limiting generalizability. Fourth, incremental predictive value beyond gestational age was not formally assessed using net reclassification improvement or integrated discrimination indices. Future multicenter studies incorporating biomarker panels, lung ultrasound scoring, and machine learning–based predictive modeling may further clarify the incremental contribution of LDH within comprehensive diagnostic algorithms.

CONCLUSION

In conclusion, serum LDH measured within the first 12 hours of life demonstrates a biologically plausible and clinically meaningful gradient across normal neonates, TTN, and RDS. Although LDH shows good early discriminatory accuracy, its elevation appears largely mediated by prematurity and pulmonary immaturity. LDH should therefore be interpreted as an adjunctive severity marker rather than a standalone diagnostic determinant. When integrated with gestational age, birthweight, and clinical assessment, LDH may enhance early risk stratification and support evidence-informed respiratory management in neonates with respiratory distress.

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REFERENCES

1. Orloff KE, Turner DA, Rehder KJ. The Current State of Pediatric Acute Respiratory Distress Syndrome. Pediatr Allergy Immunol Pulmonol. 2019;32(2):35-44.
2. Qari SA, Alsufyani AA, Muathin SH, Margoushy NE. Prevalence of Respiratory Distress Syndrome in Neonates. Egyptian J Hospital Med. 2018;70(2):257-64.
3. Edwards MO, Kotecha SJ, Kotecha S. Respiratory distress of the term newborn infant. Paediatr Respir Rev. 2013;14(1):29-37.
4. Negi R, Pande D, Karki K, Kumar A, Khanna RS, Khanna HD. A novel approach to study oxidative stress in neonatal respiratory distress syndrome. BBA Clin. 2014;3:65-9.
5. Yoon SJ, Han JH, Cho KH, Park J, Lee SM, Park MS. Tools for assessing lung fluid in neonates with respiratory distress. BMC Pediatr. 2022;22(1):354.
6. Bos LDJ, Ware LB. Acute respiratory distress syndrome: causes, pathophysiology, and phenotypes. Lancet. 2022;400(10358):1145-56.
7. Silveira Neves G, Silveira Nogueira Reis Z, Maia de Castro Romanelli R, Dos Santos Nascimento J, Dias Sanglard A, Batchelor J. The role of chest X-ray in the diagnosis of neonatal respiratory distress syndrome: a systematic review concerning low-resource birth scenarios. Glob Health Action. 2024;17(1):2338633.
8. Halvorsen CP, Olson L, Araújo AC, Karlsson M, Nguyễn TT, Khu DTK, et al. A rapid smartphone-based lactate dehydrogenase test for neonatal diagnostics at the point of care. Sci Rep. 2019;9(1):9301.
9. Mehta R, Scheffler M, Tapia L, Aideyan L, Patel KD, Jewell AM, et al. Lactate dehydrogenase and caspase activity in nasopharyngeal secretions are predictors of bronchiolitis severity. Influenza Other Respir Viruses. 2014;8(6):617-25.
10. Besiri K, Begou O, Lallas K, Kontou A, Agakidou E, Deda O, et al. Gastric Fluid Metabolomics Predicting the Need for Surfactant Replacement Therapy in Very Preterm Infants Results of a Case-Control Study. Metabolites. 2024;14(4):196.
11. Ider M, Naseri A, Ok M, Uney K, Erturk A, Durgut MK. Biomarkers in premature calves with and without respiratory distress syndrome. J Vet Intern Med. 2021;35(5):2524-33.
12. Majors CE, Smith CA, Natoli ME, Kundrod KA, Richards-Kortum R. Point-of-care diagnostics to improve maternal and neonatal health in low-resource settings. Lab. Chip. 2017;17:3351-87.
13. Sun H, Xu F, Xiong H, Kang W, Bai Q, Zhang Y, et al. Characteristics of respiratory distress syndrome in infants of different gestational ages. Lung. 2013;191(4):425-33.
14. Hermansen CL, Mahajan A. Newborn Respiratory Distress. Am Fam Physician. 2015;92(11):994-1002.
15. Moustafa A, Mohamed Ahmed M, El-Mazary A. Can Serum Lactate Dehydrogenase Differentiate between Respiratory Distress Syndrome (RDS) and Transient Tachypnea of Newborn (TTN) in Preterm Neonates? Ann Neonatol. 2024;6(2):92-101.
16. Sweet DG, Carnielli V, Greisen G, Hallman M, Ozek E, Te Pas A, et al. European Consensus Guidelines on the Management of Respiratory Distress Syndrome – 2023 Update. Neonatology. 2023;120(1):3-23.
17. An YS, Kim IU, Yang MY, Jeong H, Kim H. Serum enzymes in predicting transient tachypnea of newborn and respiratory distress syndrome. Korean J Perinatol. 2014;25(4):284-91.
18. Lee M, Lee N, Bae MH, Han YM, Park KH, Byun SY, et al. Using lactate dehydrogenase to predict the severity of respiratory distress in term newborn infants with no perinatal asphyxia. Turk J Pediatr. 2021;63(3):393-403.
19. Elfarargy MS, Al-Ashmawy GM, Abu-Risha S. Novel predictor markers for early differentiation between transient tachypnea of newborn and respiratory distress syndrome in neonates. Int J Immunopathol Pharmacol. 2021;35:20587384211000554.
20. Srinivasan S, Aggarwal N, Makhaik S, Jhobta A, Kapila S, Bhoil R. Role of lung ultrasound in diagnosing and differentiating transient tachypnea of the newborn and respiratory distress syndrome in preterm neonates. J Ultrason. 2022;22(88):e1-e5.
21. Atay FY, Kutman GK, Bidev D, Oguz SS. The Role of Cardiovascular and Inflammatory Markers in Elucidating the Etiology of Respiratory Distress in the Newborn. Haydarpaşa Numune Med J. 2019;59(4):370-4.
22. Ismail AQ, Gandhi A. Using CRP in neonatal practice. J Matern Fetal Neonatal Med. 2015;28(1):3-6.
23. De Bernardo G, De Santis R, Giordano M, Sordino D, Buonocore G, Perrone S. Predict respiratory distress syndrome by umbilical cord blood gas analysis in newborns with reassuring Apgar score. Ital J Pediatr. 2020;46(1):20.
24. Najafian B, Aminsaburi A, Fakhraei SH, Afjeh A, Eghbal F, Noroozian R. Predicting Factors of INSURE Failure in Low Birth Weight Neonates with RDS; A Logistic Regression Model. Iran J Neonatol. 2015;5(4):30-4.

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