Clinical Epidemiology and Global Health 8 (2020) 927–933

Contents lists available at ScienceDirect

Clinical Epidemiology and Global Health

journal homepage: www.elsevier.com/locate/cegh

Original article

Role of umbilical cord arterial pH and lactate in newborn assessment of term antenatal women with hypertensive disorders of pregnancy

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ARTICLE INFO

Keywords:
Hypertension
Hypoxic ischemic encephalopathy
Neonate
Pregnancy
Umbilical cord

ABSTRACT

Problem: To know role of cord arterial blood pH and lactate dehydrogenase levels in neonatal outcome assessment.

Methods: Present observational study was conducted in Obstetrics and Gynecology department of a rural tertiary center of Northern India over 6 months (July–January 2019) on 155 term (≥37-≤42 weeks) antenatal women with hypertensive disorders of pregnancy fulfilling inclusion criteria. Immediately after delivery, arterial blood sample was drawn from doubly clamped 10–12 cm long umbilical cord in pre-heparinized insulin syringe, which was sent for pH estimation and 0.5 ml in lithium heparin tube for lactate dehydrogenase levels. One- and 5-min neonatal Apgar score was noted by pediatrician. Cord blood pH and lactate levels were then compared with overall neonatal outcome. Statistical analysis was done using SPSS-22 version.

Results: Mean values of umbilical cord arterial pH and LDH was 7.2 ± 0.1 and 449.5 ± 562.9 U/L respectively. Significantly low mean cord blood pH (7.03 ± 0.12) and high LDH levels (939.74 ± 781.75 U/L) were observed in neonates of eclamptic mothers ($p = 0.00$). Mode of delivery had significant effects on cord blood parameters with significantly low pH and elevated LDH levels seen in neonates delivered by emergency LSCS for fetal distress ($p < 0.05$). Cord blood LDH levels were more significantly associated with NICU admission, neonatal morbidity and mortality. Cord blood LDH was a better predictor of neonatal outcome with 100% sensitivity and 79.73% specificity.

Conclusion: Cord arterial blood lactate dehydrogenase levels were better predictor of overall neonatal outcome.

1. Introduction

Worldwide hypertensive disorder of pregnancy (HDP) is one of major causes of perinatal and maternal morbidity and mortality.[1–3] and affects approximately 2–10% of all pregnancies.[4] HDP especially eclampsia and pre-eclampsia were found to be strongly associated with increased risk of intrapartum fetal hypoxia, severe birth asphyxia, stillbirth, and intrapartum fetal death.[5,6]

Furthermore, fetal hypoxia results whenever there is compromise in maternal oxygenation, placental perfusion, or when the oxygenated blood supply to the fetus is hampered due to any reason. As a result of inadequate oxygenation, there occurs anaerobic metabolism with production of huge amounts of organic acids, especially lactic acid. Accumulation of lactic acid in turn leads to depletion of buffer system resulting in metabolic acidosis with associated low fetal pH, fetal distress and poor Apgar score at birth.[7] The resultant fetal acidosis increases the risk of intrapartum- cerebral palsy,[8,9] hypoxic ischemic encephalopathy (HIE).[10,11]and neonatal death.[12,13]

There are various ways of detection of fetal hypoxia, of which most commonly used are Apgar score at birth, umbilical cord blood gas analysis and cord blood lactate dehydrogenase levels. Fetal acidosis results in low Apgar score at birth (< 7), low umbilical cord pH (umbilical artery cord blood pH < 7.24),[14] high umbilical base deficit or high cord blood lactate dehydrogenase levels (> 612U/L).[15] Umbilical cord blood pH has a significant effect on various fetal organ functions, even a minor change in cord blood pH can severely affect organ systems, especially nervous, and cardiovascular system with resultant fetal distress and poor Apgar score at birth.[7] The normal value of umbilical cord arterial blood pH is 7.24 ± 0.07.[14] Neonates with cord blood values lower than one to two standard deviation from mean value were associated with a substantial risk of poor Apgar score, increased neonatal intensive care unit (NICU) admission, and need for assisted neonatal ventilation.[14] Furthermore, neonatal death was found to be more likely at pH < 7.00.[16] Another important indicator of neonatal birth

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https://doi.org/10.1016/j.cegh.2020.02.026
Received 17 July 2019; Received in revised form 15 February 2020; Accepted 26 February 2020
Available online 28 February 2020

asphyxia is cord blood arterial lactate dehydrogenase (LDH) levels.[12] LDH activity is significantly linked to severity of neonatal diseases such as birth asphyxia,[12,15,17,18] HIE,[19] respiratory distress,[20] necrotizing enterocolitis.[21] and NICU admission.[12,22]

Hence, the present study was conducted with the aim to know the role of umbilical cord arterial pH and LDH levels in assessment of newborns delivered to term antenatal women with HDP.

2. Material and methods

Study Design, setting and duration: Present prospective observational study was conducted in the department of Obstetrics and Gynaecology of rural tertiary care centre of Northern India over six months (July 2018 to January 2019).

Study Sample: All one hundred fifty-five (155) term antenatal women with Hypertensive Disorders of Pregnancy, including Gestational hypertension, Pre-eclampsia, Eclampsia and Chronic hypertension reporting to the labour ward during the study period.

Inclusion criteria: Gestational age ≥37 to ≤42 weeks, singleton live pregnancy, women with HDP (Gestational hypertension/Chronic hypertension/Pre-eclampsia/Eclampsia).

Exclusion criteria: Gestation < 37 or > 42 weeks, normotensive antenatal women, multifetal gestation, intrauterine fetal death, fetus with congenital malformations, maternal drug addiction or smoking or any substance abuse, neonates born to mothers who had received opioids within 4 h prior to delivery (pharmacological depression), hemolytic disease of the newborn, previous cesarean section with scar dehiscence or scar rupture.

2.1. Definitions

Hypertension in pregnancy: It is defined as diastolic blood pressure of ≥90 mmHg on two or more occasions more than 4 h apart or a single diastolic blood pressure of > 110 mmHg.[23]

Gestational hypertension: It is new onset hypertension with systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or both at or after 20 weeks of gestation without proteinuria and or other features of preeclampsia.[24,25]

Chronic Hypertension: It is defined as blood pressure ≥140/90 mmHg before pregnancy or before 20 weeks of gestation.[26]

Preeclampsia: It is defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or both with proteinuria and, potentially other end-organ dysfunction.[24] When the systolic blood pressure becomes ≥160 mmHg or diastolic blood pressure ≥110 mmHg, or both, with epigastric pain, cerebral/visual disturbances, decreased urine output, pulmonary edema, thrombocytopenia, deranged liver functions, or intrauterine growth restriction, it is called as severe pre-eclampsia.[27]

Eclampsia: It is defined as new onset grand-mal seizures in women with preeclampsia.[23,24]

Hypoxic Ischemic Encephalopathy: It is defined as deficiency of oxygenation and blood perfusion of brain, leading to brain injury.[28] It is classified into three stages.[29]

Stage I Neonates are alert, with normal or hypertonic muscle tone, tendon reflexes are increased, but seizures are absent and the neonates usually remain active and survival is best.

Stage II Neonates are lethargic with hypotonia, tendon reflexes are increased and seizures are common. All the reflexes of baby are weak and electroencephalography (EEG) shows low voltage, periodic or paroxysmal waves. Mortality ranges between 14 and 17%.

Stage III The neonates are comatose with flaccid muscle tone; tendon reflexes are depressed or absent with frequent seizures. All neonatal reflexes are absent with EEG showing periodic or isoelectric pattern. Long term morbidity and mortality is usually very high.

Study Population: One hundred fifty-five (155) term antenatal women with HDP admitted in the labour ward of Obstetrics and Gynaecology department.

Ethical Issues: The present prospective observational study was carried out after proper Institutional Ethical Committee approval and informed written consent from all the participants.

Methodology: After obtaining informed written consent from all the randomly selected participants in their vernacular language, demographic features like age, gestation, parity, type of HDP, mode of delivery, etc were recorded on structured data collection sheet. A detailed medical and family history of all participants was taken to ensure that they fulfil the inclusion criteria for study. History of any antepartum convulsions were also recorded in un-booked cases to classify them as eclamptic, if otherwise proved. This was followed by a thorough physical examination of every subject which was recorded. The blood pressure of all the subjects at admission to the labor ward and after 2 h of rest was carefully noted with the woman in sitting position with her arm at the level of heart using an appropriately sized cuff as per the clinical guidelines given by the Society of Obstetricians and Gynecologists of Canada.[24] In order to differentiate between gestational hypertension and pre-eclampsia, around 10 ml of mid-stream urine sample of all the patients was taken in a clean container for urinary protein analysis using dipsticks and it was graded as trace to 4+ (Trace, 0.1 g/L; 1+, 0.3 g/L; 2+, 1 g/L; 3+, 3.0 g/L; 4+, 10 g/L). Furthermore, a detailed history of any pre-pregnancy or early pregnancy (< 20 weeks) rise in blood pressure was recorded to confirm chronic hypertension.

Immediately after delivery by any route (vaginal/cesarean) and before the newborn's first cry umbilical cord arterial blood sample was drawn from the double-clamped 10–12 cm long segment of the cord, as minor changes can occur in umbilical pH within 60 s of delivery,[29] and over 60 min it (both arterial or venous pH) can fall by more than 0.2 pH units.[30] When the cord is doubly clamped immediately after delivery, the pH remains relatively stable at room temperature for an hour.[31–33] The arterial blood sample thus drawn was then collected anaerobically in a pre-heparinized insulin syringe, which was sent immediately for pH estimation by blood gas analyzer at 37° Celsius and around 0.5 ml in a lithium heparin tube for the analysis of LDH levels. Apgar score of the neonates was noted at 1 and 5 min after birth by the pediatrician.

The data thus obtained included gravidity, parity, gestational age at delivery, mode of delivery, neonatal Apgar score at birth, gender and birth weight of the fetus, umbilical cord arterial blood pH and LDH values, NICU admission, neonatal complications and outcome.

2.2. Laboratory measurements

The acid-base values were analyzed immediately using Institutional Siemens arterial blood gas Analyzer machine. The normal reference value taken for arterial cord blood pH in term newborn was 7.24 ± 0.07.[14] In addition to this 0.5 ml of arterial cord blood sample collected in a lithium heparin tube for LDH analysis was transported within 5 min to the department of biochemistry, where the analysis was performed within 10 min of sampling.

Though LDH can be separated into five different isoenzymes, but in this study, we measured the total LDH activity, which is routinely considered in clinical practice. LDH was analyzed in the biochemistry department of our Institute, using the standard procedure (DGKC method), in which the test tubes containing cord blood sample were first centrifuged and then analyzed by the Erba Mannheim XL System (ERBA diagnostics Mannheim GmbH, Germany). Two reagents (R1 and R2) were used, R1 consisted of Tris buffer (pH 7.5) 100 mmol/l and Pyruvate 2.0 mmol/l and R2 was NADH 1.66 mmol/l. The activity of LDH was then detected by using the reaction given as under: Pyruvate + NADH + H+ → Lactate + NAD+.[34] in which LDH

catalysis the reduction of pyruvate to lactate oxidizing reduced nicotinamide adenine dinucleotide (NADH) to NAD. The rate of oxidation of NADH was found to be directly proportional to LDH activity in the cord blood sample and the activity was determined by rate of decrease in absorbance at 340 nm as NAD was produced. The analytic range for the equipment was 43.8–1200 U/L with a between-days coefficient of variation (CV) of 2.43%. The normal reference value for LDH taken from umbilical cord arterial blood in a term newborn was < 612 U/L.[15]

2.3. Statistical analysis

Statistical analysis of data was performed using software SPSS version 22.0. Unpaired *t*-test/Mann-Whitney U- test was used for comparison of two continuous variables and for comparison between more than two groups, ANOVA/Kruskal Wallis test was used. The qualitative variables were correlated using Chi-Square test or Fisher's exact test and a p value of < 0.05 was considered statistically significant.

3. Result

Of total 155 term antenatal women, 96 (61.9%), had Gestational hypertension, 34 (21.9%) Pre-eclampsia, 17 (11%) Eclampsia and eight (5.2%) had Chronic hypertension. The mean ± SD age of all the patients was 25.6 ± 3.1 years with a minimum of 20 years and maximum of 35 years. The mean ± SD for gestation was 38.48 ± 1.113 weeks, with a minimum of 37 weeks and maximum of 41 weeks. The various demographic features including age, gestation, gravidity, parity, type of HDP, mode of delivery and neonatal birth weight are depicted in Table 1. The comparison of type of HDP with neonatal Apgar score at 1, 5 min, umbilical cord blood arterial pH and LDH levels is depicted in Table 2. Of total 155 term antenatal women with HDP, 85 (54.8%) had normal delivery, 66 (42.6%) lower segment cesarean section (LSCS) [59 (89.4%) emergency LSCS and 7 (10.6%) elective LSCS], three (1.9%) instrumental delivery and one (0.6%) assisted breech delivery. Of 59 emergency LSCS, 40 (67.8%) were done for fetal distress and 19 (32.2%) for reasons other than fetal distress like non-progress of labor, breech in labor and previous cesarean section scar in labor, etc. Neonates born to mothers who underwent emergency LSCS for fetal distress had higher cord blood LDH levels (mean ± SD:673.98 ± 463.34 U/L vs 538.56 ± 869.31U/L) and lower arterial pH (7.12 ± 0.14 vs 7.21 ± 0.12) as compared to the neonates delivered to mothers who underwent LSCS for indications other than fetal distress. Furthermore, of all the delivered neonates 118 (76.13%) had cord blood LDH < 612 U/L and 37 (23.9%) had > 612U/L. Of these 37 neonates, 27 (73%) were delivered by emergency LSCS, two (5.4%) had instrumental delivery, one (2.7%) by elective LSCS and one (2.7%) by breech assisted vaginal delivery. Hence, neonatal umbilical cord arterial blood LDH levels were significantly associated with the mode of delivery (p = 0.000).

Three eclamptic and one pre-eclamptic antenatal women required ventilatory support for severe hypertension with pulmonary edema, of which one could not be survived and succumbed to death, but the neonates of all four survived.

Of total 155 delivered neonates, 78 (50.3%) were females and 77 (49.7%) were males. Around 78.1% and 93.5% neonates had Apgar score > 7 at one and 5 min respectively, whereas only 21.9% and 6.5% had Apgar score < 7 at one and 5 min respectively. The mean ± SD of birth weight of all the neonates was 2.7 ± 0.4 Kg with a minimum of 1.7 Kg and maximum of 3.8 Kg. The mean ± SD values of umbilical cord arterial pH and LDH was 7.2 ± 0.1 and 449.5 ± 562.9 U/L respectively. No significant correlation was observed between neonatal gender, birth weight, and cord blood arterial LDH and pH values. Of total 155 neonates, 112 (72.3%) were born healthy, 22 (14.2%) suffered from HIE I, 14 (9%) HIE II and the remaining 7 (4.5%) had HIE III. The correlation of severity of HIE with mean umbilical cord blood arterial pH and LDH levels is shown in Table 3. Of all the neonates, 112 (72.3%) required resuscitation at birth with 54 (34.8%) requiring NICU admission. There was a total of seven (4.5%) neonatal deaths. The correlation of NICU admission and neonatal death with mean umbilical cord blood arterial pH and LDH levels is depicted in Table 4. It was observed that cord blood LDH was a better predictor of overall neonatal outcome with high sensitivity and negative predictive value for neonatal morbidity and mortality as compared to pH and Apgar score at birth. The sensitivity (100% vs 85.71%), specificity (79.73% vs 59.46%) and Negative predictive value (100% vs 98.88%) of cord blood LDH levels was higher as compared to cord blood arterial pH in prediction of neonatal outcome, as shown in Table 5. The Receiver Operating Characteristic (ROC) curves for umbilical cord arterial blood pH and LDH levels with neonatal death and complications is depicted in Figs. 1 and 2.

Table 1
Demographic features.

Parameter	n	Percentage (%)
Age (Years)		
18–20	05	3.2% (05/155)
21–25	70	45.2% (70/155)
26–30	72	46.5% (72/155)
31–35	08	5.2% (08/155)
Gestation (weeks)		
≥37- < 39	98	63.2% (98/155)
≥39- < 41	51	32.9% (51/155)
≥41-≤42	06	3.9% (06/155)
Gravidity		
G1	60	38.7% (60/155)
G2	51	32.9% (51/155)
G3	28	18.1% (28/155)
G4	14	9.0% (14/155)
≥G5	02	1.3% (02/155)
Parity		
Nulliparous	75	48.4% (75/155)
Multiparous	80	51.6% (80/155)
Type of Hypertensive Disorder of Pregnancy		
Gestational Hypertension	96	61.9% (96/155)
Pre-eclampsia	34	21.9% (34/155)
Eclampsia	17	11.0% (17/155)
Chronic Hypertension	08	5.2% (08/155)
Mode of delivery		
Normal Delivery	85	54.8% (85/155)
Lower segment cesarean section	66	42.6% (66/155)
Emergency LSCS	59	89.4% (59/66)
Elective LSCS	07	10.6% (07/66)
Instrumental delivery	03	1.9% (03/155)
Breech assisted delivery	01	0.6% (01/155)
Neonatal Birth Weight (kg)		
≥1.5- < 2.5	37	23.9% (37/155)
≥2.5- < 3.5	112	72.3% (112/155)
≥3.5	06	3.9% (06/155)

4. Discussion

The present prospective observational study was conducted to compare the role of umbilical cord arterial blood pH and lactate dehydrogenase levels at birth in assessment of overall neonatal outcome

Table 2
Comparison of Type of HDP with neonatal Apgar score at 1, 5 min, umbilical cord blood arterial pH and Lactate dehydrogenase levels.

Parameters	Gestational Hypertension	Chronic Hypertension	Pre-eclampsia	Eclampsia	F	p-value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
Apgar at 1min	6.92 ± 0.87	6.63 ± 0.74	6.50 ± 1.16	5.71 ± 1.21	7.95	0.000
Apgar at 5min	8.81 ± 0.67	8.75 ± 0.71	8.38 ± 1.10	7.35 ± 1.22	14.75	0.000
Cord blood pH	7.22 ± 0.10	7.19 ± 0.10	7.15 ± 0.12	7.03 ± 0.12	17.84	0.000
Cord blood Lactate Dehydrogenase (U/L)	349.61 ± 525.09	238.26 ± 309.30	536.22 ± 443.59	939.74 ± 781.75	6.60	0.000

Table 3
Correlation of severity of Hypoxic Ischemic Encephalopathy with mean umbilical cord blood arterial pH and LDH levels.

Severity of Hypoxic Ischemic Encephalopathy	Hypoxic Ischemic Encephalopathy-I	Hypoxic Ischemic Encephalopathy-II	Hypoxic Ischemic Encephalopathy-III	F	p-value
	Mean ± SD	Mean ± SD	Mean ± SD		
Cord blood arterial pH	7.10 ± 0.10	6.99 ± 0.12	7.00 ± 0.17	3.745	0.032
Cord blood arterial LDH (U/L)	793.25 ± 630.89	1055.45 ± 732.62	1770.25 ± 896.34	5.036	0.011

Table 4
Correlation of NICU admission and neonatal death with mean umbilical cord Blood arterial pH and LDH levels.

Parameters	No Mean ± SD	Yes Mean ± SD	t	p-value	95% Confidence Interval of Difference	
					Lower	Upper
NICU Admission						
Cord arterial blood pH	7.24 ± 0.07	7.08 ± 0.14	9.484	0.000	0.124	0.19
Cord arterial blood LDH (U/L)	204.68 ± 144.76	907.46 ± 743.74	−9.20	0.000	−853.687	−551.873
Neonatal Death						
Cord arterial blood pH	7.19 ± 0.11	7.00 ± 0.17	4.222	0.000	0.102	0.281
Cord arterial blood LDH (U/L)	387.05 ± 460.57	1770.25 ± 896.34	−7.372	0.000	−1753.890	−1012.513

Table 5
Comparison of umbilical cord arterial pH and LDH in predictions of neonatal deaths.

Statistics	LDH Value (95% CI)	pH value (95% CI)
Sensitivity	100.00% (59.04%–100%)	85.71% (42.13%–99.64%)
Specificity	79.73% (72.34%–85.89%)	59.46% (51.09%–67.44%)
Positive Likelihood Ratio	4.93 (3.58–6.79)	2.11 (1.48–3.03)
Negative Likelihood Ratio	0	0.24 (0.04–1.48)
Disease prevalence	4.52% (1.83%–9.08%)	4.52% (1.83%–9.08%)
Positive Predictive Value	18.92% (14.49%–24.31%)	9.09% (6.52%–12.54%)
Negative Predictive Value	100.00%	98.88% (93.45%–99.82%)
Accuracy	80.65% (73.54%–86.54%)	60.65% (52.49%–68.39%)

in term antenatal women with HDP. Of total 155 subjects, 96 (61.9%), had gestational hypertension, 34 (21.9%) pre-eclampsia, 17 (11%) eclampsia and eight (5.2%) had chronic hypertension. The mean ± SD for age and gestation of all the patients was 25.6 ± 3.1 years and 38.48 ± 1.113 weeks respectively. In our study we observed a significant derangement of all the neonatal parameters including Apgar score at one and 5 min, cord blood arterial pH and LDH levels with severity of disease. The mean ± SD values Apgar score at one and 5 min, cord blood pH and LDH in neonates delivered to eclamptic women were 5.71 ± 1.21, 7.35 ± 1.22, 7.03 ± 0.12, 939.74 ± 781.75 U/L respectively, which was highly significant ($p = 0.000$). A similar study observed no significant difference in cord blood arterial LDH levels in neonates born to women with pregnancy-related disorders as compared to those delivered to healthy women ($p = 0.95$).[12]

In our study we observed a significant correlation between the mode of delivery and umbilical cord arterial blood pH and LDH levels. Neonates delivered by emergency LSCS, especially for fetal distress had significantly lower cord blood pH and high LDH levels ($p < 0.05$). Similar results were reported by another study which observed that maximum levels of mean arterial cord lactate were observed in neonates delivered instrumentally (5.1 mmol/L) followed by those delivered vaginally (4.3 mmol/L), emergency cesarean section (3.9 mmol/L) and minimum in neonates delivered by elective cesarean section (3.2 mmol/L).[35] Similarly, another study reported that significantly higher cord arterial blood LDH levels were found in neonates delivered by emergency caesarian section as compared to those delivered vaginally. Moreover, neonates delivered by vacuum extraction had both arterial and venous elevated cord blood LDH levels, same as with emergency cesarean section, as compared to those delivered by spontaneous vaginal delivery and elective cesarean section.[15]

Furthermore, in our study we observed a significant correlation between low cord blood pH and high LDH levels with NICU admission and neonatal death, more so with cord blood LDH levels. The mean ± SD for cord blood pH and LDH for neonates requiring NICU admission was 7.08 ± 0.14 and 907.46 ± 743.74 U/L respectively and for neonates who expired was 7.00 ± 0.17 and 1770.25 ± 896.34 U/L respectively ($p = 0.000$). A similar study also reported that cord blood lactate levels were significantly better predictor than pH for neonatal morbidity and mortality (ROC curve area:

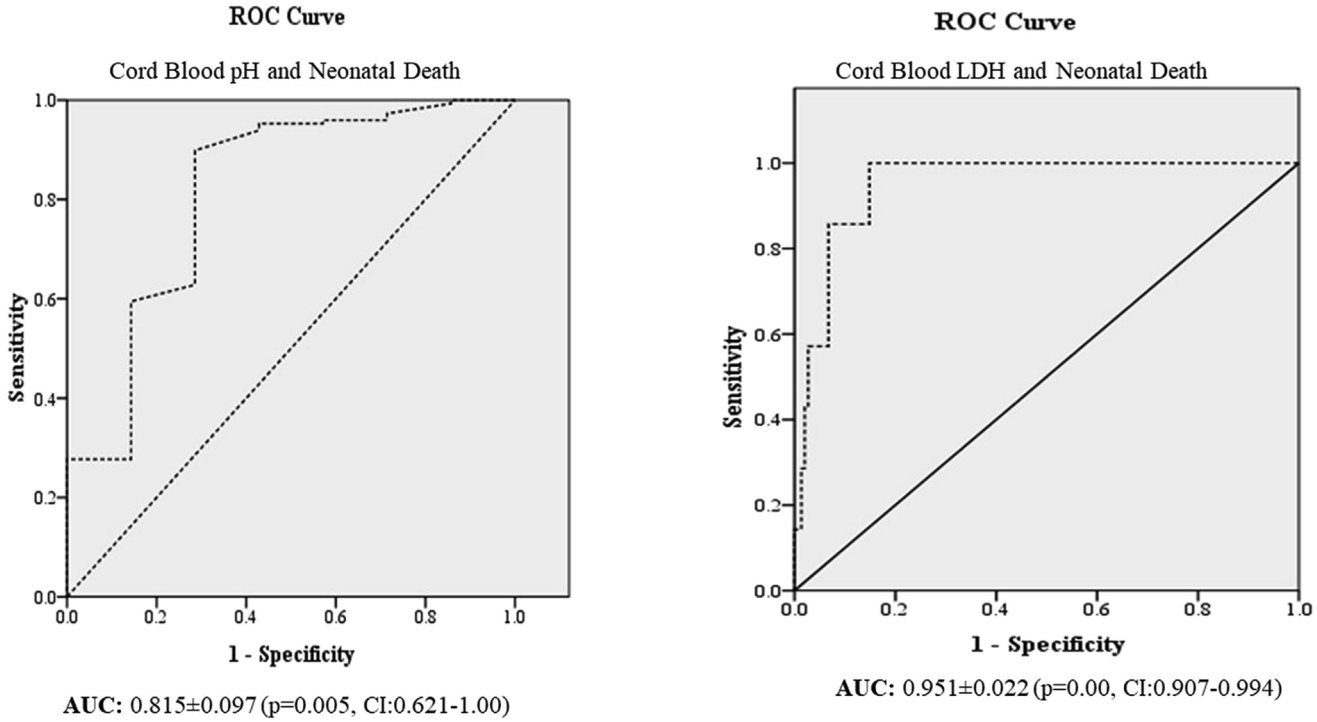

Fig. 1. Depicts Receiver Operating Characteristic curve for cord arterial blood pH and Lactate for neonatal death. The Area under curve (AUC) for cord blood lactate was 0.951 and cord blood pH was 0.815, indicating that cord arterial blood lactate is a better predictor of neonatal death.

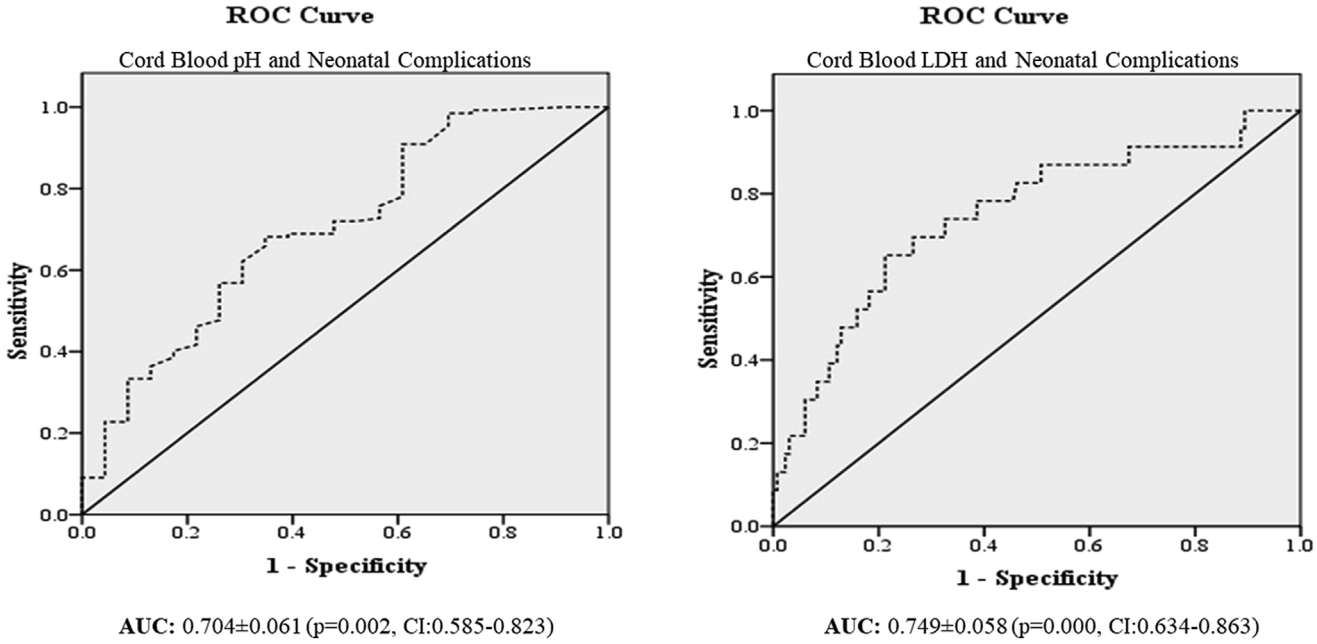

Fig. 2. Depicts Receiver Operating Characteristic curve for cord arterial blood pH and Lactate of neonates with complications. The Area under curve (AUC) for cord blood lactate was 0.749 and cord blood pH was 0.704, indicating that cord arterial blood lactate is a better predictor of neonatal complications.

0.84 vs 0.78, p = 0.03).[36] Another similar study observed that cord arterial blood lactate level was more strongly associated with admission to NICU (OR 2.91, P < 0.0001) as compared to cord blood pH (OR 2.72, P < 0.0001).[35] Similar results were reported by another study which observed that cord blood lactate is better predictor of neonatal morbidity and mortality as compared to umbilical artery pH.[37] A recent study has also reported that elevated cord blood LDH levels were significantly associated with increased risk of neonatal mortality.[38] Another similar study found that both cord blood Lactate and pH values help in distinguishing between asphyxiated and non-asphyxiated neonates, but lactate levels were observed to be have a better discriminating power.[39]

In addition to this in our study we found that the cord blood LDH levels were a better predictor of severity of HIE. The mean ± SD of cord blood LDH levels in neonates with HIE I, II and III was 793.25 ± 630.89 U/L, 1055.45 ± 732.62 U/L and 1770.25 ± 896.34 U/L respectively (p = 0.011). Similar results were reported by another study which observed that LDH was not only the best predictor of HIE (sensitivity 100% and specificity 97%) but also its long-term effects on neonates. Furthermore, they observed that poor

neonatal condition in first week of birth is strongly associated with elevated cord blood LDH levels.[22] Another similar study reported a significant rise in umbilical cord arterial blood LDH and cardiac enzyme levels (CK-MB and CK-BB) in neonates having stage 3 HIE as compared to neonates with stage 1 and 2 HIE.[38]

In our study on comparison of accuracy of umbilical cord arterial blood pH and LDH in prediction of overall neonatal outcome it was observed that LDH is a better predictor with a sensitivity of 100%, specificity of 79.73%, negative predictive value of 100% and a positive predictive value of 18.92%. The overall accuracy of cord blood LDH in prediction of neonatal death was 80.65% as compared to 60.65% for cord blood pH. Similar results were reported by another study which observed that raised cord blood LDH levels had 100% sensitivity, while CK-MB had 100% specificity for neonatal birth asphyxia. Also, they concluded that LDH levels at first 72 h of life are most accurate in differentiating asphyxiated from non-asphyxiated symptomatic neonates.[17] Another similar study observed that cord blood LDH levels are one of the best predictors of prolonged oxygen and respiratory support requirement in newborns with transient tachypnea and has a positive predictive value (PPV) of 88.9%.[20] Similar results were reported by another study which observed the sensitivities and specificities of cord blood LDH and pH as 83.9% and 74.1% vs 75.0% and 70.6%, respectively.[36] Many similar studies have observed that both cord blood lactate and pH had almost equal significance in prediction of overall neonatal outcome.[35,40] Hence, Umbilical cord arterial blood pH and LDH levels were significantly associated with poor Apgar score of newborns at birth and correlates well with NICU admission, neonatal complications and deaths in women with hypertensive disorders of pregnancy.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

There are no conflicts of interest.

Acknowledgement

I thank Dr. Namit Kant Singh for his expert advice and support.

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