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Umbilical Cord Blood Lactate as an Indicator of Fetal Hypoxia

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Abstract

Objective: This study aimed to evaluate the role of umbilical cord blood lactate levels as an indicator of intrapartum asphyxia in caesarean sections done for fetal distress at term.

Material & Methods: It was a prospective study conducted on 150 women undergoing caesarean section for fetal distress and non fetal distress indications. The participants were categorized into 3 groups of 50 women each. Group 1 included women with fetal heart rate abnormalities. Group 2 included women with meconium stained liquor and group 3 which served as control included women who had elective caesarean section. Neonatal cord blood lactate, cord blood pH and perinatal outcome were studied in all the 150 women. Statistical analysis was done by ANOVA, Tukey and Tamhane's T2 test.

Results: Significantly higher cord blood lactate values (P value <0.001) and lower pH values (P value<0.05) were noted among the neonates with both the intrapartum signs of distress as compared to controls. Cord blood lactic acidosis was present in 86% neonates of group 1, 42% neonates of group 2 and 2% neonates of group 3 while significant low pH was noted in 28% neonates of group 1, 22% neonates of group 2 and none of the neonates of group 3. The positive predictive value of cord blood lactic acidosis for intrapartum asphyxia was14% in group 1 and 47.6% in group 2 while the positive predictive value of cord blood pH in group 1 and 2 was30.7% and 90% respectively. Lactic acidosis had 100% negative predictive value in both the groups while pH had negative predictive value of 94.4% and 97.4% in group 1 and 2 respectively.

Conclusion: Fetal lactic acidosis is a reliable and accurate indicator of foetal hypoxia hence could supplement the diagnosis of genuine fetal distress but it is a poor predictor of adverse neonatal outcome.

Keywords: Cord blood lactate; Cord blood pH; Perinatal outcome; Fetal acidemia

Introduction

Birth asphyxia has long been considered as a major cause of perinatal morbidity and mortality [1]. The World health Organization (WHO) estimated that globally 4-9 million newborns suffer with birth asphyxia each year leading to approximately 1.2 million neonatal deaths which accounts for 29% of the total neonatal deaths [2]. Moreover, fetal distress is the largest contributor to the rising trend of caesarean deliveries all over the world [3].

Hence, it is of utmost importance to diagnose it accurately so as to minimize the unnecessary operative deliveries. Considering the substantial morbidity and mortality associated with birth asphyxia efforts are underway to diagnose it early and accurately [4].

Conventionally for many years the diagnosis of intrapartum hypoxia has been based on the clinical signs of fetal distress (like meconium staining of liquor, and fetal heart rate abnormalities on auscultation) and the assessment of Apgar scores at birth. However, poor specificity and low positive predictive value of these conventional methods propelled the search for a better diagnostic test [5]. In this context, the umbilical cord blood biochemical analysis attracted special attention as it is thought to mirror the intrapartum and immediate postpartum hypoxic events. As lactate levels may be considered a true reflection of the metabolic acidosis status, it may be of value in diagnosing genuine cases of intrapartum fetal hypoxia and this was the prime aim of our study.

Material and Methods

A total of 150 women with singleton live pregnancy between 37-42 weeks who underwent caesarean section under regional anesthesia in the Department of Obstetrics and Gynecology at Pt. B.D. Sharma, PGIMS, Rohtak were categorised into three groups comprising 50 women each. Group 1 included women who were diagnosed to have fetal tachycardia (fetal heart rate of>160/minute) or bradycardia (fetal heart rate of <110/minute) on intermittent auscultation by stethoscope during labour [6]. Group 2includedwomen who underwent caesarean section for moderate or heavy meconium stained liquor while women who underwent elective caesarean section served as the control and were categorised in to group 3.

The antenatal and intrapartum details of all the women were noted. Cord was doubly clamped and cut immediately after the delivery of baby during caesarean section. Cord blood was taken for estimation of lactate levels and pH in a preheparinised syringe from the clamped cord segment. Enzymatic estimation of lactate was done in plasma by colorimetric method within 30 minutes after the collection of sample. The reference range of lactate in plasma was taken as 0.5-2.22 mmol/L or 4.5-20 mg/dl [7]. The 1-min and 5-min Apgar scores, transfer to neonatal intensive care unit (NICU), neonatal hospital stay, any neonatal complication and mortality were also recorded. Intrapartum and postpartum maternal complication, if any, were a Cord blood lactate levels among the three groups was the primary outcome measure while the Apgar scores at 1 and 5 minute, cord blood pH, transfer to NICU, neonatal complications and the mean neonatal hospital stay were the secondary outcome measures of the study.

Statistical Analysis

Statistical analysis of data was performed by the SPSS 17.0 and ANOVA, Turkey multiple comparison test and Tamhane's T2 test were used to compare the three groups.

Results

As is evident from table 1 there was a significant difference in the mean age and parity of group 1 and 2 as compared to group 3 but the difference was comparable between group 1 and 2. There was no significant difference in the gestational age of women among the three groups and no correlation was seen between the gestation age and cord blood lactate values at term. Also there was insignificant difference in the time of diagnosis of fetal distress to delivery interval of the group 1 and 2 and no effect of the time of diagnosis of fetal distress to delivery interval was noted on neonatal outcome and admission to neonatal intensive care unit in our study.

	Group 1	Group 2	Group 3	P value		
	Mean ± SD	Mean ± SD	Mean ± SD	Group 1 vs Group 2	Group 1 vs Group 3	Group 2 Vs Group 3
Age (years)	23.70± 2.69	24.20± 4.33	26.28 ± 3.49	0.763	0.001	0.011
Parity	0.46 ± 0.71	0.50 ± 0.56	1.12 ± 0.87	0.832	0.001	0.004
Time of diagnosis of fetal distress to delivery interval (minutes)	94.26 ± 81.69	81.80 ± 38.36	Not studied as it was control group	0.331		

Table 1: Clinical characteristics of the subjects.

The reference range of lactate in plasma in our study was taken as 0.5-2.22 mmol/L or 4.5-20 mg/dl [7]. There were statistically significant high mean lactate levels in group 1 and 2 as compared to group 3. Group 1 also had significantly higher mean lactate values as compared to group 2 as depicted in Table 2.

Lactate level (mmol/L)	Group 1 n = 50	Group 2 n = 50	Group 3 n = 50	P value		
	Frequency (%)	Frequency (%)	Frequency (%)			
>2.22	43(86%)	21 (42%)	1 (2%)	Group 1 vs Group 2	Group 1 vs Group 3	Group 2 vs Group 3
≤2.22	7(14%)	29(58%)	49(98%)			
Range(mmol/L)	1.08-7.45	1.01-6.54	1.01-2.53			
95% CI(mmol/L)	3.42-4.36	2.49-3.45	1.47-1.71			
Mean ± SD (mmol/L)	3.89 ± 1.65	2.97 ± 1.68	1.59 ± 0.43	0.003	0.000	.000

Table 2: Neonatal cord blood lactate levels.

It may be mentioned here that neonatal cord blood pH of less than 7.20 was considered as fetal acidemia in our study [8]. Group 1 and 2 had statistically significant low mean cord blood pH values as compared to group 3 while the difference in the mean pH values of group 1 and 2 was statistically insignificant (Table 3).

Neonatal cord blood pH	Group 1 n = 50	Group 2 n = 50	Group 3 n = 50	P value		
	Frequency (%)	Frequency (%)	Frequency (%)			
< 7.20	14(28%)	11(22%)	0(0%)	Group 1 vs Group 2	Group 1 vs Group 3	Group 2 vs Group 3
≥7.20	36(72%)	39(78%)	50(100%)			
Mean ± SD	7.26 ± 0.11	7.25 ± 0.17	7.33 ± 0.04	0.909	0.014	0.004

Table 3: Neonatal cord blood pH level.

Group 3 had statistically significant higher Apgar score at 1 minute as compared to group 1(P value 0.004) and group 2(P value 0.001) while the difference was insignificant between group 1 and 2. Apgar score <7 at 5minutes has been considered as low Apgar score in our study [9]. Group 1 and 2 had significantly low mean Apgar scores at 5 minute as compared to group 3 (P value of the difference in Apgar scores between Group 1and 3 was 0.014 and group 2 and 3 was 0.002).Group 1 and Group 2 had no significant difference in their mean Apgar scores at 5 minute.

Apgar score < 7	Group 1 n = 50	Group 2 n = 50	Group 3 n = 50
	Frequency (%)	Frequency (%)	Frequency (%)
At 1 minute	23 (46%)	22 (44%)	14 (28%)
At 5 minute	5 (10%)	6 (12%)	0 (0%)

Table 4: Apgar score of newborns observed at 1 and 5min.

Group 1 and 2 had significant number of admissions to neonatal intensive care unit (NICU) as compared to group 3 as evident from table 5. Of all the NICU admissions, 6 babies from group 1, 10 from group 2 and none from group 3 had evidence of birth asphyxia and out of them, 3 from group 1 and 5 from group 2 died.

Admission to NICU	Group 1 n = 50	Group 2 n = 50	Group 3 n = 50	P value		
	Frequency (%)	Frequency (%)	Frequency (%)	Group 1 vs Group 2	Group 1 vs Group 3	Group 2 vs Group 3
Yes	13 (26%)	16 (32%)	2 (4%)	0.509	0.004	<0.001
No	37 (74%)	34 (68%)	48 (96%)			

Table 5: Status of admission to NICU.

Table 6 shows the distribution of only those patients who showed neonatal cord blood lactic acidosis. In group 1 all the 5 babies who had low Apgar scores at 5 minute had lactic acidosis, low cord blood pH and got admitted to neonatal intensive care (NICU). Three of them died of asphyxia or its associated complications. Total 12 (27.9%) neonates of group 1 needed admission to NICU out of which 6 were admitted due to birth asphyxia. In group 2 also all the 6 newborns with low Apgar scores at 5 minute had raised cord blood lactate levels and low cord blood pH. All of them were admitted to NICU of which 5 neonates died due to hypoxia or its related complications. There was no baby in the study who had evidence of hypoxia related complication but a normal lactic acid levels in cord blood. The positive predictive value of cord blood lactic acidosis for intrapartum asphyxia was 14% in group 1 and 47.6% in group 2 while the positive predictive value of cord blood pH in group 1 and 2 was 30.7% and 90% respectively. Lactic acidosis had 100% negative predictive value in both the groups while pH had negative predictive value of 94.4% and 97.4% in group 1 and 2 respectively.

	Group 1 n = 43	Group 2 n = 21	Group3 n = 1
	Frequency (%)	Frequency (%)	Frequency (%)
pH (< 7.20)	14 (32.5%)	11 (52.4%)	0 (0%)
Apgar score < 7 at 1 minute	21 (48.8%)	13 (61.9%)	0 (0%)
Apgar Score < 7 at 5 minute	5 (11.6%)	6 (28.6%)	0 (0%)
Admission to NICU	12(27.9%)	10(47.6%)	0(0%)
Neonatal mortality	3 (6.9%)	5 (23.8%)	0(0%)
Positive predictive value of cord blood lactate for birth asphyxia	14%	47.6%	-
Negative predictive value of cord blood lactate for birth asphyxia	100%	100%	-
Positive predictive value of cord blood pH for birth asphyxia	30.7%	90%	-
Negative predictive value of cord blood pH for birth asphyxia	94.4%	97.4%	-

Table 6: Association of neonatal cord blood lactate with cord blood pH, Apgar scores and neonatal Outcome.

Discussion

The present study highlights the importance of estimation of fetal blood lactate for acidosis in women who are being taken up for abdominal delivery for fetal distress. More number of higher aged women in group 3 may reflect more complicated pregnancies requiring elective caesarean births as age advances, the same has

been corroborated by Herstad et al. and Timofeev et al. [10,11]. Younger patients in group 1 and group 2 would have contributed to lower parity in two groups as compared to group 3. No correlation was seen between the gestation age and cord blood lactate values at term in our study as also stated by Watt et al. [1]. In contrast Khoshnow and Mongelli noted a weak but statistically significant positive correlation between gestational age and cord blood lactate [9]. No effect of time of diagnosis of fetal distress to delivery interval was noted on neonatal outcome and admission to neonatal intensive care unit in our study. This was also corroborated in a study conducted by Huissoud et al. [12].

Higher values of neonatal cord blood lactate and lower values of cord blood pH in fetal distress groups of the present study points towards the presence of a hypoxic state in these groups. Significant correlation of cord blood lactate and pH with intrapartum asphyxia has been demonstrated in our study and the same has been corraborated by Khoshnow and Mongelli, Varkilova et al. [9,13]. Higher number of patients with fetal lactic acidosis in group1 as compared to the group 2 in the present study suggests that passage of meconium is a weak indicator of intrapartum asphyxia as compared to non reassuring fetal heart rate. Similar results have also been reported by Yeoman et al and Blackstone and Young [14,15].

The fetal catecholamine surge which occurs as a result of hypoxia may also help in the improvement of Apgar scores by causing general neonatal arousal and this could be the reason of less number of babies with low Apgar scores out of all the babies who had lactic acidosis in our study. It could also be explained by the presence of transient acidemia during normal labor and delivery. But all the babies who had low Apgar scores at 5 minute had metabolic acidosis and need for admission to NICU suggesting that high neonatal cord blood lactate is a significant predictor of low Apgar scores and the same has been demonstrated by Khoshnow and Mongelli [9].

Absence of low values of cord blood pH in those neonates who had lactic acidosis may be due to higher sensitivity of lactate levels for detection of intrapartum asphyxia as compared to pH or non specificity of raised lactate levels to increased anaerobic metabolism [9]. Gjerris et al. suggested that lactate in arterial umbilical cord blood might be a more correct indicator of fetal asphyxia at delivery than pH [16]. However, Wiberg-Itzel et al. reported no difference in the assessment of fetal acidemia with lactate or pH analysis [17].

A total of 72.1% newborns from group 1 and 52.4% newborns from group 2 with metabolic acidosis were clinically normal in our study. It may thus be concluded that biochemical neonatal lactic acidosis may have clinically detectable manifestation of hypoxia or more frequently may result in subtle and clinically undetectable manifestation. Only those neonates who had clinical evidence of hypoxia may need intensive care or intervention and some of these babies with clinical hypoxia in the form of low Apgar scores may be at risk of neurological morbidity and mortality. Borruto et al. concluded in their study that no perfect correlation exists between the cord blood lactate level and neonatal outcome while Varkilova et al. reported a significant association between neonatal cord blood lactate and neonatal outcome [18,13].

In comparison to pH of the cord blood, the estimation of cord blood lactic acidosis has lower positive predictive value but higher (100%) negative predictive value. Hence, it can be safely concluded that women with normal umbilical cord blood lactate levels are not likely to have fetal hypoxia even though they may be having fetal heart rate abnormalities or meconium stained liquor.

Conclusion

Biochemical fetal lactic acidosis is an important indicator of fetal hypoxia which may or may not be manifesting clinically and as it has 100% negative predictive value so, those babies who don't have lactic acidosis should not be considered as having genuine fetal distress. The major limitation of our study was that it was conducted on women who have already undergone caesarean section and in future more large scales study on women in labor with clinical evidence of fetal distress with scalp blood lactate estimation may be carried out so that the benefit of diagnosing genuine fetal hypoxia goes to them too.

References

1. Watt WF, Tan KH, Yeo GS (2002) Umbilical cord lactate: A preliminary study of 130 term babies. Singapore J Obstet Gynecol 33(2): 42-48.

2. Committee on Obstetric Practice, American College of Obstetricians and Gynecologists (2005) ACOG Committee Opinion. Number 326-December 2005. Inappropriate use of the terms fetal distress and birth asphyxia. Obstet Gynecol 106(6): 1469-1470.

3. Mittal S, Pardeshi S, Mayadeo N, Mane J (2014) Trends in Caesarean delivery: Rate and indications. J Obstet Gynaecol 64(4): 251-254.

4. Low JA, Victory R, Derrick EJ (1999) Predictive value of electronic fetal monitoring for intrapartum fetal asphyxia with metabolic acidosis. Obstet Gynecol 93(2): 285-291.

5. Uzan S, Berkane N, Verstraete L, Mathieu E, Bréart G, et al. (2003) Acid base balance in the fetus during labor: pathophysiology and exploration methods. J Gynecol Obstet Biol Reprod 32(S1): S68-S78.

6. National Institute for Health and Clinical Excellence (2007) Intrapartum care: care of healthy women and their babies during childbirth. Clinical Guideline 55 RCOG Press, London.

7. Jacobs DS, Kasten BL, Demott WR, Wolfson WL (1990) Laboratory Test Handbook 2nd (Edn.) Lexi-Comp Inc, Ohio, pp. 245.

8. Ross MG, Gala R (2002) Use of umbilical artery base excess: Algorithm for the timing of hypoxic injury. Am J Obstet Gynecol 187(1): 1-9.

9. Khoshnow Q, Mongelli M (2010) Cord Blood Lactate and pH Values at Term and Perinatal Outcome: A Retrospective Cohort Study. Web med central Obstetrics and Gynaecology 1(9): WMC00694.

10. Herstad L, Klungsøyr K, Skjærven R, Tanbo T, Forsén L, et al. (2016) Elective cesarean section or not? Maternal age and risk of adverse outcomes at term: a population-based registry study of low-risk primiparous women. BMC Pregnancy Childbirth 16: 230.

11. Timofeev J, Reddy UM, Huang CC, Driggers RW, Landy HJ, et al. (2013) Obstetric complications, neonatal morbidity, and indications for cesarean delivery by maternal age. Gynecol 122(6): 1184-1195.

12. Huissoud C, Dupont C, Canoui-Poitrine F, Touzet S, Dubernard G, et al. (2010) Decision-to-delivery interval for emergency caesareans in the Aurore perinatal network. J Obstet Gynecol Reprod Biol 149(2): 159-164.

13. Varkilova L, Slancheva B, Emilova Z, Nikolov A, Metodieva V, et al. (2013) Blood lactate measurements as a diagnostic and prognostic tool after birth asphyxia in newborn infants with gestational age > or = 34 gestational weeks. Akush Ginekol (Sofiia) 52(3): 36-43.

14. Yeomans ER, Hauth JC, Gilstrap LC, Strickland DM (1985) Umbilical cord pH, PCO2, and bicarbonate following uncomplicated term vaginal deliveries. Am J Obstet Gynecol 151(6): 798-800.

15. Blackstone J, Young BK (1993) Umbilical cord blood acid-base values and other descriptors of fetal condition. Clin Obstet Gynecol 36(1): 33-46.

16. Gjerris AC, Staer-Jensen J, Jørgensen JS, Bergholt T, Nickelsen C (2008) Umbilical cord blood lactate: a valuable tool in the assessment of fetal metabolic acidosis. Eur J Obstet Gynecol Reprod Biol 139(1): 16-20.

17. Wiberg-Itzel E, Lipponer C, Norman M, Herbst A, Prebensen D, et al. (2008) Determination of pH or lactate in fetal scalp blood in management of intrapartum fetal distress, randomized controlled multicentre trial. BMJ 336(7656): 1284-1287.

18. Borruto F, Comparetto C, Wegher E, Treisser A (2006) Screening of foetal distress by assessment of umbilical cord lactate. Clin Exp Obstet Gynecol 33(4): 219-222.