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ORIGINAL ARTICLE

Non-reassuring fetal heart rate patterns in association with umbilical artery acidosis

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ABSTRACT

Objective: The main purpose of the study was to evaluate the clinical outcomes of fetuses who had intrapartum non-reassuring fetal heart rate tracings.

Material and Methods: Patients who underwent cesarean section as an emergency operation due to non-reassuring fetal heart rate patterns were included in the study. All FHR paper traces were reevaluated by an expert obstetrician, blinded to the neonatal outcomes, based on the guidelines of the NICHD workshop. Patients were placed into five groups considering the variability and accompanying deceleration type. Clinical outcomes, Apgar scores, and umbilical artery blood parameters were evaluated.

Results: The study consisted of 84 patients; Group 1, normal variability with late decelerations (n=32); Group 2, normal variability with variable decelerations (n=16); Group 3, decreased variability (n=10); Group 4, decreased variability with late decelerations (n=14); Group 5, decreased variability with variable decelerations (n=12). Groups with decreased variability and decelerations (groups 4 and 5) had higher rates of NICU admission than the groups with normal variability with decelerations (groups 1 and 2) ($p<0.05$). In the decreased variability with late decelerations group (Group 4), umbilical artery blood pH and ABE were significantly lower while lactate levels were higher than in groups 1, 2, and 3 ($p<0.001$). Among all patients, inverse correlations were shown between umbilical artery blood lactate and pH ($r=-0.734$, $p<0.001$), and also between lactate and ABE ($r=-0.581$, $p<0.001$). For the prediction of umbilical artery blood pH<7.1 and/or ABE<-12, the optimal umbilical artery blood lactate cut-off level is 7 mmol/L with a sensitivity of 88.9% and specificity of 89.3%.

Conclusion: Decreased variability in non-reassuring intrapartum fetal heart rate patterns should be considered as important as decelerations. In the evaluation of intrapartum fetal asphyxia, lactate appears to be as good a marker as pH and ABE.

Keywords: Fetal heart rate, fetal hypoxia, nonreassuring fetal status, lactate, umbilical cord blood.

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INTRODUCTION

During labor, transient but recurrent interruptions in fetal oxygenation may occur due to regular uterine contractions. It is well tolerated by many fetuses, but in some, metabolic changes due to decreased oxygenation are reflected in fetal heart rate (FHR) tracings. Detection of fetal heart rate changes that may be associated with decreased fetal oxygenation is aimed with continuous intrapartum fetal heart rate monitoring.[1] Although its effects on fetal death or long-term neurological outcomes are controversial, continuous intrapartum FHR monitoring is recommended in patients with high-risk conditions (e.g., growth-restricted fetuses, hypertensive disorders, type 1 diabetes mellitus).[2] Algorithms have been developed to identify FHR patterns that are normal, that require more attention, and that are abnormal which requires immediate delivery of the fetus.[3,4] Although normal results seem reliable in determining fetal well-being, intrapartum FHR monitoring is associated with increased cesarean rates due to high false positive rates.[5] One of the most common indications for primary cesarean delivery is non-reassuring FHR patterns.

The Apgar score provides a universally accepted, easily applicable method for revealing the status of the newborn just after birth. Although lower Apgar scores are associated with higher neonatal mortality and morbidity, it is not recommended as a prognostic tool.[6,7] Umbilical artery blood sampling provides more objective information in demonstrating fetal status at birth. An umbilical artery pH below 7.0 is defined as fetal metabolic acidemia.[8] It is also suggested that the umbilical artery pH <7.10 and ABE <-12 mmol/L threshold to identify fetuses with non-reassuring heart rate patterns that may benefit from intervention before pathological fetal acidosis and fetal damage develop.[8–10] Lactate concentration is also useful in demonstrating tissue hypoxia as a result of anaerobic metabolism.[11]

The main purpose of our study was to assess the relationship between intrapartum nonreassuring fetal heart rate tracings and early neonatal clinical findings. Secondly, to investigate the importance of umbilical artery blood lactate in the evaluation of fetal status.

MATERIAL AND METHODS

This prospective observational study was carried out at İstanbul Medeniyet University Göztepe Training and Research Hospital, Türkiye, from February 2015 through January 2016. Ethical approval was obtained from the local ethics committee, and the study was conducted in accordance with the Declaration of Helsinki. Patients who were admitted to the delivery room due to the onset of labor gave informed written consent. All patients in the delivery room were monitored with external cardiotocography (CTG) continuously, and FHR paper traces were obtained for later evaluation. Eighty-four patients who underwent cesarean section as an emergency operation due to non-reassuring FHR patterns were included in the study. The selection criteria were beyond the 34^{th} gestational weeks of pregnancy with singleton, cephalic presentation fetuses. Patients with medical disorders that might affect the fetal acid-base status, such as cardiopulmonary disease, chronic renal failure, or poorly controlled diabetes mellitus, were excluded. Pregnancies with known fetal anomalies, growth-retarded fetuses, and multiple gestations were also not included in the study. All operations were performed under general anesthesia, and premedication protocols were identical in all patients.

Comen Fetal Monitor Star 500F or Sunray SRF 618B was used as external cardiotocography to record electronic fetal heart rate tracings. The time between the last recorded non-reassuring FHR tracing and the time of cesarean section was no more than 30 minutes. All FHR paper traces were reevaluated by an expert obstetrician, blinded to the neonatal outcomes, based on the guidelines of the NICHD workshop.[3,4] We combined tracings with absent and minimal variability and labeled them as decreased variability. Variable and late deceleration definitions of the NICHD workshop were also complied with. All FHR tracings were grouped into five according to the variability and deceleration parameters: group 1, normal variability with recurrent late decelerations; group 2, normal variability with recurrent variable decelerations; group 3, decreased variability with no decelerations; group 4, decreased variability with recurrent late decelerations; and group 5, decreased variability with recurrent variable decelerations.

Immediately after delivery, the umbilical cord was double clamped and arterial blood samples were collected in a plastic syringe washed with heparin solution. Blood samples were analyzed for pH, actual base excess (ABE), and lactate within 15 minutes of delivery using a Radiometer Copenhagen ABL 510 Blood Gas System.

The study data were analyzed using IBM SPSS Statistics version 21.0 (IBM Corporation, Armonk, New York, United States). Data were presented as mean±SD and categorical parameters were presented as frequencies with group proportions. Fisher's exact test was used for pairwise group comparisons of categorical variables. For numerical variables with a normal distribution, the One-Way ANOVA test was used, and the Kruskal-Wallis test was used for variables without a normal distribution to compare more than two independent FHR pattern-based groups. Tukey and Tamhane's T2 tests were used for post-hoc analysis for variables with normal and non-normal distributions respectively. A receiver operating characteristic (ROC) analysis was performed to assess the best cut-off level of umbilical artery blood lactate which predicts the fetuses who may benefit from intervention. To evaluate the relationship between quantitative variables, Spearman's rank correlation was used. Differences were interpreted as statistically significant at $p<0.05$.

RESULTS

The study consisted of 84 participants; Group 1, Normal Variability with Late Decelerations (n=32); Group 2, Normal Variability with Variable Decelerations (n=16); Group 3, Decreased Variability (n=10); Group 4, Decreased Variability with Late Decelerations (n=14); Group 5, Decreased Variability with Variable Decelerations (n=12).

Table 1 shows the clinical characteristics of the five FHR pattern groups. The groups were similar in terms of age, gravida, parity, gestational age at delivery, and birth weights ($p>0.05$). When we compared groups according to the APGAR scores; in the Decreased Variability with Late Decelerations group (group 4), both 1- and 5-minute APGAR scores were lower than in groups 1 and 2 ($p<0.05$). First and fifth minute APGAR scores were similar in decreased variability groups (groups 3, 4, and 5) irrespective of accompanying decelerations ($p>0.05$). The Decreased Variability with Late Decelerations group (group 4) had the highest rates of meconium-stained amniotic

Table 1: Clinical characteristics of the groups of fetal heart rate pattern groups

	Group 1 Normal variability with late decelerations (n=32)	Group 2 Normal variability with variable decelerations (n=16)	Group 3 Decreased variability (n=10)	Group 4 Decreased variability with late decelerations (n=14)	Group 5 Decreased variability with variable decelerations (n=12)	p
Maternal age (years)	28.31±6.75	28.50±6.09	28.50±6.87	28.36±6.37	27.17±3.76	NS*
Gravidae (n)	1 [1–6]	1 [1–5]	1 [1–8]	2 [1–5]	1 [1–3]	NS**
Parity (n)	0 [0–2]	0 [0–3]	0 [0–3]	0 [0–3]	0 [0–1]	NS**
GA at birth (weeks)	38.5±2.0	38.6±2.0	37.9±2.6	38.5±1.7	38.5±1.9	NS*
Birth weight (gr)	3015±530	3043±560	2946±371	3928±685	2901±727	NS*
APGAR 1	8 [4–10]	8 [6–9]	6 [5–10]	4 [1–8]^{α}	6 [3–9]	**<0.001****
APGAR 5	9 [6–10]	10 [8–10]	9 [7–10]	8 [6–9]^{α}	8 [6–10]	**0.001****
MSA	10 (31.2%)	1 (6.2%)	3 (30.0%)	13 (92.9%)^{ß}	3 (25.0%)	**<0.001*****
NICU admission	4 (12.5%)	0 (0.0%)	2 (20.0%)	12 (85.7%)§	6 (50.0%)$^{¥}$	**<0.001*****

Data presented as mean±standart deviation, median [min–max], n (%). *: One-Way ANOVA; **: Kruskal-Wallis Test; ***: Fisher Exact Test; NS: Not significant; GA: Gestational age; MSA: Meconium stained amniotic fluid; NICU: Neonatal intensive care unit; α: Thamannes posthoc test showed significant difference when compared with group 1 and 2; ß: Fisher exact test showed significant difference when compared with other groups; §: Fisher exact test showed significant difference when compared with group 1–2 and 3; ¥: Fisher exact test showed significant difference when compared with group 1–2.

Table 2: Umbilical artery blood pH, lactate and actual base excess in fetal heart rate pattern groups

	Group 1 Normal variability with late decelerations (n=32)	Group 2 Normal variability with variable decelerations (n=16)	Group 3 Decreased variability (n=10)	Group 4 Decreased variability with late decelerations (n=14)	Group 5 Decreased variability with variable decelerations (n=12)	p
pH	7.30±0.08	7.31±0.05	7.26±0.11	7.15±0.13^{α}	7.24±0.04	**<0.001***
Actual base excess (mmol/L)	1.37±2.89	-1.19±2.75	0.29±4.73	-7.62±5.23^{α}	-5.25±3.60^{ß}	**<0.001***
Lactate (mmol/L)	3.19±1.84	2.61±1.53	4.48±3.21	7.76±4.20^{α}	6.39±3.04§	**<0.001***

Data presented as mean±standart deviation; *: One-Way ANOVA; a, ß: Tukey post test showed significant difference when compared with groups 1, 2 and 3; §: Tukey post test showed significant difference when compared with groups 1 and 2.

fluid among the groups (p<0.001). Groups which showed decreased variability with decelerations (groups 4 and 5) had higher rates of NICU admission than the groups that had normal variability with decelerations (groups 1 and 2) (p<0.05).

Group comparisons of the umbilical artery acid-base parameters are shown in Table 2. In group 4 (decreased variability with late decelerations), significantly lower umbilical artery blood pH and ABE levels and higher lactate concentration were observed than in groups 1, 2, and 3 (p<0.001). Significantly lower actual base excess was observed in group 5 (decreased variability with variable decelerations) than in groups 1, 2, and 3 (p<0.001). Group 5 also had higher levels of umbilical artery blood lactate concentrations compared to the groups with normal variability (groups 1 and 2) (p<0.001).

The relationship between Apgar scores and umbilical artery acid-base parameters is given in Table 3. pH and ABE were positively correlated with 1- and 5-minute Apgar scores (p<0.05), while lactate levels had a stronger negative correlation with 1- and 5-minute Apgar scores (p<0.05). The relationship between umbilical artery blood lac-

Table 3: Correlations between 1st and 5th minute Apgar scores and umbilical artery blood pH, actual base excess and lactate (n=84)

	1-minute Apgar score		5-minute Apgar score	
	r_s	p	r_s	p
pH	0.495	<0.001	0.354	0.001
Actual base excess (mmol/L)	0.352	0.001	0.262	0.016
Lactate (mmol/L)	-0.625	<0.001	-0.498	<0.001

r_s: Spearman's correlation coefficient.

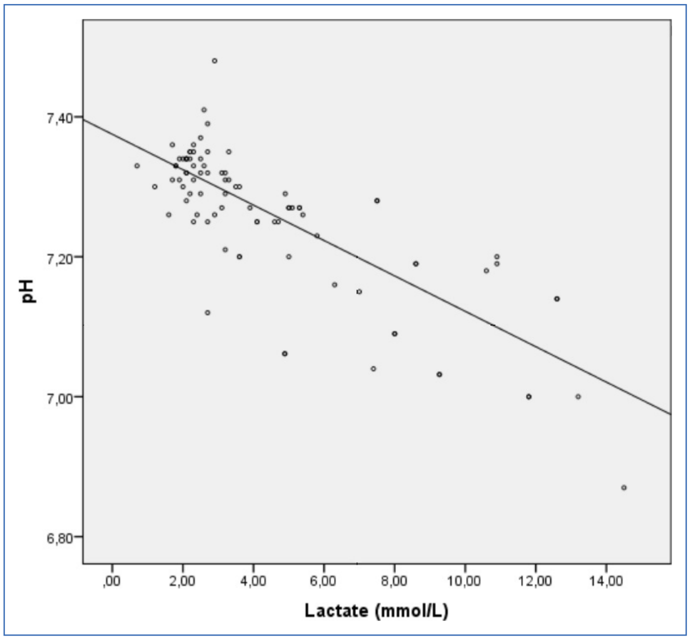

Figure 1: Scatter-dot diagram showing the relationship between umbilical arterial cord blood lactate concentrations and pH in all patients (n=84, r=-0.734, p<0.001).

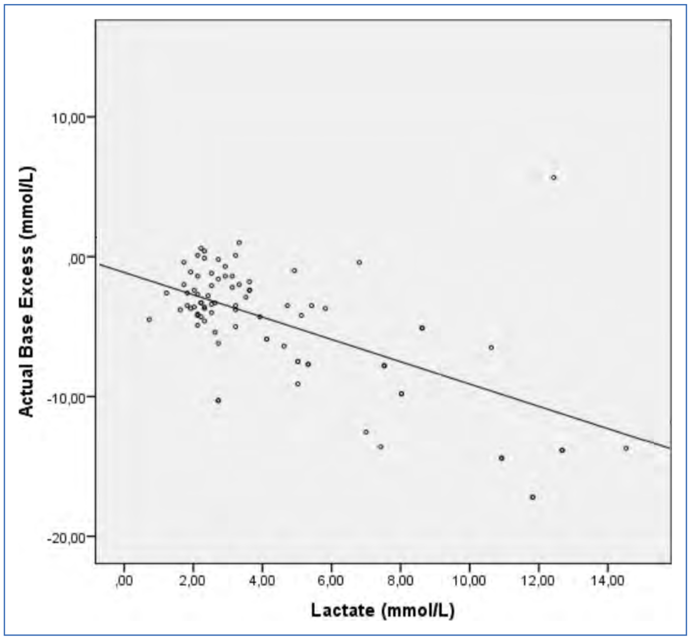

Figure 2: Scatter-dot diagram showing the relationship between umbilical arterial cord blood lactate concentrations and actual base excess in all patients (n=84, r=-0.581, p<0.001).

tate and pH - ABE is shown in Figures 1 and 2. Spearman's correlation showed a significant inverse correlation between lactate and pH (r=-0.734, p<0.001). There was also a moderate inverse correlation between lactate and ABE (r=-0.581, p<0.001).

Among 84 patients, there was only 1 newborn whose umbilical artery blood pH <7.0. Nine patients had umbilical artery blood pH <7.1 and/or ABE <-12. The ROC curve analysis was also performed to determine the best cut-off value of the umbilical artery blood lactate concentration to predict these nine fetuses (Fig. 3). The optimal umbilical artery blood lactate cut-off level of 7 mmol/L, above which the sensitivity and specificity were 88.9% and 89.3%, respectively. The area under the curve (AUC) was 0.915±0.056 (95% CI 0.805–1.00).

DISCUSSION

Intrapartum fetal heart rate monitoring aims to identify fetuses with intrapartum acidosis and prevent fetal death through timely intervention. The secondary purpose is to prevent fetal neurological damage. Although there is evidence that intrapartum fetal monitoring reduces intrapartum deaths, its contribution to long-term neurological outcomes is unknown. Intrapartum continuous electronic fetal monitoring results in increased operative vaginal delivery and cesarean rates due to high false-positive rates of intrapartum fetal monitoring.[5] Although fetal scalp blood sampling and fetal oxygen saturation have been suggested to reduce this false-positive rate, these methods are invasive.[12,13]

There are many reports in the literature showing that late decelerations are associated with fetal acidemia. In addition, decreased variability was suggested as an indicator of fetal acidemia.[14,15] Williams et al.[16] also showed that decreased variability accompanied by late decelerations is associated with lower pH, ABE values, and increased incidence of acidemia. In addition, they reported that the incidence of acidemia increased in cases of decreased variability in which decelerations were not observed. Inconsistent with this data, it has also been suggested that late, variable, and prolonged decelerations are associated with acidemia, but variability in the last 30 minutes is not.[9] In the

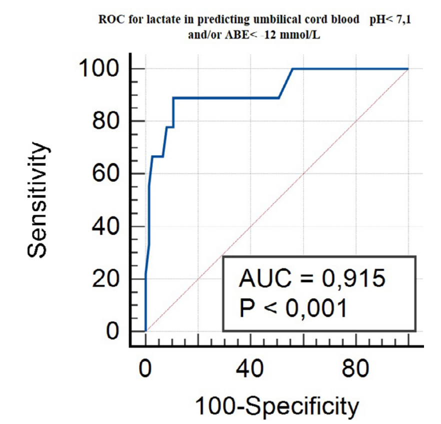

Figure 3: Receiver operating characteristic (ROC) curve for umbilical arterial cord blood lactate concentration to determine umbilical artery pH <7.1 and/or ABE <-12 mmol/L. Optimal cut-off=7 mmol/L. Area under the curve, 0.915; 95% CI: 0.805–1.00; sensitivity, 88.9%; specificity, 89.3%.

current study, late decelerations accompanied by decreased variability were associated with worse neonatal clinical outcomes compared to groups with only late or variable decelerations (with normal variability). Umbilical artery blood acid-base evaluation was also consistent with clinical findings. Decreased variability accompanied by decelerations, especially late decelerations, was associated with lower pH and ABE and higher lactate levels. It was reported in the NIHCD workshop that minimal variability or loss of variability may be the result of hypoxemia and acidosis. It occurs due to insufficient compensatory mechanisms in cases of insufficient oxygenation of the fetal brain. In this situation, decreased variability may be accompanied by recurrent decelerations.[4] Therefore, variability should be considered along with decelerations when evaluating FHR traces.

Regarding the early neonatal clinical findings, there was a positive correlation between 1- and 5-minute Apgar scores and pH and actual base excess. Additionally, there was a stronger inverse correlation with lactate concentration. In contrast to our results, pH and lactate were not significantly correlated with Apgar scores in the study reported by Hamed. They reported that the reason for this situation is that the Apgar score does not provide information about causes such as obstruction of the respiratory tract by secretions, anesthetic drugs, or cardiovascular malformations.[17] Despite the accepted criteria of pH <7.0 and ABE <-16 mmol/L in the diagnosis of fetal asphyxia, there are reports that lactate is a better marker for determining fetal status.[18]

Since the number of fetuses whose umbilical artery blood parameters meet the threshold values for fetal acidemia is only one, we could not determine an optimal cut-off level of lactate concentration for fetal acidosis. Instead, in our study, a lactate cut-off level of >7 mmol/L was shown to be a very good marker in predicting umbilical artery blood pH <7.1 and/or ABE <-12 mmol/L to identify the fetuses with non-reassuring heart rate patterns that may benefit from intervention before fetal acidosis develops. Umbilical artery blood lactate concentration was inversely related to pH and ABE. Consistent with these findings, a lactate cut-off level of 8 mmol/L was suggested to indicate intrapartum fetal asphyxia, given the inverse correlation with pH and ABE.[19]

Despite all these definitions, the positive predictive value (PPV) of intrapartum electronic fetal monitoring for acidosis is quite low. According to the current literature, neonatal acidemia was reported between 12–30% in patients with non-reassuring intrapartum fetal heart rate tracings.[20,21] In our study, the PPV of intrapartum FHR monitoring for fetal acidosis was lower than the values reported in the literature (11% for pH <7.1). This was interpreted as "obstetricians in Türkiye are not guaranteed by the laws, and there may be increased malpractice concerns, so they may act hastily in the cesarean section decision."

In addition, the presence of inter- and intraobserver variations in the evaluation of the traces is a situation that limits the reliability of the method. Nevertheless, its negative predictivity of 99% serves as a guide for the clinician for fetal well-being.[22]

Relatively small sample size and not evaluating interobserver and intraobserver variability in the assessment of fetal heart rate tracings were the main limitations of the study. Nonetheless, it provides useful information for the assessment of fetal well-being with continuous intrapartum electronic monitoring. Further large-scale studies are recommended, especially for the routine use of umbilical artery lactate concentration to identify intrapartum fetal acidosis.

CONCLUSION

Due to the high false-positive rates of intrapartum fetal heart rate monitoring for adverse neonatal outcomes, we recommend careful interpretation. Decreased variability in non-reassuring intrapartum fetal heart rate patterns should be considered as important as decelerations. In the evaluation of intrapartum fetal asphyxia, umbilical artery blood lactate appears to be as good a marker as pH and ABE.

Statement

Ethics Committee Approval: The İstanbul Medeniyet University Göztepe Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 24.02.2015, number: 2015/0015).

Author Contributions: Concept – ÖGE; Design – ÖGE; Supervision – AG; Resource – ÖGE; Materials – ÖGE; Data Collection and/or Processing – ÖGE; Analysis and/or Interpretation – AG; Literature Search – ÖGE; Writing – ÖGE; Critical Reviews – AG.

Conflict of Interest: The authors have no conflict of interest to declare.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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