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Original Research Article

Intrapartum fetal heart rate and umbilical cord gases as predictors of adverse neonatal outcome: an observational study

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ABSTRACT

Background: Perinatal asphyxia (PA) remains a leading cause of neonatal mortality and long-term neurological impairment, particularly in developing countries, with an incidence of approximately 10 per 1,000 live births. Early detection and timely intervention are crucial. Cardiotocography (CTG) is widely used for intrapartum fetal monitoring, while umbilical arterial blood gas (ABG) analysis accurately reflects the new-born's metabolic status at birth.
Methods: This cross-sectional observational study was conducted at Kasturba Hospital, Delhi, involving 360 term, singleton, low-risk pregnancies in active labor with cephalic presentation. CTG was recorded at admission, rupture of membranes (ROM), and just before delivery. Based on CTG findings, participants were grouped into a control group (n=300) with normal or indeterminate tracings, and a study group (n=60) with abnormal tracings. Paired umbilical cord blood samples were collected for pH and base deficit analysis. Fetal acidosis was defined as umbilical artery (UA) pH <7.1 and umbilical vein (UV) pH <7.2. APGAR scores at 1 and 5 minutes were recorded. NICU-admitted neonates were evaluated for hypoxic-ischemic encephalopathy (HIE) using the Thompson score on days 1, 3, and 5.
Results: Abnormal CTG at ROM and before delivery showed significant association with low UA pH, high base deficit, and low UV pH ($p<0.001$), along with higher NICU admissions. Cord gas parameters also correlated significantly with HIE severity ($p=0.024$).
Conclusions: CTG is an effective intrapartum tool, significantly associated with fetal acidosis and adverse neonatal outcomes.

Keywords: Intrapartum FHR, Cardiotocography, Thompson score, Umbilical arterial pH, Fetal acidosis, Birth asphyxia, Fetal hypoxia

INTRODUCTION

Perinatal asphyxia refers to a condition during the first and second stage of labor in which impaired gas exchange leads to fetal acidosis, hypoxemia and hypercarbia.[1] It is a serious condition associated with significant perinatal morbidity and mortality. It can lead to stillbirth or result in a severely compromised neonate, with serious systemic and neurological sequelae. Hospital-based studies in India report an incidence of 2.8% to 5.6% among institutional deliveries, accounting for approximately 24.3% of neonatal deaths.[2] Globally, the incidence ranges from 2 to 10 per 1,000 term newborns.[2] Gillam-Krakauer and Gowen reported that the incidence of birth asphyxia is approximately 2 per 1,000 live births in high-income countries, however, in low-income countries this rate can be up to ten times higher due to unavailability of efficient maternal and neonatal care.[3]

Intrapartum fetal monitoring plays a crucial role in assessing fetal well-being during labor and in identifying early signs of hypoxia. Advances in technology have

expanded the available methods for fetal surveillance, which include intermittent auscultation of the fetal heart rate (FHR), continuous electronic monitoring such as cardiotocography (CTG), biophysical profile (BPP), modified BPP, contraction stress testing, Doppler velocimetry, and more invasive assessments like fetal scalp blood pH and serum lactate measurements. FHR patterns are regulated by the autonomic nervous system, and in response to hypoxic stress, compensatory mechanisms are activated, often detectable through CTG changes. Key indicators suggestive of fetal hypoxia include bradycardia or tachycardia (FHR <100 or >160 bpm), late or persistent decelerations, and diminished beat-to-beat variability.

Confirmation of acute intrapartum hypoxic events is typically achieved through the detection of metabolic acidosis in umbilical cord blood or early neonatal blood samples. Current clinical guidelines recommend the collection of both arterial (ABG) and venous (VBG) cord blood in high-risk deliveries. While ABG reflects fetal metabolic status, VBG provides insight into the maternal-fetal exchange. Early and accurate identification of fetal asphyxia is critical in reducing perinatal morbidity and mortality and in preventing long-term complications such as hypoxic–ischemic encephalopathy (HIE), cerebral palsy, and permanent neurological impairment. There is currently no universally accepted definition for abnormal cord blood gas and lactate levels. However, acidemia is typically recognized at an umbilical arterial pH <7.2 in 7–9% of neonates, <7.1 in 1–3%, and <7.00 in 0.26–1.3%. Studies have shown that a pH value below 7.1 in the umbilical artery—defined as more than two standard deviations below the mean—is indicative of pathological academia.[4] A veno-arterial pH difference of less than 0.02 is considered within normal limits. According to the ACOG Task Force on Neonatal Encephalopathy (2013), fetal acidemia is defined by an umbilical arterial pH <7.0 and/or a base deficit of ≥12 mmol/l.[4] The Apgar score, developed in 1952 by Dr. Virginia Apgar, remains a widely accepted tool for the rapid assessment of neonatal condition at 1 and 5 minutes after birth.[5] It evaluates five parameters: heart rate, respiratory effort, muscle tone, reflex irritability, and skin color. Endorsed by both the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP), the Apgar score is primarily used to assess the new-born's immediate adaptation to extrauterine life and response to resuscitative efforts.[5] However, the neonatal resuscitation program (NRP) guidelines caution that clinical intervention should begin prior to the assignment of the 1-minute score, as it is not intended to determine the need for initial resuscitation.[5]

To assess the severity of perinatal asphyxia and its neurological impact, the Thompson scoring system is frequently utilized. HIE, a clinical syndrome resulting from severe perinatal asphyxia and secondary cerebral ischemia, is one of the leading causes of neonatal brain injury. In term infants, neurological examination at discharge remains one of the most reliable predictors of long-term outcome.[6]

The objective of this study was to evaluate the reliability of intrapartum FHR patterns and fetal acidosis in predicting neonatal outcomes, with a particular focus on the need for neonatal intensive care unit (NICU) admission. By examining correlations between specific FHR abnormalities, biochemical indicators of fetal acidosis (such as umbilical cord blood pH), and subsequent neonatal health status, this study aimed to assess the predictive value of these measures.

METHODS

This cross-sectional observational study was conducted in the labor room and NICU of a teaching hospital, Delhi, over a period of 18 months, after obtaining clearance from the Institutional Ethical Committee. All consenting singleton pregnancies between 37-40 weeks, cephalic presentation admitted in active labor (cervical dilatation ≥4 cm) were included. All complicated and high-risk pregnancies were excluded from the study. A structured questionnaire was used to collect all relevant information from the patient (mothers). The patients were subjected to intrapartum CTG in the labor room to record intrapartum FHR. The tracings were recorded at a speed of 1cm/min for 20 minutes at the time of admission, rupture of membranes, and during active labor. All FHR tracings were analysed for basic features (baseline, variability, accelerations, decelerations, and contractions) followed by overall CTG interpretation as, normal (category-I trace), indeterminate (category-II trace), and abnormal (category-III trace) according to NICHD 2008 classification.[7]

A total 360 labouring subjects were recruited and CTG was performed at admission, at rupture of membrane (ROM) and at immediately before delivery. They were later classified into 2 groups, according to CTG findings. If the CTG was category III at any time during labor, they were labelled as study group and there were 60 subjects with such findings. Subjects with CTG categories I and II were 300 in number and were labelled as controls.

An umbilical paired cord blood sample was collected soon after delivery. Before delivery of the placenta, the umbilical cord was clamped at two points, 10 cm apart, with Kocher's clamps and cut. After differentiating the umbilical vein and artery, 1 ml of umbilical cord arterial and venous blood sample was collected in a pre-heparinized syringe and analysed at 37° C by an acid blood gas analyser, within 30 minutes of sampling. pH (arterial and venous) and base excess (arterial blood) were recorded. In term infants, the lower limit of arterial cord blood pH for normal outcome is 7.1 and that for venous blood is 7.2.[8] Cord blood gas values were validated according to the methods of Westgate et al to obviate inadvertently sampling from the same vessel twice or transposing the vessels either when taking the samples.[9]

All babies were assessed with an APGAR score at 1 and 5 minutes done in labour room at the birthing stations under a radiant warmer (APGAR score as per SEAR NNPD WHO working definition) Neonates with feeble cry, or feeble tone RD, poor cry or colour were resuscitated as per prevalent NRP guidelines and APGAR at 1 and 5 minutes were noted in these cases too. Delayed cord clamping and cord milking was not attempted in any of the neonates. Neonates needing admission were transported on oxygen as per institutional NICU protocol.

Babies admitted to NICU were evaluated for: HIE by using Thompson score on day 1 (6 hours), day 3 and day 5 of life; neonatal seizures that were due to any other cause ex metabolic, and birth trauma; and outcome of babies in form of discharge or death.

The data were entered in Microsoft Office Excel spreadsheet, and analysis was done using statistical package for social sciences (SPSS) version 23.0. Relevant statistical analysis was done by relevant tests and p value of <0.05 was considered statistically significant.

RESULTS

Out of total 300 controls enrolled in the study; the mean age was 25 years (range 16–40 years). Median age was 25 years (interquartile range 22–28 years) with a standard deviation of ±4.35 years. Out of total 60 cases in the study group, the mean age was 26 years (18–37 years). Median age was 25 years (interquartile range 23–29 years) with a standard deviation of ±4.82 years. There was no significant association between maternal age and FHR **(**abnormal CTG (p value=0.1714, $\chi 2$=3.5275).

Out of the total 300 controls, 163 (54.33%) were primigravida, while out of the 60 cases in the study group, 32 (53.33%) were primigravida. The association between FHR (abnormal CTG and parity was not significant (p value=0.8871, $\chi 2$=0.0201).

In the control group, 206 (68.67%) patients underwent vaginal delivery compared with 32 (53.33%) patients in the study group (Table 1). The number of patients undergoing LSCS in the study group 28 (46.67%) had a significant association with an abnormal CTG (p value=0.022, $\chi 2$=5.247) confirming the belief that an abnormal FHR (abnormal CTG) leads to higher incidence of LSCS (Table 1). Lower segment caesarean section was performed for various causes like fetal distress, protracted descent and dilatation, arrest of descent and dilatation and fetal distress.

Birth asphyxia is more commonly associated with meconium-stained amniotic fluid (MSL). The liquor was clear in 241 (80.33%) in the control group compared with 24 (40%) in the study group (Table 1). There was a significant association (p value <0.0001, $\chi 2$=80.74) between development of MSL and FHR (abnormal CTG).

Neonatal outcome

Out of a total of 360 mother's enrolled in the study, there was no foetal /perinatal death or still birth. There were 194 male babies and 166 female babies and sex was not found to be confounding factor for study outcome (Table 1; p value=1.000).

According to weight distribution; no baby in control and study group was in very low birth weight or extremely low birth weight category. 271 out of 300 i.e. 90.33% (control) and 56 out of 60 i.e. 93.33% (study) neonates had normal weight. 21 (7%) of control group and 4 (6.67%) of study group neonates had low birth weight. 8 (2.67%) neonates in the control group and no neonate in study group was in large for gestational age category infants were large for gestational age (LGA). The association between FHR (abnormal CTG and neonatal weight was not significant (p value=0.4622, $\chi 2$=0.5405).

Resuscitation was required in 13 (4.32%) out of 300 control deliveries and 34 (57.63%) out of 60 live neonates born to study group mothers.

Out of 300 control group mothers' neonates 4 (1.33%) neonates had respiratory distress for which they were admitted to the NICU and were put on oxygen by hood. Of 60 live neonates born to study mothers, there were no deaths, and 33 (55%) neonates were admitted to the NICU. Out of these 8 (13.3%) required ventilation, 9 were put on CPAP (15%) and 16 (26.66%) were put on oxygen hood NICU admission had a significant association (p value <0.00001).

On comparing the CTG findings at admission, with acidaemia it was observed that the association between abnormal FHR (CTG) at admission and acidemia as measured by umbilical artery pH (p value 0.32), umbilical artery base deficit (p value=1.000), and umbilical vein pH (p value=1.000) was not significant. However, abnormal FHR (CTG) at ROM has a significant association with acidemia as measured by umbilical artery pH (p value=0.0015), umbilical artery base deficit (p value <0.00001), and umbilical vein pH (p value <0.00001). Similarly, abnormal FHR (CTG), at just before delivery, has a significant association with acidemia as measured by umbilical artery pH (p value <0.00001), umbilical artery base deficit (p value <0.00001), and umbilical vein pH (p value <0.00001) (Tables 2-4).

On comparing the CTG with APGAR score it was concluded that CTG at ROM had strong association with APGAR at 1 min (p value 0.0000139) while CTG at admission and CTG before delivery did not correlate significantly with APGAR at 1 minute. CTG at ROM (p value=0.05) as well as CTG before delivery (p value=0.036) had a significant association with 5-minute APGAR, while CTG at admission did not have any significant association with 5-minute APGAR (p value=0.546) (Table 5).

Thompson score was abnormal in 14 neonates on day 1. It was mild in 11 and moderate in 3. None of these neonates had abnormal maternal CTG at admission. Correlation of CTG at ROM and before delivery was higher with abnormal Thompson score (Table 6).

On comparing the diagnostic accuracy of CTG at different time with respect to acidaemia by different parameters, it was concluded that CTG at rupture of membrane has the best accuracy with specificity and NPV of abnormal CTG in predicting acidaemia by umbilical venous pH at ROM more than 95% but had low sensitivity and PPV (Table 7).

Table 1: Parameters of new-borns as per CTG category.

Parameters	CAT I and II (controls) n=300 (%)	CAT III (study) n=60 (%)	P value
B weight			
LBW	21 (7)	4 (6.67)	0.437
Normal	271 (90.33)	56 (93.33)	
LGA	8 (2.67)	0	
Sex			
Male	151 (50.33)	43 (71.66)	1.000
Female	149 (49.66)	17 (28.33)	
Colour of liquor			
Clear	241 (80.33)	24 (40)	0.001
GR I MSL	38 (12.67)	11 (18.33)	
GR II MSL	20 (6.67)	12 (20)	
GR III MSL	1 (0.33)	13 (21.67)	
Admission to NICU			
Mothers side	296 (98.67)	36 (60)	<0.00001
NICU admission	4 (1.33)	24 (40)	
Mode of delivery			
Vaginal delivery	206 (68.67)	32 (53.33)	0.032
LSCS	94 (31.33)	28 (46.67)	

Table 2: Relationship of acidemia by umbilical artery pH with CTG at different time.

Variables	Acidemia (%)	Normal (%)	P value
CTG at ADM			
CAT I and II (n=352)	17 (4.83)	335 (95.17)	0.32
CAT III (n=8)	0	8 (100)	
CTG at ROM			
CAT I and II (n=347)	14 (4.03)	333 (95.97)	0.0015
CAT III (n=13)	3 (23.08)	10 (76.92)	
CTG before DEL			
CAT I and II (n=301)	6 (1.99)	295 (98.01)	<0.00001
CAT III (n=59)	11 (18.64)	48 (81.36)	

Table 3: Relationship of acidemia of umbilical artery by base deficit with CTG at different time.

Variables	Acidemia (%)	Normal (%)	P value
CTG at ADM			
CAT I and II (n=352)	12 (3.41)	340 (96.59)	1.000
CAT III (n=8)	0	8 (100)	
CTG at ROM			
CAT I and II (n=347)	12 (3.46)	335 (96.54)	<0.00001
CAT III (n=13)	4 (30.77)	9 (69.23)	
CTG before DEL			
CAT I and II (n=301)	6 (1.99)	295 (98.01)	<0.00001
CAT III (n=59)	10 (16.95)	49 (83.05)	

Table 4: Relationship of acidemia by umbilical vein pH with CTG at different time.

Variables	Acidemia (%)	Normal (%)	P value
CTG at ADM			
CAT I and II (n=352)	25 (7.1)	327 (92.9)	1.000
CAT III (n=8)	1 (12.5)	7 (87.5)	
CTG at ROM			
CAT I and II (n=347)	21 (6.05)	326 (93.95)	<0.00001
CAT III (n=13)	5 (38.46)	8 (61.54)	
CTG before DEL			
CAT I and II (n=301)	8 (2.66)	293 (97.34)	<0.00001
CAT III (n=59)	18 (30.51)	41 (69.49)	

Table 5: Relation of CTG with APGAR score.

Variables	APGAR at 1 min (%)			P value	APGAR at 5 min (%)			P value
	>7	4-6	0-3		>7	4-6	0-3	
CTG at ADM								
CAT I and II (n=352)	343 (97.44)	9 (2.55)	0	0.5456	351 (99.7)	1 (0.28)	0	1.000
CAT III (n=8)	7 (87.5)	1 (12.5)	0		8 (100)	0	0	
CTG at ROM								
CAT I, II (n=347)	339 (97.69)	8 (2.31)	0	0.0000	347 (100)	0	0	0.05
CAT III (n=13)	9 (69.24)	4 (30.76)	0	139	11 (84.62)	2 (15.38)	0	
CTG before DEL								
CAT I, II (n=301)	296 (98.33)	5 (1.67)	0	0.546	288 (95.68)	12 (3.99)	1 (0.33)	0.036
CAT III (n=59)	52 (88.14)	7 (11.86)	0		48 (81.36)	11 (18.64)	0	

Table 6: Relation of HIE scoring by Thompson score with CTG.

Variables	Normal (%)	Mild (1-10) (%)	Moderate (11-14) (%)	Severe (15 and above)	P value
CTG at ADM					
CAT I and II (n=352)	343 (97.44)	7 (1.99)	2 (0.57)	0	1.000
CAT III (n=8)	3 (37.5)	4 (50)	1 (12.5)	0	
CTG at ROM					
CAT I, II (n=347)	344 (99.14)	3 (0.86)	0	0	0.05
CAT III (n=13)	2 (15.38)	8 (61.54)	3 (23.08)	0	
CTG before DEL					
CAT I, II (n=301)	301 (100)	0	0	0	0.05
CAT III (n=59)	45 (76.27)	11 (18.64)	03 (5.09)	0	

Table 7: Diagnostic accuracy of CTG at different time with respect to acidaemia by different parameters.

Parameters	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
CTG at ROM				
Umbilical artery pH	17.65	97.08	23.08	95.97
UA base deficit	25	97.38	30.77	96.54
Umbilical vein pH	19.23	97.6	38.46	93.95
CTG before del				
Umbilical artery pH	64.71	86.01	18.64	98.01
UA base deficit	62.5	85.76	16.95	98.01
Umbilical vein pH	69.23	87.72	30.59	97.34

DISCUSSION

One of the major causes of neonatal mortality and morbidity is intrauterine fetal compromise in the form of fetal hypoxia. Many methods came up over the years to assess fetal compromise *viz.* auscultation, CTG, cord blood gas analysis, and scalp blood pH. CTG is a continuous recording of the fetal heart rate obtained via an ultrasound

transducer placed on the mother's abdomen. CTG is widely used in pregnancy as a screening method to assess fetal wellbeing and identification of babies with acute or chronic fetal hypoxia or at risk of developing such hypoxia. Cord blood gases analysis came to be known when James et al reported in 1958 that gas analysis of blood samples obtained from a clamped umbilical cord could reflect fetal hypoxia can give an indication of preceding fetal hypoxic stress.[10] Both intrapartum FHR monitoring and umbilical blood pH as the methods of fetal surveillance in labour indicate foetal hypoxia and compromise. A total of 360 women were enrolled in the study, and it was observed that maternal age (p value=0.1714) and parity (p value=0.887) did not exhibit any discernible impact on FHR. Similarly, factors such as baby's birth weight (p value=0.437) and the sex of the baby (p value=1.000) were also determined to lack significant associations with abnormal FHR. These findings suggest that, in this cohort, maternal age, parity, birth weight, and baby's sex do not seem to be influential factors in the occurrence of abnormal FHRs. The observations are similar to study by Salma and Girotra et al.[2,11] The study revealed strong associations between abnormal FHR and certain factors. Meconium staining of liquor (p value=0.001), mode of delivery (p value=0.032) and NICU admission (p value <0.00001) demonstrated significant correlations with abnormal FHR. Similar associations have also been observed by Indiramani et al for Meconium-stained liquor.[12] Our results were in agreement with study by Medeiros et al, and Bhagwani et al for NICU admissions.[13,14] Cardiotocography FHR was recently found to be an independent predictor of fetal acidosis and indicator for caesarean delivery by Ranjana et al and Behuria et al.[15,16]

When examining cases where FHR was categorized as III by CTG at the rupture of membranes, a noteworthy association with academia was observed, as evidenced by umbilical artery pH (p value=0.0015), umbilical artery base deficit (p value <0.00001), and umbilical vein pH (p value <0.00001). The similar association was also observed by Chandrika et al and Sharmin et al.[17,18] Abnormal FHR just before delivery also exhibited a significant association with acidemia across all three methods (p value <0.00001), while abnormal CTG at admission did not show a strong correlation with acidemia (p value=1.000). This is in contrast to some studies which show interventions in the form of caesareans was higher in patients with abnormal CTG at admissions by Astruc et al.[19] In terms of neonatal outcomes, a more pronounced association was observed between abnormal FHR at rupture of membranes and just before delivery and poorer APGAR scores at 1 minute (p value=0.05), APGAR at 5 minutes (p value=0.036), and Thompson score of neonates (p value=0.05). These data suggest the intrapartum FHR variability is physiologically relevant and can be used for predicting the acidemia and Apgar scores at birth of the new-born infants without severe cases of morbidity and from uncomplicated pregnancies.

The study demonstrated a high diagnostic accuracy of FHR monitoring at the rupture of membranes, with a specificity of 97.08% and a notably high negative predictive value (NPV) of 95.97%. In comparison, CTG conducted just before delivery exhibited a better sensitivity of 64.71%. Based on these findings, one may recommend to prioritize the performance of CTG at the rupture of membranes, since the findings in this study show the closest association between CTG at ROM and neonatal hypoxia. But this may not be the complete picture of this complex situation. Kundu et al concluded in their study on CTG interpretation in last 60 min before delivery with neonatal outcome; that an estimation of the pH value and consequently of neonatal outcome on the basis of a present CTG seems to be difficult.[20] The purpose of intrapartum CTG is to identify fetal hypoxia caused by acute or subacute events during labor, enabling timely medical intervention to help prevent complications like hypoxic-ischemic encephalopathy, cerebral palsy, and neonatal death.

Limitations

This study has few limitations. A single-center, observational study involving only term, low-risk pregnancies limits generalizability. Key intrapartum confounders such as oxytocin use, labor duration, and delivery mode were not fully controlled. Outcomes were assessed only in the immediate postpartum period, without long-term follow-up. Lastly, unequal group sizes may have impacted statistical comparisons.

CONCLUSION

Intrapartum FHR monitoring and cord blood gas analysis remain important tools in assessing fetal well-being and predicting neonatal outcomes. While CTG is widely used, its subjectivity and limited predictive value especially in isolation underscore the need for careful interpretation. Cord blood gas analysis, particularly umbilical arterial pH, offers a more objective measure of fetal metabolic status at birth and has shown stronger associations with adverse outcomes, including low Apgar scores and higher Thompson scores, which are indicative of neonatal encephalopathy severity. However, variability in sampling technique and timing may affect its accuracy. This study supports the complementary use of both CTG and cord blood analysis to improve the early identification of at-risk neonates and guide timely interventions. Future research should include larger, more diverse populations, standardized protocols, and long-term follow-up to better understand their combined predictive value for both immediate and longer-term neonatal outcomes.

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