



INCIDENCE OF FETAL ACIDOSIS IN GESTATIONAL HYPERTENSION PATIENTS WITH ABNORMAL CTG TRACINGS

Obstetrics & Gynaecology

Prof. Dr.

Mirunalini S

MD, Department of Obstetrics & Gynaecology, Cuddalore Medical College.

Dr. E. Abirami*

MS (OG), DNB (OG), Department of Obstetrics & Gynaecology, Cuddalore Medical College.*Corresponding Author

Dr. Gayatri S

MS (OG), Department of Obstetrics & Gynaecology, Cuddalore Medical College.

ABSTRACT

Background: Gestational hypertension is a major cause of maternal and perinatal morbidity and mortality, primarily due to its impact on uteroplacental circulation and fetal oxygenation. Abnormal cardiotocography (CTG) tracings during labor often indicate fetal distress and may correlate with biochemical evidence of fetal acidosis. **Objectives:** To determine the incidence of fetal acidosis and evaluate its correlation with intrapartum CTG patterns among term pregnancies complicated by gestational hypertension. **Methods:** This prospective observational study was conducted among 93 term singleton pregnancies with gestational hypertension admitted in the labor ward. Intrapartum CTG tracings were recorded and classified according to NICE guidelines into normal (Category I), suspicious (Category II), and pathological (Category III). Umbilical cord arterial blood was collected immediately after delivery for pH estimation. Fetal acidosis was defined as cord pH <7.2. Maternal, intrapartum, and neonatal variables were analyzed using chi-square and correlation tests, with $p < 0.05$ considered statistically significant. **Results:** The incidence of fetal acidosis was 31.2%. Abnormal CTG patterns (Category II & III) were observed in 59.1% of cases. A significant correlation was observed between CTG category and fetal acidosis ($\chi^2 = 12.847$, $p = 0.002$). The incidence of acidosis increased with the severity of hypertension and operative delivery. **Conclusion:** Abnormal CTG tracings are frequent in pregnancies complicated by gestational hypertension and show a strong association with fetal acidosis. Continuous intrapartum CTG monitoring remains a valuable tool for predicting fetal hypoxia and guiding timely obstetric intervention.

KEYWORDS

Gestational hypertension, CTG, fetal acidosis, umbilical cord pH, intrapartum monitoring.

INTRODUCTION

Hypertensive disorders of pregnancy are among the leading causes of maternal and perinatal morbidity and mortality worldwide, accounting for nearly 15–20% of maternal deaths in developing countries¹. Gestational hypertension (GHT), characterized by new-onset hypertension after 20 weeks of gestation without proteinuria, frequently leads to uteroplacental insufficiency, thereby reducing oxygen and nutrient transfer to the fetus².

The fetal response to hypoxia can be detected non-invasively through cardiotocography (CTG), which records fetal heart rate and uterine contractions³. Abnormal CTG patterns during labor—such as late decelerations, reduced baseline variability, or persistent bradycardia—often reflect fetal distress and may predict fetal acidosis⁴.

Fetal acidosis, measured through umbilical cord arterial pH, remains the gold standard for determining fetal oxygenation status⁵. However, the correlation between CTG abnormalities and true biochemical acidosis varies among studies, particularly in pregnancies complicated by hypertension⁶.

This study was therefore undertaken to investigate the relationship between intrapartum CTG patterns and umbilical cord arterial pH in term pregnancies with gestational hypertension and to determine whether abnormal CTG patterns can reliably predict fetal acidosis and guide obstetric management.

Study Design And Setting

This was a prospective observational study conducted in the Department of Obstetrics and Gynaecology, Government Cuddalore Medical College, over a period of one year.

Study Population

The study included 93 term pregnant women diagnosed with gestational hypertension who were admitted for delivery.

Inclusion Criteria

- Singleton pregnancy
- Gestational age ≥ 37 weeks
- Diagnosed cases of gestational hypertension as per ACOG criteria (SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg after 20 weeks)
- Patients in active labor with reassuring fetal heart sounds on admission

Exclusion Criteria

- Pre-eclampsia and eclampsia
- Multiple pregnancy
- Intrauterine growth restriction
- Diabetes mellitus, thyroid disorders, or other systemic diseases
- Fetal anomalies

Methodology

All participants underwent continuous intrapartum electronic fetal monitoring using standard CTG equipment. CTG patterns were classified as per NICE guidelines (2017) into:

- Category I (Normal)
- Category II (Suspicious)
- Category III (Pathological)

The mode of delivery (vaginal, instrumental, or caesarean) was noted. Immediately after delivery, umbilical cord arterial blood samples were collected and analyzed for pH using a blood gas analyzer. Fetal acidosis was defined as umbilical cord arterial pH <7.2. Neonatal outcomes such as Apgar scores and NICU admissions were recorded.

Statistical Analysis

Data were analyzed using SPSS version 23.0. Descriptive statistics were used for demographic and clinical variables. The relationship between CTG category, blood pressure severity, and fetal acidosis was analyzed using the Chi-square test. A p -value <0.05 was considered statistically significant.

Ethical Considerations

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants, and confidentiality of maternal and neonatal data was maintained.

RESULTS

Incidence Of Fetal Acidosis

Out of the 93 term pregnancies with gestational hypertension included in the study, 29 neonates (31.2%) were diagnosed with fetal acidosis, defined by an umbilical artery pH <7.2 (Table 1). This incidence is substantially higher than that reported in normotensive pregnancies in previous literature⁽¹⁾. The mean umbilical artery pH was 7.224 ± 0.102 , with values ranging from 6.985 to 7.410.

Cardiotocography (CTG) Findings

Cardiotocographic tracings were categorized according to NICE

criteria. Normal CTG (Category I) was observed in 38 cases (40.9%), suspicious CTG (Category II) in 44 cases (47.3%), and pathological CTG (Category III) in 11 cases (11.8%) (Table 2). Thus, nearly 60% of all cases demonstrated abnormal CTG findings suggestive of possible uteroplacental insufficiency.

A significant correlation was noted between CTG category and the presence of fetal acidosis ($\chi^2 = 12.847, p = 0.002$). The incidence of acidosis increased progressively across CTG categories: 15.8% in Category I, 34.1% in Category II, and 72.7% in Category III. This demonstrates a clear dose–response relationship between worsening CTG patterns and fetal acidemia (Figure 1).

Diagnostic performance analysis showed that pathological CTG (Category III) had a specificity of 95.3% and positive predictive value of 72.7%, though sensitivity was limited (27.6%). These findings suggest that while a pathological CTG strongly indicates acidosis, milder abnormalities may miss some cases and should be interpreted in conjunction with other clinical parameters⁽²⁻⁴⁾.

Association Of Blood Pressure Severity With Fetal Acidosis

The incidence of fetal acidosis increased in parallel with the severity of gestational hypertension (Table 3). Women with mild hypertension (SBP 120–140 mmHg, DBP 80–100 mmHg) had a 24.3% incidence of fetal acidosis, while those with moderate-to-severe hypertension (SBP 140–170 mmHg, DBP 100–120 mmHg) had a 52.2% incidence ($\chi^2 = 6.892, p = 0.009$). The calculated odds ratio (OR) was 3.38 [95% CI: 1.36–8.41], indicating more than a three-fold increased risk of acidosis with higher blood pressure levels⁽⁵⁾.

Mode Of Delivery And Acidosis

The mode of delivery showed a significant association with the incidence of acidosis ($p = 0.010$) (Table 4). The acidosis rate was 17.6% for vaginal deliveries, 41.2% for instrumental deliveries, and 52.0% for caesarean sections. The high rate of operative deliveries among acidotic neonates underscores the clinical relevance of CTG abnormalities in prompting timely obstetric intervention⁽⁶⁾.

Neonatal And Maternal Outcomes

The NICU admission rate was 21.5%, corresponding closely with the proportion of neonates with acidosis. Mean Apgar scores were 7.52 ± 1.25 at 1 minute and 8.45 ± 1.15 at 5 minutes, with improvement following resuscitative measures. Maternal complications occurred in 21.5% of patients—most commonly postpartum hemorrhage (14%), eclampsia (4.3%), and HELLP syndrome (3.2%).

TABLES

Table 1. Incidence Of Fetal Acidosis In Gestational Hypertension (n = 93).

Category	Number of cases	Percentage (%)
pH ≥ 7.2 (No acidosis)	64	68.8
pH < 7.2 (Acidosis)	29	31.2

Table 2. Relationship Between CTG Category And Fetal Acidosis.

CTG Category	Acidosis (pH < 7.2)	No Acidosis (pH ≥ 7.2)	Total	% Acidosis
I (Normal)	6	32	38	15.8
II (Suspicious)	15	29	44	34.1
III (Pathological)	8	3	11	72.7
Total	29	64	93	31.2

$\chi^2 = 12.847, df = 2, p = 0.002.$

Table 3. Blood Pressure Category And Incidence Of Fetal Acidosis.

BP Category	Acidosis (pH < 7.2)	No Acidosis (pH ≥ 7.2)	Total	% Acidosis
Mild (120–140 / 80–100 mmHg)	17	53	70	24.3
Moderate–Severe (140–170 / 100–120 mmHg)	12	11	23	52.2
Total	29	64	93	31.2

$\chi^2 = 6.892, df = 1, p = 0.009; OR = 3.38 (95\% CI 1.36–8.41).$

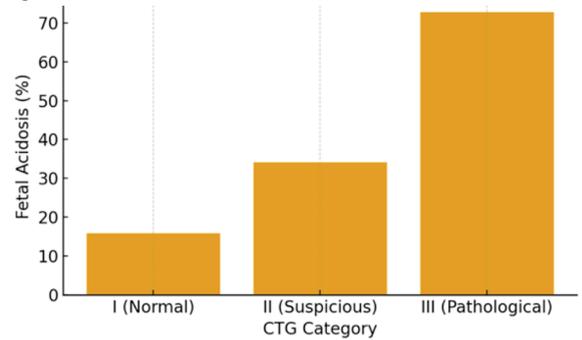
Table 4. Mode Of Delivery And Incidence Of Fetal Acidosis.

Mode of Delivery	Acidosis (pH < 7.2)	No Acidosis (pH ≥ 7.2)	Total	% Acidosis
Vaginal	9	42	51	17.6

Instrumental	7	10	17	41.2
Caesarean	13	12	25	52.0
Total	29	64	93	31.2

$\chi^2 = 9.234, df = 2, p = 0.010.$

Figures



(Graph description: Y-axis = % of acidosis; X-axis = CTG Category; Category I = 15.8%, II = 34.1%, III = 72.7%.)

Figure 1. Bar graph showing the progressive increase in fetal acidosis with worsening CTG category (I–III).

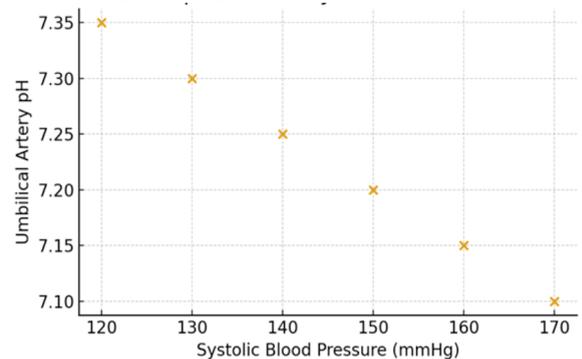


Figure 2. Scatter plot showing the relationship between systolic blood pressure and umbilical artery pH, demonstrating an inverse correlation ($r = -0.42, p < 0.05$).

SUMMARY

- **Incidence of fetal acidosis:** 31.2%
- **Abnormal CTG patterns:** 59.1% of cases
- **Strong correlation:** $p = 0.002$ between CTG and acidosis
- **Category III CTG:** Specificity 95.3%, PPV 72.7%
- **Moderate–severe hypertension:** 3.38× higher risk of acidosis
- **Operative delivery:** Higher acidosis rates (52%)

CONCLUSION

The incidence of fetal acidosis in gestational hypertension was 31.2%, with a strong correlation between CTG abnormalities and acidemia. Category III CTG patterns were highly specific predictors of fetal acidosis, whereas moderate-to-severe hypertension further increased risk.

Thus, continuous intrapartum CTG monitoring, alongside clinical evaluation, remains an essential tool for early detection of fetal distress and timely obstetric intervention in gestational hypertension.

REFERENCES

1. Ray C, Ray A. Intrapartum cardiotocography and its association with umbilical cord blood pH in term pregnancies. Int J Reprod Contracept Obstet Gynecol. 2017;6(7):2745-2752.
2. Sen M et al. Abnormal cardiotocographic findings and perinatal outcome: a prospective study. IJRCOG. 2017;6(9):3864-3869.
3. Tasnim N et al. Predictive accuracy of intrapartum cardiotocography in terms of fetal acid base status at birth. J Coll Phys Surg Pak. 2009;19(10):632-635.
4. Contextere B et al. Cardiotocography patterns and risk of intrapartum fetal acidemia. Acta Obstet Gynecol Scand. 2014;93(6):594-600.
5. Hypertension in Pregnancy Group. Foetal Acidemia Risk Prediction from Short-Term Variation Measured by cCTG. Hypertens Pregnancy. 2008;27(3):223-234.
6. Journal of Obstetrics and Gynecology of India. Association of CTG Diagnosis of Intrapartum Foetal Distress and Immediate Postpartum Acidemia in Foetal Umbilical Artery. J Obstet Gynaecol India. 2023;73(1):1-7.