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Complete List of Authors:	Galli, Letizia; St. George's University Hospitals NHS Foundation Trust, ; Unit of Obstetrics and Gynecology, Azienda Unità Sanitaria Locale di Reggio Emilia - IRCCS, Dall'Asta, Andrea; University Hospital of Parma, Italy, Obstetrics and Gynecology Whelehan, Virginia; St George's University Hospitals NHS Foundation Trust Archer, Abigail; St George's University Hospitals NHS Foundation Trust Chandraharan, Edwin
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TITLE PAGE

Intrapartum cardiotocography patterns observed in suspected clinical and subclinical chorioamnionitis in term fetuses.

AUTHORS

Letizia Galli[1,2,3], Andrea Dall'Asta[3,4], Virginia Whelehan[1], Abigail Archer[1], Edwin Chandraharan[1]

INSTITUTIONS

[1]St George's University Hospitals NHS Foundation Trust, London, United Kingdom.

[2]Unit of Obstetrics and Gynecology, Azienda Unità Sanitaria Locale, Istituto di Ricovero e Cura a Carattere Scientifico (IRCCS), Azienda Unità Sanitaria Locale - Reggio Emilia, Italy.

[3]Department of Medicine and Surgery, Obstetrics and Gynecology Unit, University of Parma, Parma, Italy.

[4]Department of Surgery and Cancer, Institute of Reproductive and Developmental Biology, Imperial College London, United Kingdom.

ADDRESS FOR CORRESPONDENCE

Dr. Letizia Galli, MD

St. George's University Hospitals NHS Foundation Trust, Blackshaw Road, London SW17 0QT, United Kingdom

Unit of Obstetrics and Gynecology, Azienda Unità Sanitaria Locale, Istituto di Ricovero e Cura a Carattere Scientifico (IRCCS), Azienda Unità Sanitaria Locale - Reggio Emilia, Italy

Department of Medicine and Surgery, Obstetrics and Gynecology Unit, University of Parma, Italy

Tel: +390521702434

E-mail: letizia.galli1986@gmail.com

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Abstract

Aim

To evaluate the CTG features observed in suspected intrapartum clinical and subclinical chorioamnionitis in term fetuses according to the recently suggested criteria for the pathophysiological interpretation of the fetal heart rate and their correlation with perinatal outcomes.

Methods

Retrospective analysis of non-consecutive CTG traces. "CTG chorioamnionitis" diagnosed either based on a persistent rise in the baseline for the given gestation or on a persistent increase in the baseline fetal heart rate during labor >10% without preceding decelerations and in the absence of maternal pyrexia. Perinatal outcomes were compared among cases with no sign of chorioamnionitis, in those with only "CTG chorioamnionitis" and in those who developed clinical chorioamnionitis.

Results

2105 CTG traces analyzed. 356 fulfilled the CTG criteria for chorioamnionitis. Higher rates of APGAR < 7 at 1 and 5 minutes (21.6% vs 9.0% and 9.8% vs 2.0%, respectively, p <0.01 for both) and lower umbilical artery pH (7.14 $\pm$ 0.11 vs 7.19 $\pm$ 0.11, p <0.01) and an over 5-fold higher rate of NICU admission (16.6% vs 2.9%, p <0.01) were noted in the "CTG chorioamnionitis" group. Differences in the incidence of abnormal CTG patterns were noted between cases with clinical chorioamnionitis (89/356) and the "CTG chorioamnionitis group" (267/356).

Conclusions

Intrapartum CTG features of suspected chorioamnionitis are associated with adverse perinatal outcomes.

Keywords

Fetal heart rate, inflammation, intrapartum fever, CTG pathophysiology, fetal heart monitoring.

INTRODUCTION

The term chorioamnionitis refers to an acute inflammation of the amniotic membranes and the chorion of the placenta (1). Scientific evidence suggests that intrapartum chorioamnionitis is a major cause of non-hypoxic fetal compromise and adverse perinatal outcomes (2,3,4,5,6) being associated with a 5-fold higher risk of cerebral palsy (7). The diagnosis of chorioamnionitis is based on the clinical criteria described by Gibbs et al., which include maternal pyrexia and at least one among maternal or fetal tachycardia, uterine fundal tenderness, and purulent or foul amniotic fluid (8,9). Nevertheless, epidemiologic data suggest that up to 90% of cases of chorioamnionitis confirmed on pathology specimen of the membranes and the placenta occur in women with no overt sign of clinical (i.e. subclinical) chorioamnionitis (10,11). This illustrates the fact that chorioamnionitis is primarily a fetal disease and not a maternal disease, therefore signs and symptoms may not be consistently present in the mother. Conversely, maternal pyrexia may occur due to epidural anesthesia in a vast majority of cases (12,13,14), in the absence of histopathological evidence of chorioamnionitis (11,15).

Cardiotocography (CTG) is a test for fetal hypoxia and represents the most widely used tool for the intrapartum monitoring of the fetal wellbeing. Previous studies evaluated the relationship between CTG patterns and clinical or subclinical chorioamnionitis but could not demonstrate any role of the CTG in the diagnosis of chorioamnionitis (16-20). One of the reasons for these negative findings was attempting to correlate the CTG features that occur in intrapartum hypoxia (i.e. decelerations) to chorioamnionitis, which is a non-hypoxic pathway of fetal neurological injury. In addition, in these studies baseline tachycardia was defined as above 160 beats per minute (bpm), however term fetuses with a strong vagal dominance may not increase the baseline heart rate up to 160 bpm or above.

The recently suggested criteria for the pathophysiological interpretation of the fetal heart rate (21) offer a unique opportunity to re-evaluate the association between chorioamnionitis and CTG abnormalities. According to the pathophysiology interpretation of the CTG, an increase in the fetal heart rate without preceding decelerations may reflect a 'non-hypoxic' cause of fetal compromise. Due to the poorly developed fetal blood-brain barrier and resultant impact of fetal systemic inflammatory response syndrome (FSIRS) on the brain, the autonomic and somatic nervous system centres may be affected. Therefore, abnormal CTG features such as reduced variability and lack of accelerations and cycling may be used as markers of chorioamnionitis. In this study we evaluated these "non-hypoxic" CTG characteristics and the perinatal outcomes within a selected cohort of fetuses with a suspicion of chorioamnionitis based on an increase in the baseline fetal heart rate without decelerations and in the absence of preceding maternal dehydration, tachycardia or pyrexia.

METHODS

This was a retrospective study which included a non-consecutive series of women with non-anomalous singleton term pregnancy who delivered at St. George's University Hospitals NHS Foundation Trust, London, between 2014 and 2016.

According to the inclusion criteria of the study all women were in active labor, which was defined by means of a fully effaced, ≥3 cm dilatated cervix coupled with ≥3 contractions in 10 minutes recorded at tocography (22,23), and had ruptured membranes, which allowed continuous intrapartum CTG recording by internal transducer. At inclusion, all cases were at term pregnancy, which was defined by a gestational age between 37^{+0} and 41^{+6} weeks as determined by first trimester crown-rump length measurement, had cephalic presenting fetus and normal CTG according to the classification by the International Federation of Gynecology and Obstetrics (FIGO)(24). As a prerequisite for inclusion fetal growth was appropriate based on growth scan performed beyond 30 weeks or based on symphysis-fundal distance assessment performed at 36 to 37 weeks and there was no history of maternal complications of the pregnancy such as preeclampsia or other hypertensive disorders of the pregnancy and diabetes.

All CTG traces were systematically evaluated by one single investigator (LG) with specific training on CTG interpretation and analyzed according to the FIGO guidelines and the newly developed interpretation of the CTG using fetal pathophysiology (21,25,26) in terms of baseline fetal heart rate (FHR) from labor onset until delivery and for CTG patterns including loss/absence of accelerations (i.e. abrupt increase — of the FHR above the baseline with onset to peak in less than 30 seconds and amplitude of more than 15 beats per minute (bpm) and lasting more than 15 seconds but less than 10 minutes), loss/absence of cycling (i.e. absence of alternative periods of active and quiet sleep indicating altered fetal behavioral state), loss/absence of variability (i.e. the oscillation in the FHR signal evaluated as the average bandwidth amplitude of the signal in 1-minute CTG segments),

baroreceptor-mediated or “variable” decelerations (i.e. characterized by a rapid fall in the FHR without any delay and a rapid recovery to the original baseline FHR and secondary to the compression of the umbilical cord), chemoreceptor-mediated or “late” decelerations (i.e. characterized by a gradual and slow recovery to the original baseline FHR even after cessation of uterine contractions and secondary to the accumulation of carbon dioxide and metabolic acids during hypoxia) and the presence of saltatory pattern (i.e. characterized by an increased variability with bandwidth value exceeding 25 bpm and lasting more than 30 minutes) or sinusoidal patterns (i.e. a regular, smooth, undulating signal, resembling a sine wave, with amplitude of 5-15 bpm and a frequency of 3-5 cycles per minute, lasting for more than 30 minutes, which coincides with the absence of accelerations) (21,24). CTG traces were double checked by the supervisor (EC) to ensure inter-observer agreement.

According to the pathophysiological interpretation of the CTG, an increase >10% in the fetal heart rate without ongoing decelerations in the absence of preceding maternal tachycardia or pyrexia reflects a ‘non-hypoxic’ cause of fetal compromise such as fetal inflammatory response secondary to chorioamnionitis (21). Therefore, “CTG chorioamnionitis” was suspected either based on a persistent rise in the baseline FHR above the 95th percentile for the given gestation according to previously published reference ranges (27), in the absence of maternal dehydration and pyrexia, or based on the persistent increase in the baseline FHR >10% during labor, in the absence of preceding “repetitive” decelerations and of any other CTG indicator of hypoxia (24), compared to the baseline FHR recorded during the first 60 minutes of the CTG trace following the diagnosis of active labor. This 10% cut-off for raised baseline FHR was chosen as evidence of chorioamnionitis based on the fact that a 1°C rise in the body temperature increases the heart rate by approximately 10% (21,28,29,30). Only apyrexial women showing the aforementioned CTG features were considered at risk of chorioamnionitis and included for the study purposes. Clinical chorioamnionitis was

diagnosed in those women who eventually showed any sign of chorioamnionitis according to the Gibbs' Criteria (1,8), while cases showing CTG features suspicious for chorioamnionitis without any evidence of maternal pyrexia or other signs or symptoms were labelled as "subclinical chorioamnionitis".

Cases where maternal pyrexia or tachycardia were transitory or were considered to be secondary to maternal dehydration, epidural analgesia and maternal urinary or respiratory tract infection or in which the diagnostic criteria for chorioamnionitis (1,8) were fulfilled before the onset of the CTG features suggestive of chorioamnionitis were excluded, as were cases in which the raised baseline FHR and/or the 10% increase in the baseline FHR was associated with preceding repetitive decelerations.

Information concerning maternal demographics and intrapartum and neonatal outcomes were retrieved from a dedicated electronic database for all the included cases.

Approval for this retrospective study was granted by the St. George's Research & Enterprise Office. Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) version 20 (IBM Inc., Armonk, NY, USA). Data were shown as mean $\pm$ standard deviation or as number (percentage). Categorical variables were compared using the Chi-square or Fisher exact test, while comparison of continuous variables included T test for independent sample and 2-tailed *t* tests. $p < 0.05$ was considered as statistically significant.

RESULTS

Overall, 2105 CTG traces were analyzed of which 356 (16.9%) fulfilled the CTG criteria for suspected chorioamnionitis. Pregnancy features and perinatal outcomes of cases with and without CTG suspicion of chorioamnionitis are summarized in Table 1. Within the "CTG chorioamnionitis" group, a significantly higher rate of nulliparae was noted (78.1% vs 67.6%, p <0.01), delivery occurred at significantly later gestation ($40^{+3} \pm 1^{+2}$ vs $40^{+0} \pm 2^{+0}$, p <0.01) and neonatal weight was significantly higher (3524 ± 469 vs 3384 ± 494, p <0.01) compared to cases with no CTG suspicion of chorioamnionitis. Within this latter group a significantly lower rate of obstetric intervention was noted (44.8% vs 69.9%, p <0.01). Fetuses showing intrapartum CTG features suggestive of chorioamnionitis also showed significantly higher rates of APGAR <7 at 1 and 5 minutes (21.6% vs 9%, p <0.01 and 9.8% vs 2.0%, p <0.01, respectively) and MSAF (38.2% vs 2.1%, p<0.01). Additionally, the "CTG chorioamnionitis" group showed lower umbilical artery pH (7.14 ± 0.11 vs 7.19 ± 0.11, p <0.01) but not higher rate of arterial pH less than 7.0 (p 0.20). NICU admission rate was over five-fold higher in fetuses showing CTG features suggestive of chorioamnionitis (16.6% vs 2.9%, p <0.01) (Table 1). There was no case of intrapartum stillbirth or neonatal death.

Persistent loss of accelerations and the presence of chemoreceptor-mediated decelerations and baroreceptor-mediated decelerations were the most common abnormal CTG findings in the "CTG chorioamnionitis" group and were observed in 64.3%, 63.5% and 46.6%, respectively; persistently reduced variability and loss of cycling occurred in 30.9% and 33.1%, respectively, while saltatory and sinusoidal patterns were noted in 13.8% and 6.7%, respectively (Figure 1).

Within the "CTG chorioamnionitis" group, 89 (25%) fetuses eventually developed signs or symptoms of clinical chorioamnionitis, the remaining 267 (75%) being labelled as "subclinical chorioamnionitis" group. Subgroup analysis of the CTG patterns showed a significantly higher incidence in the loss of cycling (46.1% vs 28.8%, p <0.01) and persistently reduced variability (40.4% vs 27.7%, p 0.02) in the

"clinical" compared to the "subclinical chorioamnionitis" groups (Table 2). With regard to the relationship between abnormal CTG features, MSAF and neonatal outcomes loss/absence of variability was the only CTG abnormality which was significantly associated with all the considered adverse perinatal outcomes and also with lower umbilical cord arterial and venous pH ($p < 0.01$ for both) but not with MSAF (Table 3). Loss/absence of accelerations and loss/absence of cycling were more common in those fetuses who experienced all the considered poor perinatal outcomes, however loss/absence of cycling was not associated with MSAF (p 0.17). Chemoreceptor-mediated decelerations were significantly related to low APGAR scores at 1 and 5 minutes ($p < 0.01$ and 0.01 respectively) and MSAF ($p < 0.01$), while the presence of saltatory or sinusoidal patterns was variably associated with low APGAR at 1 and 5 minutes, MSAF fluid and NICU admission. Finally, no association was found between baroreceptor-mediated "variable" decelerations and any of the considered adverse perinatal outcomes within the study group.

DISCUSSION

Data from our study suggest that an inappropriate (i.e. higher than expected) baseline fetal heart rate for the given gestational age and a persistent increase of the baseline in the absence of predisposing factors (i.e. suggestive of chorioamnionitis according to a pathophysiological interpretation of the CTG) are significantly associated with obstetric interventions, MSAF and adverse neonatal outcomes. Furthermore, a significant relationship between abnormal CTG patterns, clinical and subclinical chorioamnionitis and adverse neonatal outcomes has been demonstrated.

To our best knowledge, this is the first study that determined the features observed on the CTG trace in suspected chorioamnionitis based on the application of fetal pathophysiology without the use of CTG guidelines (24,31,32). These are based on "pattern recognition" and are primarily aimed at the detection of intrapartum hypoxia (21). On the other hand, chorioamnionitis is a fetal disease which causes fetal compromise through a "non-hypoxic" (i.e. inflammatory) pathway, therefore the CTG trace may not show the features described in standard guidelines on CTG interpretation. It is vital to appreciate that a post term fetus with an ongoing chorioamnionitis may increase its heart rate from 120 bpm to 155 bpm secondary to inflammatory response. Therefore, it may still be classified as normal according to CTG Guidelines which consider a baseline FHR between 110-160 bpm as the normal range for all fetuses at term, irrespective of their gestational age.

A fetus who is exposed to intrapartum hypoxia would attempt to protect its myocardial workload by a reflex decrease in the FHR, termed as a deceleration (21,25,26,33). According to our hypothesis, unlike in hypoxia, where the increase in the catecholamine-mediated FHR occurs following the decelerations to protect the myocardium (21,33), in chorioamnionitis the increase of the baseline FHR is secondary to inflammatory mediators and raised metabolic rate secondary to ongoing inflammation and occurs in the absence of preceding decelerations.

Our study suggests that fetuses with CTG intrapartum features of chorioamnionitis have a higher incidence of poor Apgar score both at 1 and 5 minutes. This may be explained by the effect of inflammation on the muscle tone as well as on the fetal tracheo-bronchial tree, which may result in fetal congenital pneumonia and resultant low Apgar scores at 1 and 5 minutes. The significantly increased incidence of MSAF in fetuses with CTG features suggestive of chorioamnionitis may be explained by the fact that a fetus not only inhales the infected amniotic fluid into the lungs (i.e. develops congenital pneumonia and FSIRS) but also swallows the infected amniotic fluid leading to the onset of gastroenteritis and passage of meconium.

The occurrence of metabolic acidosis so defined by umbilical artery cord pH <7.00 was not significantly higher in fetuses showing CTG features suggestive of chorioamnionitis, however there was a trend towards lower umbilical arterial and venous pH within this group. This may be explained by the fact that in infection there is an increased metabolic demand leading to higher oxygen consumption compared to non-infected cases. Therefore, chorioamnionitis may lead to accelerated fall in pH during labor resulting in umbilical cord metabolic acidosis at birth (21,33,36,37).

With regard to the observed CTG features, our study suggests that cases showing clinical features of chorioamnionitis have an increased likelihood of developing absence of cycling and loss of baseline variability compared to those with subclinical chorioamnionitis. This may be explained by the detrimental effect of increased maternal temperature on the fetal hypothalamic thermo-regulatory centre, with subsequent alteration of the fetal sleep-wake cycles manifesting with loss of cycling as well as depression of the fetal autonomic nervous system, which is associated with loss of baseline variability (21,38). The presence of baroreceptor-mediated and chemoreceptor-mediated decelerations was not associated with lower umbilical arterial and venous cord pH in cases of clinical chorioamnionitis, however the presence of chemoreceptor-mediated late decelerations was related to an increased rate of MSAF. It is well-known that late decelerations occur due to

uteroplacental insufficiency (21,24,33). In chorioamnionitis, due to the presence of inflammation and placental villitis, the gaseous exchange across the placenta may be impaired leading to the onset of late decelerations.

Our study also shows that fetuses with features of suspected chorioamnionitis on the CTG trace have an increased likelihood of NICU admission. This may be secondary to poor postnatal adaptation as a result of ongoing inflammation and congenital pneumonia.

The main strength of our study is represented by its original design, in which we first attempted to move away from the traditional CTG guidelines (24) and analyzed CTG according to a pathophysiological approach in order to demonstrate the feasibility of the intrapartum CTG suspicion of chorioamnionitis and detect its CTG features within a wide cohort of laboring women at term pregnancy. Additionally, optimal CTG recording was ensured by internal transducer in all cases and all CTG traces were systematically evaluated by the same two practitioners who had dedicated training on pathophysiological interpretation of the CTG.

We acknowledge that our study has limitations. In first instance, this is a retrospective analysis of CTG traces. Information concerning the group-B-Streptococcus (GBS) status and additional risk factors for intrauterine infection was not addressed, nevertheless this was a cohort of women with uncomplicated term pregnancy in which the prevalence of GBS is considered to be relatively constant (39,40). In the UK, routine screening for GBS is not carried out according to the current national guidelines (32). Additionally, it may be argued that the CTG traces in the non-chorioamnionitis group were not analyzed, hence their features were not compared to those with chorioamnionitis. However, the aim of our study was to use a pathophysiological approach to determine the CTG characteristics of chorioamnionitis, and we believe that a comparative study with the 'non-chorioamnionitis' group can be performed only when the CTG features of chorioamnionitis have been determined.

Another limitation is that placentae were not routinely sent for pathological examination, hence the correlation with histology could not be evaluated. However, the results of histopathological examination are not available to clinicians involved in everyday labour ward practice, therefore the purpose of our study was to determine the perinatal outcomes in fetuses with CTG features of suspected chorioamnionitis. Additionally, our dataset did not include data on inflammatory markers, such as white blood cells, which may confirm the diagnosis of clinical chorioamnionitis but are not tested during labor unless intrapartum infection is suspected. However, it is important to point out that increased inflammatory markers during labor are not specific indicators of chorioamnionitis.

The diagnostic criteria for "CTG chorioamnionitis" may also be subject of controversy, however evidence from our research has shown that when evaluating fetuses with no CTG features of hypoxia, those with CTG features suspicious for chorioamnionitis had worse perinatal outcomes compared to those with "appropriate" baseline FHR and, of note, almost one in four cases with "CTG chorioamnionitis" eventually showed clinical evidence of chorioamnionitis so defined according to the Gibbs' criteria. Finally, we empirically chose a 10% increase in the baseline FHR as suspicious for chorioamnionitis, however we appreciate that fetal inflammatory response may not consistently elevate the baseline heart rate above the 10% in all cases.

In summary, fetuses with CTG features suggestive of chorioamnionitis have significantly higher rates of adverse perinatal outcomes and an over five-fold higher rate of NICU admission compared to fetuses with no suspicion of chorioamnionitis at CTG analysis. Additionally, different CTG features were observed between clinical and subclinical chorioamnionitis. Therefore, we believe that inappropriate baseline for the given gestational age and a persistent rise in the baseline fetal heart rate >10% without proceeding decelerations on the CTG trace in the absence of maternal pyrexia or

tachycardia should prompt suspicion of chorioamnionitis. In these cases, close intrapartum monitoring is warranted in order to avoid poor perinatal outcomes.

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Figure legends

Figure 1 – Algorithm for the management of suspected chorioamnionitis based on the cardiotocography (CTG) features by the application of fetal pathophysiology.

FHR: fetal heart rate; MSAF: meconium stained amniotic fluid.

Table 1 – Pregnancy features and perinatal outcomes in the suspected cardiotocography (CTG) chorioamnionitis and in the non-suspected chorioamnionitis group.

	Non-suspected chorioamnionitis **N 1749**	**Suspected CTG chorioamnionitis** **N 356**	**P**
Parity	Nulliparae N 1183 (67.6%) Pluriparae N 566 (32.4%)	Nulliparae N 278 (78.1%) Pluriparae N 78 (21.9%)	<0.01
Gestation at delivery	$40^{+0} \pm 2^{+0}$	$40^{+3} \pm 1^{+2}$	<0.01
Induction of labor	Yes N 803 (45.9%) No N 946 (54.1%)	Yes N 176 (49.4%) No N 180 (50.6%)	0.22
Mode of delivery	SVD N 964 (55.2%) ID N 584 (33.4%) CS N 199 (11.4%)	SVD N 107 (30.1%) ID N 157 (44.1%) CS N 92 (25.8%)	<0.01
Birthweight	3384 ± 494	3524 ± 469	<0.01
APGAR < 7 I MIN	157 (9.0%)	77 (21.6%)	<0.01
APGAR < 7 V MIN	35 (2.0%)	35 (9.8%)	<0.01
Meconium stained amniotic fluid	37 (2.1%)	136 (38.2%)	<0.01
Mean UA PH	7.19 ± 0.11	7.14 ± 0.11	<0.01
Mean UV PH	7.24 ± 0.12	7.22 ± 0.13	0.20
PH UA <7.00	24 (1.4%)	9 (2.5%)	0.20
NICU admission	51 (2.9%)	59 (16.6%)	<0.01

UA – Umbilical artery

UV – Umbilical vein

NICU – Neonatal intensive care unit

SVD – Spontaneous vaginal delivery

ID – Instrumental delivery

CS – Caesarean delivery

Data presented as Number (percentage), mean ± standard deviation or median (range)

Table 2 – Suspected cardiotocography (CTG) chorioamnionitis group: comparison of the CTG features between clinical and subclinical chorioamnionitis.

CTG features	**Suspected CTG chorioamnionitis all cases N 356**	**Clinical chorioamnionitis N 89**	**Subclinical chorioamnionitis N 267**	**p**
Absence or loss of accelerations	229 (64.3%)	63 (70.8%)	166 (62.2%)	0.14
Absence or loss of cycling	118 (33.1%)	41 (46.1%)	77 (28.8%)	<0.01
Absence or loss of variability	110 (30.9%)	36 (40.4%)	74 (27.7%)	0.02
Baroreceptor decelerations	166 (46.6%)	42 (47.2%)	124 (46.4%)	0.90
Chemoreceptor decelerations	226 (63.5%)	53 (59.6%)	173 (64.8%)	0.37
Saltatory pattern	49 (13.8%)	10 (11.2%)	39 (14.6%)	0.42
Sinusoidal pattern	24 (6.7%)	5 (5.6%)	19 (7.1%)	0.63

Table 3 – Suspected cardiotocography (CTG) chorioamnionitis group: correlation between abnormal CTG features, meconium stained amniotic fluid (MSAF) and neonatal outcomes.

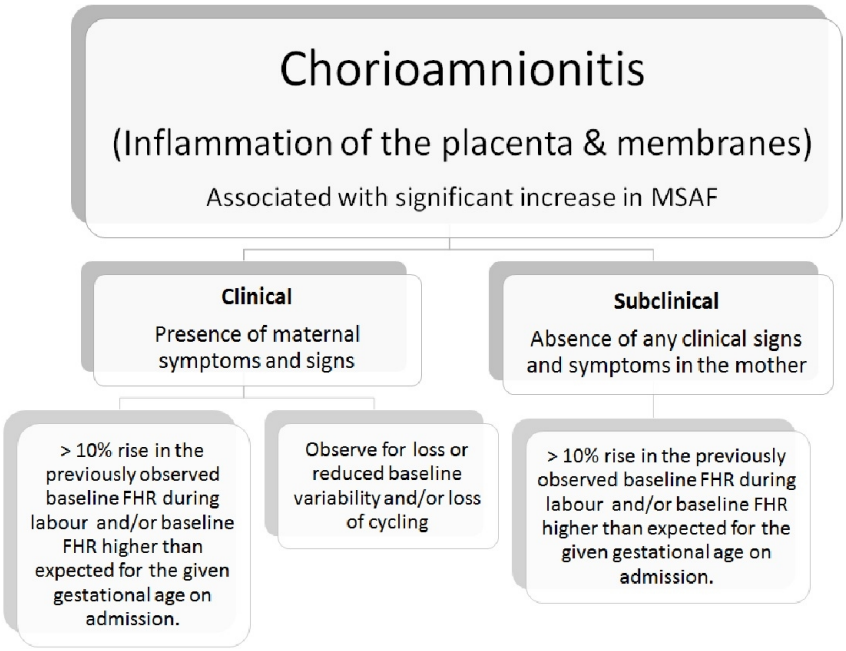
CTG features	MSAF			APGAR <7 at I min			APGAR <7 at V min			MEAN UA pH			MEAN UV pH			NICU admnission		
	Yes	No	p	Yes	No	p	Yes	No	p	Yes	No	p	Yes	No	p	Yes	No	p
Absence or loss of accelerations	99/229 43.2%	37/127 29.1%	<0.01	69/229 30.1%	9/127 7.1%	<0.01	33/229 14.4%	2/127 1.6%	<0.01	7.12±0.11	7.19±0.01	<0.01	7.20±0.14	7.26±0.12	0.01	54/229 23.6%	5/127 3.9%	<0.01
Absence or loss of cycling	51/118 35.7%	85/238 43.2%	0.17	53/118 44.9%	25/238 10.5%	<0.01	27/118 22.9%	8/238 3.4%	<0.01	7.12±0.12	7.16±0.11	0.04	7.19±0.14	7.24±0.12	<0.01	43/118 36.4%	16/238 6.7%	<0.01
Absence or loss of variability	44/110 40%	92/246 37.3%	0.64	43/110 39.1%	35/246 14.2%	<0.01	28/110 25.4%	7/246 2.8%	<0.01	7.11±0.12	7.16±0.11	<0.01	7.18±0.14	7.24±0.13	<0.01	33/110 30%	26/246 10.6%	<0.01
Baroreceptor decelerations	66/166 39.8%	70/190 36.8%	0.57	43/166 25.9%	35/190 18.4%	0.08	18/166 10.8%	17/190 8.9%	0.55	7.13±0.11	7.16±0.11	0.14	7.22±0.13	7.22±0.14	0.78	23/166 13.8%	36/190 18.9%	0.20
Chemoreceptor decelerations	99/226 43.8%	37/130 28.5%	<0.01	60/226 26.5%	18/130 13.8%	<0.01	29/226 12.8%	6/130 4.6%	0.01	7.13±0.11	7.16±0.12	0.09	7.21+0.13	7.22+0.14	0.64	43/226 19%	16/130 12.3%	0.10
Saltatory pattern	25/49 51%	111/307 36.2%	0.047	17/49 34.7%	61/307 19.9%	0.02	9/49 18.3%	26/307 8.5%	0.03	7.12±0.12	7.15±0.11	0.34	7.21+0.13	7.22±0.14	0.87	14/49 28.5%	45/307 14.6%	0.02
Sinusoidal pattern	11/24 45.8%	125/332 37.7%	0.42	11/24 45.8%	67/332 20.1%	<0.01	4/24 16.6%	31/332 9.3%	0.24	7.13±0.12	7.14±0.11	0.66	7.19±0.15	7.21±0.13	0.72	8/24 33.3%	51/332 15.4%	0.02

MSAF – meconium stained amniotic fluid

UA – Umbilical artery

UV – Umbilical vein

NICU – Neonatal intensive care unit



Frequency of abnormal intrapartum findings in cases of CTG suggestive for chorioamnionitis

- Persistent loss of accelerations (64.3%)
- Chemoreceptor-mediated decelerations (63.5%)
- Baroreceptor-mediated decelerations (46.6%)
- Persistently reduced variability (30.9%)
- Loss of cycling (33.1%)
- Saltatory pattern (13.8%)
- Sinusoidal pattern (6.7%)

- MSAF 38.2%

Figure 1 – Algorithm for the management of suspected chorioamnionitis based on the cardiotocography (CTG) features by the application of fetal pathophysiology.

344x353mm (96 x 96 DPI)