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Editorial

A pragmatic approach to recognize intrapartum chorioamnionitis

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ABSTRACT

Chorioamnionitis is a significant contributor to non-hypoxic fetal compromise, increasing the risk of neonatal encephalopathy and cerebral palsy. This paper highlights the limitations of traditional diagnostic methods for chorioamnionitis, which rely on histopathology, microbiology, and clinical signs, and emphasizes the importance of recognizing the fetal inflammatory response using intrapartum cardiotocography (CTG). By understanding the physiological pathways of inflammation, clinicians can identify characteristic CTG patterns. This pragmatic approach allows for earlier detection and intervention, potentially improving perinatal outcomes. The paper calls for a re-evaluation of clinical management practices and advocates for a multifaceted approach that incorporates CTG findings into the diagnosis of chorioamnionitis to prevent adverse neonatal outcomes.

Why is this relevant?

It is well understood that neonatal neurological damage can result from various factors other than hypoxia, and more often there are multiple contributing factors [1,2]. Evidence of infection in labor is reported in 15.6 % of these cases and can contribute to neonatal encephalopathy [3]. In the United Kingdom (UK), the Each Baby Counts project identified that approximately 74 % of term babies born with encephalopathy were potentially avoidable and therefore, there is an opportunity for improvement [4].

Intrapartum chorioamnionitis is a significant contributor to non-hypoxic fetal compromise and poor perinatal outcomes, with a considerable increase in the risk of cerebral palsy associated with it [5,6]. Most importantly, when infection and hypoxia occur together, they work synergistically, leading to increased vulnerability to periods of hypoxic-ischemic stress during labor, further raising the risk of encephalopathy and cerebral palsy [1,5].

Chorioamnionitis occurs most frequently in the setting of preterm birth but can be present in 3.8 % of deliveries at term and up to 5.1 % in post-term deliveries [2]. According to the 2022 Mothers and Babies: Reducing Risk through Audits and Confidential Enquiries across the UK (MBRRACE-UK) perinatal mortality surveillance report, infection was the primary cause of death in 6.6 % of neonatal deaths, a rate of 0.11 per 1,000 live births[7]. In 2019–21 pregnancy-related infections accounted for 6 % of maternal deaths including 3 cases following chorioamnionitis at term [8].

Intrapartum cardiotocography (CTG) is the most widely used tool to assess fetal well-being during labor, intended to detect signs of fetal hypoxic ischemia, prompting a timely medical intervention that would help prevent hypoxic-ischemic encephalopathy and reduce perinatal mortality [9]. But can CTG help us identify chorioamnionitis?

There are now recognized CTG patterns that should raise the suspicion of underlying inflammation/infection. These changes are easily understood once we comprehend the physiology behind the inflammatory pathway [10–13]. This paper aims to help obstetricians and midwives to recognize fetal inflammatory response on the CTG.

What is chorioamnionitis?

Clinical perspective

Clinical chorioamnionitis is usually diagnosed based on the presence of maternal fever, and 2 or more of the following signs: maternal tachycardia (>100 bpm), fetal tachycardia (>160 bpm), leukocytosis (>15,000/mm^3) uterine tenderness, purulent or malodorous vaginal discharge [14].

Intrapartum fever is relatively common, particularly in women who receive epidural analgesia, with rates ranging from 15 % to 25 % [15]. However, only about 5.4 % of these cases seem to be related to infection, and the majority, 94.5 % to 96.2 %, are attributable to epidural analgesia [16]. Interestingly, epidural-related fever is exclusive of women in labor (it is not described in nonpregnant surgical population, such as orthopedic patients, nor in elective cesarean delivery). Increased levels of inflammatory markers such as IL-6 and IL-10 have been measured in women in labor after epidural placement. Furthermore, some studies showed that prophylactic anti-inflammatory glucocorticoids could reduce epidural-related maternal fever. This may be explained by an inflammatory response induced by labor itself [15,16].

Typically, the amniotic cavity is a sterile compartment. There are two main pathways in which it can be invaded by microorganisms: ascending from the lower genital tract or hematogenous dissemination through the placenta, with the former being the most frequent route. Disruption of the anatomical structures that protect against ascending infections can increase the risk of chorioamnionitis, such as premature

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rupture of membranes (PROM), short cervix, cerclages, and multiple digital examinations [2,14].

Chorioamnionitis may also cause myometrial dysfunction. It has been associated with prolonged labor and higher cesarean section rates due to failure to progress. Some authors have attempted to explain this using a bimodal theory. In the initial phases of chorioamnionitis, proinflammatory cytokines stimulate the onset of labor or an increase in uterine contractility/tachysystole; at later stages, if the inflammatory process affects the myometrium, there is a significant decline in uterine contractility, associated with prolonged labor, lack of response to oxytocin augmentation and risk of postpartum bleeding secondary to uterine atony [14]. Zackler et al. identified that this impaired uterine activity appears 2 h after the diagnosis of chorioamnionitis by the onset of fever [17].

Although it is not classically considered a sign of chorioamnionitis, the presence of meconium in the amniotic fluid is associated with inflammation/infection. Meconium-stained amniotic fluid can be a physiological phenomenon in term fetuses, being more common with advanced gestational age, with about 6 % at 37 weeks to 25 % at 41 weeks. It is a sign of gastrointestinal maturation [18]. However, it can also be associated with chorioamnionitis or hypoxia, increasing the risk of poor neonatal outcome. In fetal life, amniotic fluid is present in the fetal gut due to normal swallowing and in the tracheal bronchial tree due to fetal breathing movements. In the case of microbial invasion of the amniotic cavity, the swallowed amniotic fluid can trigger an inflammatory response leading to increased peristalsis with a higher chance of passing meconium [19]. In addition, meconium stimulates bacterial growth and inhibits bacteriostatic activity of amniotic fluid, increasing the risk of chorioamnionitis [18,20].

Histopathology perspective

Chorioamnionitis is an acute placental inflammation that occurs in response to an infectious or non-infectious damaging stimulus. It is characterized by diffuse infiltration of neutrophils in the chorion and amnion. When the inflammation extends to the fetal compartment, thus the umbilical cord, it is known as acute funisitis, which is the histological manifestation of fetal inflammatory response syndrome (FIRS). Acute chorioamnionitis is the most common infection-related diagnosis in labor and the most frequent diagnosis in placental pathology reports [2,14].

In placental histopathology, the inflammatory reaction is usually classified into maternal and/or fetal inflammatory response. The extension of the neutrophil invasion (stage) is more important than the intensity (grade) [2]. Some degree of inflammatory response in the chorion is commonly observed in spontaneous deliveries with good outcomes. However, funisitis is associated with more intense fetal and intraamniotic inflammation, indicated by higher levels of interleukin-6 (IL-6) in fetal plasma and amniotic fluid, and poorer neonatal outcomes [19].

Microbiology and biochemical perspective

Microbial invasion of the amniotic cavity refers to the presence of microorganisms in amniotic fluid, identified through cultivation or molecular microbiological methods applied to samples retrieved by amniocentesis. On the other hand, intraamniotic inflammation refers to an inflammatory response in the amniotic cavity, which can be identified by the presence of inflammatory cells like neutrophils or by increased levels of inflammatory markers, such as interleukin-6 (IL-6) or matrix metalloproteinase (MMP-8) [14].

Intraamniotic infection refers to the combination of microbial invasion and the resulting intraamniotic inflammation. When there are microorganisms without evidence of inflammation this is most likely caused by contamination of specimen [14].

When intraamniotic inflammation occurs without the presence of microorganisms, it is known as sterile intraamniotic inflammation. Interestingly, this is observed in 40–60 % of acute chorioamnionitis [2]. Sterile intraamniotic inflammation is thought to result from other non-infectious causes or infections that failed to be detected using current cultivation methods and molecular microbiological techniques [14].

As mentioned earlier, the primary pathway of intra-amniotic infection is the ascending route. The microorganisms most commonly found in the amniotic fluid are *Ureaplasma species* (spp.), *Gardnerella vaginalis*, *Mycoplasma hominis, Streptococcus agalactiae, Staphylococcus aureus,* and *Bacteroides* spp. The higher the microbial load, the more intense is the intraamniotic inflammatory response. Among these, genital mycoplasmas are the most frequently detected microorganisms in the amniotic cavity. These organisms are not regularly cultured in obstetrics or neonatal medicine, and standard antimicrobial regimens are not effective in treating them. Notably, only a fraction of patients diagnosed with histologic chorioamnionitis have proven intraamniotic infection [14].

Both microbial and nonmicrobial triggers can cause an inflammatory response in the amniotic cavity, the mother, and the fetus, characterized by the following biochemical markers:

Amniotic cavity: Proinflammatory cytokines like MMP-8 and IL-6 are increased [2,14]. Neutrophils are the most common subset of leukocytes in the amniotic fluid. In patients with intraamniotic inflammation, cytokine concentration changes in the amniotic fluid are comparable between those with intraamniotic infection and those with sterile inflammation. Amniotic fluid concentrations of MMP-8 or IL-6 are excellent tests for the detection of inflammation and predict poor pregnancy and neonatal outcomes, particularly in preterm labor with intact membranes and PROM [2].

Mother: Maternal plasma concentrations of pyrogenic cytokines such as IL-6, are higher in patients with clinical chorioamnionitis. Previous studies have failed to demonstrate that C-reactive protein (CRP) is an effective early marker to detect chorioamnionitis [21].

Fetus: FIRS is defined as an elevated fetal plasma IL-6 concentration, which correlates with adverse outcomes. In such cases, funisitis and increased IL-6 levels in the umbilical cord are observed, and there is a higher incidence of neonatal sepsis [2,14]. The severity of funisitis correlates with fetal plasma IL-6 concentrations (an indicator of the severity of FIRS) and amniotic fluid IL-6 [14].

Maternal blood cultures and other microbiology results are not available straight away and become especially useful in the post-partum period to guide antibiotic therapy in case of sepsis.

How to recognize chorioamnionitis on the CTG?

When exposed to microbial invasion or non-infection-related stimuli, the fetus deploys an inflammatory response [19].

At an initial stage, the inflammatory response leads to an increase in metabolic rate which leads to an increase in fetal heart rate (FHR) [13]. Physiological guidelines have defined that this should be recognized by an increased baseline for gestational age or >10 % raise in baseline during labor [10]. As the baseline FHR is determined by the fine-tuning of the autonomic system, it is understood that a preterm baby is likely to have a baseline on the upper limit as the parasympathetic system is not yet fully developed. Conversely, a term baby is more likely to have a baseline on the lower limit of the normal range. Intrapartum FHR between 150 and 160 bpm at or after 40 weeks was shown to be associated with a higher incidence of labor complications, including maternal intrapartum pyrexia [22]. Sukumaran et al, in a retrospective study of placental confirmed chorioamnionitis, showed that all cases of maternal pyrexia were preceded by fetal tachycardia, defined as >10 % increase in baseline FHR over time, and in 43,8% of cases, the increase in baseline did not reach the 160 bpm cut-off [23]. Galli et al. also showed that tachycardia defined according to physiological guidelines was associated with lower APGARs, lower umbilical arterial pH and a fivefold increase in Neonatal Intensive Care Unit admission [11].

Peripheral vasoconstriction is a fetal compensatory mechanism to

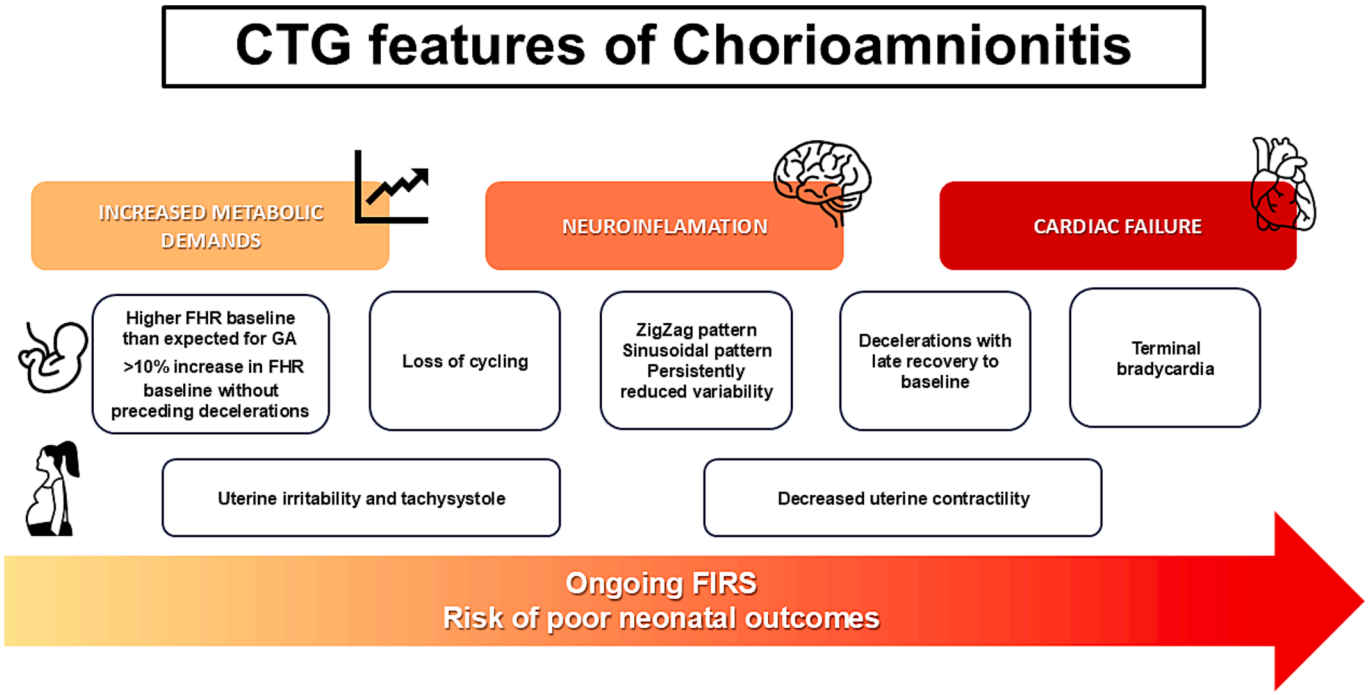

Fig. 1. CTG features associated with chorioamnionitis and progression of these changes during the fetal inflammatory response cascade. **Abbreviations:** CTG, cardiotocography; FHR, fetal heart rate; GA, gestational age; FIRS, fetal inflammatory response syndrome.

intrapartum hypoxia, redistributing blood from the periphery to the central organs such as the heart, brain, and adrenals. However, in the presence of chorioamnionitis, bacterial toxins, and inflammatory mediators induce vasoparalysis and peripheral vasodilatation, compromising this protective mechanism [13].

It is also important to note that the fetus has a poorly developed fetal blood–brain barrier and with the ongoing FIRS the integrity of the autonomic system is affected leading to a malfunction in the calibration between the sympathetic and parasympathetic influence on the FHR [13,19]. This is reflected on the CTG with loss of cycling at the initial stages. The progression of neurological dysfunction leads to changes in fetal variability with persistently reduced variability at a higher baseline, periods of increased variability (ZigZag pattern) and sinusoidal patterns [11,13,23,24]. Previous research showed that absence of cycling is associated with intrapartum maternal pyrexia and a higher chance of poorer perinatal outcomes measured by Apgar 7 at 5 min, despite no association with fetal acidosis [12]. Di Pasquo et al. described intrapartum CTG findings as suggestive of fetal inflammation (SOFI), defined by a persistent increase in the baseline FHR $>$ 10 % without any preceding decelerations, with or without any of the following: a) reduced variability; b) increased variability/ZigZag pattern; c) absence of cycling; d) sinusoidal pattern. These findings were associated with higher levels of IL-6 in the umbilical arterial blood. Changes in variability, attributed to neuroinflammation were associated with a further increase in IL-6 levels indicating worsening FIRS [25].

Decelerations are commonly seen in normal labor with good outcomes because they are the first compensatory mechanism to hypoxic stress in labor. In chorioamnionitis, decelerations can also be seen but, these are most typically decelerations with a slow recovery to the baseline developing after the onset of fetal tachycardia [11,23,24]. Yamaguchi-Goto et al. analyzed CTG traces from women with confirmed chorioamnionitis who had infants with cerebral palsy and demonstrated that 49 % were delivered following bradycardia and the majority of the remaining exhibited other CTG features like decreased variability, recurrent late decelerations, prolonged decelerations, sinusoidal pattern, and severe variable decelerations. These same features were present in the bradycardia group before it occurred [24].

Terminal bradycardia is a sign of heart failure that can occur in terminal stages of both inflammatory and hypoxic insults, with myocarditis and myocardial metabolic acidosis resulting in impaired contractility and cardiac arrest [13,19].

Discussion

The cascade of the fetal inflammatory response syndrome can be recognized on the CTG, allowing a much earlier recognition of chorioamnionitis, and giving clinicians the opportunity for a prompt intervention and better management of labor (Fig. 1).

In a woman with a term pregnancy, with intact or rupture of membranes, with or without fever, an increase in baseline FHR (higher than expected for gestational age or an increase above 10 % during labor) without preceding decelerations, the diagnosis of CTG chorioamnionitis should be considered. Changes in variability (reduced at a higher baseline or increased/Zigzag pattern) are likely to happen at later stages and are linked with neuroinflammation and higher levels of IL6 in the umbilical cord. Recently, Chandraharan et al. developed the “Chorio Duck Score”, to identify signs of chorioamnionitis and help clinicians in the process of decision-making and timely intervention [13].

Management must consider the general clinical picture, stage of labor, rate of progression, parity, presence of meconium and maternal fever. For example, in a primiparous woman in early stages of labor with meconium-stained fluid and an increase in baseline FHR, a cesarean section should be recommended. On the other hand, with similar CTG changes, a multiparous, 8 cm dilated, progressing without oxytocin most likely can have a safe vaginal delivery without the risks associated with cesarean delivery.

In the authors’ opinion, in the presence of persistent changes in variability (reduced at a higher baseline or increased/Zigzag pattern), delivery should be expedited, irrespective of parity or stage of labor. Regarding second stage of labor, the authors’ advocate for active management in view of recent evidence that in women with neuraxial analgesia, delayed pushing is associated with higher incidence of clinical chorioamnionitis and low umbilical cord pH, without changing mode of delivery [27].

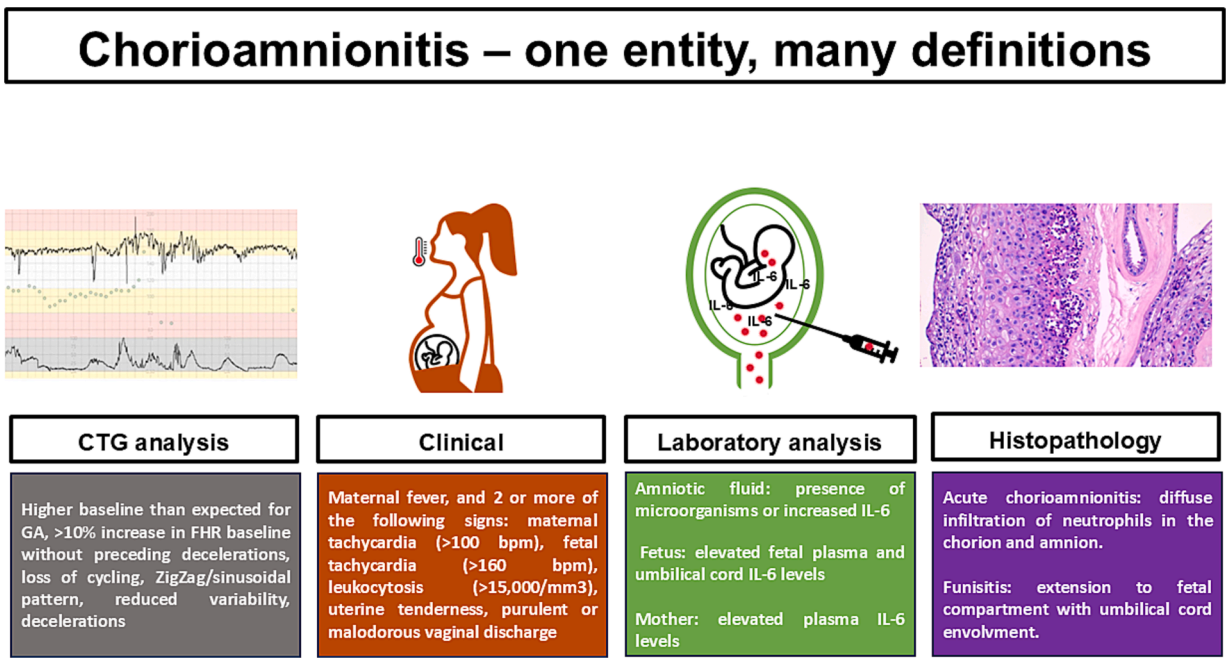

Fig. 2. Chorioamnionitis can be defined in different ways depending on the perspective it is approached: CTG analysis, clinical, laboratory analysis and histopathology. **Abbreviations:** CTG, cardiotocography; GA, gestational age; FHR, fetal heart rate; IL-6, interleukin-6.

In FIRS the metabolic demands are increased and the fetal capacity to tolerate hypoxia is reduced, with an exponential risk of encephalopathy when both conditions – inflammation and hypoxia- are present. Oxytocin increases the frequency, duration and strength of contractions, interrupting blood flow and reducing oxygenation time and should therefore be avoided in cases of chorioamnionitis.

Chorioamnionitis is an inflammatory response, not necessarily associated with infection. At the moment, there is not enough evidence to start antibiotics with the CTG diagnosis of chorioamnionitis, without maternal fever or other risk factors [26]. Further studies are needed to clarify the role of antibiotics in labor. In addition, the analysis of proinflammatory cytokines in amniotic fluid or in maternal blood, is promising to individualize the management of this patients.

Comparing with the other diagnostic perspectives, CTG is the only tool that clinicians have available to assess fetal well-being in real-time during labor (Fig. 2).

Clinical chorioamnionitis is based on the presence of fever, which is more often associated with epidural anesthesia rather than infection; conversely, chorioamnionitis has been shown to be present in women who do not develop intrapartum fever.

Histopathology has been classically considered the gold standard, but it is a late diagnosis, unavailable during labor management and only available days or even weeks after.

Analysis of amniotic fluid is promising, as it allows the differentiation between intra-amniotic inflammation and intra-amniotic infection. However, it has the disadvantages of an amniocentesis, cultures take several days and currently, there are no biochemical established cut-off values and their utility is based on concentration progression over time [19].

Future research should focus on the optimal interval between CTG changes and time to deliver, the value of antibiotics, and the role of inflammatory markers to predict maternal and neonatal outcomes.

Conclusion

CTG allows an earlier recognition of chorioamnionitis and the monitoring of the cascade of fetal inflammatory response in real-time. The increase in baseline FHR without preceding decelerations should be considered the first sign of inflammatory response, and changes in variability a sign of disease progression. Management depends on the overall picture including stage of labor, rate of labor progression and presence of meconium.

CRediT authorship contribution statement

Susana Pereira: Writing – review & editing, Writing – original draft, Validation, Supervision, Conceptualization. **Paulina Costa:** Writing – review & editing, Writing – original draft, Investigation. **Mohamed Oumar:** Writing – review & editing, Writing – original draft, Investigation. **Itziar Garcia-Ruiz:** Writing – review & editing, Writing – original draft, Supervision, Investigation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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