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The Intrapartum CTG Changes Associated with Chorioamnionitis and Maternal Sepsis: A Case Series

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ABSTRACT

Maternal sepsis is a preventable yet leading cause of maternal and neonatal morbidity and mortality. Infection-induced inflammatory changes during labor can alter fetal heart rate (FHR) patterns on cardiotocography (CTG), serving as early indicators of fetal compromise before clinical deterioration. This case series presents five women diagnosed with chorioamnionitis or maternal sepsis during labor, highlighting specific CTG changes and their correlation with maternal and neonatal outcomes. Across cases, recurrent abnormalities included fetal tachycardia, loss of cycling, reduced baseline variability, and recurrent decelerations, often preceding fever or laboratory confirmation. Predominant pathogens included Group B Streptococcus (GBS), Staphylococcus aureus, and Streptococcus pyogenes. Timely initiation of antibiotics and appropriate obstetric intervention yielded favorable maternal outcomes, with minimal neonatal morbidity. Findings suggest that CTG may serve as a non-invasive early-warning tool for sepsis-related compromise. Further prospective studies are warranted to validate CTG-based sepsis markers and integrate them into clinical practice.

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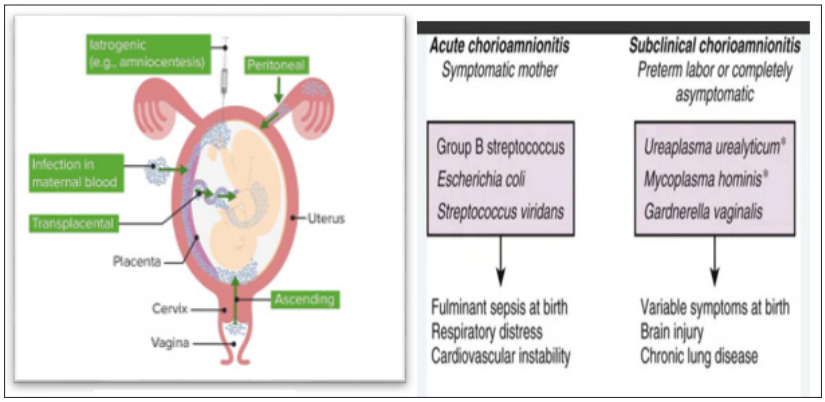
Introduction

Maternal sepsis remains a leading preventable cause of maternal and neonatal morbidity and mortality worldwide. Chorioamnionitis and systemic maternal infections are frequent intrapartum complications that may lead to sepsis and fetal compromise. Cardiotocography (CTG), widely used for intrapartum fetal surveillance, provides continuous assessment of fetal well-being but is often interpreted solely for hypoxic patterns. Recent studies suggest that infection-related CTG changes may precede overt clinical signs, serving as early indicators of fetal and maternal distress [1,2]. However, regional evidence remains scarce, particularly in the Middle East and North Africa (MENA) region. This study presents five intrapartum cases complicated by maternal infection or sepsis to characterize CTG alterations, clinical progression, and outcomes.



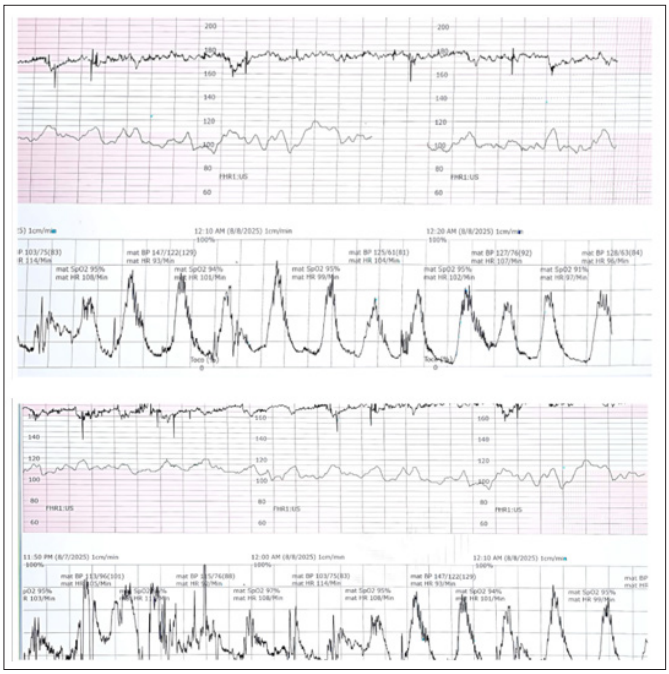
Transmission Causal Organism

Methods

This retrospective case series was observed at DAE Hospital and included five women who presented with intrapartum infection or sepsis. Maternal demographic data, obstetric history, laboratory investigations, microbiological findings, CTG patterns, and clinical outcomes were reviewed. CTG tracings were assessed for baseline FHR, variability, accelerations, and decelerations following the Royal College of Obstetricians and Gynaecologists guidelines [3]. Maternal laboratory parameters (white blood cell count, C-reactive protein, procalcitonin) and culture results were correlated with CTG changes. Management followed institutional protocols emphasizing early antibiotic therapy, supportive measures, and delivery planning based on maternal and fetal conditions.

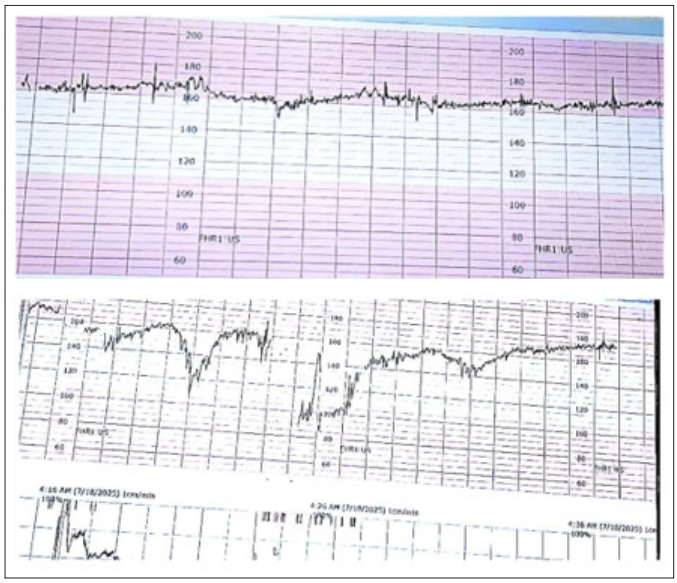
Results

Case 1



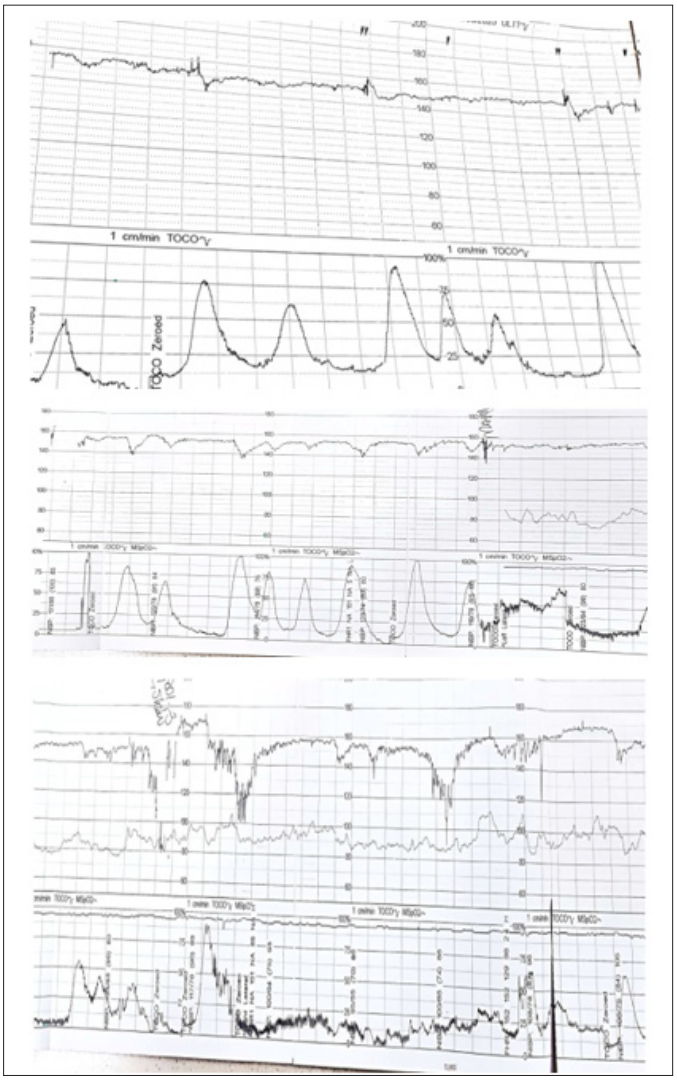
A 31-year-old primigravida at 40+6 weeks with diet-controlled gestational diabetes and GBS colonization presented with spontaneous rupture of membranes (SROM) for 20 hours. Labor was augmented but complicated by pathological CTG with fetal tachycardia and late decelerations. She developed intrapartum fever and underwent emergency cesarean section for fetal distress and failure to progress. She experienced combined atonic and traumatic postpartum hemorrhage (QBL 1767 mL) with preoperative hemoglobin of 12.0 g/dL. Treatment included methergine, carboprost, and misoprostol, followed by ICU monitoring and intravenous ceftriaxone with metronidazole for 48 hours. GBS was isolated on culture, and the patient recovered well.

Case 2



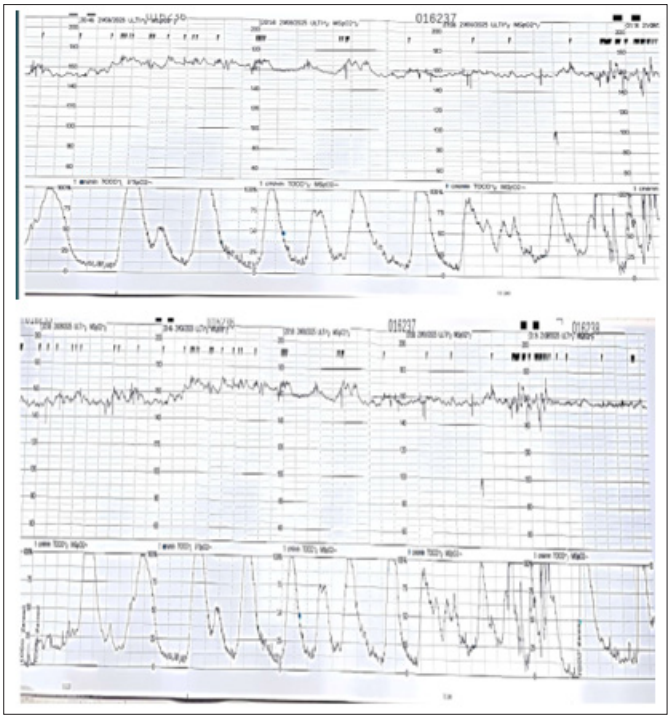
A 26-year-old primigravida with gestational diabetes was admitted for induction of labor at 39+1 weeks for a large-for-gestational-age fetus. After Propess and prostin induction, she developed fever (38.4°C) with pathological CTG showing recurrent decelerations. A Grade 1 cesarean section revealed acute chorioamnionitis without funisitis. Uterine swabs grew heavy GBS colonies. Laboratory results showed mildly elevated lactate (20.6 mmol/L) and CRP (20 mg/L). She was treated with intravenous antibiotics and had an unremarkable recovery, being discharged on day three.

Case 3



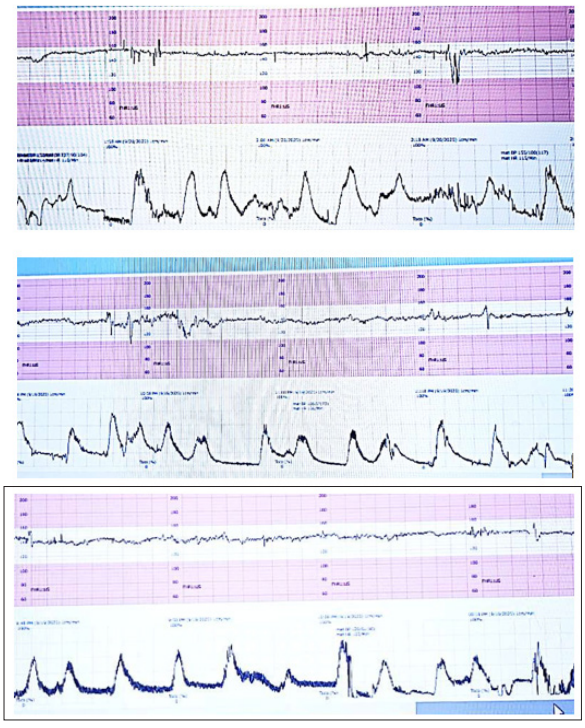
A 44-year-old G3P2 woman with an IVF pregnancy at 40+2 weeks presented with abdominal pain and SROM. CTG showed non-reactive tracing with reduced variability and fetal tachycardia (165–175 bpm). She developed maternal tachycardia (110–115 bpm) and low-grade fever (37.6°C). A Grade 1 cesarean section was performed. Investigations revealed leukocytosis (WBC 17.8→24×10⁹/L) and elevated CRP (53→393 mg/L). Staphylococcus aureus was isolated from fetal membranes, and fetal CRP improved from 44 to 7.1 mg/L after treatment. Both mother and neonate recovered following ceftriaxone and metronidazole therapy.

Case 4



A 31-year-old G5P4 woman at 37 weeks presented with high-grade fever (39.6°C), abdominal pain, and diarrhea. She was hypotensive (71/38 mmHg) and required noradrenaline infusion. Laboratory findings showed leukocytosis (14.6→29.8×10⁹/L), rising CRP (9.3→221 mg/L), and elevated procalcitonin (4.6→0.58 µg/L upon recovery). CTG revealed fetal tachycardia with intermittent late decelerations. She underwent augmentation and delivered vaginally. Blood cultures isolated Streptococcus pyogenes. She was treated with meropenem and supportive ICU care, recovering fully by postpartum day 14.

Case 5



A 29-year-old primigravida at 40+2 weeks was induced for reduced fetal movement and placental calcification. Two hours after CTG abnormalities, fetal tachycardia and variable decelerations, maternal fever (38.4°C) developed. An emergency cesarean section was performed for failure to progress and fetal distress. Umbilical cord was found wrapped around the neck and limbs. Laboratory findings showed WBC 21×10⁹/L and CRP 52 mg/L. Swabs grew GBS. Both mother and neonate recovered well following antibiotic therapy.

Across these five cases, fetal tachycardia and reduced baseline variability consistently appeared before maternal fever or laboratory confirmation. GBS was the predominant pathogen, though other organisms such as Staphylococcus aureus and Streptococcus pyogenes were also observed. Prompt initiation of antibiotics and timely obstetric interventions were associated with favorable maternal and neonatal outcomes. These findings highlight CTG as an early, non-invasive indicator of potential sepsis-related compromise.

Discussion

This series reinforces evidence that infection-related CTG abnormalities, particularly fetal tachycardia, reduced variability, and recurrent decelerations, may serve as early indicators of maternal infection and sepsis. The physiological mechanism involves maternal inflammatory cytokine release, leading to placental hypoperfusion and fetal hypoxia [4]. Inflammatory mediators can also cross the placenta, triggering a fetal inflammatory response and autonomic dysfunction reflected as altered CTG patterns [5]. The findings are consistent with Sukumaran et al. who reported similar associations between histologic chorioamnionitis and abnormal CTG traces [2].

Although CTG is a valuable screening tool, interpretation must be integrated with maternal clinical assessment and laboratory results. The overlap with non-infectious causes of fetal tachycardia, such as maternal dehydration or epidural analgesia, limits specificity. However, as demonstrated here, CTG changes often preceded clinical or laboratory confirmation of infection, suggesting potential for earlier recognition and intervention.

In the MENA region, limited research and underreporting of maternal sepsis hinder the development of evidence-based monitoring frameworks [6]. This case series emphasizes the importance of incorporating CTG findings into broader sepsis surveillance strategies, ideally within a multidisciplinary care model [7,8].

Conclusion

Intrapartum CTG abnormalities such as fetal tachycardia, reduced variability, and recurrent decelerations should prompt consideration of infection or sepsis when other etiologies are excluded. Timely antibiotic administration and clinical vigilance are critical to improving outcomes. Future research should aim to establish CTG-based diagnostic algorithms and prospective validation in larger populations.

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