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Feto-Maternal Tachyarrhythmias Complicating Pregnancy with Acute Pyelonephritis, Fetal Growth Restriction, And Oligohydramnios: A Case Report and Review of Literature

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Abstract

Background: Feto-maternal tachyarrhythmias are uncommon but clinically significant complications during pregnancy. They are frequently associated with maternal systemic infections, placental insufficiency, or underlying cardiac structural and electrical abnormalities. Prompt clinical recognition and a highly coordinated, multidisciplinary approach are vital to mitigating perinatal morbidity and optimizing maternal and neonatal outcomes.

Case presentation: We report the case of a 34-year-old unbooked Nigerian primigravida who presented late in the third trimester with symptoms of a severe urinary tract infection. She was diagnosed with acute pyelonephritis, which was uniquely complicated by concurrent persistent maternal sinus tachycardia and fetal tachyarrhythmia. The pregnancy was further compromised by co-existing uterine fibroids, oligohydramnios, and suspected fetal growth restriction.

The woman required prolonged inpatient management involving intravenous antibiotic therapy, close cardiology co-management with rate-limiting agents, and intensive fetal surveillance. Due to worsening fetal parameters and a non-reassuring biophysical profile at term, an emergency caesarean section was performed. This resulted in the delivery of a live male neonate with low birth weight but excellent Apgar scores.

Conclusion: This case highlights the complex pathophysiological interplay between maternal focal infections, systemic inflammatory cardiovascular stress, placental insufficiency, and fetal rhythm disturbances. It highlights the critical need for vigilant cardiotocographic and Doppler monitoring, alongside aggressive multidisciplinary intervention, in pregnancies complicated by simultaneous feto-maternal tachyarrhythmias.

Keywords: fetal tachyarrhythmia; maternal tachycardia; acute pyelonephritis; fetal growth restriction; oligohydramnios; pregnancy complications

Introduction

Feto-maternal tachyarrhythmias are uncommon but clinically significant complications during pregnancy. They are frequently associated with maternal systemic infections, placental insufficiency, or underlying cardiac structural and electrical abnormalities [1]. Cardiac rhythm disturbances during pregnancy present unique diagnostic and therapeutic challenges [1, 2]. Managing these conditions requires balancing gestational physiological cardiovascular adaptations against the potential teratogenic or adverse haemodynamic risks that antiarrhythmic therapies pose to the fetus [2]. Maternal tachyarrhythmias are typically secondary to underlying cardiac pathology, but they can also be precipitated by acute systemic infections, severe anemia, or endocrine disruptions [3].

Conversely, fetal tachyarrhythmias are rare, occurring in less than 1% of pregnancies. However, they carry a high risk of severe complications, including hydrops fetalis, intrauterine growth restriction, and perinatal death [4]. The simultaneous occurrence of both maternal and fetal tachyarrhythmias is an exceptional clinical scenario. It usually points to shared precipitating factors, such as infection-mediated maternal fever, increased circulating catecholamines, transplacental inflammatory cytokines, or severe uteroplacental dysfunction [5]. Acute pyelonephritis

remains a leading cause of non-obstetric antepartum hospitalization. It is well-documented to cause severe maternal morbidity and precipitate adverse fetal events, including preterm labour, chorioamnionitis, and acute fetal distress [6].

Maternal tachyarrhythmias complicate approximately 1% to 2% of all pregnancies, with sinus tachycardia being the most frequently documented rhythm disturbance [3]. The maternal cardiovascular system in pregnancy is already in a high-output state. It is highly sensitive to infection-related systemic inflammation, endotoxemia, and increased sympathetic drive, all of which lower the threshold for tachyarrhythmias [7].

Acute pyelonephritis affects roughly 1% to 2% of pregnant patients, predominantly in late pregnancy. It carries a significant risk of maternal sepsis, acute respiratory distress syndrome (ARDS), and low birth weight secondary to either preterm delivery or growth restriction [6,8].

Fetal tachyarrhythmia is clinically defined as a sustained fetal heart rate (FHR) exceeding 160 beats per minute. While often idiopathic or driven by intrinsic cardiac conduction defects like re-entrant supraventricular tachycardia (SVT), it can be triggered by extrinsic stressors [4,9]. These include maternal hyperthermia, localised or systemic intrauterine infection, transplacental passage of chronotropic drugs, and hypoxia [9].

When combined with chronic uteroplacental insufficiency, manifested as fetal growth restriction and oligohydramnios, the fetus has very little reserve. This increases its vulnerability to hypoxia-induced autonomic imbalances and abnormal heart rate patterns [10]. Additionally, the presence of uterine fibroids during pregnancy can disrupt local placental perfusion, exacerbate growth restriction, and significantly increase the likelihood of operative obstetric delivery [11].

This case report outlines the intricate management of an unbooked primigravida where acute pyelonephritis coexisted with dual feto-maternal tachyarrhythmias and underlying placental insufficiency.

Case presentation

A 34-year-old unbooked Nigerian primigravida presented to our tertiary facility at an estimated gestational age of 33 weeks and 4 days, calculated from an early first-trimester dating ultrasonography. She gave a one-week history of progressive, severe dysuria and frequency. Notably, she reported no subjective fever, haematuria, abnormal vaginal discharge, or drainage of liquor.

On physical examination, the patient appeared acutely ill and anxious, though she was anicteric and well-hydrated. Her initial vital signs revealed a marked tachycardia with a regular pulse rate of 122 bpm and a elevated blood pressure of 130/80 mmHg. Her respiratory rate was 20 breaths per minute, and she was afebrile at presentation (temperature: 36.8°C). Abdominal examination revealed a symphysis-fundal height (SFH) of 28 cm, which was significantly discordant with her dates, suggesting fetal growth restriction. The uterus was irritable, and a notable, non-tender nodular mass measuring approximately 6 cm by 4 cm was palpated on the left fundal aspect, consistent with uterine fibroids. Marked bilateral renal angle tenderness was elicited upon percussion.

Urinalysis demonstrated significant pyuria and nitrituria, and a presumptive diagnosis of acute pyelonephritis in an unbooked primigravida with fetal growth restriction and uterine fibroids was made. The woman was admitted for inpatient care. Empirical intravenous antibiotic therapy was initiated with ceftriaxone (1 g every 12 hours) aligned with institutional protocols for severe antepartum urinary tract infections [6]. Urine culture later confirmed *Escherichia coli* sensitive to third-generation cephalosporins.

An urgent obstetric ultrasonography and Doppler assessment were performed. The scan confirmed a viable singleton fetus in a cephalic presentation. However, it revealed a sustained, regular fetal tachyarrhythmia with a baseline fetal heart rate fluctuating between 174 and 185 bpm, without signs of fetal hydrops. Umbilical artery doppler indices showed an elevated systolic/diastolic (S/D) ratio and a high pulsatility index, falling near the 95th percentile for gestational age, indicating increased placental vascular resistance.

Because the maternal tachycardia (118–126 bpm) persisted despite aggressive intravenous hydration, antipyretics, and targeted antibiotic therapy, a cardiology consultation was requested. A 12-lead electrocardiogram (ECG) confirmed sinus tachycardia with no ischaemic changes, pre-excitation syndromes, or structural conduction defects. An echocardiogram revealed a structurally normal heart with a hyperdynamic left ventricle (ejection fraction: 68%).

To control maternal heart rate and optimize diastolic filling time, the cardiology team initiated oral beta-

blocker therapy with metoprolol (25 mg every 12 hours), which is considered safe for gestational use under close surveillance [12].

Over the next few weeks of her admission, the patient showed significant clinical improvement. Her urinary symptoms resolved, her inflammatory markers normalized, and her maternal heart rate stabilized within a reassuring range of 84 to 92 bpm. Following maternal rate control and the resolution of the pyelonephritis, the fetal heart rate gradually decelerated from its tachyarrhythmic peaks, stabilizing at a high-normal baseline of 155 to 160 bpm.

However, serial obstetric growth scans and fetal surveillance revealed a deteriorating intrauterine environment. By 37 weeks, repeat ultrasonography demonstrated severe oligohydramnios, with an amniotic fluid index (AFI) of 4.2 cm. Estimated fetal weight fell below the 3rd percentile for gestational age, confirming severe fetal growth restriction [10].

Due to the worsening oligohydramnios and non-reassuring umbilical artery doppler indices, the multidisciplinary team opted for a planned delivery via emergency cesarean section at 38 weeks and 3 days of gestation, avoiding the stress of prolonged labor induction on a compromised fetus.

A live male neonate weighing 1.8 kg (very low birth weight for term) was successfully delivered. The neonate demonstrated excellent immediate extrauterine adaptation, with Apgar scores of 7 and 10 at 1 and 5 minutes, respectively. The amniotic fluid was scant but clear.

The mother's postoperative recovery was uneventful. Her cardiovascular status remained stable, and her metoprolol dose was progressively tapered and discontinued by the fifth postoperative day under cardiology guidance. Both mother and neonate were discharged home in stable condition on the seventh postoperative day, with scheduled outpatient follow-ups in the neonatology, cardiology, and postnatal clinics.

Discussion

This case illustrates the complex relationship between maternal localized infections, maternal-fetal hemodynamics, and placental dysfunction. Acute pyelonephritis is a potent trigger for systemic inflammatory cascades. The release of bacterial endotoxins and endogenous pyrogens drives a hypermetabolic state, marked by maternal sinus tachycardia [6,7].

In this woman, the persistent maternal tachycardia likely triggered a corresponding fetal tachyarrhythmia. This was likely driven by a combination of transplacental inflammatory cytokines, maternal catecholamine transfer, and borderline fetal hypoxia caused by pyelonephritis-induced uterine irritability [5,9].

What makes this case particularly complex is that these acute infectious and arrhythmic stressors occurred alongside chronic placental insufficiency, as evidenced by the severe growth restriction and oligohydramnios [10]. The elevated umbilical artery Doppler indices indicated poor placental perfusion. This significantly reduced the fetus's ability to tolerate acute cardiovascular stress. Furthermore, the presence of the uterine fibroids may have compounded this issue, potentially altered local myometrial blood flow and contributed to the restricted fetal growth [11].

Early, decisive collaboration between obstetricians, cardiologists, and neonatologists was central to the successful outcome of this pregnancy. Aggressive antibiotic therapy eliminated the primary infectious trigger, while targeted beta-blockade using metoprolol effectively managed maternal sinus tachycardia without causing adverse fetal bradycardia or conduction blocks [12].

Ultimately, realising that the fetus was in a compromised state with no reserve, marked by severe oligohydramnios and persistent growth restriction, justified timely delivery via caesarean section, preventing intrauterine demise [10].

Conclusion

Feto-maternal tachyarrhythmias are rare but serious complications in obstetrics, especially when they occur alongside acute maternal infections and underlying placental insufficiency. This case underlines the importance of a high index of clinical suspicion, immediate source control for maternal infections, and meticulous fetal surveillance using serial Doppler and fluid volume assessments. Managing these complex high-risk pregnancies through a synchronised, multidisciplinary approach is essential to achieving positive maternal and neonatal outcomes.

Declarations

Author contribution

MEN, ACI, GUE and ROE were involved in conceptualisation, manuscript writing. JJ, BON, EIO, HCU, DUN, CO, CGO and ACE participated in

manuscript revision. All authors approved the final copy for submission to the journal.

Ethical approval

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Informed Consent

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Data availability statement

Not applicable

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