Case Report

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A case of sustained fetal atrial flutter at 25-week gestation: diagnostic challenges and therapeutic strategies

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Introduction: Sustained fetal tachycardia is an uncommon phenomenon in gestations (approximately 0.1%). Atrial flutter (AF) accounts for 10–30% of fetal tachyarrhythmias, which is characterized by a rapid atrial rate estimated at 300–600 beats per minute, associated with variable degrees of atrioventricular conduction.

Case presentation: A 29-year-old asymptomatic woman, pregnant for the third time, was diagnosed with a male fetus at 25 weeks gestation exhibiting sustained atrial flutter with rapid ventricular response (2:1 and 1:1 AV block). Maternal digoxin reduced the fetal ventricular rate to 120 bpm, mitigating hydrop risk. The infant was delivered via cesarean at 35 weeks, presenting with low blood pressure (80/50 mmHg) and an irregular pulse (160 bpm). The electrocardiogram showed AF; intravenous amiodarone was administered, and the rhythm successfully converted to a normal sinus rhythm.

Clinical discussion: Fetal arrhythmias in pregnant women require detailed assessment and treatment, including maternal history, electrocardiogram, and renal function assessment. The approach to treatment involves the use of transplacental antiarrhythmics, where digoxin is considered the first line of treatment. Other options include sotalol and flecainide. The mother should be monitored for side effects, with follow-up in the postpartum period for the infant.

Conclusion: Fetal AF may occur in the second trimester and requires an increased awareness of this life-threatening arrhythmia. Whatever the gestational age, early recognition of fetal tachycardia is important due to the potential for adverse and life-threatening outcomes.

Keywords: amiodarone, case report, digoxin, fetal atrial flutter, rapid ventricular response

Introduction

Fetal arrhythmias indicate that the fetus has an abnormal heart rate or rhythm; they are classified into tachycardia and bradycardia or can be a combination of them[1,2]. They can be benign and do not affect the fetus or may be dangerous and lead to life-threatening consequences such as atrioventricular block, supraventricular or ventricular tachycardia (SVT or VT), and atrial flutter (AF). Fetal tachycardia is defined when the heart rate is >180 beats per minute (bpm); it can be sustained (when it lasts >50% of the examination time) or intermittent[3]. SVT (66%) and AF are the most common tachycardia types; many other types, such as permanent junctional reciprocating tachycardia and atrial ectopic contractions, can be found in rare cases[2,4]. AF, which can be a lethal condition, forms one-third of tachyarrhythmias; it is diagnosed when the atrial rate is 300–600 bpm with diverse atrioventricular conduction block, resulting in a slower ventricular rate of 220–240 bpm[5-7]. It can be found in fetuses with congenital heart malformations, myocarditis, and SSA autoantibodies, after surgeries, or in structurally normal hearts[5,7,8]. Many life-threatening complications can appear in fetuses with AF, such as hydrops, fetal heart failure, neurological defects, and death[5,9]. Therefore, rapid diagnosis and treatment are crucial. This paper describes a case of a fetus with AF that was discovered at 25 weeks of gestation during a routine check-up without any obvious causes and aims to explain the pathophysiology of this arrhythmia, diagnostic approaches, its prognosis, and the different therapeutic strategies suggested, hoping to increase awareness of these cases.

Case presentation

A 29-year-old pregnant woman came to an obstetrics and gynecology specialist at 25 weeks gestation for a routine check-up. The doctor noted an abnormal fetal heart rhythm and referred the patient to our specialist clinic. The patient came to our clinic without any symptoms or signs and did not observe any unusual findings during the whole pregnancy. The patient was pregnant for the third time without any complications in the recent or previous pregnancies. She underwent two caesarean sections before. The medical, drug, allergic, and psychosocial histories were within the normal range. Vital signs of our patient showed a low blood pressure of 100/70 mmHg; other findings were within the normal range. The physical examination of the other systems was unremarkable. The laboratory investigations were

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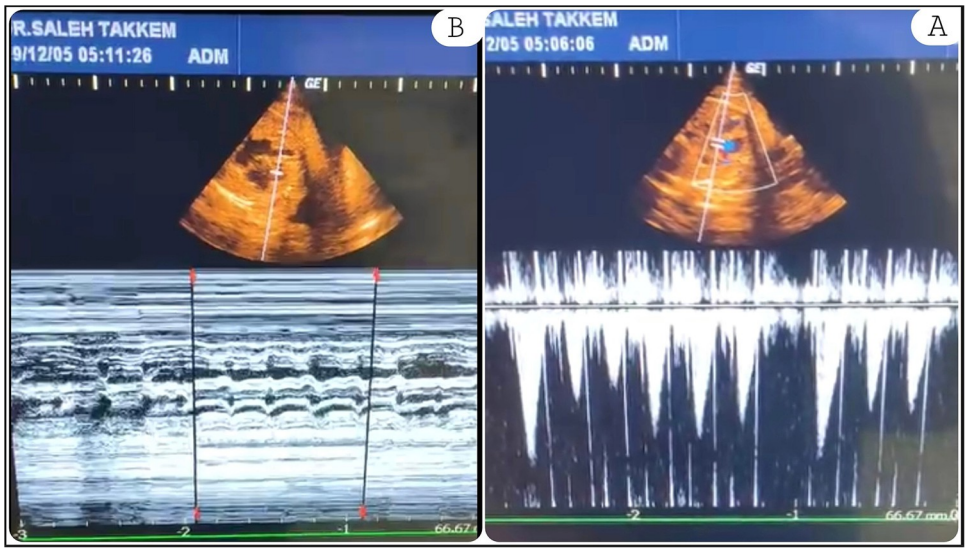

Figure 1. (A) Doppler imaging of the left ventricular outlet in the fetus shows irregular waves consistent with responsive atrial flutter with altered transmission. (B) M-mode image showing the speed of atrial flutter.

normal except for a low haemoglobin rate of 10.5 g/dl. The ultrasound imaging scan revealed a male fetus at 25 weeks of gestation, suffering from sustained atrial flutter with rapid ventricular response with a 2:1 alternating with 1:1 atrioventricular conduction (AVC) block, an atrial rate of 350 bpm, and a ventricular rate of 170 bpm (Fig. 1). The fetus was at risk of hydrops because of the low heart output. Oral digoxin was administered to the mother to control and adjust the speed of the fetus' flutter response until delivery, where a loading dose of 0.5 mg for two days was followed by 0.25 mg every day (the digoxin serum level was evaluated every week). As a result, the mean ventricular response in the fetus lowered to 120 bpm. The child was born at 35 weeks of gestation by caesarean section because of gynecological reasons and two previous caesarean sections. The child was male, weighing about 3,000 g. The Apgar score in the first and the fifth minutes was 10. The newborn who was diagnosed with AF was admitted to the neonatal intensive care unit for further cardiac evaluation. His vital signs were abnormal (low blood pressure of 80/50 mmHg and irregular pulse of 160/bpm). All of his laboratory tests were normal. The echocardiography scan showed the presence of patent ductus arteriosus and patent foramen oval, measuring 2 mm, which were completely diapered after a month of birth. The electrical cardiography (ECG) of the newborn revealed AF with a narrow QRS, an atrial rate of 350 bpm, and a ventricular rate of 170 bpm (Fig. 2). Therefore, he was treated with amiodarone intravenously for 24 hours, where the loading dose was 5 mg/kg IV over 30–60 min, and the maintenance dose was 0.005 mg/kg/min IV infusion. Immediately after the treatment, the neonatal heart converted to a normal sinus rhythm, and the heart rate lowered to 110 bpm. The newborn was discharged from the hospital on the 22nd day of life in a good state. In addition, amiodarone was also given for a month to prevent recurrence with a serum level of 5 mg/kg/day. The follow-up for two years was uneventful without any recurrence of AF.

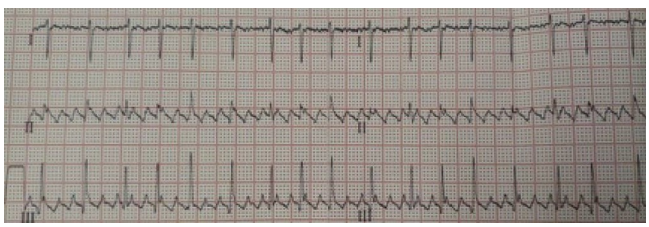

Figure 2. The ECG of the newborn after delivery shows atrial flutter waves (sawtooth).

Discussion

Fetal arrhythmias occur in 1–2% of pregnancies. It can be classified into three main categories: (1) irregular rhythms with a normal fetal heart rate (FHR) due to premature beats or conduction abnormalities; (2) tachyarrhythmias (FHR > 180 bpm); and (3) bradyarrhythmias (FHR < 110 bpm)[10] (Fig. 3). Sustained fetal tachycardia is an uncommon finding in pregnancies (approximately 0.1%)[8]. It can be classified based on the heart's electrophysiology into (1) sinus tachycardia (ST), (2) atrial tachycardia (which includes AF and atrial ectopic tachycardia), (3) conduction system tachycardia, and (4) VT (Fig. 3); this classification plays a significant role in clinical practice. In addition, it can be simplified into three categories: ST, SVT (containing atrial and conduction systems), and VT[3]. AF and SVT are the two most prevalent types of fetal tachyarrhythmia[11], while VT is the rarest[3]. AF represents 10–30% of fetal tachyarrhythmias and is characterized by a rapid atrial rate of approximately 300–600 bpm, associated with variable degrees of AVC like 2:1, 3:1, or 4:1, resulting in a slower ventricular rate of 220–240 bpm, because the ventricles are unable to match the extremely rapid atrial rates in a 1:1 fashion[6]. Many studies indicate that AF typically manifests in the third trimester, likely due to the enlarged atrial size observed in 27–30 weeks of gestation, making it more susceptible to atrial extrasystoles. In addition, they suggest that before the 30 weeks of gestation, the atrial was small

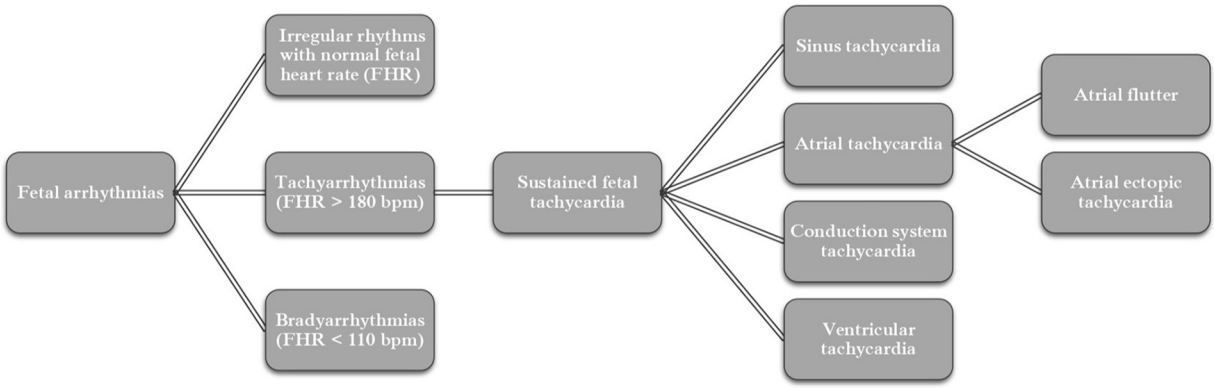

Figure 3. Classification of fetal arrhythmias and sustained fetal tachycardia.

and immature, so it could not preserve a sustained atrial fetus macroreentrant circuit[3,7]. In our case, AF was detected in the 25th week of gestation. AF can be a result of congenital heart malformations (which include atrioventricular septal defect, Ebstein's malformation, hypoplastic left heart syndrome, and pulmonary atresia), myocarditis, or SSA autoantibodies[7,8]. In addition, AF may arise secondary to iatrogenic causes like cardiac surgery performed within atria or in fetuses with structurally normal hearts[5]. Sustained fetal tachycardia may lead to significant complications, including fetal heart failure, nonimmune hydrops, and polyhydramnios. On the other hand, mirror syndrome, also referred to as Ballantyne's syndrome, may progress in a pregnant woman as a maternal complication with sustained fetal tachycardia (it includes fetal hydrops and maternal preeclampsia)[3]. One of the most important complications is hydrops fetalis (HF)[4]. Many risk factors encourage HF, such as tachyarrhythmia, characterized by rapid and incessant heart rhythm, occurring at a younger gestational age[9]. HF may lead to severe and harmful consequences like long-term neurological complications and fetal demise (about 35% of intrauterine mortality)[2]. Due to the limited availability of noninvasive fetal electrocardiography, like fetal magnetocardiography (FMCG), which is until now a very expensive method, the assessment of antenatal rhythms predominantly depends on the sequence of atrial and ventricular systolic mechanical events observed via echocardiography, which includes M-mode and Doppler techniques[1,3,12]. The use of M-mode imaging to record atrial and ventricular systolic wall motions, in combination with Doppler cardiac ultrasound/echocardiography to evaluate the timing and sequence of blood flow events associated with atrial and ventricular contractions, provides effective tools for assessing anatomical structure and rhythm in the fetal heart, ultimately enabling the specialist in the diagnosis of arrhythmias[1,3]. Postnatally, the diagnosis of cardiac rhythm depends on ECG[3]. Newborn ECG shows rapid, irregular atrial activity (280–500 bpm), characterized by the presence of an atrial flutter wave (referred to as a "sawtooth" appearance) in leads II, III, aVF, and V1[5]. It is recommended that any pregnant woman and her fetus suffering from FA undergo a comprehensive evaluation including maternal cardiac history, physical examination, review of maternal medications (with a focus on potential QTc prolonging agents like antiemetics and antibiotics), baseline ECG, assessment of renal and hepatic function, electrolyte levels, and consultation with an adult cardiologist[8]. The successful management of fetal tachycardia depends on the determination of FHR and type of arrhythmias, then giving the mother the transplacental drugs immediately[2,3]. The main objectives of treatment are to either suppress the arrhythmia or, if unsuccessful, to decrease the ventricular rate[1]. The fetal arrhythmia may be corrected by antiarrhythmic drugs, transesophageal pacing, or electrical cardioversion, which is especially helpful if conventional pharmacotherapy is ineffective. Delivery is considered a good choice in near-term fetuses, where pharmacological treatments are contraindicated[3,5]. Antiarrhythmic drugs can be used in transplacental (given to the mother orally or intravenously) or through the umbilical cord in the case of HF[3]. The optimal management of fetal tachycardias remains uncertain, leading to a lack of standardised clinical guidelines. Consequently, a series of treatment protocols have been reported in the literature, such as digoxin, flecainide, sotalol, amiodarone, verapamil, and propafenone[2,8,13]. Digoxin is the most widely used first-line medication because of its safety, long-term employment in pregnancies, widespread use in medical centres, and its ability to decrease the heart rate. In addition, sotalol and flecainide were administered as second- and third-line therapy[2,8]. However, the optimal serum level target for digoxin remains uncertain, where the widely recognised clinical practice range for digoxin target levels is 1.0 to 2.0 ng/mL, primarily due to concerns about potential toxicity at higher levels[14]. In contrast, some researchers and studies encourage using flecainide or sotalol as first-line medications[2,8]. Amiodarone is recommended for fetal arrhythmias only as a third-line therapeutic option due to its toxic side effects and inability to cross the placenta as readily as does sotalol[8,15]. The optimal pharmacological intervention for fetal tachycardia with hydrops is still controversial in the literature. Several studies have raised concerns regarding the efficacy of digoxin as a standalone treatment for HF due to its poor placental transfer. Conversely, some studies have indicated that sotalol is a successful medication for fetal AF, where a high fetal-to-maternal ratio allows sotalol to completely cross the placenta[16]. The therapeutic protocol should be continued until the baby is born[2]. Also, prophylaxis treatment should be given for a few months after delivery to prevent relapse; after infancy, there is no need for any treatment. Brain ultrasound imaging must be done after delivery to assess the newborn's brain and rule out hypoxic changes[5]. Minor symptoms like nausea, dizziness, or vomiting have been reported as maternal adverse events of antiarrhythmic drugs. Despite being rare, severe complications of medication

administration have also been described, including Mobitz type II AV block after combination therapy involving sotalol and digoxin and maternal atrial fibrillation following flecainide treatment, both of which resolved spontaneously after discontinuation of the therapy, which emphasises the importance of careful maternal monitoring[8].

Conclusion

Fetal AF is an unexpected finding during the second trimester of gestation. Therefore, this paper aims to emphasize the importance of having a high suspicion of this serious rhythm disorder in the fetus that comes with tachycardia regardless of gestational age, because of its adverse and life-threatening consequences. In addition, early diagnosis and intrauterine treatment can control the AF response and reduce morbidity and mortality associated with complications arising from AF like hydrops and neurological defects.

Ethical approval

Ethics clearance was not necessary since the university waives ethics approval for publication of case reports involving no patients' images, and the case report does not contain any personal information. The ethical approval is obligatory for research that involves human or animal experiments.

Consent

We have obtained written informed consent from the parents (the legally authorized representative) for the publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author's contribution

A.Q.: writing – review & editing, writing – original draft, data curation. K.T., A.S.S.: Writing – review & editing, Writing – original draft. S.T.: writing – review & editing, data curation, supervisor. A.Q.: submitted the final manuscript. All authors read and approved the final manuscript.

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Data availability statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Methods

The work has been reported in line with the SCARE 2023 criteria[17].

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used ChatGPT in order to paraphrase some sentences. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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