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CASE REPORT

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Successful management of fetal atrial flutter at term pregnancy with postnatal electrocardioversion

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Abstract

Fetal atrial flutter is a lethal tachyarrhythmia with a 10% mortality rate. Diagnosis is made with echocardiography, and management should be multidisciplinary with obstetricians, fetal cardiologists, and specialist neonatologists.

KEYWORDS

obstetrics and gynaecology, cardiovascular disorders

1 | INTRODUCTION

Fetal atrial flutter (AF) is an uncommon condition accounting for about 30% of all fetal tachyarrhythmias. It is associated with structural heart anomalies and hydrops, with 10% fetal mortality rate. This case demonstrates a successfully managed atrial flutter at term with postnatal electrocardioversion using a multidisciplinary team (MDT) approach.

It is a serious and threatening form of tachyarrhythmia, and associated with fetal mortality and neurological complications. The incidence of fetal tachyarrhythmia in less than 1% of pregnancies, and the two commonest forms are AF (10%-30%) and supraventricular tachycardia (SVT) (66%-90%).[1]

Fetal AF is characterized by a rapid regular atrial contraction (300-600 beats/minute).[2] This makes the ventricles unable to respond to this rapid speed in a 1:1 fashion leading to a 2:1 or variable atrioventricular (AV) block. It commonly develops in the third trimester and should not be confused with fetal tachycardia or distress. The proposed underlying mechanism causing fetal AF is the re-entrant circuit causing premature atrial impulses.[3] Diagnosis is mainly by fetal echocardiography which demonstrates the cardiac rhythm best with M-mode function, while also assessing any features of cardiac compromise or associated structural anomalies or changes. The outcome of fetal tachyarrhythmia is dependent on the presence of hydrops and cardiac disease, and not the type of tachyarrhythmia.[4] Treatment is individualized with factors like gestational age, structural heart problems, and hydrops taken into significant consideration. Early detection and treatment improve clinical outcome significantly.[5]

We present a case of fetal atrial flutter in a 24-year-old low-risk pregnancy at 37 weeks gestation. There were no fetal hydrops or associated cardiac disease, and she had an urgent caesarean section. Neonatal sinus rhythm was restored with single electrocardioversion and some course of digitalization.

2 | CASE REPORT

A 24-year-old primigravida booked low risk in a different hospital. Her booking blood results were normal with hemoglobin level of 144 g/L, and she had a normal anomaly scan at 20 weeks gestation. She had regular uneventful antenatal midwife-led care and had no significant past medical history of note. At each clinic visit, the fetal heart rate (FHR) was normal in keeping with gestational age.

At 37 weeks gestation, she attended her routine antenatal care, also with first episode of reduced fetal movement. There was difficulty in detecting the FHR both by the hand-held

fetal Doppler and cardiotocographic (CTG) machine as the heartbeat was too fast. A quick bedside ultrasound scan showed FHR of 220 beats per minute (bpm). The maternal pulse was 100bpm with blood pressure of 128/76 mmHg. She was quickly transferred by ambulance to the Obstetric Unit of the nearest general hospital. A quick transabdominal ultrasound scan done by the Obstetric team showed live active singleton gestation with ventricular FHR of 260 bpm using pulse wave doppler (PW), and an impression of fetal SVT was made. The maternal temperature was normal, and other noncardiogenic causes of fetal tachycardia were ruled out. There was a discussion to go for emergency cesarean section; however, this was forestalled by specialist advice from the pediatric cardiologist in a tertiary hospital who advised for the patient to be transferred by ambulance to the University teaching hospital for specialist review and to provide adequate neonatal care after delivery.

On arrival at the tertiary hospital 2 hours later, obstetric ultrasonography showed estimated fetal weight compatible with her gestation, with normal liquor volume and normal anterior placentation. Fetal echocardiography done by the fetal cardiologist revealed an atrial heart rate of 480bpm and ventricular rate (VR) of 240bpm using the M-mode function. There was a mild tricuspid valve regurgitation, but no obvious cardiac structural anomaly, atrial enlargement, or hydrops noted. A clinical impression of fetal AF at 480bpm with 2:1 nodal AV block was made. There was a quick MDT discussion between the fetal cardiologist, on-call neonatologist, and the obstetric team. Options of management and their risks and benefits were discussed with the patient including antenatal medical treatment with drugs like digoxin and sotalol, or abdominal delivery with neonatal management. The high VR of 240bpm and the potential risk of developing imminent hydrops were discussed. The patient was not keen on medical management, and since the pregnancy was term, the team agreed on abdominal delivery and postnatal treatment to which the patient consented. She had an urgent cesarean section under regional block. The outcome was a live female neonate who weighed 3530 g at birth with favorable Apgar score although with initial poor color. The cord gases (PH) were normal.

The baby required some continuous positive airway pressure (CPAP) for support for a few minutes as the oxygen saturation was 70% and was transferred immediately to the neonatal intensive care unit (NICU). Electrocardiography (ECG) done showed ventricular heartbeat of 235bpm (Figure 1), with persistent AF. A single synchronized direct-current cardioversion of 4 joules following ketamine sedation reverted the arrhythmia to sinus rhythm at 175 bpm. Loading dose of digoxin was started (15 mcg/kg) followed later by maintenance dose of 10 mcg twice daily with serum digoxin level monitored. A neonatal echocardiography done on the day of delivery showed a patent foramen ovale (PFO) and mild tricuspid regurgitation with otherwise normal ventricular function.

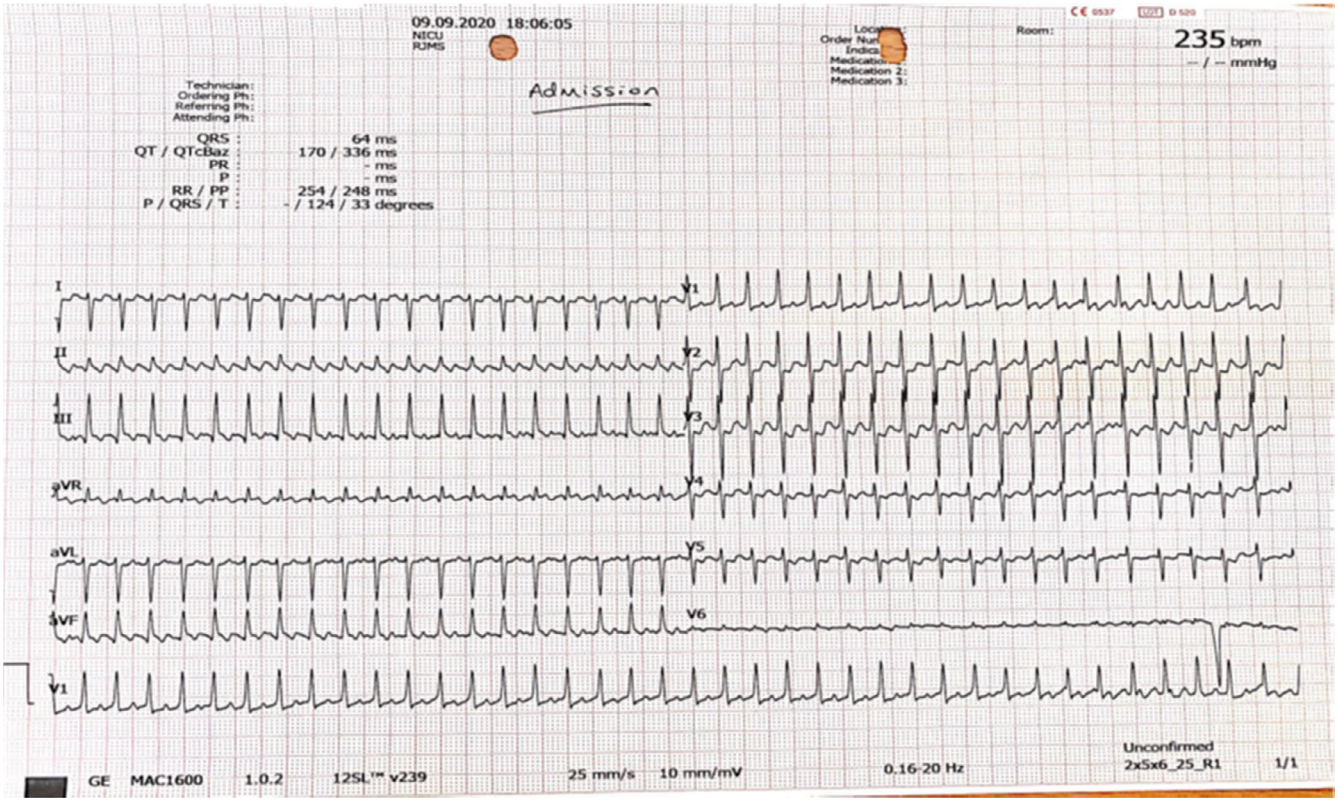

FIGURE 1 Postnatal ECG precardioversion showing the neonate still in Atrial flutter with the ventricular heartbeat rate of 235 bpm

The baby's mother had a good postoperative recovery and was well debriefed with follow-up plan made. There was no neonatal recurrence of AF while on admission, and on day 4 neonatal life, the baby was discharged back to the NICU of the general hospital where she was monitored for 3 days. She was then discharged home on same dose of digoxin for prophylaxis, and the last serum digoxin level before discharge was normal at 1.5 μg/L. Follow-up echocardiogram done 8 weeks after discharged showed similar findings as before with normal ventricular function. The baby was to be followed up every few months with an echocardiogram and review, and there has been no concern so far. Her development has been normal up to the point of writing this article at the age of 4 months.

3 | DISCUSSION

Fetal heart rate monitoring remains an important part of antenatal care, and as seen in our case, the tachycardia was first detected by routine fetal heart rate check.[5] Fetal AF accounts for roughly 25-30 of all fetal tachyarrhythmias and is associated with variable AV conduction. There are congenital structural anomalies that may occur with AF including hypoplastic left heart syndrome, atrioventricular septal defect, pulmonary atresia, and Ebstein's anomaly. Both SVT and fetal AF have similar incidence of hydrops fetalis averaging 40% and similar overall mortality rate of 10%. The mortality rate in fetal AF with hydrops, however, may be up to 30%. Studies have shown that hydropic fetuses with fetal AF have higher VR than the nonhydropic ones, but no difference in the atrial rates.[6]

Most cases of fetal AF occur in the third trimester as seen in our case, with the median age presentation of 32 weeks, although it can occur at midgestation too. The pathophysiology of fetal AF is believed to be due to intra-atrial macroreentry circuit which the atria develop a critical capacity to support at 27-30 weeks, hence the onset of AF in the third trimester.[4] The ventricles are protected during fetal AF by the AV node which is not part of the intra-atrial re-entrant circuit. This is achieved by variably blocking the AV conduction, with 2:1 AV block present in over 80% of patients with fetal AF.[4,7] This finding is in keeping with our patient who had 2:1 block. Persistent tachyarrhythmia may result in fetal hydrops or heart failure, and fetal hydrops develops when the VR above 230bpm lasts over 12 hours. There was a potential risk of hydrops in our case with the VR of 240bpm, and this was taken into consideration in decision-making. Early features of cardiac compromise include atrioventricular valvular regurgitation and bilateral atrial enlargement, while decreased systolic function and cardiomegaly indicate later changes. The findings of subcutaneous oedema, pulmonary effusion, pericardial effusion, and ascites are echocardiographic features of hydrops.[6] In our case, there was only mild tricuspid regurgitation.

The diagnosis of fetal AF is made by fetal echocardiography. Fetal magnetocardiography (fMCG) is a noninvasive technique used in the diagnosis of complex tachyarrhythmia, but it is not available for routine clinical practice.[2,6] With echocardiography, both the atria and ventricles should be clearly identified in relation to the spine and descending aorta as landmarks. The focus should not be to get the VR only in fetal tachyarrhythmia using the PW button as was done in the referring hospital. With the M-mode across respective atrium and ventricle, their respective rates (cardiac rhythm) are measured which gives the variable conduction pattern also. It is very important to make the right diagnosis before proceeding to treatment especially in low resource setting or general hospitals without pediatric cardiologist services. In our case, the initial diagnosis was thought to be fetal SVT before the patient was referred. This was because of inadequate assessment with no proper echocardiography done. This is one of the major reasons of writing this report for educational purposes. It is known that up to 20% of fetal tachyarrhythmia is associated with cardiac anomaly, hence the need to also involve a pediatric cardiologist to perform a thorough fetal echocardiography before deciding on management option.[3,8] This informs the need of MDT management with neonatologists, pediatric cardiologist, and obstetricians. At term pregnancies, the babies should not just be delivered based on fetal distress without these considerations, as the outcomes may be untoward. This is well exemplified in this case.

Based on the points highlighted above, the antenatal management of fetal AF depends on several factors including fetal gestational age, presence of fetal hydrops or features of heart failure, and associated structural heart disease. The risk of hydrops in fetal AF is said to be more generally with a VR of over 210 bpm, and more rapid onset of hydrops with VR of over 230bpm. There was no hydrops in our case even though the ventricular heart rate was 240 bpm.[6] The development of associated cardiac anomaly is associated with poorer neonatal outcome, hence the advantage of early detection and treatment. The major aim of treatment is the prevention or resolution of hydrops either by VR control or conversion to sinus rhythm. Although prenatal treatment of fetal AF with transplacental antiarrhythmic medications is the most common documented method of treatment, this is not always the case.[9] At term or late preterm gestation, delivery of the fetus with postnatal treatment is often considered as a better choice.[3,10] This obviates the adverse effects of the medication on the mother and the risk of transplacental treatment on the fetus. This explains the basis of our postnatal treatment modality.

The commonly used antiarrhythmic drugs include digoxin as first choice and sotalol, flecainide, amiodarone, verapamil,

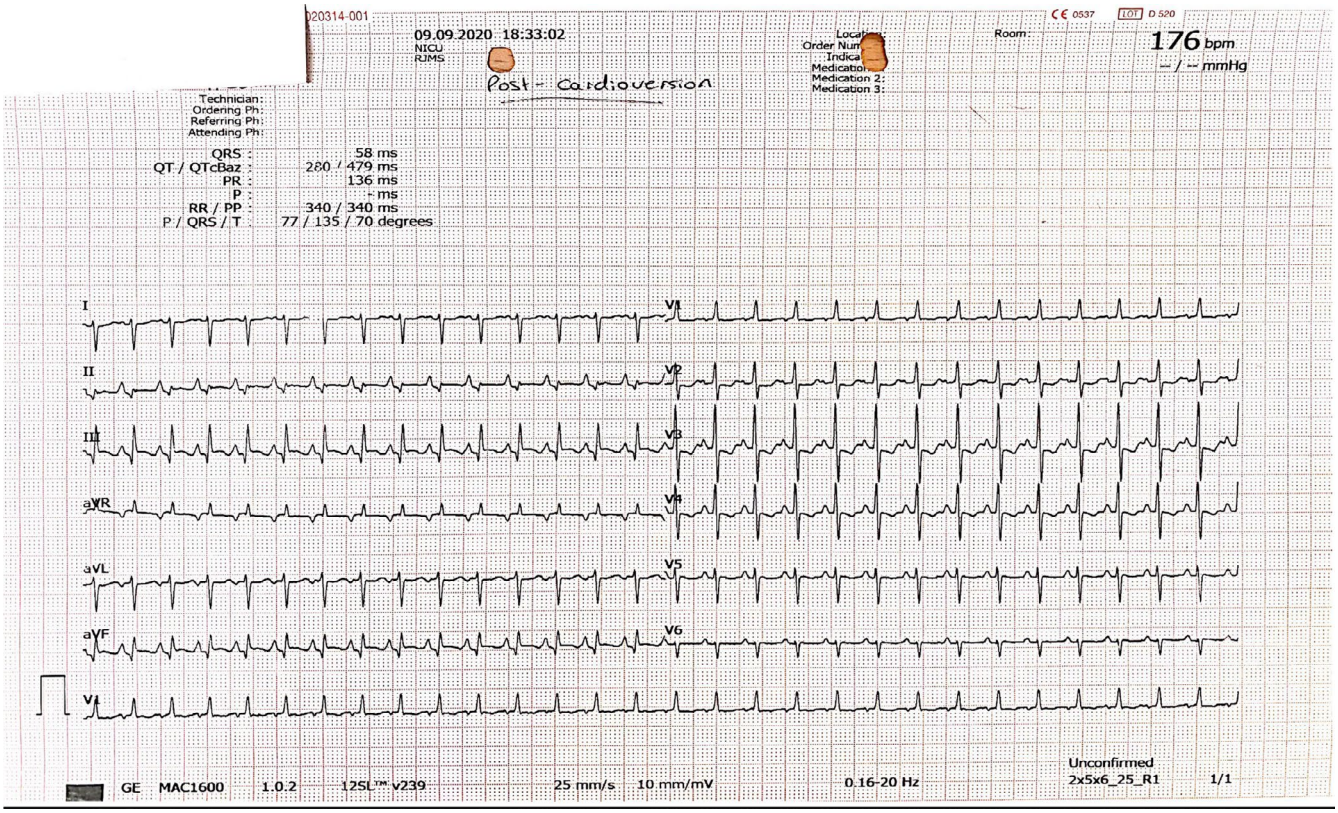

FIGURE 2 Postnatal ECG postcardioversion with sinus rhythm achieved with ventricular rate of 176 bpm

procainamide as second choice.[2,11] Studies show that there is a significantly better response to sinus rhythm prenatally when digoxin is used in nonhydropic fetuses (80%), compared with 43% in hydropic fetuses.[8,12]

It is important to remember that spontaneous conversion to sinus rhythm sometimes happen in fetal AF postnatally, although this was not observed in our case.[13] The ECG done postnatally confirmed the neonate was still in AF (Figure 1), hence the use of synchronized cardioversion which successfully resulted in sinus rhythm (Figure 2). Digoxin was continued as an antiarrhythmic prophylaxis. After sinus rhythm is achieved postnatally, one may monitor to see whether there is recurrence with AF before instituting prophylaxis, or electively treat for 6 months to 1 year.[10] However, the risk of AF recurrence is very rare beyond the neonatal period. In our case, digoxin was given for the first 28 days and to be continued for the first 6 months at least with follow-up.

4 | CONCLUSION

Fetal AF is a serious and threatening fetal tachyarrhythmia with an associated general mortality rate of 10%. Adequate diagnosis, awareness of association including fetal hydrops and cardiac anomaly, and MDT involvement often ensure optimal outcome. Postnatal cardioversion is a successful way of achieving sinus rhythm, and antiarrhythmic prophylaxis is often necessary especially for the neonatal period. It is essential that fetal AF is not managed as "fetal distress" by general obstetricians and midwives. Cases should be managed in centers with pediatric cardiologist expertise.

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CONFLICT OF INTEREST

No conflict of interest declared.

AUTHOR CONTRIBUTIONS

Dr Nnadozie Igbokwe, the lead author: wrote the manuscript, did literature review, got patient perspective and consent, and did the final editing. Dr Aisha F Ibrahim: summarized the clinical case notes and edited the final script. Dr Samy Mutalab: contributed to the discussion and key message. Dr Oonagh Cleland: suggested the case report and approved the final manuscript.

ETHICAL APPROVAL

A written informed consent was obtained from the patient.

DATA AVAILABILITY STATEMENT

All data used and references available online.

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