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

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Abstract

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 multidisciplinary team approach.

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Abstract

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 multidisciplinary team approach.

Key Message

At term pregnancy, emergency delivery and postnanal electrocardioversion with prophylactic digitalisation is a successful treatment option for fetal AF. Clinicians should be aware of AF and its diagnosis, and not just manage it as fetal distress.

Introduction

Fetal AF is a serious and threatening form of tachyarrhythmia, as it may cause fetal hydrops 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 characterised 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 or 2:1 conduction. 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 reentrant circuit causing premature atrial impulses.[3] The use of M-mode ultrasound establishes the diagnosis followed by fetal echocardiogram to rule out structural heart anomalies. The outcome of fetal tachyarrhythmia is dependent on the presence of hydrops and cardiac disease, and not the type of tachyarrhythmia.[4] Treatment is individualised with factors like gestational age, structural heart problems and hydrops taken into significant consideration. Early detection and treatment improved 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 was no fetal history of hydrops or cardiac disease and she had emergency caesarean section. Neonatal sinus rhythm was restored with single electrocardioversion and some course of digitalisation.

Case Report

A 24-year-old primigravida, booked low risk in a different hospital. Her booking blood results ultrasound scan were normal with Haemoglobin 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 by both 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 260bpm with an impression of fetal SVT made. There was a discussion to go for emergency caesarean section however this was forestalled by specialist advice from the paediatric cardiologist in a tertiary hospital who advised for the patient to be transferred by ambulance to the University teaching hospital 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 cardiac ultrasound done by the paediatric cardiologist revealed an atrial heart rate of 480bpm, and ventricular rate of 240bpm using the M-mode function. There was a mild tricuspid valve regurgitation, but no obvious cardiac structural anomaly or hydrops noted. A clinical impression of fetal AF at 480bpm with 2:1 nodal AV block was made. Options of management were discussed with the patient including medical antenatal treatment or

abdominal delivery with neonatal management. Since the pregnancy was term, a joint agreement with the obstetric team was for urgent abdominal delivery and postnatal treatment to which the patient consented. She had an emergency caesarean section under regional block. The Outcome was a live female neonate who weighed 3530 grams at birth with favourable Apgar score although with poor colour.

The baby required some continuous positive airway pressure (CPAP) for support for a few minutes as the oxygen saturation was at 70% and was transferred immediately to the neonatal intensive care unit (NICU). ECG done showed ventricular hear beat of 235bpm **(Figure 1),** with persistent AF**.** A single synchronised 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 10mcg 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.

The baby's mother had a good post-operative 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 1.5ug/L. Follow up echocardiogram done 8 weeks after discharged showed similar findings as before with normal ventricular function. The baby is 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.

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 mortality rate of 10%. Studies have shown that hydropic foetuses with fetal AF have higher ventricular rates than the non-hydropic 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. 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. Fetal AF could easily be diagnosed by clinicians using the M-mode ultrasound with the apical four-chamber heart view. 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 ventricular rate only in fetal tachyarrhythmia using the power Doppler button. With the M-mode across both either atrium or ventricle, their respective rates could be measured and 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 paediatric cardiologist services. In our case, the initial diagnosis was thought to be fetal SVT before the patient was referred. This is because no simultaneous atrial reading was taken. This is one of the major reasons of writing this report for educational purposes. Secondly, it is known that up to 20% of fetal tachyarrhythmia is associated with cardiac anomaly, hence the need to perform fetal echocardiography before deciding on management option.[3,8] This informs the need of multidisciplinary team management with neonatologists, paediatric 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 with a ventricular rate of over 210 bpm. There was no hydrops in our case even though the ventricular heart rate was 260 bpm.[6] The development of associated cardiac anomaly is associated with poorer neonatal outcome, hence the advantage of early detection and treatment. 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 foetus 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 foetus. This explains the basis of our postnatal treatment modality.

The commonly used antiarrhythmic drugs include digoxin as first choice and sotalol, flecainide, amiodarone, verapamil, procainamide as second choice.[2,11] Studies show that there is a significantly better response to sinus rhythm prenatally when digoxin is used in non-hydropic foetuses (80%), compared with 43% in hydropic foetuses.[8,12]

It is important to remember that spontaneous conversion to sinus rhythm does 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 synchronised 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 if 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.

Conclusion

Fetal AF is a serious and threatening second commonest fetal tachyarrhythmia with an associated mortality rate of 10%. Adequate diagnosis, awareness of association including fetal hydrops and cardiac anomaly, and multidisciplinary team involvement often ensure optimal outcome. Postnatal cardioversion a successful way of achieving sinus rhythm and antiarrhythmic prophylaxis is often necessary especially for the neonatal period. It is essential fetal AF is not managed as 'fetal distress' by general obstetricians and midwives. Call for help when in doubt.

Author contribution

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 summarised 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.

Conflict of interest

No conflict of interest declared.

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Ethical consideration

A written informed consent was obtained from the patient.

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