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(CASE REPORT)

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Transplacental digoxin therapy for treatment of fetal supraventricular tachycardia

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Abstract

Fetal supraventricular tachycardia (SVT) is the most common form of primary fetal tachycardia. It is a known cause of non-immune hydrops and can lead to significant perinatal morbidity and mortality. We report a case of a 27-year-old primigravida with a diagnosis of fetal supraventricular tachycardia. Her fetus was treated transplacentally with maternal oral digoxin leading to reversion to sinus rhythm. She was delivered by caesarean section and baby had an uneventful neonatal period.

Keywords: Arrhythmia; Tachycardia; Supraventricular tachycardia; Digoxin; Hydrops; Hydrops fetalis

1. Introduction

Fetal tachycardia is a rare complication during pregnancy and is said to occur when the fetal heart rates exceeds 180 beats per minute[1]. The most common form of primary fetal tachycardia is fetal supraventricular tachycardia (SVT) and it is a known cause of non-immune hydrops[1,2]. Hydrops fetalis complicates about 40% of fetuses with SVT.[3] When complicated by hydrops, it is associated with significant perinatal morbidity and mortality[4,5]. Perinatal mortality from fetal hydrops is as high as 35% compared with 0% to 4% in non hydropic fetuses.[6] The risk of developing hydrops fetalis is related to the gestational age at onset and the duration of the SVT. The younger the gestational age at onset, the higher the risk of developing fetal hydrops[2,6]. Again, the longer the duration of the disease, the more the risk of developing fetal hydrops. SVT is characterized by a 1: 1 atrioventricular activity of the FHR exceeding 200 beats per minute(bpm), and atrial flutter when the atrial rate is in excess of ventricular rate[2,4]. We present a case of fetal SVT that was managed successfully in our facility using maternal digoxin therapy. The justification for this therapy and a review of relevant literature are also presented.

2. Case Presentation

The patient is a 27-year-old primigravida who was noted to have persistent fetal tachycardia of between 204 and 220 beats per minute on her routine ante natal clinic visit at the gestational age of 29 weeks. The patient had no personal or family history of cardiac disease, diabetes or thyroid dysfunction. She had no history of use of alcohol, tobacco, caffeine or recreational drugs and all her routine tests were normal.

She was sent to the radiology department for Doppler ultrasonography. She was scanned with B mode, M mode, colour and spectral Doppler ultrasonography (see Fig 1 and Fig 2). On the initial scan the fetus was found to have a heartbeat of 218 beats per minute. The colour and spectral Doppler show normal colour flow in the umbilical artery and the

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spectral tracing show normal resistive index of 0.48 and pulsatility index of 0.63. On B mode scan, the heart shows normal four chamber view and normal outflow tracts. There is no evidence of subcutaneous edema, pleural effusion or ascites.

A diagnosis of fetal supraventricular tachycardia was made. The patient was counselled on the condition and placed on digoxin tablets. She was started on 625 microgrammes of digoxin and maintained on 250 microgrammes twice daily.

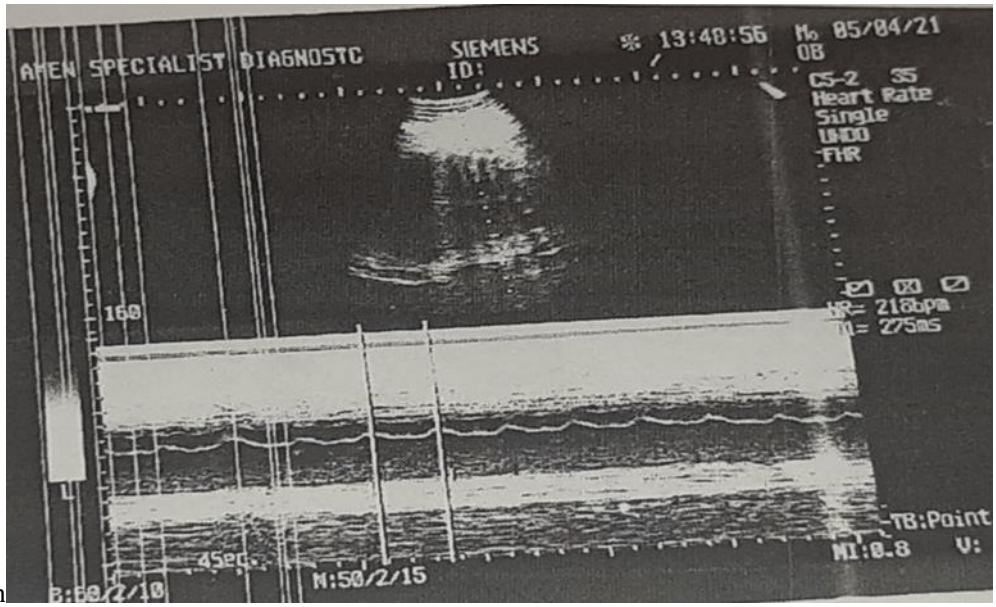

Figure 1 M Mode Tracing On Initial Scan

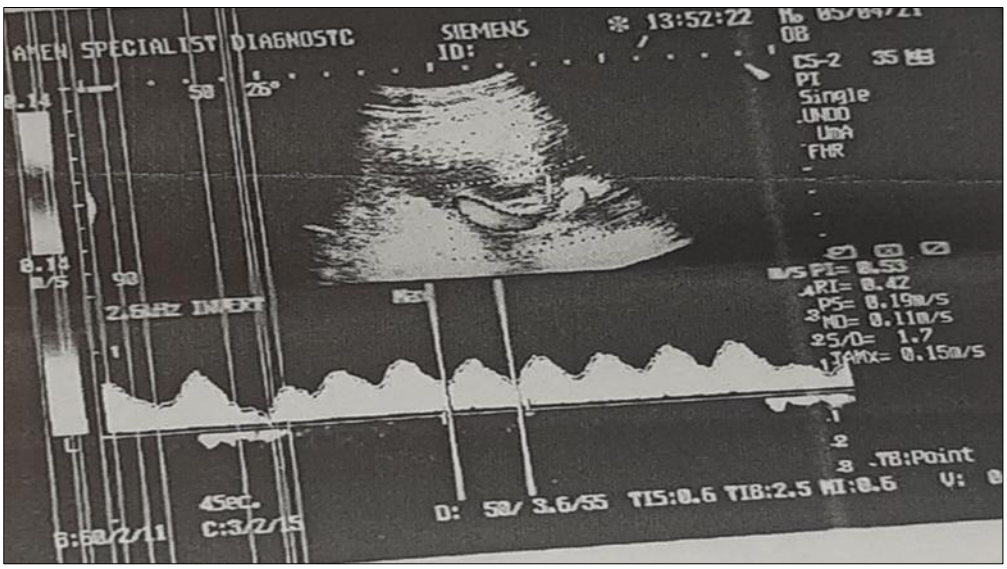

Figure 2 Spectral Tracing On Initial Scan

On a repeat scan 3 days following medical treatment, the baseline fetal heart rate was between 158 and 172 beats per minute and by 7 days following treatment, there was further reduction of fetal heart rate to 139 to 156 beats per minute. The spectral and colour Doppler features remained within normal limit. On B mode scan, the heart was unremarkable.

She was continued on digoxin tablets 250 microgrammes twice daily till delivery. She was seen in the ante natal clinic weekly till delivery and the fetal heart rate remained normal. At 38 weeks of gestation, she had an elective caesarean

section with the delivery of a male baby with Apgar scores of 8 at the first minute and 10 at 5th minutes and birth weight of 3.09kg. He was examined by the attending pediatrician and had no obvious congenital structural abnormalities following birth. Baby remained healthy during the neonatal period. The baby was healthy 6 weeks after delivery with no evidence of cardiac arrhythmia. The parents were counselled extensively on the need for regular follow up, they were subsequently referred to the pediatricians.

3. Discussion

Fetal arrhythmias occur in about 1% of pregnancies however, majority of them are benign intermittent atrial contractions and require no treatment[3,7]. A concomitant structural heart defect is present in 5–10% of cases[8], however in majority of cases, also as seen in our patient, it is an isolated abnormality. The less common sustained fetal tachyarrhythmias are associated with significant fetal and neonatal morbidity and mortality[7,9]. Supraventricular tachycardia (SVT) along with atrial flutter (AF) are the most common types of sustained fetal tachycardia. Excessive Caffeine, smoking, illicit drugs usage, fetal cardiac malformation and extracardiac malformations like diaphragmatic hernia may contribute to frequent fetal premature atrial contractions which may progress to tachyarrhythmia[10]. Our patient did not have any of these. The fetus had no obvious structural abnormality following in utero ultrasound assessment or physical examination after birth. Various mechanisms for occurrence of SVT have been described as atrioventricular (AV) reentrant, intraatrial reentrant and AV nodal reentrant[3,10,11]. In a study by Ko et al., these three types of SVTs accounted for 73%, 14% and 13% of the cases[11].

The diagnosis of SVT is dependent on clinical presentations and radiological findings. Fetal SVT has a wide spectrum of presentation ranging from the intermittent fetal tachycardia with no haemodynamic effects to persistent fetal tachycardia with high output cardiac failure leading to hydrops fetalis[2]. Routine ante natal auscultation of the fetal heart rate can serve as a screening method to detect fetal tachycardia, however using the M-mode or Doppler in ultrasound[12], the fetal cardiac rhythm can be assessed by determining the relationship between atrial and ventricular activities[2,13]. Fetal echocardiogram is necessary for identification of concomitant structural heart disease, detailed assessment of myocardial function and the degree of hemodynamic compromise[2,13]. Doppler magnetocardiogram (MCG) and Doppler myocardial deformation analysis are the newer methods used for the diagnosis[3,13,14].

Important considerations during treatment includes gestational age, mechanism of arrhythmias, FHR and risk of prematurity and degree of compromise (usually the presence of hydrops)[2,10]. In the term fetus, or preterm fetus in which fetal lung maturity can be demonstrated (usually > 34 weeks of gestation) urgent delivery and direct therapy are provided to the newborn[10]. Vaginal delivery can be considered if the fetus is non hydropic. Prior to this time, cases of intermittent SVT with no signs of cardiac failure are followed with weekly fetal ECG but more intensive monitoring (daily or 2 times weekly) is indicated if the SVT is sustained[13]. Those with hemodynamic compromise (e.g., hydrops fetalis) will require hospitalization. In sustained SVT, rapid and permanent conversion to sinus rhythm using anti-arrhythmic drugs is needed to prevent or resolve congestive heart failure. If reversion to sinus rhythm and resolution of hydrops is achieved prior to birth, neonatal outcomes is improved[5].

The mainstay of treatment is fetal rhythm control and conversion to sinus rhythm via transplacental medical interventions to the mother which was reported over 40 years ago[2,15]. There is no clear consensus regarding the best drug-treatment regimens for fetal SVT[5,13,16], however digoxin, sotalol, flecainide, amiodarone, other antiarrhythmic agents or a combination thereof have been described as successful interventions to treat fetal SVT and AF in multiple studies[6,7,9]. Combination therapy is preferred as first-line treatment when fetal hydrops is present[5]. In our patient, digoxin monotherapy was used for the treatment. It is the first-line drug of choice in treating fetal SVTs in many centres [2,3,13], while in some other centers, flecainide or procainamide are used as first-line agents however their use may be limited due to the higher pro-arrhythmic activity and fetal-negative inotropic actions, both of which may exacerbate hydrops fetalis[3,13]. However, due to its safety and side effect profile and relatively rapid response time, digoxin is the preferred initial treatment[17].

When using digoxin, maternal serum level of digoxin should be monitored to avoid toxicity, which can present as nausea, vomiting and blurring of vision[2]. Despite its effectiveness in the treatment of fetal SVT, amiodarone is usually reserved for refractory cases because of its unfavourable neonatal side effect profile (notably neonatal hypothyroidism) and the need for careful maternal monitoring[5,10]. In such refractory cases where conversion to sinus rhythm is not achieved with several maternally administered antiarrhythmic drugs, direct fetal therapy can be considered[10]. These include intravascular, intraumbilical, intra-amniotic, intraperitoneal, intramuscular and intracardiac treatment of the fetus[4]. Direct fetal therapy has been reported to be effective in rapid resolution of fetal SVT[10,18], however it poses a significant risk to the fetus and should always be administered as an adjunct to maternal therapy[10]. Our patient had no need for direct fetal therapy.

After successful anti-arrhythmic treatment, dosage is maintained until delivery with weekly monitoring of the fetal cardiac rhythm. If the heart rate is controlled, cesarean section is not indicated due to the rhythm disorder only[2]. Our patient chose to have a caesarean section, she vehemently refused the option of vaginal delivery. Following delivery, some babies will still need treatment usually with a B-blocker for at least 6 to 12 months[13]. In some fetuses, SVT may resolve spontaneously in utero, during delivery, or after birth while others may only require short-term administration of antiarrhythmics[3]. Some of these cases, however, tend to recur later on, especially during adolescence[3]. Our patient's baby had no need for continuous treatment as the sinus rhythm was maintained even after birth.

4. Conclusion

Fetal supraventricular tachycardia is an uncommon pathology which requires a high index of suspicion for its identification. Early diagnosis and initiation of treatment afford the potential for excellent outcomes. Some babies however, may require more prolonged assessment and therapy with occasional recurrence seen even during adolescence.

Compliance with ethical standards

Acknowledgments

The authors would like to thank the woman whose management was discussed here.

Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

Statement of ethical approval

The management of the patient and the reporting of this case followed ethical principles.

Statement of informed consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understood that her names and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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