

A Fetus with Hydropic Change Secondary to Fetal Supraventricular Tachycardia

MC YAM, TY LEUNG, TK LAU, RYT SUNG

Abstract

Supraventricular tachycardia (SVT) is a well-recognised cause of non-immune hydrops fetalis, which is associated with a high incidence of perinatal mortality. The arrhythmia can be diagnosed accurately during prenatal life. We report here a case of fetal hydrops secondary to SVT at 20 weeks of gestation successfully treated with transplacental flecainide. The controversy on the types of drugs used and the optimal route of administration were discussed.

Key words

Flecainide; Hydrop fetalis; Supraventricular tachycardia

Introduction

Fetal tachyarrhythmia is a rare but serious disease. Without treatment, it may progress to fetal cardiac failure, hydrops fetalis and in utero fetal death. With the rapid advancement in fetal echocardiographic equipment and techniques, such arrhythmia can be diagnosed accurately during prenatal life by using M-mode echocardiogram and doppler.¹ Prenatal control of the tachyarrhythmia can be achieved by either transplacental or direct fetal treatment with a wide-range of antiarrhythmic drugs.² We report a 20 weeks gestation hydropic fetus secondary to supraventricular tachycardia (SVT) who was successfully treated by transplacental flecainide.

Case Report

A 37 years old woman was referred to our clinic during first trimester. Upon her request, a chorionic villus sampling was performed at 12 weeks gestation and was normal. During a routine ultrasound scan at 20 weeks gestation, fetal tachycardia, scalp edema and ascites were detected. There was no other structural abnormality identified. Fetal echocardiogram revealed normal fetal cardiac structure and satisfactory cardiac contractility. There was monotonous fetal tachycardia with heart rate up to 259/min. The atrial/ventricular contraction ratio was 1:1 as confirmed by M-mode echocardiogram (Figure 1). The diagnosis of fetal supraventricular tachycardia with hydropic change was made. The patient was otherwise healthy.

The patient was started on flecainide from 50 mg twice daily and gradually increased to 150 mg twice daily over a 9-day period. The fetal heart rate was gradually brought down to 220/min (day 4 after flecainide), 200/min (day 8), 190/min (day 9) and 140/min (day 10). By day 11, the basal heart rate was normal at 140/min but there was also intermittent fetal bradycardia down to 80/min. Flecainide was then decreased to 100 mg twice daily until term. The scalp edema of the fetus resolved nine days after treatment while the ascites took four weeks to disappear. The mother was monitored with regular ECG. She tolerated the drug well without any complication and there was no recurrence of fetal SVT, nor fetal bradycardia. Flecainide was stopped after delivery.

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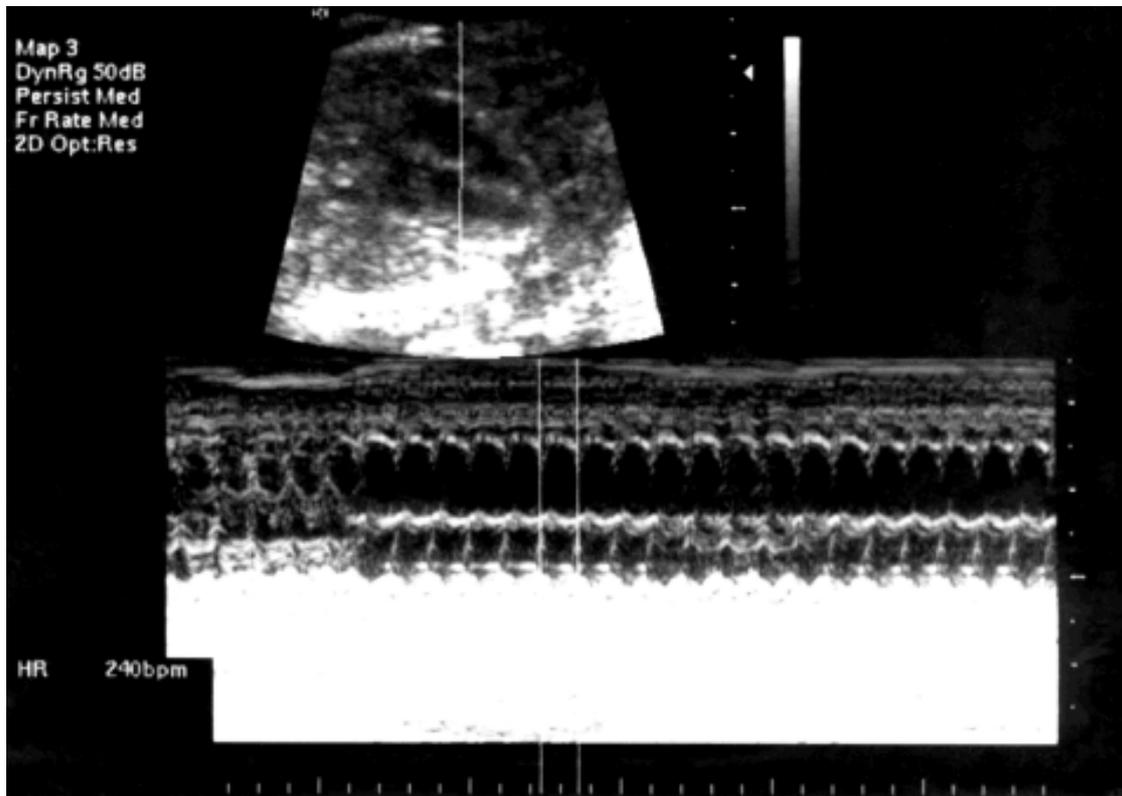


Figure 1 M-mode echocardiogram showing supraventricular fetal tachycardia, with typical regular fast fetal heart rate with 1:1 atrial/ventricular ratio.

The patient had a normal vaginal delivery at 39 weeks of gestation. The baby was a boy, weighed 3.6 kg, with Apgar score of 9 and 9 at 1 and 5 minutes respectively. Postnatal echocardiogram was normal and the ECG showed sinus rhythm without any pre-excitation. The baby was put on conservative treatment and regularly reviewed at outpatient clinic. There was no attack of SVT after 18 months of follow-up.

Discussion

Non-immune hydrops fetalis is a rare but serious disorder. There are many possible causes for non-immune hydrops. Associated perinatal mortality is high unless the underlying cause could be identified and treated appropriate in utero or ex utero if the fetus is maturity enough for delivery. With complete prenatal and postnatal evaluation, precise diagnosis can be arrived in approximately 85% of cases.³ Fetal tachyarrhythmia is one of the recognised causes of non-immune fetal hydrops. In Simpson and Sharland's

series, the mortality was up to 56% if the arrhythmia was not controlled prenatally.⁴ Fetal tachy-arrhythmia with supraventricular tachycardia (65%) and atrial flutter (17%) accounting for more than 80% of cases of hydrops-related fetal arrhythmia.⁵ The predominant mechanism of supraventricular tachycardia in the fetus is believed to be due to atrioventricular reentrant tachycardia,⁶ and the problem in our patient may have similar mechanism as there was absence of pre-excitation on the postnatal ECG. With advances in ultrasound technology, it is now possible to diagnose accurately the type of arrhythmia using M-mode echocardiogram to compare the relation between atrial and ventricular contraction.¹ Cross-sectional echocardiography allows identification or exclusion of other associated structural abnormality, which may be present in 5-10% of cases.⁴

There is a general consensus that fetal SVT complicated with fetal hydrops should be managed either by in utero therapy if the fetus is too premature for delivery, which reduces the perinatal mortality rate from 56% to 9%,⁴ or by immediate delivery if the pregnancy is of reasonable

maturity, in particular if beyond 34 weeks. However, whether there is a need to initiate in utero therapy in cases of SVT without accompanying hydrops or cardiac decompensation is controversial. Arguments against initiation of treatment are that treatment is not essential in those tolerating SVT well and that any drug treatment might be associated with significant maternal side-effects. However, precise prediction of the natural course of this condition in an individual is difficult and treatment could be more difficult if fetal hydrops develops. Therefore, some would suggest initiation of treatment if SVT is persistent even without accompanying hydrops. In case treatment is not initiated, the fetus should be monitored closely for early signs of deterioration when treatment is definitely indicated.

More controversial is the choice of antiarrhythmic drug. Due to the small case number in each treatment center, no randomized control trial is available to properly evaluate and compare the relative efficacy of drugs currently used. Available information were based on descriptive studies. The most common drug used in managing fetal SVT without hydrops is digoxin given orally to the mother, which converted SVT to sinus rhythm with high success rate.⁷ When this failed, the addition of verapamil, or substitution of digoxin with flecainide will achieve better control up to 83%.⁴ In case hydrops has developed, digoxin is probably not the drug of choice because placental transfer of digoxin is known to be poor in such circumstances.^{8,9} Flecainide does cross the placenta effectively, even when the fetus is hydropic. Use of flecainide controlled the rhythm more rapidly and caused resolution of hydrops more frequently than the combination of digoxin and verapamil. Although there was concern about the negative inotropic effects of flecainide on the fetus, we did not observe any fetal myocardial depression during the treatment period as monitored by regular fetal echocardiogram. On the other hand, there was a need, irrespective of drug used, to monitor the maternal cardiac status when antiarrhythmics were prescribed. As in our case, the maternal ECG was regularly monitored by cardiologists and no unwanted effect was found throughout the treatment period.

As an alternative to transplacental treatment, antiarrhythmics could be given either to the amniotic cavity or directly to the fetus by intramuscular or intravenous injection. This approach minimises the potential maternal side effects, but requires invasive procedures, which carries the risks of inducing abortion, preterm labour, rupture of

membranes and infection, and repeated invasive procedures may be required. In the series reported by Hansmann et al.,⁹ two of their thirteen fetuses developed cardiac arrest during direct fetal treatment and multiple injections of antiarrhythmic drugs were often required. Balancing these potential risks, we considered that maternal oral treatment was probably safer than direct therapy.

If fetal SVT could be controlled successfully, the prognosis in general is very good with low incidence of recurrence. In our case, the baby was successfully delivered at term. He remained in normal cardiac rhythm, and long-term prophylactic antiarrhythmic drug was not required.

Conclusion

Fetal SVT is a rare cause of hydrops fetalis with significant mortality and morbidity. Oral maternal flecainide is a rapid and effective drug treatment for this condition and the outcome was excellent.

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