CASE REPORT

Fetal supraventricular tachycardia, treating the baby by targeting the mother

Aysha Husain,¹ Zakariya Hubail,² Rashed Al Banna^{3,4}

¹Department of Internal Medicine Cardiology Unit, Salmaniya Medical Complex, Manama, Bahrain

²Department of Pediatric, Salmaniya Medical Complex, Manama, Bahrain

³Department of the Internal Medicine, Cardiology Unit, Salmaniya Medical Complex

⁴Arabian Gulf University, Medical College, Internal Medicine department, Manama, Bahrain

Correspondence to

Dr Rashed Al Banna, abdullarashed@yahoo.com

SUMMARY

Fetal supraventricular tachycardia (SVT) is the most common form of fetal tachycardia. If started early in pregnancy, it can cause non-immune fetal hydrops. Echocardiography is the preferred method for the diagnosis with simultaneous pulsed Doppler recording from the superior vena cava and ascending aorta. Transplacental therapy with digoxin is the most common way of treatment. We present a case of fetal SVT detected at 26 weeks of pregnancy. Digoxin therapy restored the rhythm initially, but later paroxysms of fetal SVT persisted necessitating the addition of second antiarrhythmic medication which was discussed with the parents. The couple chose to proceed for premature delivery at 32 weeks.

BACKGROUND

Fetal supraventricular tachycardia (SVT) is the most common form of fetal tachycardia. This was our first experience with such disease in our institute. Diagnosis and treatment imply a challenge to the physicians involved. Multidisciplinary approach is mandatory between paediatric cardiologist, adult cardiologist and obstetrician.

CASE PRESENTATION

A 26-year-old lady in her second pregnancy was referred to our hospital for investigation and treatment of fetal tachycardia noted at 26 weeks of gestation during her routine antenatal visit. She gave a history of loss of fetal movements for 1-day duration.

The pregnancy course was uneventful and fetal growth was normal. There was no history of fever, thyrotoxicosis or excessive consumption of caffeine. Her medical and obstetric history was unremarkable. She gave birth to a healthy baby boy 3 years previously.

Vital signs were heart rate 98 bpm and blood pressure 130/80 mm Hg. Cardiovascular examination was normal.

INVESTIGATIONS

The cardiotocograph, which is a technical means of recording the fetal heart beats and uterine contractions during pregnancy, showed a persistent fetal tachycardia with a heart rate 200 bpm.

Ultrasound examination revealed a single live active male fetus appropriate for gestational age. There was no pleural or pericardial effusion with normal amniotic fluid index.

The fetal echocardiogram revealed a structurally normal heart, situs solitus (normal positions of

thoracic and abdominal organs), levocardia (the heart is on the correct side of the body (to the left)) normal chamber sizes, normal valves, normal great arteries and veins and normal function. The fetal heart rate (FHR) at that time was 123–140 bpm and was consistent with a normal sinus rhythm, normal atrioventricular sequential contraction, with a 1:1 relationship between the atrial and ventricular activities; normal mechanical PR interval as measured from mitral inflow-left ventricular outflow (figure 1) and from simultaneous pulmonary veins-right pulmonary artery pulsed Doppler interrogation (figure 2).

A week later, the obstetrician noted recurrence of fetal tachycardia. The repeated fetal echocardiogram did show rapid heart rate at 195–210 bpm. The superior vena cava–aorta (SVC–Ao) Doppler recording is shown in Figure 3. The time interval between atrial contraction and ventricular contraction (V–A interval) was longer the time interval between ventricular and atrial contractions (V–A interval). This is indicative of a re-entrant circuit, such as atrioventricular re-entry tachycardia. The mitral inflow–outflow Doppler pattern showed a single inflow signal. This is less useful in determining the rhythm, as it can be normally seen at rapid heart rates, owing to E–A waves' fusion. Simultaneous pulsed Doppler recording from the SVC and ascending aorta is most informative in these situations. M mode recording of atrial and ventricular movements was also consistent with the above findings. Since the heart rate was not very high, only observation and close follow-up was

Figure 1 Fetal echocardiography still image of simultaneous mitral inflow and aortic outflow obtained via five-chamber view. The heart rate was 143/min. The positive signal is due to aortic outflow and the negative signals are due to mitral inflow. The mechanical PR interval was 95 ms. Ao, aortic outflow; E, mitral E wave; A, mitral A wave.

To cite: Husain A, Hubail Z, Al Banna R. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-008515

Figure 2 Simultaneous pulse Doppler recording during sinus rhythm, from the right pulmonary artery (positive signal) and right pulmonary vein (negative signal). The atrial contraction is denoted by an indentation in the pulmonary vein signal. The mechanical PR interval in this recording was 92 m/s.

recommended. The tachycardia, however, persisted over the next 2 weeks, and in view of her gestational age and so it was decided to start transplacental antiarrhythmic therapy.

TREATMENT

The condition was fully discussed with couple who agreed on treatment.

The mother underwent further investigations to ensure safety of digoxin therapy. Her basic laboratory investigations including urea, electrolytes and creatine were within the normal range. 12-Lead ECG was normal along with her transthoracic echocardiography.

She was started on digoxin 0.25 mg 8 hourly for 2 days, which caused the fetal heart to normalise to 140–160 bpm. During the whole course of treatment, maternal serum digoxin, urea, electrolytes and ECG were monitored.

On the third day, the dose of digoxin was reduced to 0.25 mg once daily that caused a recurrence of fetal tachycardia with heart rate 200 bpm. The maternal serum digoxin was within the normal limit.

Further escalation of digoxin dose versus changing to other antiarrhythmic medication has been discussed with the couple. It has been decided to increase the dose of digoxin as the initial response was favourable.

The dose of digoxin increased to 0.5 mg twice a day for 2 days. FHR came down to 140 bpm, but unfortunately on the third day of escalating the dose, the mother started to experience symptoms of digitalis toxicity. She had nausea, vomiting and epigastric pain. A 12-lead ECG showed normal sinus rhythm with 67 bpm and inverted tick sign suggestive of digoxin therapy. Her serum digoxin was elevated (3.06 mmol/l). FHR was found to be normal. The digoxin therapy was withheld for 1 day and then resumed a day after with a dose of 0.25 mg twice daily. Further monitoring of FHR was normal, ranging between 129 and 143 bpm. She was discharged on the same digitalis dosage with close out patient follow-ups.

OUTCOME AND FOLLOW-UP

After discharge from the hospital, in one of the follow-up studies, short paroxysms of tachycardia at 210/min were noted, alternating with sinus rhythm 130/min. On further follow-up, this pattern persisted; although no overt heart failure was noted, the ventricles started to dilate. She was compliant with her medication and her serum digoxin level was within the therapeutic range.

The option of trying antiarrhythmic medications or premature delivery of the baby was discussed with the couple. They chose to go for delivery by caesarean suction.

The mother was admitted on the second day and scheduled for elective caesarean suction at 32 weeks of gestation. She received doses of dexamethasone to enhance fetal lung maturity. Lower segment caesarean section was performed and the male baby was delivered. The initial heart rate was 205/min, but came down to 130–140 shortly thereafter with no intervention.

He stayed in the neonatal intensive care unit for heart rate monitoring and because of neonatal jaundice. During his stay, there was no recurrence of SVT. Fetal echocardiogram was normal. His weight was increasing normally and he was discharged from the hospital after 1 week with follow-ups in the paediatric cardiology outpatient department.

DISCUSSION

Fetal tachyarrhythmia was first described by Hyman¹ using phonocardiography. Even though the fetal heart is affected, the main mode of treatment is transplacental by administering the drug to the mother. In 1970s, however, Silber *et al*² were the first to notice the association between fetal SVT and mortality from non-immune hydrops fetalis.

Fetal SVT is defined as 1:1 atrioventricular activity of the FHR exceeding 200 bpm. It accounts for 60–80% of the fetal tachyarrhythmias with prevalence ranging from 1/1000 to 1/25 000 pregnancies.^{3 4}

There are confounding factors which can be maternal, such as excessive caffeine consumption, smoking and illicit drugs abuse. Fetal factors are particularly congenital defects like diaphragmatic hernia contributing to frequent fetal premature atrial contractions, which may progress to unrelenting tachyarrhythmia.⁵

The clinical presentation of fetal SVT has a wide spectrum. It can be intermittent with no haemodynamic effects to persistent type with high output cardiac failure leading to hydrops fetalis.⁶

The risk to develop hydrops fetalis is related to the age of the fetus (the younger being more susceptible) and the duration of the SVT.³ SVT was diagnosed as early as 13 weeks of gestation.⁷

To diagnose fetal cardiac rhythm, echocardiography is essential. M mode and Doppler echocardiography are used to assess the relation between atrial and ventricular activation. Apart from aiding in assessing the fetal cardiac rhythm, echocardiogram is a must to exclude coexistence of structural heart disease.⁸

Doppler positions for rhythm interpretation include foramen ovale and hepatic veins for the detection of atrial rate, while the left ventricular outflow for detecting the ventricular rate.³ A more sophisticated method for rhythm analysis is the one proposed by Fouron *et al*,⁹ which studied the Doppler flow velocities of SVC and aorta simultaneously. According to this method, they divided fetal SVT into three groups: the short ventricular-atrial tachycardia (A–V) caused mainly by fast accessory pathway re-entry, long V–A tachycardia interpreted as atrial ectopic tachycardia or paroxysmal junctional reciprocating tachycardia and atrial flutter with variable A–V block. They proposed treatment plan for each type that proved its efficacy.⁹

In atrial flutter, there is more atrial activity than ventricular activity, usually in a 2:1 ratio. This can be easily demonstrated by M mode. In ventricular tachyarrhythmia, the dissociation of atrial and ventricular contraction can be demonstrated by M mode and Doppler. The fetus in this case did not have either of them.

Treatment decision should be based on bridging the gap between maternal risk and fetal benefits. To choose the best treatment option, one must consider the following factors:

Figure 3 Simultaneous pulse Doppler recording from the superior vena cava (SVC) and ascending aorta. The positive signal is caused by SVC blood flow, while the negative signal is caused by aortic flow. The A wave reversal is seen as negative signal, moving away from the heart. Note that the AV interval is longer than the VA interval. Ao, aortic outflow; D, SVC D wave (diastole); S, SVC S wave (systole); A, SVC A wave reversal (atrial contraction).

gestational age, mechanism of arrhythmias, FHR and risk of prematurity and degree of compromise (usually the presence of hydrops).^{6,8}

The experience with transplacental drug therapy started from late 1970s with propranolol, verapamil and digoxin.^{10–12} Invasive methods with umbilical cord adenosine administration started in the mid 1990s.¹³

Digoxin is the first choice of treatment for fetal SVT. Transplacental approach with this drug resulted in 50% conversion rates to sinus rhythm in non-hydropic fetuses with SVT. However, the rate goes down to 15–25% in fetal SVT complicated by hydrops fetalis or ventricular dysfunction. This is probably attributed to the low transplacental transfer of this drug.¹⁴ Digoxin was used by Strasburger¹⁵, intramuscularly with a dose of 88 µg/kg fetal non-hydropic weight to enhance the delivery of this drug.

Maternal serum level of digoxin should be monitored to avoid toxicity, which can present as nausea, vomiting and blurring of vision.

The second line of treatment includes amiodarone, sotalol and flecainide. They can be used as monotherapy or in addition digoxin.³

Delivery can be normal if the fetus is maintained on sinus rhythm, which is the most likely outcome with fetal SVT.^{8,16} Moreover, if SVT was not controlled, then intrauterine delivery is warranted especially from 28 weeks of pregnancy after completed lung maturity.¹⁷

Learning points

- Fetal SVT is a cause of non-immune hydrops fetalis.
- Simultaneous pulsed Doppler recording from the superior vena cava and ascending aorta is the preferred echocardiographic method in diagnosis.
- Transplacental therapy with digoxin is the most common modality of treatment.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- Hyman AS. Irregularities of the fetal heart: a phonocardiographic study of the fetal heart sounds from the fifth to eighth months of pregnancy. *Am J Obstet Gynecol* 1930;20:332–47.
- Silber DL, Durnin RE. Intrauterine atrial tachycardia, associated with massive edema in a newborn. *Am J Dis Child* 1969;117:722–6.
- Perles Z, Gavri S, Rein AJT, et al. Tachyarrhythmias in the fetus: state of the art diagnosis and treatment. *Prog Pediatr Cardiol* 2006;22:95–107.
- Silver LE, Platt LD, Santulli TV Jr, et al. Resolution of hydrops fetalis despite persistent fetal tachycardia. *J Ultrasound Med* 2001;20:1141–5.
- Rana YS, Sodhi B, Kochar SPS, et al. Successful digoxin therapy of fetal supraventricular tachycardia. *S Asian Fed Obstet Gynecol* 2009;1:44–6.
- Shand AW, Dickinson JE, D'Orsogna L. Refractory fetal supraventricular tachycardia and obstetric cholestasis. *Fetal Diagn Ther* 2008;24:277–81.
- Porat S, Anteby EY, Hamani Y, et al. Fetal supraventricular tachycardia diagnosed and treated at 13 weeks of gestation: a case report. *Ultrasound Obstet Gynecol* 2003;21:302–5.
- Darragh R. Fetal tachycardia's. *ACC Curr J Rev* 1999;63–6.
- Fouron JC, Fournier A, Proulx F, et al. Management of fetal tachyarrhythmia based on superior vena cava/aorta Doppler flow recordings. *Heart* 2003;89:1211–16.
- Teuscher A, Bossi E, Imhof P, et al. Effect of propranolol on fetal tachycardia in diabetic pregnancy. *Am J Cardiol* 1978;42:304–7.
- Wolff F, Breuker KH, Schlensker KH, et al. Prenatal diagnosis and therapy of fetal heart rate anomalies: with a contribution on the placental transfer of verapamil. *J Perinat Med* 1980;8:203–8.
- Kerenyi TD, Gleicher N, Meller J, et al. Transplacental cardioversion of intrauterine supraventricular tachycardia with digitalis. *Lancet* 1980;2:393–4.
- Kohl T, Tercanli S, Kececioglu D, et al. Direct fetal administration of adenosine for the termination of incessant supraventricular tachycardia. *Obstet Gynecol* 1995;85:873–4.
- Oudijk MA, Ruskamp JM, Ambachtsheer BE, et al. Drug treatment of fetal tachycardias. *Paediatr Drugs* 2002;4:49–63.
- Strasburger JF. Fetal arrhythmias. *Prog Pediatr Cardiol* 2000;11:1–17.
- Jaeggi ET, Carvalho JS, De Groot E, et al. Comparison of transplacental treatment of fetal supraventricular tachyarrhythmias with digoxin, flecainide, and Sotalol: results of a nonrandomized multicenter study. *Circulation* 2011;124:1747–54.
- Hagen A, Schmid O, Hopp H, et al. Management of fetal tachycardia. *Ultrasound Obstet Gynecol* 2003;22(S1):160.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow