

Fetal Supraventricular Tachycardia - A Case Report

E B S Soh MRCOG, S Raman, FRCOG, P M K Chia, MRCOG, Department of Obstetrics and Gynaecology, University of Malaya Medical Centre, 50603 Kuala Lumpur

Summary

A gravid patient with fetal supraventricular tachycardia is presented. A review of this rare condition and the present recommended mode of therapy are discussed.

Key Words: Fetal supraventricular tachycardia

Clinical Presentation

Madam S L, a 26-year-old housewife in her second pregnancy, presented at 37 weeks and 5 days of gestation. Her early antenatal follow-up with a private Obstetrician had been uneventful. She had an unremarkable past obstetric and medical history.

She was referred because of fetal tachycardia. The attending Obstetrician was initially unable to detect the fetal heart on cardiocography and an ultrasound examination revealed a rapid fetal heart rate of 220-230 beats per minute. There was no apparent maternal cause that could have resulted in fetal tachycardia.

On arrival at the Labour Ward, the mother was comfortable and afebrile. Systemic examination was unremarkable. Examination of the abdomen revealed a term-sized uterus with a singleton fetus in longitudinal lie and cephalic presentation. Auscultation confirmed fetal tachycardia. The cardiocograph (CTG) was unable to record the fetal heart.

An ultrasound examination showed an active baby in cephalic presentation. The placenta was in the posterior upper segment and the liquor volume was normal. A fetal echocardiogram and M-mode study

confirmed the fetal heart rate to be 210-220 beats per minute. However, no structural cardiac defect was found on detailed examination. Ultrasonic examination of the rest of the fetus was also normal. There was no evidence of fetal cardiac failure or hydrops. Diagnosis of fetal supraventricular tachycardia (SVT) was made. (The M mode fetal echocardiogram is displayed on Figure 1).

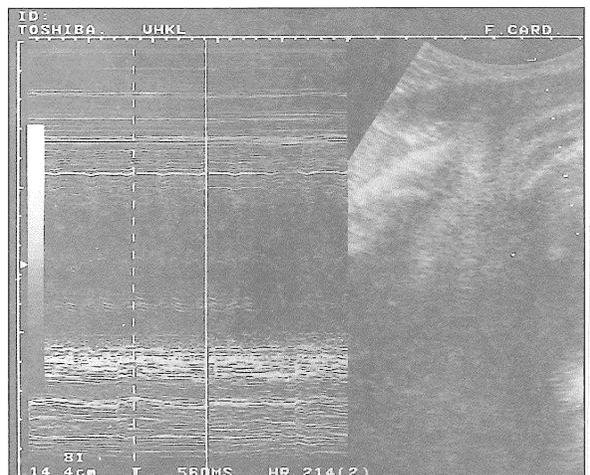


Fig. 1: M-mode ultrasound showing the fetal heart rate

The problem was discussed with the couple and the mother was started on oral Digoxin 0.25 mg. This resulted in reduction of the fetal heart rate to 140 beats per minute two hours later. A CTG examination done was reactive. Five hours later the patient went into spontaneous labour and delivered uneventfully a healthy 3.2kg baby girl with Apgar scores of 9 at one minute and 10 at five minutes.

The baby was well with a heart rate of 120-140 beats per minute for the first two days. She was feeding well and an echocardiogram revealed a persistent ductus arteriosus (PDA) which was most probably physiological at this stage. On the third day of life, the supraventricular tachycardia recurred. The baby's heart rate was at 280 beats per minute (long lead 2 ECG strip displayed on Figure 2). This was cardioverted with intravenous Adenosine at 0.1 mg/kg. She was also

started on oral Digoxin in a tapering dose. She later developed several other episodes of SVT which were reverted by adenosine as well. By the seventh day the baby's heart rate was regular at 120 beats per minute (Figure 3). She was discharged well on a maintenance dose of Digoxin at 0.25 µg twelve hourly. On follow-up one month later the Digoxin dose was tapered down to 0.25 µg daily. This dose was maintained at the last review at three months of life. The baby has been well so far.

Discussion

Fetal tachycardia is uncommon and occurs in 0.4 - 0.6% of all pregnancies¹. Supraventricular tachycardia (SVT) is the most common of these tachycardias. Short periods of SVT are probably of no significance.

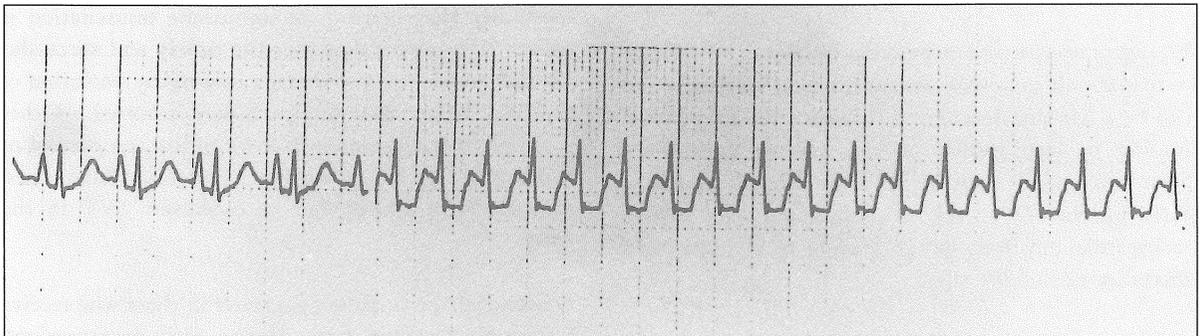


Fig. 2: ECG showing sinus rhythm converting to supraventricular tachycardia (SVT) spontaneously

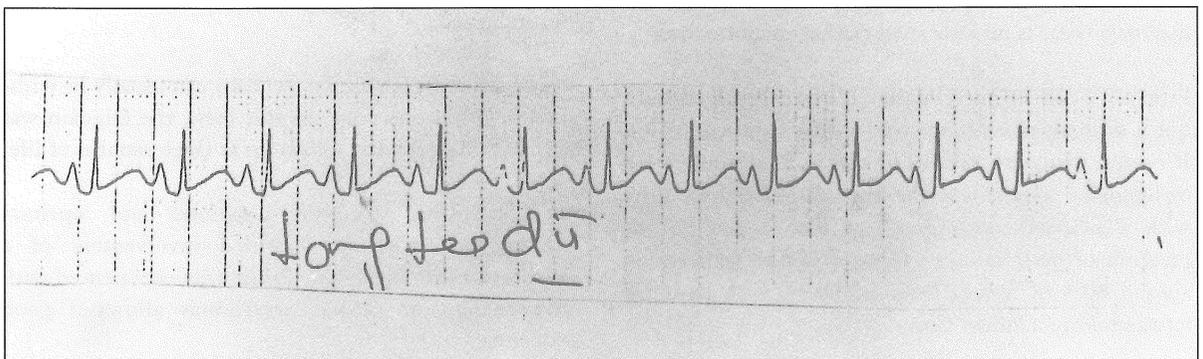


Fig. 3: ECG showing sinus rhythm at Day 7 of life

CASE REPORTS

However, persistent SVT can lead to high output cardiac failure, leading to hydrops fetalis.

Fetal SVT is defined as fetuses with a persistent heart rate of over 180 beats per minute without any variability, with a 1:1 atrioventricular conduction (on M mode ultrasound)². Fetal echocardiography with M-mode and/or two-dimensional Doppler will diagnose fetal SVT.

In this patient, the initial fetal heart rate could not be detected by the cardiotocograph (CTG) because of the rapid heartbeat with alternating rates. The ultrasound technique of recording detects the Doppler shift of the fetal heart movements, rather than its electrical activity. To produce a tracing, it averages out the fetal heart rate over 1.2 seconds. In rapid tachycardia like in this fetus, the machine was unable to summate the very rapidly changing heart rates to produce a signal, hence the absence of CTG recordings.

Fetal supraventricular tachycardia (SVT) can be difficult to manage. It causes significant neonatal morbidity and can be a life-threatening condition for the fetus; often leading to fetal cardiac failure, hydrops and death. Sustained fetal SVT and hydrops have been diagnosed as early as the 27th week of gestational age. Fetal tachycardia and heart failure leading to hydrops has a mortality rate of 20-50%².

The predominant mechanism for fetal supraventricular tachycardia is atrioventricular (AV) re-entrant tachycardia (i.e. a 1:1 atrioventricular (AV) conduction). Atrioventricular block in-utero and accessory AV connections are the other mechanisms³. In the majority, there is no associated cardiac malformation.

Fetal SVT can be successfully treated transplacentally, but is difficult when congestive cardiac failure develops. It is most frequently treated by maternal administration of Digoxin. Digoxin is the only anti-arrhythmic drug with a positive inotropic effect that is safe in the presence of heart failure. Control can be achieved in around 80% of fetuses before delivery, with hydropic fetuses taking a longer time.

The drug of second choice is often flecainide acetate, especially when Digoxin therapy has failed. Flecainide is a potent Class 1c anti-arrhythmic drug for atrial, junctional and ventricular arrhythmias. It is effective and safe in babies and children. It should not be the drug of first choice in atrial flutter. Other than this precaution, both Digoxin and flecainide do not produce significant fetal or maternal secondary effects.

There has even been a report of direct fetal administration of adenosine via the umbilical vein for the termination of incessant supraventricular tachycardia⁴. Adenosine terminates SVT re-entry tachycardia safely and effectively. Normalisation of fetal cardiac rhythm will lead to resolution of fetal hydrops. The quicker the control of rhythm, the better is the condition of the neonate at delivery and may even delay the onset of premature labour. Resolution of hydrops takes 1-2 weeks after rhythm control. If tense ascites is present in hydropic babies, peritoneal drainage can improve morbidity after delivery, as immediate resuscitation is easier. This report describes the timely and successful institution of maternal therapy leading to resolution of the SVT before fetal cardiac failure occurred. Other anti-arrhythmic agents such as quinidine, propranolol or verapamil, with or without Digoxin, have been prescribed for the mother to cardiovert SVT in the fetus².

Gestational age at deliver is greater in those who receive intrauterine management despite early presentation³. Previously, termination of the pregnancy by induction of labour or Caesarean section was recommended for fetal SVT². However, the present opinion is not to abbreviate the gestation. Caesarean deliveries are also not indicated.

Post-partum treatment for the baby can usually be withdrawn within one year. In this baby, the Digoxin was maintained at the last follow up at three months of life.

In conclusion, accurate diagnosis and optimal management with a combined involvement of a paediatric cardiologist and obstetrician experienced with management of cardiac arrhythmias allows a good outcome.

References

1. Southhall D P, Richard J, Hardwick R P, Shinebourne E A, Gibbons G L D, Thelwall-Jones H, De Swiet M, Johnston P G B: Prospective study of fetal heart rate and rhythm patterns. *Arch. Dis. Child.* 1980; 55: 506.
2. Bergmans M G M, Jenker G J, Kock H: Fetal SVT. Review of literature. *Obstet. Gynaecol. Surv.* 1985; 40: 61-8.
3. Naheed Z J, Strasburger J F, Deal B J, Benson D W Jr, Gidding S S: Fetal tachycardia: mechanism and predictors of hydrops. *Am. Coll. Cardio.* 1996; 27: 1730-40.
4. Kohl T, Tercanli S, Kececioglu D, Holzreue W. Direct fetal administration of adenosine for the termination of incessant supraventricular tachycardia. *Obstet. Gynaecol.* 1995; 85: 873-4.