Intermittent fetal tachycardia and fetal hydrops

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Abstract

A case is reported where fetal hydrops was noted 10 days after an initial observation of intermittent fetal tachycardia at 31 weeks. A diagnosis of supraventricular tachycardia was made and a successful conversion to sinus rhythm was achieved with maternally administered flecainide, with subsequent resolution of the hydrops. The fetus required no further treatment in pregnancy or at follow up. The mother suffered no side effects of the treatment.

Fetal tachyarrhythmias are rare occurrences but can be fatal if not investigated and promptly treated. Although treatment has considerably improved fetal survival, the choice of anti-arrhythmic agent is still controversial.

We describe a case of a fetus presenting initially with an intermittent tachyarrhythmia, the importance of which was not realised, which subsequently became a continuous supraventricular tachycardia resulting in fetal hydrops. The fetus was successfully treated with flecainide, a relatively new and effective treatment of tachyarrhythmias with associated fetal hydrops.

Case report

A healthy 29 year old woman, at 31 weeks in her second pregnancy, was noted to have an intermittent fetal tachycardia at a routine antenatal visit while on holiday. No further investigations, treatment, or referral was advised. Ten days later, when seen at her local hospital antenatal clinic, the fetal tachycardia was again noted, and an ultrasound scan revealed fetal hydrops. The patient was referred to the regional tertiary centre for further management. Ultrasound assessment revealed severe fetal hydrops with bilateral pleural, pericardial, and peritoneal effusions, and a tachycardia of approximately 300 beats/minute. The rate reverted to normal on one occasion during the initial assessment but this was short lived. M mode echocardiography confirmed a tachycardia of 300 beats/minute with a 1:1 relationship between atrial and ventricular conduction, consistent with a diagnosis of supraventricular tachycardia. A detailed ultrasound scan revealed a symmetrically enlarged heart occupying approximately half of the fetal thorax. There were no structural (especially cardiac) abnormalities and no stigmata of chromosomal anomaly. The fetus's size was normal for gestation. Doppler recording from the umbilical artery was also normal (pulsatility index=1.0).

The fetal supraventricular tachycardia was treated by maternal administration of flecainide, initially as an intravenous infusion (2 mg/kg) over 30 minutes followed by 100 mg three times a day orally. Fetal cardiography was used to monitor the fetal heart rate until sinus rhythm was well established. This took 12 hours. Standard and 24 hour maternal electrocardiograms were normal during flecainide treatment. Drug concentrations in the mother were measured twice a week and were in the therapeutic range. The hydrops resolved within 11 days. The oral flecainide was discontinued and there was no recurrence of the supraventricular tachycardia.

The mother was discharged home with a portable Doppler heart rate monitor (Fetal Dopplex, Huntleigh Technology) to use four times daily to exclude a recurrence of the tachycardia. She was followed up in her district hospital with weekly scans to exclude hydrops. There was no recurrence of the arrhythmia and she was admitted at 39 weeks' gestation for induction of labour. She had an uneventful labour and a vaginal delivery of a 3840 g live infant girl with normal Apgar scores. The placenta weighed 1020 g. Cardiological assessment of the infant at birth and at the 6 week follow up visit was normal.

Discussion

Fetal tachyarrhythmias are rare with a reported incidence of one in 10-25 000 pregnancies.¹ This is probably an underestimate since many cases are asymptomatic and undiagnosed. While not all tachyarrhythmias result in fetal hydrops, the high mortality associated with fetal hydrops (20-50%) makes it imperative that these cases are promptly referred for investigation.²

All tachyarrhythmias with associated fetal hydrops require active treatment. It has been suggested that treatment should also be given to those preterm fetuses with a tachyarrhythmia but no signs of fetal hydrops because of the high risk of hydrops developing subsequently (70%), and the lack of a reliable way of predicting the development of fetal hydrops from the type, duration or rate of the arrhythmia.¹ However, with more cases being reported the effectiveness of treatment of tachyarrhythmias in the absence of hydrops is being questioned.^{1 3 4} With Doppler flow studies of the fetal aorta being reported as being useful in the prediction of onset of fetal hydrops,⁵ there would appear to be a place for surveillance and withholding drug treatment in those fetuses with a tachyarrhythmia but no signs of fetal hydrops, remembering

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that all antiarrhythmic drugs have important side effects.

The choice of the optimum drug for treating fetal tachyarrhythmias is controversial. This is reflected by the fact that at least seven different drugs have been reported. Digoxin has been the drug most frequently described but its efficacy, especially in the presence of fetal hydrops, has been seriously questioned.^{1 3} Flecainide, a potent class 1c antiarrhythmic agent, was chosen to treat this patient because of its recently reported high efficacy in those fetuses with severe hydrops.² It is a relatively new antiarrhythmic agent, having been released in the UK in 1983. It has proved to be highly effective against both atrial and ventricular tachyarrhythmias in adults.⁶ In pregnancy it is quick acting with an 80% placental transfer,² which compares favourably with the 40% of digoxin and 10–20% of verapamil.⁴ It is recommended that administration is stopped once sinus rhythm is achieved and the hydrops has resolved.² If the tachyarrhythmia recurs, continued treatment may be required. As with many antiarrhythmic agents it has a negative inotropic effect and hence, in a severely compromised myocardium, either maternal⁶ or fetal,² there is a risk of death. Although there has not been fetal death attributed to the use of flecainide alone, the numbers are too small at this stage to draw any conclusions about its safety. Therefore, at present, it should be reserved for the treatment of supraventricular tachycardias causing fetal hydrops, in those centres with the necessary

paediatric cardiological and obstetric experience. This experience should include the ability to be able to diagnose correctly the arrhythmia, counsel the mother about the potential risks, and monitor both mother and fetus during and after treatment.

Finally, it is not normal practice in most antenatal clinics to record the fetal heart rate; indeed, some professionals may not listen to the fetal heart at each visit although the overall incidence of fetal dysrhythmias is reported as 2% and therefore not that uncommon.⁷ This case serves to emphasise the need to maintain this simple clinical screening method and to refer the case promptly if a fetal arrhythmia is suspected.

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