Short reports

Fetal cardiac arrhythmia: antepartum diagnosis of a case of congenital atrial flutter

K J ANDERSON, S C SIMMONS, AND K A HALLIDIE-SMITH

Heatherwood Hospital, Ascot, and Hammersmith Hospital, London

SUMMARY A case of antepartum atrial tachyarrhythmia was detected in the 36th week of pregnancy. Cardiotocograph recordings done twice daily enabled close surveillance of the fetal condition after oxytocin challenge testing had failed to show evidence of hypoxia. After a diagnosis of fetal cardiac arrhythmia had been made, elective caesarean section in the 40th week of pregnancy resulted in delivery of an infant in atrial flutter and cardiac failure. Both these problems were soon resolved by cardioversion and subsequent treatment with digoxin. Cardiac catheterisation showed no underlying cardiac abnormality. Increasing use of antenatal cardiotocography may show that intrauterine tachyarrhythmias are more common than had generally been believed.

Case report

A 30-year-old, para 1, gravida 2, blood group O-negative woman, was admitted to Heatherwood Hospital at 35 weeks' gestation because of cardiotocographic evidence of pronounced fetal tachycardia. Her only previous delivery had been in 1976, when induction of labour at 41 weeks' gestation had resulted in a healthy boy weighing 3595 g.

During the current pregnancy there had been no problems until 35 weeks' gestation when the woman drew attention to the fact that fetal movements were diminished. At this hospital all inpatients and any outpatient considered to have a higher than normal fetal risk are monitored at least daily by antepartum cardiotocography. Cardiotocograph tracing showed a fetal heart rate of 180 beats/minute with reduced variability in the baseline but not in the beat-to-beat pattern (Fig. 1). After the woman was admitted an oxytocin challenge test failed to show any fetal heart rate deceleration, thereby excluding fetal hypoxia (Fig. 2). Thereafter the fetus remained active and there was no clinical evidence of hypoxia or fetal growth retardation. Repeated cardiotocograph tracings, at least twice daily, showed the same pattern with the baseline tachycardia being consistently increased at between 180 and 200 beats/minute. On any one tracing however, there was very little variation in the fetal heart rate baseline variability (Figs 1 and 2). Serum oestriol and human placental lactogen assays were within normal limits and x-ray film of the fetus was normal. In view of the overall stability of the fetal condition it was decided to allow the pregnancy to proceed to term.

An elective caesarean section was performed at

Fig. 1 Antenatal cardiotocograph at 35 weeks' gestation.

Fig. 2 Stress cardiotocograph at 35 weeks' gestation.

39 weeks' gestation after the onset of spontaneous labour.

A boy weighing 4020 g was delivered at term with Apgar scores of 9 at one minute and at five minutes. The placental weight was 660 g. A rapid and irregular heart rate of about 200 beats/minute was noted. Ten minutes after delivery, variable cyanosis, hypotonia, and tachypnoea became evident. Arterial pulses were of small volume and hepatomegaly was noted. There was splitting of the second heart sound and a loud apical gallop rhythm, but no murmur was evident. After immediate transfer to Hammersmith Hospital, an electrocardiogram (ECG) showed the baby to be in atrial flutter with varying 2:1 and 3:1 block with a dominant right ventricle and a normal axis. Chest x-ray films showed a large heart with selective enlargement of the right atrium and right ventricle. Arterial blood gases in room air showed P_{aO_2} of 51 mmHg (6.78 kPa), P_{aCO_2} of 30 mmHg (3.99 kPa), and pH of 7.32.

A diagnosis of atrial flutter with a low cardiac output and early cardiac failure was made and at age 12 hours the baby electrically defibrillated to sinus rhythm with 8 joules. He remained in sinus rhythm and the cyanosis slowly settled.

Cardiac catheterisation studies at 13 days showed no evidence of a shunt at any level. Viral studies gave negative results.

The baby was started on oral digoxin immediately after cardioversion. Digoxin was continued for the next 4 months and there were no cardiovascular abnormalities at either the 4- or 12-month follow-up.

Discussion

Persistent fetal tachycardia may not always signify fetal hypoxia, although this diagnosis must always be considered first. In this infant the features that led to the suspicion of fetal hypoxia were the persistently

reduced variability in the baseline fetal heart rate and the absence of spontaneous accelerations. However, an absence of decelerations and the presence of satisfactory beat-to-beat fetal heart rate variability on repeated cardiotocograph recordings, and on oxytocin challenge testing, is substantial evidence against a diagnosis of fetal hypoxia. A provisional diagnosis of fetal cardiac tachyarrhythmia was made and the possibility of an underlying fetal cardiac malformation considered.

An ECG has to be of an exceptional quality if an antepartum diagnosis of cardiac arrhythmia on abdominal fetal recording is to be made. Diagnosis of the exact cause of fetal cardiac arrhythmia by abdominal fetal ECG recording is technically extremely difficult.¹

The first case of atrial flutter diagnosed *in utero* was reported by Blumenthal *et al.*² in 1968. There has been only one antepartum ECG diagnosis of atrial flutter reported and definitive diagnosis required neonatal ECG confirmation.¹ Two other cases have been recorded on ECG using a fetal scalp electrode intrapartum.^{3,4} A further 4 cases of atrial flutter were suspected on auscultation findings alone⁵⁻⁷ bringing the total to 8 cases for atrial flutter recognised antepartum. In these last 4 cases a definitive diagnosis was made in the neonate and cardiotocograph recordings were made during labour, but not antepartum.

Caesarean section was chosen in this case, not because of fetal hypoxia, but because of concern that the pronounced fetal tachycardia might be aggravated by the stress of labour resulting in precipitation or deterioration of fetal congestive cardiac failure. The difficulty in recognising early signs of fetal hypoxia on the cardiotocograph from those persistent abnormalities already present before labour was a factor in favour of elective caesarean section. There was also a great element of maternal anxiety present

which would certainly have been made worse by the presence of a cardiotocograph at the bedside during labour.

Congenital atrial flutter is a potentially serious arrhythmia and of the 8 reported cases which were suspected antepartum, one was associated with hydrops fetalis,⁷ and one with tetralogy of Fallot.⁸ The main risk of atrial flutter in the fetus is that of any underlying cardiac malformation plus the risk of fetal congestive cardiac failure. The associated heart block occurring *in utero* in this case was fairly constant compared with the variable block detected on neonatal ECG recordings.¹ Immediate availability of paediatric intensive care facilities is essential for appropriate management of fetal cardiac arrhythmias, but this baby showed no evidence of acidosis at delivery.

All but one of the 8 recorded cases of atrial flutter diagnosed antepartum required cardioversion, or digoxin, or a combination of both, to control the arrhythmia in the neonate.^{1 5 6 8} A regimen of cardioversion followed by digitalisation as used in this case is the accepted treatment.¹ The alternative of digoxin followed by DC cardioversion has potential drawbacks including secondary cardiac arrhythmia and aggravation of cardiac failure due to delay in version.^{1 5 6 8}

Prognosis for cardioversion of atrial flutter in the neonate is good in the absence of a cardiac lesion.

Furthermore, the prognosis is better if the diagnosis is made antepartum or at birth, than if it develops later in infancy.¹

References

- Shenker L. Fetal cardiac arrhythmias. *Obstet Gynecol Survey* 1979; **34**: 561-72.
- Blumenthal S, Jacobs J C, Steer C M, Williamson S W. Congenital atrial flutter; report of a case documented by intrauterine electrocardiogram. *Pediatrics* 1968; **41**: 659-61.
- Symonds E M. Fetal cardiac arrhythmias and fetal acid base status. *Aust NZ J Obstet Gynaecol* 1972; **12**: 170-5.
- Komáromy B, Gaál J, Lampé L. Fetal arrhythmia during pregnancy and labour. *Br J Obstet Gynaecol* 1977; **84**: 492-6.
- Levkoff A H. Perinatal outcome of paroxysmal tachycardia of newborn with onset *in utero*. *Am J Obstet Gynecol* 1969; **104**: 73-9.
- Herin P, Thoren C. Congenital arrhythmias with supra-ventricular tachycardia in the perinatal period. *Acta Obstet Gynaecol Scand* 1973; **52**: 381-6.
- Van der Horst R L. Congenital atrial flutter and cardiac failure presenting as hydrops foetalis at birth. *S Afr Med J* 1970; **44**: 1037-9.
- Pearl W. Cardiac malformations presenting as congenital atrial flutter. *South Med J* 1977; **70**: 622-4.

Correspondence to Dr K J Anderson, Department of Obstetrics and Gynaecology, Mercy Maternity Hospital, Clarendon Street, East Melbourne, Victoria 3002, Australia.

Received 15 July 1980

Pregnanediols and breast milk jaundice

J F MURPHY, I HUGHES, E R VERRIER JONES, S GASKELL, AND A W PIKE

Department of Child Health, and Tenovus Institute, Welsh National School of Medicine, Cardiff

SUMMARY Samples of breast milk collected from mothers of infants with breast milk jaundice were analysed for 5 β -pregnane-3 α , 20 β -diol, and other pregnanediols using gas chromatography-high resolution mass spectrometry. None was detected in any of the specimens and therefore it is unlikely to be the inhibitory factor in bilirubin conjugation. The plasma osmolalities of the infants, which were determined at the onset of jaundice, were within normal limits.

Breast milk jaundice is a well-known clinical entity. Arias *et al.*¹ reported that breast milk from mothers of jaundiced infants inhibited glucuronyl transferase

activity *in vitro*. The effect was subsequently attributed to a steroid, 5 β -pregnane-3 α , 20 β -diol, isolated from the inhibitory breast milk.² The steroid was shown to inhibit glucuronyl transferase *in vitro* and to produce jaundice when given to newborn infants. Ramos *et al.*³ however, did not observe jaundice in infants given pregnanediol, nor could they identify the steroid in breast milk. Using human liver tissue, Adlard and Lathe⁴ showed that 5 β -pregnane-3 α , 20 β -diol did not inhibit bilirubin conjugation, and Hargreaves and Piper⁵ demonstrated inhibition of bilirubin glucuronide secretion from rat liver. Recent editions of standard paediatric textbooks continue to cite 5 β -pregnane-3 α , 20 β -diol as the inhibitory factor in breast milk jaundice.