Intrauterine supraventricular tachyarrhythmias and transplacental digitalisation

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SUMMARY Six newborn infants with intrauterine supraventricular tachyarrhythmias (five cases of atrial flutter and one of supraventricular tachycardia) are described. Transplacental digitalisation was attempted in three cases. Supraventricular tachycardia associated with hydrops fetalis, detected in a fetus at a gestation of 31 weeks, was successfully converted to normal sinus rhythm eight days after the mother began treatment with digoxin. The serum concentration of digoxin in cord blood almost equalled the maternal concentration in three cases.

In the remaining three cases treatment with digitalis was effective in converting tachyarrhythmias to sinus rhythm after delivery.

With maintenance digoxin therapy, the prognosis of fetal tachyarrhythmias seems to be good, once conversion to sinus rhythm has been accomplished.

Fetal tachyarrhythmias have been cited as possible causes of non-immune hydrops fetalis and intrauterine fetal death.¹ Transabdominal fetal electrocardiography is fraught with difficulty and therefore of limited value in the analysis of fetal cardiac arrhythmias. On the other hand, fetal echocardiography is a non-invasive means of not only diagnosing fetal cardiac structural anomalies and rhythm disturbances but also detecting associated congestive heart failure in utero.²⁻⁵

A number of authors have reported success with transplacental pharmacological treatment to convert intrauterine supraventricular tachyarrhythmias to a normal sinus rhythm.³⁻⁶⁻¹⁴ In such cases fetal echocardiography is a useful tool for serial monitoring of cardiac rhythm and evaluation of antiarrhythmic treatment.

There have been only a few reports on fetal

tachyarrhythmias and transplacental pharmacological treatment in several cases.¹⁵⁻¹⁷ This report describes the clinical course of six newborns with intrauterine supraventricular tachyarrhythmias and, in three cases, the results of transplacental digitalisation.

Patients and methods

Between 1977 and 1985 six newborn infants with intrauterine supraventricular tachyarrhythmias were admitted to our hospitals and were given antiarrhythmic treatment (Table 1).

In three cases, after the obstetricians had detected fetal tachycardia, fetal echocardiography was performed, and the mothers received antiarrhythmic agents.

Fetal echocardiography was performed trans-

Table 1 *Clinical findings of the six cases*

Case No	Sex	Gestational age on recognition (weeks)	Fetal heart rate (beats/min)	Tachyarrhythmias	Congestive heart failure
1	M	39 (19 hours antepartum)	180-200	Atrial flutter	(+)
2	M	40 (two hours antepartum)	200	Atrial flutter	(+)
3	M	38 (intrapartum)	210	Atrial flutter	(+)
4	M	30	240	Atrial flutter	Hydrops fetalis
5	M	41 (one day antepartum)	80-200	Atrial flutter	(-)
6	F	31	300	Supraventricular tachycardia	Hydrops fetalis

abdominally with a 5 MHz linear transducer. An M mode study was performed to evaluate disturbances in cardiac rhythm after two dimensional echocardiographic analysis of the cardiac structures—cardiac chambers, interventricular and interatrial septa, and atrioventricular and semilunar valves, which could be readily obtained.

The P wave of the surface electrocardiogram represented A wave of the fetal atrioventricular valves, and the QRS wave complex was shown as atrioventricular closure or semilunar valve opening. The cardiac phases could be presumed by motion of the cardiac walls or valves.

The diagnosis of the fetal arrhythmias was made mainly by the motion of these cardiac structures.

Five of the six newborn infants were boys, and all were full term (38–41 weeks' gestation).

Results

In four cases the fetal tachyarrhythmia was first recognised shortly before birth or during delivery.

In case 2 fetal tachycardia with a heart rate of 200 beats/minute was first noted at 40 weeks' gestation. In the belief that the fetus was distressed the obstetrician performed caesarean section. An elec-

trocardiogram recorded immediately after birth showed atrial flutter with 2:1 atrioventricular conduction.

In case 5 a fetal irregular rhythm with a rate of 80 to 200 beats/minute was first noted one day before birth. A fetal echocardiogram showed atrial flutter with 2:1 to 5:1 atrioventricular conduction. Hydrops fetalis was not detected.

In cases 4 and 6 hydrops fetalis with massive ascites, pleural effusion, and subcutaneous oedema was present at gestational weeks 30 and 31, respectively. Case 6 had no oedema on delivery, however, because the supraventricular tachycardia had reverted to normal sinus rhythm before delivery as a result of transplacental digitalisation.

Table 2 Serum concentration of digoxin in cases 4, 5, and 6

Case No	Gestational age (weeks)	Digoxin dose on delivery (mg/day)	Serum concentration of digoxin (ng/dl)	
			Maternal blood	Cord blood
4	30	0.5 (oral)	1.00	0.62
5	41	0.5 (intravenous)	1.20	1.30
6	31	0.25 (oral)	0.30	0.36

Fig. 1 Clinical course of case 6.

Fetal echocardiography disclosed atrial flutter with 2:1 atrioventricular conduction at a heart rate of 240 beats/minute in case 4 and supraventricular tachycardia with 1:1 atrioventricular conduction at a heart rate of 300 beats/minute in case 6.

Shortly after birth, cases 1, 2, and 3 had evidence of mild cardiomegaly on chest radiographs and moderate hepatomegaly. No signs of congestive heart failure were observed in case 4 in the perinatal period.

Structural abnormalities were ruled out by post-natal echocardiography and electrocardiography in all cases.

Treatment with digoxin was begun immediately after diagnosis of supraventricular tachycardia. Emergency injections of digoxin were administered to three infants (cases 1, 2, and 3) after delivery. The atrial flutter stopped 20 minutes after injection of digoxin in case 1 but persisted for two days in case 2 and five days in case 3.

Transplacental digitalisation was attempted in three cases (cases 4, 5, and 6). In case 4 fetal tachyarrhythmia was detected at 30 weeks' gestation and hydrops fetalis developed at 34 weeks. Transplacental digitalisation was attempted and atrial flutter converted briefly to a sinus rhythm. Atrial flutter returned, however, and persisted, without improvement of hydrops fetalis. Caesarean section was performed at 36 weeks' gestation. The serum concentration of digoxin in cord blood is shown in Table 2. Successful electrical cardioversion to a sinus rhythm was achieved immediately after birth. In case 5 fetal tachyarrhythmia was detected at 41 weeks' gestation. Although the mother was digital-

ised for two days, atrial flutter persisted, but it spontaneously converted to sinus rhythm, however, 10 minutes after normal delivery.

Case 6. The details of the clinical course of case 6 are as follows (Fig. 1).

At 31 weeks' gestation a 27 year old primigravida noticed a sudden decrease in fetal movement. Her obstetrician noted fetal tachycardia and referred the patient to our hospital.

Fetal echocardiography performed on the same day revealed fetal tachycardia of 300 beats/minute with 1:1 atrioventricular conduction (Fig. 2). There was no cardiac structural abnormality, and scalp oedema and ascitic fluid were absent.

Digoxin (0.25 mg twice a day intravenously) was administered to the mother. On the day after admission fetal echocardiograms showed persistent supraventricular tachycardia and a small amount of ascitic fluid and pleural effusion. On the sixth day massive ascites, pleural effusion, and subcutaneous oedema were present (Fig. 3). A brief episode of normal heart rate (130 beats/minute) occurred on the seventh day, when the maternal serum digoxin concentration was 1.02 ng/ml. Intravenous administration of digoxin was changed to oral administration on the eighth day, and the normal sinus rhythm returned to tachycardia. Shortly after intravenous digitalisation was restarted, the tachycardia converted to normal sinus rhythm, which continued thereafter.

Fetal echocardiography showed complete resolution of hydrops fetalis one week after normal sinus rhythm was established. The mother complained of

Fig. 2 Two dimensional and M mode echocardiogram from case 6 on the first hospital day. Rapid motion of the mitral valve shows atrial and ventricular rates of 300 beats/minute. No pleural effusion was present. LA: Left atrium; RV: right ventricle; RA: right atrium; MV: mitral valve.

Fig. 3 Two dimensional and M mode echocardiogram from case 6 on the sixth hospital day. Massive pleural effusion was present. LA: Left atrium; RV: right ventricle; RA: right atrium; LV: left ventricle; MV: mitral valve, PE: pleural effusion.

palpitations on the 13th day of admission to hospital, and her electrocardiogram showed Wenckebach second degree atrioventricular block and digitalis effect of ST-T. These phenomena were considered to be due to digitalis intoxication. Maternal serum digoxin concentration was 1.29 ng/ml. Digoxin intoxication subsided after the oral dosage was reduced to 0.25 mg once a day. The patient was discharged at this dosage, and there was no recurrence of fetal tachycardia. A girl weighing 3791 g was delivered vaginally at 40 weeks' gestation.

The serum concentrations of digoxin in the umbilical cord blood and maternal blood on delivery were 0.36 ng/ml and 0.30 ng/ml, respectively. The infant's electrocardiogram showed Wolff-Parkinson-White syndrome (type A) and no structural defect was observed by echocardiography. Prophylactic digoxin has been administered for one year without recurrences of supraventricular tachycardia.

Discussion

Before the era of fetal echocardiography, tachyarrhythmias occurring in utero were often interpreted as fetal distress, and often caesarean sections or prematurely induced labour were performed.¹⁸

Recent development and improvement of fetal echocardiography has made it possible to study fetal cardiac structure, monitor fetal rhythm disturbance, and administer appropriate transplacental pharmacological treatment.¹⁵

Newburger *et al* reported that congestive heart failure was evident, at birth or shortly after birth, in 62% of infants with intrauterine supraventricular tachycardias.¹⁵ Deaths due to intrauterine tachycardias have also been reported.^{3 5 11}

To our knowledge, successful pharmacological cardioversion has been achieved in about 10 cases (Table 3).^{3 6-14} In most of the successful cases,

Table 3 Reported cases of successful transplacental pharmacological cardioversion

	Year of publication	Gestational week	Tachyarrhythmia	Antiarrhythmic agent
Teuscher ¹⁴	1978	34	Tachycardia (> 200 beats/min)	Propranolol
Kerenyi	1980	29-30	Supraventricular tachycardia	Digoxin
Lingman ¹²	1980	29	Supraventricular tachycardia	Digoxin
Harrigan ⁷	1981	26	Tachycardia (> 260 beats/min)	Digoxin
Dumesic ⁶	1982	26	Tachycardia (> 240 beats/min)	Digoxin
				Propranolol
				Procainamide
Kleinman ⁴	1982	27	Supraventricular tachycardia	Digoxin
Heaton ⁸	1982	30	Supraventricular tachycardia	Digoxin
Kleinman ¹¹	1983	35	Supraventricular tachycardia	Digoxin
Iliff ¹⁰	1983	36	Tachycardia	Digoxin
Hirata ⁹	1985	34	Atrial flutter	Digoxin
Present study	1985	31	Supraventricular tachycardia	Digoxin

digoxin was administered to the mothers because it has been confirmed that digoxin crosses the placenta and that the fetal serum digoxin concentration approximates to that of the mother.¹⁹ A few cases of fetal tachycardias treated successfully with propranolol¹⁵ or procainamide⁶ have been reported. In newborn babies, however, the possible side effects of propranolol include depressed Apgar scores, hypoglycaemia, and episodes of bradycardia,²⁰ and therefore propranolol is not the drug of first choice for fetal supraventricular tachycardias. The effects of other drugs, such as procainamide and verapamil,¹⁰ have not been confirmed, because they have been administered to only a few patients.^{6 9 14}

In three cases of intrauterine tachyarrhythmias we attempted to convert atrial flutter and supraventricular tachycardia to normal sinus rhythm by administering digoxin. In case 6 fetal supraventricular tachycardia recovered on administration of digoxin to normal sinus rhythm in utero and did not recur until birth.

Although digoxin prevented atrial flutter temporarily in case 4, atrial flutter recurred and the mother underwent caesarean section at 36 weeks' gestation.

It is easier and safer to treat supraventricular tachycardia after than before delivery, because we have better methods for postpartum treatment, including electrical cardioversion and precise pharmacological monitoring. When an immature fetus with supraventricular tachycardia has a poor chance of extrauterine survival, however, due to immaturity, transplacental pharmacological cardioversion should be attempted.

Because of the potential risk of congestive heart failure in utero, we attempted transplacental digitalisation in case 5, but extrauterine treatment might have been more appropriate as the fetus was full term.

Once fetal atrial supraventricular tachyarrhythmia had been converted to normal sinus rhythm, no patient in our series experienced recurrent tachyarrhythmia during the first year of life with preventive administration of digoxin. As the prognosis of these supraventricular tachyarrhythmias seems to be good, preventive treatment is recommended for a minimum of one year.

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