Non-immune hydrops foetalis due to foetal tachyarrhythmia—management with transplacental amiodarone

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INTRODUCTION

Non-immune hydrops foetalis (NIH) can result from a variety of causes and has a poor outcome with reported overall survivals ranging from 10% to 27.3%.^{1,2} An exception is NIH due to supraventricular tachycardia which is amenable to therapy if picked up early. We report the successful management of a foetus with NIH due to supraventricular tachycardia treated with transplacental amiodarone at our centre.

CASE REPORT

A 29-year second gravida woman was detected to have polyhydramnios and hydrops foetalis with persistent foetal tachycardia (FHR > 220/min) on obstetric ultrasound done at 30 weeks gestation. The mother's blood group was B RhD positive. Features of hydrops foetalis included foetal ascites, right-sided pleural effusion, generalised subcutaneous oedema with skin fold thickness of 0.6 mm (normal skin fold thickness < 0.5 mm) and irregular cardiac activity (HR = 130/min) with sudden increases to 270/min. Repeat ultrasound done a week later showed persistence of polyhydramnios with foetal hydrops. The woman was transferred to our centre.

At our centre, repeat ultrasound confirmed the above findings. Foetal echocardiography revealed supraventricular tachycardia and pericardial effusion but no structural cardiac disease (Figure 1).

The mother was started on intravenous amiodarone (150 mg bolus over 30 min followed by 1 mg/min for next 6 hours followed by 0.5 mg/min for the next nine hours) and followed up

with oral amiodarone 200 mg eight hourly with close monitoring. Subsequent repeat ultrasound examinations showed resolution of pericardial effusion, pleural effusion and ascites over the next week (Figure 2) with a normal foetal heart rate. Amiodarone was continued orally until she delivered and then gradually tapered off. Repeat foetal echocardiography done 48 hours after the parenteral amiodarone showed resolution of tachyarrhythmia (HR 130–140/min).

The woman delivered a healthy male neonate at 36 weeks period of gestation through elective caesarean section. The birth weight was 2250 g and APGAR scores 7/10 and 9/10 at 1 and

Figure 1 Foetal echocardiograph showing a pericardial effusion marked (PE).

Figure 2 Foetal echocardiograph depicting right atrial (RA) and right ventricle (RV) dilatation.

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5 min respectively. There were no obvious congenital anomalies. Observation in the neonatal intensive care unit for 72 hours ruled out any tachyarrhythmia. The heart sounds were normal and there was no murmur. Chest radiograph and electrocardiogram were normal. Echocardiography done at 48 hours of life showed no tachycardia, no pericardial effusion and there was no structural heart disease. The baby was roomed in with the mother and discharged on day six of life. Thyroid function tests were normal and the baby was followed up monthly for thyroid dysfunction.

On review at three months age, the infant was healthy with heart rate between 130–140/min and a normal echocardiography. Parents have been advised regular review for the infant until two years of age.

DISCUSSION

Foetal tachycardia is defined as foetal ventricular heart rate in excess of 180/min and includes sinus tachycardia, supraventricular tachycardia and atrial flutter.³ If undiagnosed and untreated, foetal tachycardia may lead to low-output cardiac failure and hydrops, thereby increasing foetal morbidity and mortality.^{4–6}

Ultrasonography can diagnose both, the abnormal foetal heart rate as well as the hydrops.^{5,6} However, since foetal tachyarrhythmias are amenable to transplacental as well as direct foetal pharmacologic therapy, it is essential that the pregnant woman be transferred to a tertiary care centre with facilities for foetal echocardiography and therapy.⁷ This was done for our patient.

Foetal echocardiography is essential to diagnose the exact nature of tachycardia, to rule out underlying structural defects and to monitor response to therapy.^{3,8} Our patient was diagnosed to have supraventricular tachycardia (SVT) without any underlying structural cardiac defects.

Transplacental therapy of foetal SVT is achieved by the administration of anti-arrhythmic agents to the mother. Digoxin is the usual first-line drug while sotalol, flecainide and amiodarone have been used as second-line drugs.^{3,4,9} In a review of published studies, out of 314 cases of foetal SVT, Krapp and colleagues reported digoxin to be significantly less effective in the treatment of SVT in foetuses with hydrops (24.6% success) as compared to foetuses without hydrops (65.4% success).⁹ Another study on refractory cases of foetal tachycardia reported a success rate of 93% in the treatment of foetal SVT in hydropic foetuses using amiodarone.¹⁰ We used amiodarone as a drug for the first-line of treatment of our patient since hydrops had already developed.

Amiodarone acts by prolonging the repolarisation of the myocardium and is a Class III anti-arrhythmic agent. Transplacental therapy with amiodarone has been proven effective and serious

maternal side effects have not been reported.¹¹ We resorted to intravenous maternal administration of the drug with close monitoring for maternal side effects like thrombocytopenia and photosensitive skin rash. After 48 hours of start of therapy, the foetal heart rate normalised with regression of hydrops over the next few days. There were no maternal side effects of amiodarone in our patient and no thyroid dysfunction in the baby.

The aim of reporting this case is to highlight the fact that foetal tachycardia can lead to cardiac failure and non-immune hydrops in the foetus. Early recognition and prompt in-utero transfer of such patients to a tertiary care centre can enable transplacental therapy with monitoring. This will considerably improve outcomes in an otherwise serious foetal condition.

CONFLICTS OF INTEREST

None.

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