Prenatal Diagnosis and Management of Fetal Atrial Flutter: A Case Report

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Fetal atrial flutter (AF) occurs in less than 1% of all pregnancies. It seems to have a poor prognosis, due in part to its association with structural heart disease and with development of hydrops fetalis. Early prenatal detection and treatment are essential to improve the outcome. We present a case of fetal AF coexistent with 2:1 atrioventricular block treated with intrauterine digoxin and quinidine. Under the impression of impending heart failure, a male infant who was small for gestational age was delivered by cesarean section. The infantile heart was restored to a sinus rhythm after a single electrocardioversion and a short period of digitalization. He did not need anti-arrhythmic medication. He has demonstrated normal development up to the time of writing (2 years old). The prenatal cardioechographic findings, modality of treatment, and outcome are discussed.

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KEY WORDS: • prenatal • atrial flutter • digitalization • ultrasound

INTRODUCTION

Fetal tachyarrhythmias occur in 1% to 2% of all pregnancies [1]. Fetal atrial flutter (AF) is the second most common fetal tachyarrhythmia among congenital arrhythmias diagnosed prenatally [2]. Recognition of fetal AF has increased in the past two decades because of the common use of obstetric ultrasound, prenatal heart rate monitoring, and close observation of high-risk obstetric patients [3]. AF is associated with re-entrant supraventricular tachycardia, atrial septal defect, hypoplastic left heart syndrome, Ebstein’s malformation of the tricuspid valve, and cardiomyopathy [4]. Fetal hydrops is present in 38.6% of all fetuses with AF, especially in re-entrant supraventricular tachycardia, when the ventricular rate exceeds 210 bpm [5]. AF seems to have a poor prognosis, due in part to its association with structural heart disease (in up to 20% of cases) and with the development of hydrops fetalis [6]. Early detection and treatment are essential to improve outcome. We present the case of a patient with fetal AF who received both intrauterine and postnatal treatment.

CASE REPORT

A healthy, 32-year-old female, gravida 3, para 0, had uneventful prenatal examinations until 34 weeks of gestation, when fetal tachycardia was found; she was then referred to our hospital. M-mode ultrasound revealed an atrial rate of 462 bpm and a ventricular rate of 231 bpm. AF with 2:1 atrioventricular block was diagnosed (Figs. 1–3). High-dose

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rapid digitalization (digoxin 0.25 mg intravenously, iv, every 6 hours, q6h) was prescribed. Three days later, quinidine (200 mg oral q6h) was added due to persistent fetal tachycardia. Despite medical treatment with digoxin and quinidine for 1 week, fetal cardioechogram showed persistent AF with tricuspid regurgitation and pericardial effusion. Cesarean section was arranged at 36 weeks of gestation for impending heart failure. A 2,120 g male infant was born with Apgar scores of 7 at 1 minute and 9 at 5 minutes.

Neonatal electrocardiogram revealed AF and 2:1 atrioventricular block. Parenteral digitalization (digoxin 20 + g iv) was commenced approximately 1 hour after delivery. As persistent arrhythmia and frequent episodes of hypotension were noted, cardioversion was performed, leading to a sinus rhythm of 120 bpm. Postnatal cardiogram showed a 3.2 mm patent ductus arteriosus, a 5 mm atrial septum defect, and a 67% left ventricular ejection fraction. After cardioversion, a maintenance dose of digoxin (6 µg q12h iv) was given for 1 day, after which, as there was no further attack of arrhythmia, the dose was tapered. Since discharge, the infant was followed up closely without medication at pediatrics clinics, and has demonstrated normal development up to the time of writing (at 2 years of age).

**DISCUSSION**

AF accounts for one-fifth to one-third of all fetal tachyarrhythmias [7]. It is defined as a rapid regular atrial rate of 300 to 600 bpm accompanied by variable degrees of atrioventricular block, resulting in slower ventricular rates [3]. In M-mode ultrasound, the atrial rate is faster than the ventricular rate, and there is intermittent (often 2:1) atrial-to-ventricular conduction.

Prenatal management and prognosis of a fetus with AF depend on the gestational age at onset, and the development of heart failure or hydropic change. Where there is no evidence of heart failure, or if the tachycardia is intermittent, it may be reasonable to observe and deliver the affected fetus near term [2,7,8]. If the AF can lead to heart failure and there is a high risk of intrauterine or neonatal death, aggressive treatment is necessary. Our case had persistent tachycardia with pericardial effusion and was a candidate for prenatal therapy. Krapp et al reported successful treatment of non-hydropic fetuses
with AF in 50% to 55% of cases with digoxin alone [5]. If AF persists, then consideration should be given to the addition of flecainide or verapamil under close monitoring. The prognosis of the hydropic fetus is considerably worse and less responsive to therapy than that of the non-hydropic fetus. Fetuses that die in utero have associated fetal hydrops, structural cardiac defects, or both. Lisowski et al reported a success rate with anti-arrhythmic drugs (of which digoxin and flecainide are the first choices for fetal tachycardia) of 43.3% and 80% in hydropic and non-hydropic groups, respectively [8].

Our patient did not receive direct fetal therapy with digoxin because the pregnancy was near term and the fetus did not respond to initial transplacental therapy. Postnatal direct treatment may achieve a sinus rhythm with fewer potential adverse effects to the mother and the fetus. After birth, a normal sinus rhythm was achieved successfully through electrocardioversion. Further prospective studies are needed to disentangle the effects of preterm delivery versus intrauterine management of fetal AF with hydrops.

REFERENCES