Fetal Tachyarrhythmia with 1:1 Atrioventricular Conduction. Adenosine Infusion in the Umbilical Vein as a Diagnostic Test

Tiago L. L. Leiria, Gustavo G. de Lima, Rejane F. Dillenburg, Paulo Zielinsky

Porto Alegre, RS - Brazil

This is the report of a case of fetal tachyarrhythmia with 1:1 atrioventricular conduction detected by pre-natal echocardiography in a fetus at 25-weeks gestation. Adenosine infusion via cordocentesis was performed as a diagnostic test to differentiate between atrioventricular nodal reentrant supraventricular tachyarrhythmia and atrial flutter. After infusion, transient 2:1 atrioventricular dissociation was obtained and the diagnosis of atrial flutter was made. Transplacental therapy with digoxin and amiodarone was then successfully used.

The diagnosis of fetal tachyarrhythmias is a challenge, and it is based on the use of echocardiography [1,2]. Fetal supraventricular tachyarrhythmias can be divided into two types according to their arrhythmogenic mechanism: those dependent on the atrioventricular node and those that are independent from it [3]. The early tachycardias are of the reentrant type mediated by an accessory pathway or nodal reentrant tachycardias. The second type includes atrial flutter (the most frequent), atrial fibrillation, multifocal atrial tachycardia, sinus nodal reentrant tachycardia, and sinus tachycardia. Recently, adenosine has been used to treat fetal arrhythmia [4,5]. It is a puridic nucleoside that slows the conduction velocity within the atrioventricular node, thus stopping the reentrant circuit. As a diagnostic agent, it can be used to differentiate between supraventricular tachycardias by reentry in the atrioventricular node by an accessory pathway from a 1:1 atrial flutter.

Case Report

A 31-year-old white Caucasian female in her 25th week of gestation was seen at the Fetal Cardiology Unit of the Instituto de Cardiologia do Rio Grande do Sul because, during a routine obstetric visit, fetal tachycardia was diagnosed and the heart rate was described as being over 200bpm.

Fetal echocardiography showed a supraventricular tachycardia with 1:1 atrioventricular conduction and a heart rate over 300bpm (fig. 1). The fetus was hydropic and no structural cardiac abnormality was detected. It was then decided to give a transplacental attack dose of digoxin (1mg/day).

On the third day of treatment, significant deterioration was observed, with the development of heart failure, detected on the echocardiogram by the presence of ascites, as well as pleural and pericardial effusions. Therefore, adenosine, via cordocentesis, was given to promote immediate and transient decrease of the heart rate; it went from 340bpm to 170bpm, with a 2:1 atrioventricular block, which allowed the diagnosis of atrial flutter to be made (fig. 2).

Because transplacental therapy is often ineffective in the presence of fetal hydrops, amiodarone via cordocentesis (15mg/kg) was given and the arrhythmia disappeared. Because atrial flutter recurred during the procedure, the fetus was given digoxin (0.03mg/kg) as well as a new dose of amiodarone (15mg/kg), with reversion to sinus rhythm. Occasional episodes of atrial flutter of short duration were observed 10 hours after the procedure. Transplacental therapy with intravenous amiodarone (1.3g/day), in addition to oral digoxin (0.25mg/day), was initiated. A third cordocentesis was performed a week later, with reversal of the arrhythmia to sinus rhythm with the administration of 15mg/kg of amiodarone.

After five days, signs of fetal hydrops could no longer be seen. Digoxin was withdrawn on the 15th day and a daily oral dosage of 200mg of amiodarone was instituted for the mother. Follow-up fetal echocardiograms showed sinus rhythm with a heart rate of 130bpm until the patient was discharged.

Discussion

The diagnosis of fetal tachyarrhythmia is suggested when the fetal heart rate is over 200bpm [1]. Fetal tachyarrhythmias occur in 0.4-0.6% of pregnancies and frequently they may be associated with heart failure and fetal hydrops, with consequent high rates of mortality [5-7]. M-mode fetal echocardiography is a very useful method for defining the diagnosis of fetal arrhythmias because it allows the identification of the temporal relationship between atrial and ventricular contractions [8]. Depending on this relationship, the tachyarrhythmias can be divided into two groups: the group with atrioventricular dissociation and that with 1:1 atrioventricular conduction [3].

Instituto de Cardiologia do Rio Grande do Sul/Fundação Universitária de Cardiologia
Mailing address: Paulo Zielinsky - Instituto de Cardiologia do RS – Unidade de Pesquisa - Av. Princesa Isabel, 395 - 90620-001 - Porto Alegre, RS - Brazil

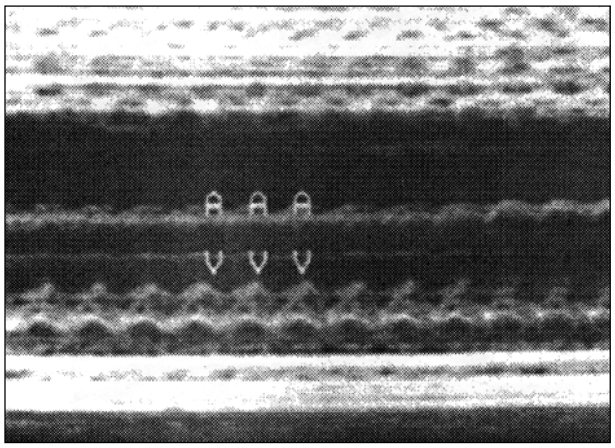

Fig. 1 – M-mode fetal echocardiogram showing atrial flutter with 1:1 atrioventricular conduction (heart rate >300bpm). A- atrial systole; V- ventricular systole.

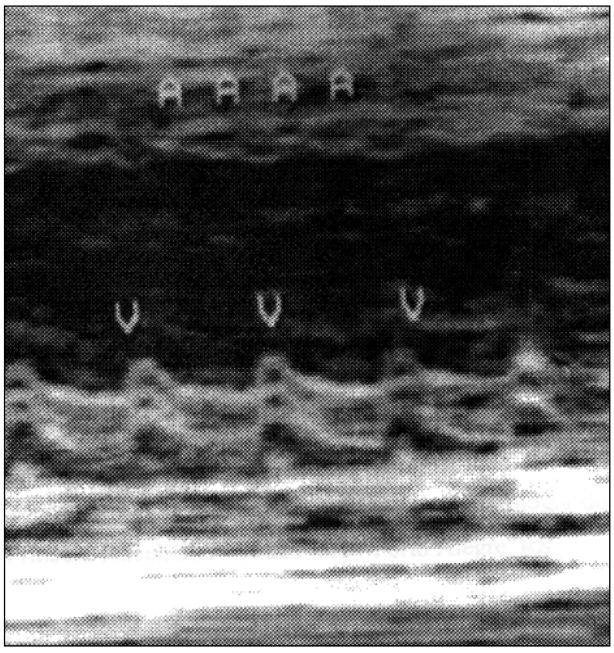

Fig. 2 – Fetal atrial flutter with a transient 2:1 atrioventricular block after direct infusion of adenosine via cordocentesis. A- atrial systole; V- ventricular systole.

The atrioventricular node is an integral part of the arrhythmogenic circuit, both in the atrioventricular reentries and in nodal reentries. Because of that, these arrhythmias are classified as nodal-dependent. However, in atrial tachycardia, flutter, and fibrillation, the AV node is not involved in the arrhythmogenic circuit. Because of the irregular pattern and because of its rare occurrence in the normal fetal heart, atrial fibrillation is usually not part of the differential diagnosis. Thus, the so-called nodal-independent arrhythmias, with a regular rhythm and a higher probability of occurrence in the prenatal period, are atrial tachycardia or flutter. Atrial flutter is suspected when the atrial contraction rate is over 300bpm, but in atrial tachycardia the heart rate is usually between 200 and 280bpm.

We have described a case of fetal supraventricular tachycardia causing hydrops, with a regular and fast 1:1 atrioventricular conduction. Adenosine was used to depress the atrioventricular conduction and cause atrioventricular dissociation, thus allowing the diagnosis of atrial flutter to be made and the adequate therapy to be initiated. The administration of adenosine is a useful diagnostic tool for establishing the differential diagnosis between supraventricular tachycardia (mediated by an accessory pathway or by nodal reentry) and atrial flutter with a 1:1 atrioventricular conduction.

References

1. Allan LD, Anderson RH, Sullivan ID, Campbell S, Holt DW, Tynan M. Evaluation of fetal arrhythmias by echocardiography. Br Heart J 1983; 50: 240-5.
2. Soyer DJ. Atrial Flutter in the Human Fetus: Diagnosis, Hemodynamic Consequences and Therapy. J Cardiovasc Electrophysiol 1996; 7: 989-98.
3. Naheed ZJ, Strasburger JF, Deal BJ, Benson W, Gidding SS. Fetal tachycardia: mechanisms and predictors of hydrops fetalis. J Am Coll Cardiol 1996; 27: 1736-40.
4. Blanch G, Walkinshaw SA, Walsh K. Cardioversion of fetal tachyarrhythmia with adenosine. Lancet 1994; 344: 1646.
5. Bergmans MGM, Jonker GJ, Kock HCLV. Fetal supraventricular tachycardia. Review of the literature. Obstet Gynecol Surv 1985; 40: 61-8.
6. Engelen AD, Weijtens O, Brenner JI, et al. Management outcome and follow up of fetal tachycardia. J Am Coll Cadiol 1994; 24: 1371-5.
7. Zielinsky P, Dillenburg RF, de Lima GG, Zimmer LP. Taquiarritmias supraventriculares no feto: experiência de uma unidade de referência em cardiologia. Arq Bras Cardiol 1998; 70: 337-40.
8. Zielinsky P. Distúrbios do ritmo cardíaco fetal. Detecção e conduta pré-natal. Arq Bras Cardiol 1996; 66: 83-6.