Management of Fetal Tachyarrhythmias

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Introduction

Fetal tachyarrhythmias, although relatively rare, are an important cause of fetal morbidity and mortality [1–4]. They are defined as a fetal heart rate ≥ 180 bpm [2], but most often are in excess of 200 bpm [4]. Although sustained tachycardia (present ≥ 12 consecutive hours) [5] has been known to be a risk factor for fetal congestive heart failure [3], intermittent tachycardias can also be associated with hydrops [6]. The majority of fetal tachycardias are due to supraventricular tachycardia (SVT), which shows 1:1 atrioventricular (AV) contraction sequence, abrupt onset and termination, and minimal (within 10 bpm range) or no heart rate variability. Almost 90% of SVTs in fetuses are due to AV reentrant tachycardia via the common type of accessory pathway [3]. SVTs due to AV nodal, permanent junctional, or multiple reentrant pathways, as well as due to automatic ectopic focus confined to atrium, are much less common during fetal age [3,7,8,9]. However, atrial flutter due to intra-atrial reentrant mechanism is common next to AV reentrant SVTs [2]. In atrial flutter, the atrial rate ranges from 300 to 500 bpm with variable AV block, leading to a ventricular rate ranging from normal to 300 bpm. The ventricular rate is often irregular in contrast to minimal variability seen in SVTs. Junctional ectopic tachycardia and ventricular tachycardia are rare in fetal life, are characterized by ventricular rate in excess of the atrial rate, and are often difficult to distinguish from each other.

Opinion statement

Fetal tachyarrhythmias are an important cause of fetal morbidity and mortality. The majority of fetal tachyarrhythmias are due to atrioventricular reentrant type of supraventricular tachycardia and atrial flutter. Fetal echocardiography remains the main tool of diagnosing and discerning the mechanism of tachyarrhythmia. The goals of therapy for fetal arrhythmias are to restore sinus rhythm, resolve heart failure, and postpone delivery before term. Although there is no anonymity in the approach to the drug treatment of fetal tachycardia, digoxin is the most commonly employed first-line antiarrhythmic drug for supraventricular tachycardia. In digoxin nonresponders, flecainide (± digoxin) controls tachyarrhythmia with high conversion rate. A combination of digoxin and sotalol has proved effective therapy for atrial flutter, but the proarrhythmic side effect of sotalol on the fetus has been a concern. Amiodarone has emerged as a second-line treatment after digoxin failure in nonhydropic fetuses and the most effective treatment for drug-refractory fetal tachycardia accompanied by hydrops. Both the fetus and mother should be closely monitored for the response and adverse effect of the treatment. The antiarrhythmic treatment for supraventricular tachycardia should be continued after birth and during infancy due to the high incidence of postnatal recurrence.

DIAGNOSIS

Currently, fetal tachycardias are diagnosed by M-mode, pulsed Doppler, or color flow encoded M-mode fetal echocardiography. The M-mode, with the cursor oriented across atrial and ventricular cavities, reveals the relationship of atrial and ventricular contractions and their individual rate of contraction (Fig. 1). Using M-mode echocardiography, a recent study has differentiated fetal SVTs according to the ventriculoatrial (VA)
The majority of SVTs had short VA times, which were characteristic of AV reentry via the common type of accessory pathway. The long VA time was typical of permanent junctional reentrant tachycardia or atrial ectopic tachycardia. Alternatively, pulse Doppler interrogation with a wide sample placed at mitral-aortic continuity demonstrates the rate of and relationship between inflow and outflow. Thus, it can reveal the diagnosis of SVT versus atrial flutter with AV block (Fig. 2). More recently, a new technique, fetal magnetography, has demonstrated an effective, noninvasive means of analyzing complex fetal arrhythmia, which can potentially overcome some of the shortcomings of fetal echocardiography [7••,10,11]. It can aid in the differential diagnosis of ventricular tachycardia with 1:1 conduction versus SVTs and ventricular tachycardia with AV dissociation versus junctional ectopic tachycardia [7••]. In addition to discerning the mechanism of the fetal tachycardia, a thorough fetal echocardiographic evaluation should be undertaken to evaluate the cardiac structure, function, associated cardiac defects, hemodynamics, and presence of fetal hydrops. Most fetuses with tachyarrhythmia have structurally normal hearts. Structural malformations of the heart are seen in up to 5% of the fetuses, which most frequently include Ebstein anomaly of the tricuspid valve, AV canal defect, hypoplastic left heart syndrome, and rhabdomyoma [1,2,4]. Ventricular dysfunction can occur and the severity of tachycardia-induced ventricular dysfunction is reflected by the degree and persistence of AV valve incompetence and alternations of venous blood flow (pulsatile reversal of blood flow) in the inferior vena cava and ductus venosus [12,13]. Fetal hydrops, characterized by the presence of any two or more of the following signs—skin edema, ascites, pericardial effusion, and pleural effusion—carries a much higher mortality than in the nonhydropic fetus.

**INDICATIONS FOR HOSPITALIZATION**

Mothers carrying fetuses with incessant tachycardia, tachycardia and major heart defects, or hydrops should be hospitalized to initiate the treatment with a loading dose of antiarrhythmic agent, for close monitoring for adverse effects, and to modify the treatment, including using direct fetal therapy if necessary. The goal is to achieve the control of fetal cardiac rhythm and rate without losing time. Continuous cardiac telemetry, serial electrocardiogram (ECG) to assess the cardiac effects of the drug, serum chemistries, and repeated serum drug concentrated in the mother should be obtained. Both the mother and fetus should be closely observed for any adverse effect of the treatment. Once the fetal cardiac rhythm or rate is controlled and maintenance therapy is instituted, the mother can be discharged for regular outpatient follow-up.

**Treatment**

- In the absence of a fully known natural history and well-designed trial about the management strategy for fetal tachyarrhythmia, there is no anonymity in the approach to the treatment of fetal tachycardia [14•]. However, fetal tachyarrhythmia, if untreated, can result in 8% to 30% of fetal and neonatal