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Perinatal management and long-term cardiac outcome in fetal arrhythmia

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ABSTRACT

Background: Cardiac arrhythmias are commonly observed in the fetus, however, may have major consequences for fetal development and post natal life.
Aims: To evaluate the perinatal management and cardiac outcome of fetuses with tachy- or bradyarrhythmia.
Study design: Perinatal management, outcome and long-term cardiac follow-up were evaluated retrospectively in consecutive fetuses with cardiac arrhythmias.
Results: Forty-four fetuses were diagnosed: supraventricular tachycardia (SVT, n = 28), atrial flutter (AF, n = 7) and atrioventricular block (AVB, n = 9). The overall incidence of cardiac anomalies was 18% mainly in the AVB group; hydrops was present in 34%. Direct or transplacental fetal anti-arrhythmic medication was given in 76%. Mortality was 6% in SVT/AF and 78% in the AVB group, respectively. AF resolved in all patients. In the SVT group, Wolff–Parkinson–White (WPW) syndrome was present in 21%, diagnosed at birth or later in life. After the age of one year about 90% of patients in the SVT group remained asymptomatic and free of drugs (median follow-up 76 months).
Conclusions: Mortality rate is low in patients with fetal SVT and AF but high in patients with AVB. Related morbidity includes WPW-syndrome and congenital cardiac anomalies. Electrocardiographic screening is recommended in all fetal SVT cases before adolescence since WPW-syndrome may occur later in life.
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1. Introduction

Fetal arrhythmias are common in clinical practice with a frequency ranging from 1% to 3% of all pregnancies. Most of these arrhythmias reflect transient, isolated atrial ectopic beats. However, sustained episodes of tachy- or bradyarrhythmia do occur and can lead to congestive heart failure, hydrops, fetal or neonatal demise, or severe neurologic morbidity in survivors [1–7].

The most common forms of fetal tachycardias are supraventricular tachycardia (SVT) and atrial flutter (AF). The majority of fetal SVTs are atrioventricular re-entrant tachycardias (AVRT) caused by the presence of an accessory atrioventricular myocardial pathway [8]. If indicated, anti-arrhythmic drugs can be given transplacentally or directly to the fetus. In SVT and AF, various anti-arrhythmic drugs are used, including digoxin, flecainide, sotalol and amiodarone [9].

More than three quarters of fetal bradycardia cases are caused by complete atrioventricular block (AVB). AVB in the absence of structural heart disease is mostly autoimmune mediated by maternal anti-Ro (SS-A) or anti-La (SS-B) antibodies [10]. In AVB, transplacental steroid treatment may reduce the effects of inflammation and fibrosis of the conduction system caused by maternal antibodies [11]. Complete AVB in the presence of complex congenital heart disease (CHD) has a poor prognosis [12,13].

Elective delivery by cesarean section can be performed in the third trimester of pregnancy to start direct neonatal therapy (anti-arrhythmic drugs, radiofrequency catheter ablation or pacemaker therapy). The goal of pre- and postnatal treatment of tachycardia is to achieve sinus rhythm or to reduce the fetal heart rate in order to prevent heart failure or death. In most cases of fetal tachycardia, medication can be stopped within the first year after delivery. However, in cases of fetal and neonatal SVT recurrences can be expected in approximately 30% of patients later in life [14].

The aim of this study was to evaluate the perinatal management and long-term cardiologic outcome of fetuses with tachy- or bradyarrhythmia diagnosed at our center.

2. Material and methods

2.1. Patients

We searched both our antenatal and neonatal databases for infants with in utero cardiac arrhythmia, diagnosed between January 1990

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and December 2005 at the Leiden University Medical Center, which is a tertiary fetal referral center. Arrhythmias included both tachy- and bradyarrhythmias. Sinus tachycardias, transient sinus bradycardias, premature atrial or ventricular contractions and ventricular tachycardias were excluded. In this time period the management protocol included complete work up with ultrasound examination and consultation of the pediatric cardiologist. Fetal ultrasound included detailed anatomic imaging of the fetal heart to diagnose or exclude cardiac defects. Routine karyotype was obtained in fetuses with suspected structural heart disease.

2.2. Fetal diagnosis and therapy

SVT as a result of AVRT was diagnosed if there was a 1:1 atrioventricular conduction observed at a rate of 200–400 bpm. AF was diagnosed when the atrial rate was 300–450 bpm. Ventricular rates in AF depended on the degree of atrioventricular conduction block, usually 200–250 bpm. The highest (peak) fetal heart rate was noted to give an indication of the severity of the tachycardia. AVB was classified as second degree or complete AVB based on M-mode evaluation and Doppler-flow measurements. Fetal hydrops was defined as a fluid collection visible on ultrasound in two or more cavities of the fetal body [15]. Maternal serum antibody titers (anti-cardiolipin antibodies, anti-Ro (SS-A) and anti-La (SS-B) were obtained in case of a heart block.

Anti-arrhythmic therapy was started when arrhythmias were sustained or associated with hemodynamic compromise prior to 34 weeks' gestation. After 34 weeks' gestation, such cases were delivered. A baseline electrocardiography (ECG) of the mother was obtained before the treatment started and maternal cardiac monitoring was conducted during the loading period to detect early signs of toxicity. During the study period, the following drugs were used: digoxin, sotalol, flecainide, amiodarone and adenosine. Digoxin was administered to the mother in adjusted oral doses to maintain a maternal serum therapeutic level of 1–2 ng/mL (loading dose 2×0.75 mg, maintenance 0.25–0.5 mg, maximum 0.75 mg/daily). Flecainide (oral dose 200–400 mg daily) and sotalol (oral dose 2×80–160 mg daily) were used as secondary agents. Amiodarone was administered by combined direct fetal intravenous and maternal oral and intravenous route. Direct fetal amiodarone therapy consisted of amiodarone on the basis of estimated fetal weight (10 mg/kg). In some cases adenosine was given intravenously (0.1 mg/kg) as direct fetal therapy just before amiodarone therapy. Drugs to treat AVB that were given to the mother included ritodrine (intravenously 9 mg/h), dexamethasone (4 mg daily) and fenoterol (intravenously 150 μg/h).

After birth, if tachycardia or AVB was present, ECGs were made to confirm antenatal diagnosis. In all SVT and AF cases, ECGs were made during sinus rhythm to detect ventricular preexcitation (deltawave) caused by anterograde conduction through an accessory pathway (WPW-syndrome).

2.3. Long-term cardiac follow-up

With the approval of the protocol by the institutional review board of the Leiden University Medical Center, family physicians were contacted by letter to explain the aims and nature of the study. After consultation with the family physician, parents were sent an explanatory letter asking their permission to review all medical records and their cooperation for testing the children. All cases were evaluated by a pediatric cardiologist and included medical history, physical examination and ECG. Additional studies, i.e. 24 hour-Holter monitoring, exercise-test and ECG were performed if children were symptomatic. Long-term neurodevelopmental outcome was also assessed and has been reported separately [16].

3. Results

3.1. Study population characteristics

During the 16-year study period, 44 pregnancies were referred to our center because of sustained fetal tachy- or bradyarrhythmia. Fig. 1 shows the overall outcome of the 44 fetuses with arrhythmia. Perinatal characteristics of the study population are presented in Table 1.

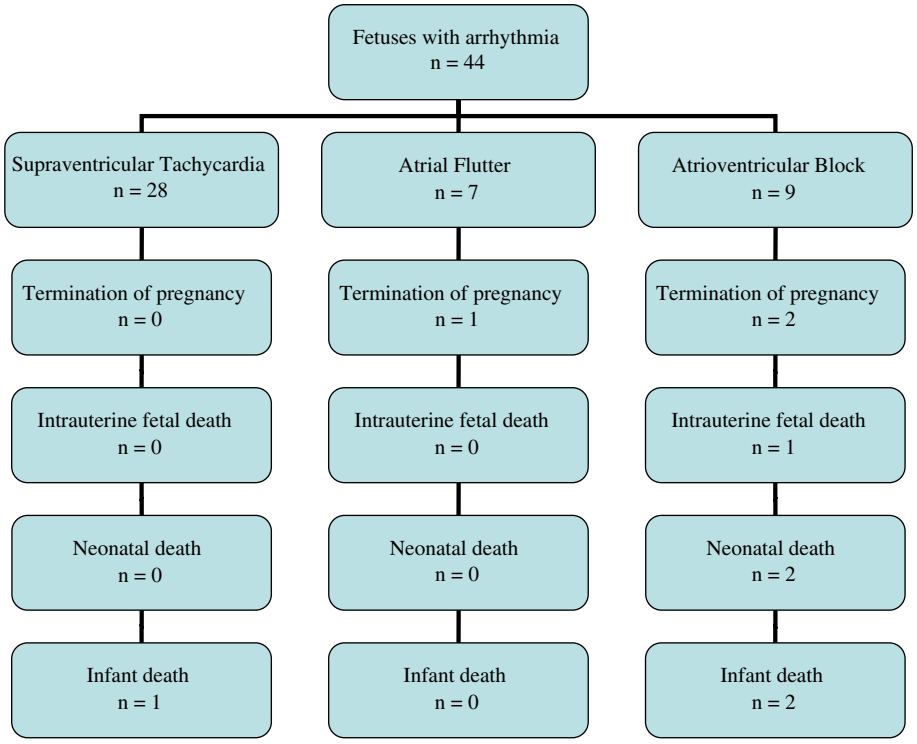

Fig. 1. Flowchart showing the outcome of the 44 studied fetuses with tachy- or bradyarrhythmias.

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