We are reporting a case of fetal supraventricular tachycardia (SVT) which converted from blocked premature atrial contractions (PAC). It has been treated by verapamil in utero. We suggest that fetal PACs are usually benign phenomena which resolve spontaneously, but require some follow-up to exclude the development of SVT. SVT is rare but complicated by fetal congestive heart failure or even fetal death.

Keywords: Tachycardia, supraventricular; Atrial premature complexes

Fetal arrhythmias are noted in only 1-2% of all pregnancies and can be categorized by rate and regularity. Almost all arrhythmias fall into one of three categories: irregular, tachycardic, or brady-cardic. Premature atrial contractions (PACs) are the most irregularity which is usually benign phenomena but timely prenatal pharmacotherapeutic intervention is generally advised to return to an adequate heart rate when it turns into tachycardia. Fetal therapy is sometimes difficult and often unsuccessful. These tachycardias can be treated in utero and proposed protocols for drug management are described. A close fetal and maternal monitoring during treatment and a team approach is advised.

Case Report

A pregnant woman had been referred at the 29+2 weeks of gestation because of the fetal arrhythmia. The fetal echocardiographic examination showed occasional ‘skipped beats’ due to isolated blocked PACs. It was happening in the every 2 beats intermittently (Fig. 1). Structural cardiac abnormality was not associated. This patient revisited at 30+6 weeks. The fetal heart rate was 228-234 (beats per minute, BPM) considered as supraventricular tachycardia (SVT) at this time (Fig. 2).

We started maternal digitalization to get down the fetal heart rate. Digoxin had been loaded 0.25 mg per 8 hours intra-venous, followed by 0.25 mg per 8 hours per-os. It maintained digoxin serum level 1.36-1.46 ng/mL (recommended 1.0-2.5 ng/mL). Fetal heart rate was still fast after one week digitalization. We needed to do other treatment because of the theoretical worry of the impairing transplacental passage of drugs in the presence of hydrops [1]. We rapidly switched the medication to verapamil at 33+4 weeks before the fetus developed hydrops. The woman took verapamil 60 mg per 8 hours per-os. The heart rate was still fast (216 BPM) but skipped beats were observed in every 2 to 5 beats, sometimes in 14 beats at 34+0 weeks (Fig. 3). The heart beats returned into mostly normal range (151 BPM) and had some PACs at 35+0 weeks. The heart rate kept normal (143 BPM) and no more PACs
The patient kept taking the medicine and the baby has been delivered spontaneously at 40+4 weeks (Male 3,340 g). The new-born has normal heart beats without any medication after birth (Fig. 4). He is well in the age of 20 months now without medication.

**Discussion**

PACs are the most common cause of an irregular fetal heart rhythm. PACs have the potential risk of changing to severe forms of fetal arrhythmia such as SVT or atrial flutter that can lead to cardiac failure and fetal hydrops [2]. Tachyarrhythmia may develop in up to 1% of fetuses with this condition [3]. The most frequent mechanism of the SVT is atrioventricular re-entry. This might be greater in prenatal life, since a higher incidence of accessory pathways has been demonstrated in the immature.

Drug therapy is usually aimed at slowing conduction at the atrioventricular node. Choice of the appropriate drug and route of administration to achieve a rapid therapeutic level in the fetus and early detection of maternal and fetal complications determine successful intrauterine antiarrhythmic treatment of the fetus. Conversion to normal sinus rhythm seems to occur more easily in the absence of fetal hydrops [4]. Treatment had been started before the fetus progress to hydrops in this case. Many therapeutic protocols for the antiarrhythmic therapy of fetal tachyarrhythmia...