CASE REPORT
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A FETAL SUPRAVENTRICULAR TACHYCARDIA WHICH CONVERTED FROM BLOCKED PREMATURE ATRIAL CONTRACTIONS

Ji-Hyun Lim, MD[1], Jin-Young Choi, MD[1], Hye-In Sung, MD[1], Ilwoon Ji, MD[1], Heon-Seok Han, MD[2]
Departments of [1]Obstetrics and Gynecology, [2]Pediatrics, Chungbuk National University College of Medicine, Cheongju, Korea

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 bradycardic. 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 arrythmia. 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

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Corresponding author: Ilwoon Ji, MD
Department of Obstetrics and Gynecology, Chungbuk National University College of Medicine, 410 Sangbong-ro, Heungdeok-gu, Cheongju 361-763, Korea
Tel: +82-43-269-6053 Fax: +82-43-275-7359
E-mail: iwji@chungbuk.ac.kr

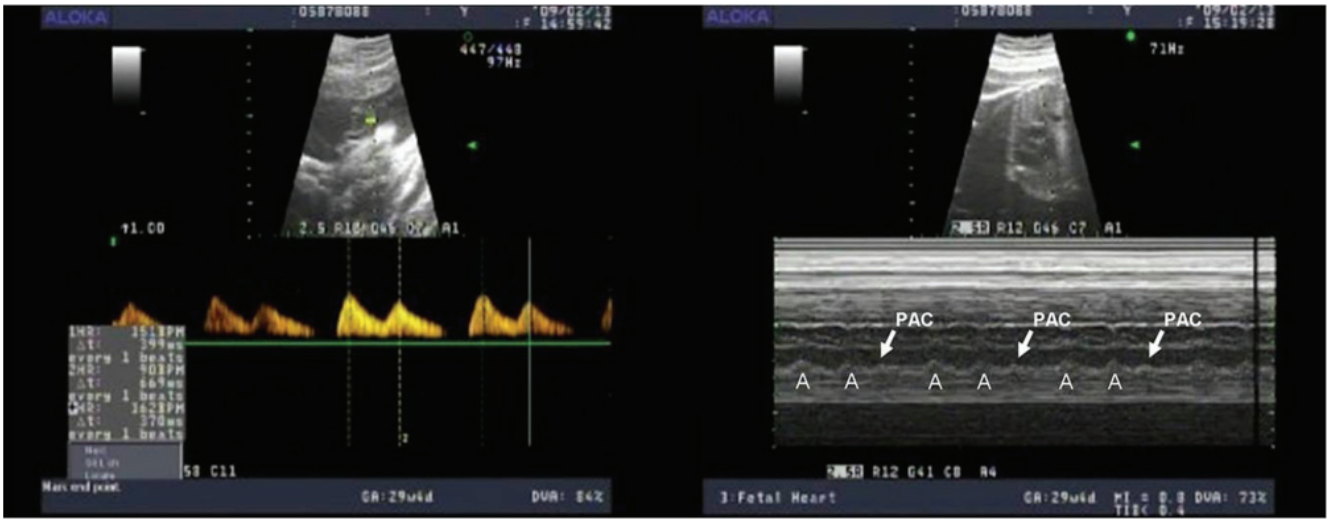

Fig. 1. Pulse Doppler shows skipped beats and M-mode reveals blocked premature atrial contractions (PAC).

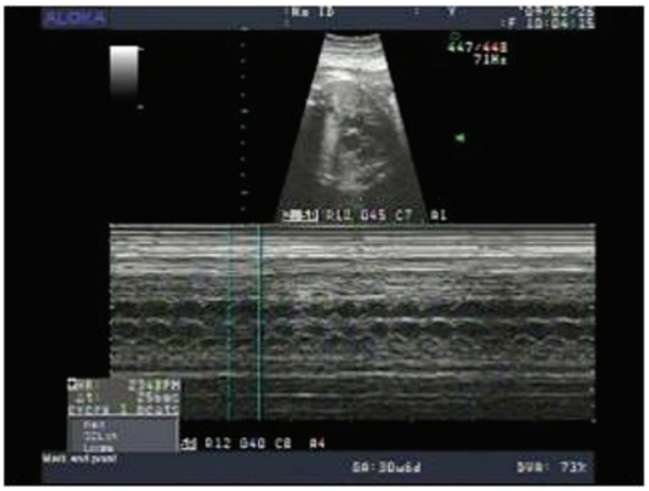

Fig. 2. M-mode shows supraventricular tachycardia which beats 234 per minutes.

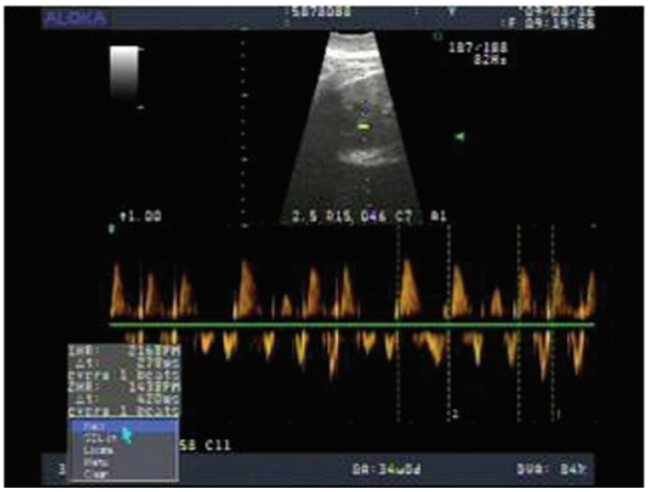

Fig. 3. Heart rate is 216 beats per minute but skipped beats in every 2 to 5 beats.

at 37+0 weeks.

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

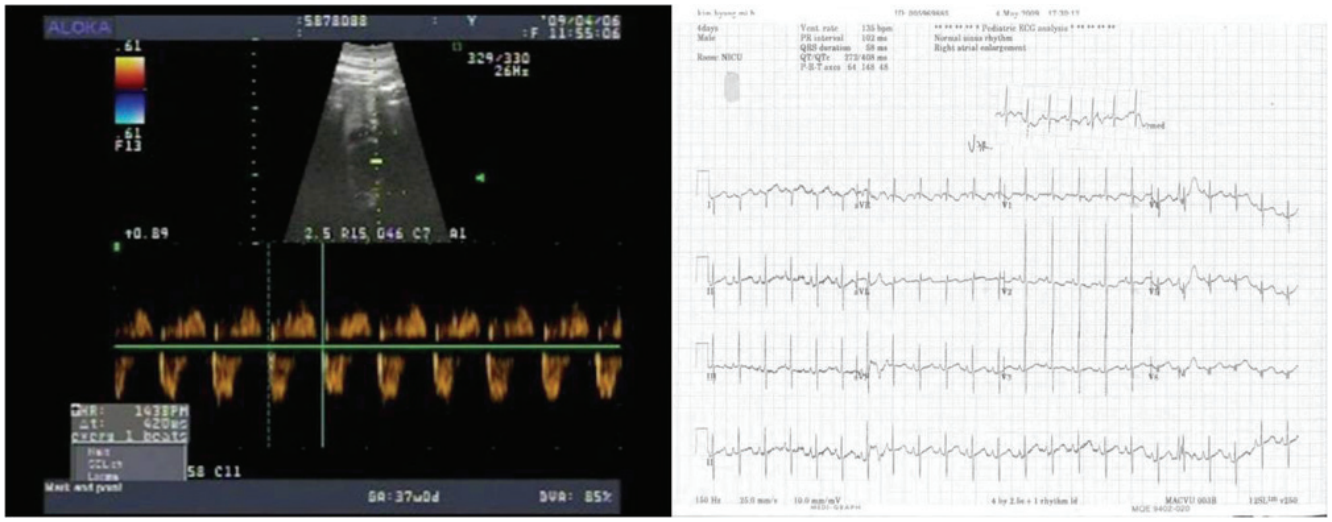

Fig. 4. Fetal heart rate returned normal (143 beats per minute [BPM]) by Verapamil and the baby has normal regular heart rate (135 BPM) without medication.

are currently used. Digoxin is generally used as agent of first choice. Serum levels in the fetus range from 70% to 100% of the maternal serum level, with normal transplacental passage in the absence of hydrops. Owing to the increase of glomerular filtration rate toward term, the elimination half-life of digoxin consequently decreases resulting in a higher dosage of digoxin for adequate loading and maintenance therapy [5]. Flecainide, sotalol, and amiodarone are provided for the second-line therapy. Verapamil is a calcium-channel blocker. It has been used widely in the adult tachycardia. Verapamil acts on the sinoatrial and atrioventricular nodes. Verapamil depresses atrioventricular (AV) nodal conduction and prolongs functional refractory periods. Verapamil does not alter the normal atrial action potential or intraventricular conduction time, but depresses amplitude, velocity of depolarization and conduction. Through this action, it interrupts re-entrant pathways and slows the ventricular rate. Verapamil may shorten the antegrade effective refractory period of the accessory bypass tract. Acceleration of ventricular rate has been reported in patients with atrial fibrillation and a coexisting accessory AV pathway following administration of verapamil [6]. Verapamil is rarely used in the fetus. It may induce hypotention or A-V blockade. It should not be used with $MgSO_4$. Verapamil, however, is familiar with us to use and easy to get. Fortunately, we treated fetal SVT successfully by verapamil without complications as others who have used it [7]. Verapamil still may be useful if it is used in caution.

PAC might be associated with fetal structural cardiac abnormality in up to 2%, SVT in 5% to 10% [8]. A full anatomical survey including a detailed survey of the fetal heart should be performed.

The majority of fetuses tolerate fetal SVT. However, it may be guessed that in fetuses with paroxysmal tachyarrhythmia, cease of cerebral autoregulation may lead to severe impairment of the maintenance of constant cerebral perfusion, especially in fetuses before 32 week' gestation [9]. Therefore, rapid and persistent control of fetal SVT may prevent fetal neurological damage. We suggest that fetal PACs are usually benign phenomena which resolve spontaneously, but require some follow-up to exclude the development of SVT.

In fetuses with SVT that were treated in utero, the postnatal outcome may be complicated by a recurrence of tachyarrhythmia in approximately 50%. By maturation of the infant's conduction tissue, the probability of late recurrence decreases. Only in 10-20% of infants may the tachycardia persist beyond the first year of life [10]. For the vast majority, normal long-term development can be expected after in utero and/or postnatal cessation of tachyarrhythmia.

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차단된 조기심방수축으로부터 전환된 태아의 심실위빈맥 1예

충북대학교 의과대학 [1]산부인과학교실, [2]소아청소년과학교실
임지현[1], 최진영[1], 성혜인[1], 지일운[1], 한헌석[2]

태아의 조기심방수축은 저절로 소실되는 경우가 많다. 그러나 간혹 심실위빈맥으로 전환되기도 한다. 심실위빈맥은 드물게 울혈성 심부전을 유발하고 태아를 사망에 이르게 할 수 있다. 그러므로 태아의 조기심방수축도 적절한 추적관찰이 필요하다. 저자들은 차단된 조기심방수축으로부터 전환된 태아의 심실위빈맥 예를 경험하고 치료하였기에 문헌 고찰과 함께 보고하는 바이다.

중심단어: 빈맥, 심실위, 조기심방수축