

Fetal Supraventricular Tachycardia With And Without Non Immune Hydrops

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Citation

T Atasaral, B Vurall?, M Osmana?ao?lu, E Dilber, H Bozkaya. *Fetal Supraventricular Tachycardia With And Without Non Immune Hydrops*. The Internet Journal of Gynecology and Obstetrics. 2008 Volume 11 Number 1.

Abstract

Objective: To report two cases of fetal supraventricular tachycardia (SVT) with and without non immune hydrops and review the literature. **Case Reports:** Case I: A 21-year-old woman has been referred to our clinic for hydrops fetalis at 35 weeks of gestation with fetal tachycardia. Fetal echocardiogram showed a symmetrically enlarged heart with fetal heart rate 246/min. An immediate caesarean section was performed and she had an 3600 g boy with apgar scores 1 and 2, at 1 and 5 min, respectively. Despite intensive cardiopulmonary resuscitation, the infant died. Case II: A 23-year-old woman, at 34 weeks in her first pregnancy, was noted to have a fetal tachycardia at non-stress test and was referred to our university hospital for further management. Fetal growth corresponded with gestational age on ultrasound examination. Fetal echocardiogram revealed interatrial septal aneurysm, a secundum atrial septal defect of 6-7 mm size, and tricuspid insufficiency with satisfactory cardiac contractility and with heart rate 240/min confirmed by M-mode echocardiogram. The patient was started on Sotalol 120 mg twice daily and was hospitalized at 38 w 4 D. Sotalol was stopped before delivery. She had a normal vaginal delivery as a live male weighed 3350 g. Postnatal echocardiogram was normal and the ECG showed sinus rhythm. **Conclusions:** New larger prospective and multicentric studies are needed which would focus on the definitive treatment of fetal SVT and taken a close collaboration between obstetricians and paediatric cardiologists into consideration on follow-up mother and fetus.

It's presented at the VI. National Gynecology and Obstetrics Congress, Antalya, May 14-19, 2008.

INTRODUCTION

Hydrops fetalis is a condition in the fetus characterized by an accumulation of fluid, or edema, in at least two fetal compartments, including the subcutaneous tissue, pleura, pericardium, or in the abdomen. The cause of non-immune hydrops fetalis may be secondary to maternal and placental disorders but, generally it is associated with fetal disorders. The common etiological factors of non-immune hydrops fetalis are cardiovascular abnormalities, infectious disease and aneuploidy in Europe (6).

The normal fetal heart rate is between 120 and 180 beats per minute and depends on gestational age and degree of fetal activity. Fetal dysrhythmias are diagnosed in at least 2 % of pregnancies during routine ultrasound scanning (1). The vast majority being intermittent extrasystoles which have little clinical relevance (2). Less than 10 % of referrals are due to sustained tachy or bradyarrhythmias and this rhythm disturbances in the fetus are clinically important (3).

Tachycardia in the fetus is defined as intermittent or sustained increase in heart rate of 180 beats/min (4).

Fetal SVT is defined by a 1:1 atrioventricular conduction in which the atrial contraction precedes the ventricular contraction. Heart rates in SVT most commonly range from 200-300 bpm, is either paroxysmal or incessant in nature and associated with fetal hydrops in 36 - 64 % (2,3,4,5).

Supraventricular tachycardia (SVT) is a well-recognised cause of non-immune hydrops fetalis, which is associated with a high incidence of perinatal mortality.

CASE REPORT I

A 21 years old woman, gravida 2, para 1 has been referred to our clinic for hydrops fetalis at 35 weeks of gestation and fetal tachycardia was detected on doppler ultrasonography examination. On ultrasound scan, fetal biparietal diameter was 89.3 mm: 36 weeks, femur length: 70.8 mm: 36 weeks 2 day, fetal tachycardia, bilateral pleural, pericardial effusions, ascites, scalp edema and a symmetrically enlarged heart were detected. There was no other structural abnormality identified. The mother was hospitalized with a suspect of

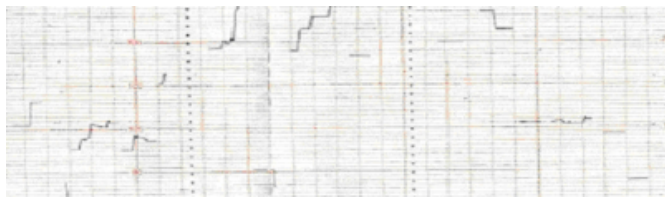
fetal supraventricular tachycardia with hydropic change. Her family and pregnancy historics were unremarkable. Fetal echocardiogram showed a symmetrically enlarged heart and fetal tachycardia with heart rate 246 beats/min. The atrial/ventricular contraction ratio was 1:1 as confirmed by M-mode echocardiogram. Because of the pregnancy was of reasonable maturity, at 36 weeks gestation, an immediate caesarean section was performed and she had an 3600 g boy with apgar scores 1 and 2, at 1 and 5 min, respectively. Despite intensive cardiopulmonary resuscitation, the infant died. A postmortem fetal autopsy was refused by her family.

CASE REPORT II

Case II: A 23-year-old woman, at 34 weeks in her first pregnancy, was noted to have a fetal tachycardia at non-stress test and was referred to our university hospital for further management (Figure 1). No structural abnormalities were seen on ultrasound examination and

Figure 1

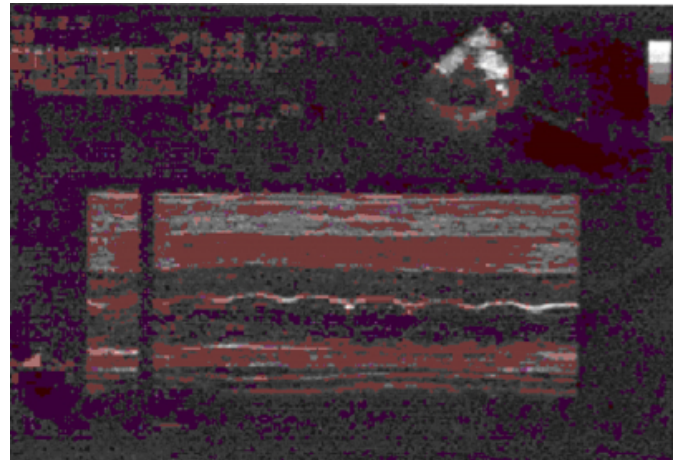
Figure 1: Non-stress test of the case II which could not be able to draw accurately because of the high fetal heart rate up 200 beats/min.



fetal growth corresponded with gestational age. Fetal echocardiogram revealed interatrial septal aneurysm, a secundum atrial septal defect of 6-7 mm size, and tricuspid insufficiency with satisfactory cardiac contractility and with heart rate 240/min. The atrial/ventricular contraction ratio was 1:1 as confirmed by M-mode echocardiogram (Figure 2).

Figure 2

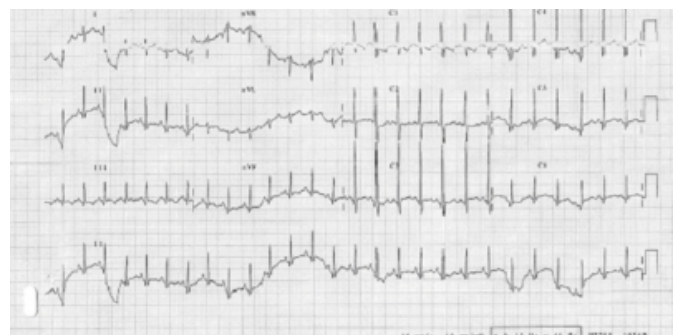
Figure 2: M-mode echocardiogram of the case II showing supraventricular fetal tachycardia, with regular fast fetal heart rate with 1:1 atrial/ventricular ratio.



Following a maternal ECG, the patient was started on Sotalol (Talozin tablet, 80 mg, Adeka, Turkey) 120 mg twice daily. The basal fetal heart rate was brought down to 140/min (day 1 after sotalol), Therefore Sotalol was then decreased to 80 mg twice daily and the patient was discharged on follow up with fetal echocardiography and NST daily until term. The patient was hospitalized at 38 w 4 D, Sotalol was stopped before delivery and she had a normal vaginal delivery as a male weighed 3350 g with Apgar score of 7 and 9 at 1 and 5 minutes, respectively. Postnatal echocardiogram was normal and the ECG showed sinus rhythm with heart beat 163 beats/min (Figure 3).

Figure 3

Figure 3: After treatment and delivery, the neonate's ECG of the case II



DISCUSSION

Supraventricular tachycardia has one-to-one AV conduction, and fetal heart rate is between 200 and 300 beats per minute. Fetal echocardiography allows correct diagnosis of the dysrhythmia, underlying mechanism and exclusion of structural abnormalities ([456]). In comparison to SVT, atrial

flutter has more rapid regular atrial rate of 300-600 beats per minute accompanied by variable degrees of atrioventricular (AV) conduction block (AV block ratio is usually 2:1) resulting in slower ventricular rates. Atrial flutter is the result of an intra-atrial electrical macro-re-entrant circuit around fixed or functional anatomical conduction barriers ([4]).

Although the precise identification of the mechanism underlying a tachyarrhythmia is important since the pharmacological approach can be quite different, we differentiated supraventricular tachycardia from atrial flutter; however, because of paediatric cardiologist's low clinical experience, the differentiation of sub-type of supraventricular tachycardia could not be able accurately defined. Since the most common mechanism of fetal SVT is an atrioventricular re-entry tachycardia (AVRT) caused by the presence of an accessory pathway between atrium and ventricle and underlies around 90 % of fetal SVT (7), we focused on the diagnosis of AVRT.

Fetal supraventricular tachycardia is an important cause of fetal morbidity and mortality. Hydrops fetalis has been shown to be the most important factor in determining the outcome of tachyarrhythmias. As was seen in the presented case 1, it's associated with a mortality risk of 35 % and in cases without significant heart failure with a mortality risk of 0-4 % (6).

The fetus with supraventricular tachycardia requires urgent cardiac and obstetric evaluation. There are three approaches to management: no intervention but close monitoring, antiarrhythmic drug therapy, and delivery (6,8,9). Abstention of treatment with close pregnancy monitoring is a valid option if the fetus presents with intermittent brief runs of tachycardia in the absence of haemodynamic impairment. If there is persistent tachycardia or circulatory compromise, prompt intervention should be instituted to prevent congestive heart failure and fetal death (6,8,9). Nevertheless, the choice of treatment is contentious as there are no controlled data that document the superiority of any antiarrhythmic drug treatment for fetal tachyarrhythmia. There is, however, considerable non-randomized experience in the transmaternal treatment of fetal SVT with a number of antiarrhythmic agents including digoxin, procainamide, flecainide, sotalol and amiodarone (6,8,9).

Maternal digoxin therapy is suitable for atrial flutter and AVRT, however sotalol is effective in treating digoxin-refractory fetal tachyarrhythmias and has been proposed as

first-choice therapy for atrial flutter (8,9). In the presence of fetal hydrops, transplacental digoxin transfer is poor and effective fetal drug levels decreases while placental transfer ratio of flekanid and sotalol are 80 % and for this reason superior to digoxin therapy. In addition in treating digoxin-refractory SVT's including AET and PJRT flekainid and sotalol therapies are more successful. Sotalol is a β -blocking agent with additional anti-arrhythmic properties and mild negative inotropic effect. Placental transfer is quick and almost complete, with fetal levels being almost identical to those of maternal plasma. Sotalol is effective in treating digoxin-refractory fetal tachyarrhythmias (9) and has been proposed as first-choice therapy for atrial flutter. Side effects and pro-arrhythmic risk are dose related (9). Despite initial safety concerns, no statistical difference was found in mortality related to its use in SVT when compared with that of other studies (9). Close maternal monitoring of QT interval on the ECG, and of electrolyte levels, especially during the initiation process, is recommended (9). Sotalol is usually started orally at 80 or 160 mg twice a day (9). An incremental dosage scheme of 80 mg per three days, starting with 80 mg twice daily to a maximum of 160 mg three times a day, has been proposed as a means of minimizing complications (9,10). If a fetus presents with significant SVT after 35 weeks of gestation, expedited delivery followed by postnatal conversion is an option (11). The choice between immediate delivery or drug therapy should be a balanced assessment of gestational age and lung maturity, the circulatory changes present in the fetus, available neonatal facilities for postnatal management and maternal choice. If the fetus with SVT is not improving or if there is circulatory compromise, prompt intervention should be instituted to prevent congestive heart failure and fetal death. Moreover, in the presence of fetal hydrops, maternal-fetal digoxin transfer is poor and effective fetal drug levels may not even be obtained at near-toxic maternal levels. The direct fetal route via cordocentesis is used for acute treatment especially in the setting of severe hydrops (9). In our first case fetal hydrops occurred secondary to SVT and because of the pregnancy was of reasonable maturity, a cesarean section was performed. However, the neonate died because of severe congestive heart failure.

New larger prospective and multicentric studies are needed which would focus on the definitive treatment of fetal supraventricular tachycardia and taken a close collaboration between obstetricians and paediatric cardiologists into consideration on follow-up mother and fetus.

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