

RESEARCH LETTER

Hemodynamic effect of fetal supraventricular tachycardia on the unaffected twin

Sanjeev Aggarwal*, Susan Czaplicki and Kavitha Chintala

Division of Cardiology, The Carman and Ann Adams Department of Pediatrics, Children's Hospital of Michigan, Wayne State University, 3901 Beaubien Blvd, Detroit, USA

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Fetal arrhythmias are diagnosed in 1 to 3% of pregnancies, of which supraventricular tachycardia (SVT) constitutes 10% (Reed, 1989). Sustained fetal SVT with a heart rate more than 220 beats per minute (bpm) can result in congestive heart failure and nonimmune hydrops, which is associated with a poor outcome (Simpson and Sharland, 1998). SVT in twin pregnancy is infrequently reported and poses a therapeutic and ethical dilemma in regard to the effect of treatment on the unaffected twin. To date, there is no evidence that fetal SVT in one twin affects hemodynamics of the other twin who is in normal rhythm. We report for the first time, a case in which fetal SVT in one twin was associated with early signs of congestive heart failure, in the other twin, with subsequent echocardiographic improvement following control of the SVT in the first twin.

A 26-year-old gravida 2, para 0 African-American woman with a twin pregnancy at 27 (3/7) weeks of gestation was referred to us for evaluation of fetal tachycardia in one of the twins. The course of pregnancy was uneventful until then. Specifically, there was no history of hypertension, diabetes or infections. She was a nonsmoker and denied use of alcohol or drugs. During a regular prenatal visit, auscultation revealed audible arrhythmia in twin A with heart rate in the range of 200 bpm. Ultrasound revealed monochorionic diamniotic twins of similar size (estimated fetal weight of twin A, 992 g and twin B, 959 g). There were no other congenital defects. A fetal echocardiogram utilizing M mode and Doppler techniques revealed that Twin A had SVT at a rate of 230 bpm with 1:1 conduction to ventricles. Cardiac anatomy was normal. The size and contractile function of the ventricles were normal and there was no mitral or tricuspid regurgitation. There was no evidence of pericardial effusion or congestive heart failure as assessed by the cardiovascular profile (Huhta, 2004).

Fetal echocardiography of Twin B revealed normal cardiac structural anatomy. There was mild cardiomegaly with a cardiothoracic circumference ratio of

0.6. The left ventricular shortening fraction was normal. The right ventricle was mildly dilated and there was mild to moderate tricuspid regurgitation. The tricuspid valve was morphologically normal (Figure 1). Twin B had a heart rate of 144 bpm with regular sinus rhythm and normal 1:1 atrio-ventricular conduction.

Oral digoxin therapy was initiated on the mother in the hospital with a starting dose of 250 mcg orally every 8 h for 24 h, followed by 250 mcg once a day. SVT in twin A converted to normal sinus rhythm by the 3rd day of treatment. Repeat fetal echocardiography performed one week later revealed a heart rate of 130 to 140 bpm without any premature atrial contractions or intermittent tachycardia in twin A. Interestingly, twin B had a significant improvement in the right ventricular dilation and the tricuspid valve regurgitation had resolved. The heart rate of Twin B remained in the range of 140 to 150 bpm. Digoxin therapy was continued until delivery and the twins were monitored weekly. There was no recurrence of arrhythmia in Twin A or of hemodynamic changes in Twin B. The patient delivered normally at 38 weeks of gestation. Electrocardiogram (ECG) performed on both the neonates after birth revealed normal sinus rhythm with normal 1:1 Atrioventricular (AV) node conduction. The neonatal echocardiograms in both the twins confirmed normal anatomy and ventricular function.

Fetal arrhythmias are fairly frequent occurrences and are often initially suspected by an irregular heartbeat on auscultation or fetal Doppler. A fetal echocardiogram is required to confirm the diagnosis and type of arrhythmia and to evaluate the complications. Fetal SVT comprises 10% of fetal arrhythmias and is defined as fetal heart rates of 200 to 300 bpm with 1:1 atrio-ventricular conduction (Owen and Cameron, 1997).

Fetal arrhythmias in multiple pregnancy can be interesting and challenging since it raises a moral and therapeutic dilemma for treatment. Apart from consequences of drug therapy to the mother, consequences to the other fetus need to be considered before initiating therapy. Also, the question of whether dysrhythmia in one twin affects the hemodynamics of the other twin arises. In order to find an answer, we reviewed all available literature for fetal arrhythmias in multiple pregnancy. We found five case reports, all of which discuss aspects of management of fetal SVT in multiple pregnancy.

*Correspondence to: Sanjeev Aggarwal, Division of Cardiology, The Carman and Ann Adams Department of Pediatrics, Children's Hospital of Michigan, Wayne State University, 3901 Beaubien Blvd, Detroit, USA. E-mail: ssanjeev@dmc.org

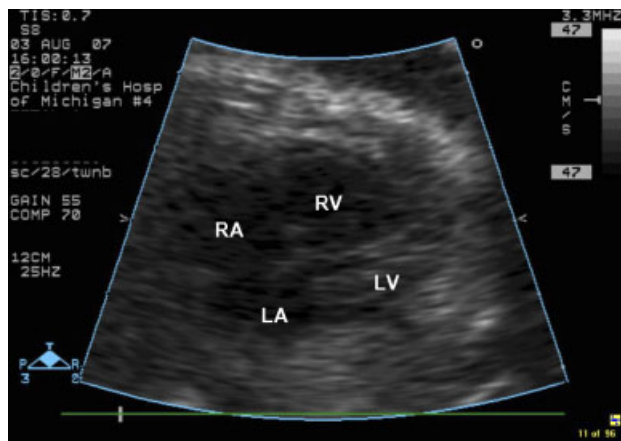


Figure 1—Horizontal four-chamber view of the heart demonstrating dilated right atrium and right ventricle

Tanawattanacharoen *et al.* reported the successful use of digoxin in the management of fetal SVT in one twin (Tanawattanacharoen *et al.*, 2005), while there are two reports of successful use of transplacental flecainide for SVT and hydrops in one of the twins (Edwards *et al.*, 1999; Gerli *et al.*, 2006). Jones *et al.* reported successful use of transplacental digoxin to control fetal SVT in one fetus among triplets similar to our case report (Jones and Garmel, 2001). There is one report where simultaneous SVT occurred in both twins and oral digoxin was successful in treating both fetuses (Shima *et al.*, 2004 #3). None of these reports mention any hemodynamic effects on the unaffected fetus.

Fetal SVT is known to cause heart failure and subsequent hydrops in the affected fetus. The earliest echocardiographic sign of impending cardiac failure is the dilatation of right atrium and right ventricle (Huhta, 2004). Tricuspid valve regurgitation occurs as a result of increased right ventricular wall stress (Huhta, 2004). Interestingly, in our case, both these findings were noted in twin B who remained in sinus rhythm as confirmed by continuous monitoring during the 2-day hospitalization as well as by fetal echocardiography. In contrast, Twin A, who was in SVT, did not manifest any heart failure. We excluded all other causes of heart failure in twin B including twin to twin transfusion syndrome which is in any case unlikely in twins of similar weights (Barrea *et al.*, 2005). That heart failure in Twin B was induced by SVT in Twin A was also supported by the fact that all signs of heart failure resolved when SVT in Twin A was controlled with drug therapy.

The fetal ventricles at baseline have poor compliance and impaired diastolic relaxation. Occurrence of SVT leads to a marked decrease in the ventricular filling times and this leads to increased systemic venous pressure. The exact reason why this occurred in Twin B is unknown, but we speculate that an imbalance in the venous pressure in the presence of SVT in twin A led to a net transfer of blood from twin A to twin B. The resultant hemodynamic sequel is similar to that seen in twin to twin transfusion syndrome in the presence of unbalanced arterio-venous anastomosis in a monochorionic placenta (Bajoria, 1998). In both

conditions, the recipient may develop cardiomegaly and decreased cardiac function. Besides, hormonal activation during SVT could have had repercussion on the twin in sinus rhythm. To our knowledge, this is the first case in which altered hemodynamics in one twin appeared to be caused by SVT in the other.

The optimal management of fetal SVT remains controversial. Gestational age, duration of the arrhythmia and presence or absence of hydrops are important determinants of the management strategies for fetal SVT. Since it is difficult to assess whether SVT in a particular fetus is intermittent or sustained and since fetal hydrops can occur in both, most centers now treat all fetal SVTs (Simpson and Sharland, 1998). The most common first line medical management is transplacental administration of digoxin, which, as a monotherapy, is successful in converting 60 to 70% of SVT in non-hydropic fetuses (Simpson and Sharland, 1998). In our case, the fetal SVT in one twin converted to normal rhythm after 3 days of starting digoxin. *Drug therapy for the treatment of fetal arrhythmias may be complicated by adverse effects to both mother and fetus. Presence of multiple gestation poses a serious concern and ethical issues due to adverse effects of medication and/or intervention on the unaffected fetuses. However, there were no ethical problems in the decision for treatment in our case because both twins had cardiovascular problems for which digoxin was the drug of first choice, resulting in clinical improvement, although via different mechanisms.* There were no side effects of digoxin either on the mother or the other unaffected twin.

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