Neonatal Arrhythmia
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Citation

ABSTRACT
Arrhythmias are seldom observed in the newborn period and rarely lead to serious consequences. Because they may be a continuation of fetal arrhythmias, newborn arrhythmias are different from those occurring at later ages. Here we describe a case of a newborn presented with tachycardia at birth. A female baby of 1950 grams born by emergency cesarean section for fetal distress at 36 weeks of gestation. Fetal tachycardia of 251 bpm was detected prenatally. Electrocardiography showed supraventricular tachycardia (SVT). Hematological and biochemical tests done were within normal limits. Echocardiography revealed normal anatomy with severe tachycardia, dilated chambers with moderate to severe TR with moderately reduced ventricle function. For persisting SVT intravenous adenosine was administered with no significant decrease in heart rate, then continuous intravenous amiodarone infusion was started resulting in a transient decrease in heart rate, however again increased, hence baby was started on intravenous digoxin which responded well. Repeated echocardiography showed normal cardiac chambers and function. Baby was discharged on maintenance oral digoxin and was gradually weaned and stopped after 12 months of age. Neonatal arrhythmias is not an uncommon condition in newborns, however it should be early recognized and evaluated for a better outcome of the baby. Although the frequency of arrhythmias in the newborn period is not high, SVT are the most frequently observed arrhythmias in this period.

KEY WORDS
Fetal tachycardia, Neonatal arrhythmias, Supraventricular tachycardia

INTRODUCTION
Cardiac arrhythmias are often diagnosed in fetuses and newborns, occurring in 2% of the pregnancies and, in the neonatal period, the incidence varies between 1% and 5%.1 Arrhythmias observed in the newborn period rarely lead to serious consequences. Long-term tachycardia and bradycardia attacks induced by neonatal arrhythmias may lead to heart failure and hydrops fetalis.2 Neonatal arrhythmias can be discovered incidentally after birth, during evaluation for other conditions, or they may be a continuation of fetal arrhythmias, hence newborn arrhythmias are different from those occurring at later ages.3 Here we report a newborn who presented with non-specific symptoms, had tachycardia and ECG showed features suggestive of SVT.

CASE REPORT
A female baby of 1950 grams was born to a non-consanguineous parents, gravida two mother by emergency cesarean section for fetal distress at 36 weeks of gestation. The other sibling is alive, healthy and no other family members had any known cardiac disease. There was no apparent maternal cause that could have resulted in fetal tachycardia. Fetal tachycardia of 251 bpm was detected prenatally. Baby was found to be mild tachypneic (60/min), heart rate of 260 bpm with mean blood pressure of 35 mmHg, soft systolic murmur heard in left parasternal border. Baby was kept on nasal CPAP for respiratory distress and weaned gradually after 48 hours, Electrocardiography (ECG) showed SVT with narrow QRS complex, no δ waves and normal T waves at a rate of 260 bpm, with suspicious
Case Note

For persisting SVT intravenous adenosine was administered with no significant decrease in heart rate, then continuous intravenous amiodarone infusion was started resulting in a transient decrease in heart rate (180 bpm) however again increased to the range of 240-260 bpm, hence baby was started on intravenous digoxin which responded well. Baby gradually remained in sinus rhythm with the heart rate ranging from 120-160 bpm and was weaned to oral Digoxin after 1 week. Repeated echocardiography showed normal cardiac chambers and function. Transcranial ultrasonography revealed no ischaemic, haemorrhagic or ventricular alterations. Baby was discharged on maintenance oral digoxin (5 mcg/kg/day) and was gradually weaned and stopped after 12 months of age. The baby has been well so far, at present 18 months old with no further episodes of arrhythmia with normal growth and development. The subsequent ECG records have been normal.

DISCUSSION

Fetal arrhythmias are categorized under three groups which are tachyarrhythmia, bradyarrhythmia and irregular cardiac rhythm. If fetal heart rate is above 180 bpm, it is tachyarrhythmia; if it is below 100 bpm, then it is bradyarrhythmia. Fetal tachyarrhythmia incidence in pregnant is reported between 0.4 and 0.6%. The reason of 70-80% of fetal tachyarrhythmias which are one of the major causes of fetal distresses is the SVT. Serious cardiac defects such as ventricular septal defect, aortic stenosis, coarctation of aorta, cardiac tumor, left atrial isomerism and Ebstein anomaly may be seen in cases with fetal tachyarrhythmia. Hydrops fetalis is one of the most significant factors for estimating perinatal outcomes. There is the risk of congestive heart failure and mortality risk at the rate of 27%. Fetal SVT which is one of the most common reasons of fetal tachyarrhythmias is the cardiac arrhythmia in which fetal heart rate is 220-300 bpm and AV conduction is 1:1. Although fetal SVT is observed generally at 2nd and 3rd trimester, it may also be seen in first trimester. In case that it takes longer than 12 hours, it may cause heart failure, non-immune hydrops fetalis, preterm labor or fetal losses. SVT is the most common symptomatic fetal cardiac arrhythmia. AV re-entry tachycardia is the most common mechanism, occurring in more than 90% of fetal SVTs. Other, rare mechanisms for SVT in fetuses are AF or fibrillation, automatic tachycardia, and permanent junctional reciprocating tachycardia. In fetuses that have SVT, the characteristic heart rate is 240 to 260 bpm. In fetuses that have AF, the characteristic atrial rate is 300 to 500 bpm, with varying ventricular response rates. Ultrasonography currently is the most commonly used diagnostic tool to analyze heart rhythm in a fetus. A detailed study includes definition of cardiac structure, rhythm, function, hemodynamics, and presence of fetal hydrops. Structural malformations of the heart are seen in up to 5% of fetuses that have SVT, which
most frequently include Ebstein anomaly of the tricuspid valve, AV canal defect, hypoplastic left heart syndrome, or rhabdomyoma. Fetal SVT is associated with a fetal and neonatal mortality rate of 8% to 30%. Preterm delivery of an infant who has hydrops and SVT is associated with high rates of morbidity and mortality.11,11 The indications to treat fetal SVT are prematurity or evidence of severe hemodynamic compromise of the fetus, such as hydrops.10 The therapeutic goal is rate control (in AF) or complete control of the arrhythmia. Digoxin is the most frequently used monotherapy in mothers whose fetuses do not have hydrops but do have either SVT or AF.10 Sotalol or digoxin is the first-line medication to treat fetal AF. The treatment aim is either to suppress the arrhythmia or, if this is not achieved, to slow the ventricular rate to a more normal heart rate. If AF persists to birth, sinus rhythm can be restored by transesophageal overdrive pacing or synchronized electrical cardioversion. Neonatal recurrence of AF is unusual and long-term treatment is rarely required.7 SVT is a relatively common tachyarrhythmia in the neonatal intensive care unit. It may be recurrent or occasionally persistent, but rarely is it life-threatening. Acute termination of SVT is critical in patients who develop signs and symptoms of hemodynamic instability, including lethargy, pallor, poor perfusion, hypotension, acidosis, and signs of cardiac failure. SVT in early infancy is dangerous and potentially fatal if not treated early and appropriately. In neonates, AV re-entrant tachycardia is common.13 The heart in a neonate with SVT may be structurally normal or there may be congenital heart disease (Ebstein’s anomaly and L-transposition of great arteries). Congenital heart disease was detected in 28% and conduction defects and L-transposition of great arteries). Congenital heart disease was detected in 28% and conduction defects were recognized and evaluated for a better outcome of the baby. However, management of the infant with circulatory compromise, or with a history of fetal hydrops, antenatal polypharmacy, prematurity, or congenital heart disease is more difficult. There is a risk of death from the underlying problem or due to inappropriate or inadequate treatment, and an understanding of the mechanisms of tachycardias is important to make a logical management plan. Diagnoses and treatments of fetal tachyarrhythmias are very important due to the fact that they may cause fetal distress. Neonatal arrhythmias is not an uncommon condition in newborns, however it should be early recognized and evaluated for a better outcome of the baby. Although the frequency of arrhythmias in the newborn period is not high, SVT are the most frequently observed arrhythmias in this period. Although the long term prognosis for newborn arrhythmias may be good, patients should be closely monitored.

REFERENCES


