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CASE REPORT

Management of Fetal Supraventricular Tachycardia: Three Cases at a Single Institution

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Abstract

Background: Fetal tachycardia complicates 1-2% of pregnancies, and a large percentage of these arrhythmias are supraventricular. Sustained fetal tachycardia can have serious complications for the fetus, including cardiovascular failure, nonimmune fetal hydrops, and fetal death. Management of fetal tachycardia depends on multiple factors. Here we present three different cases of fetal supraventricular tachycardia (SVT) and their management at a single institution.

Cases: Case 1 is a 25-year-old G2P1001 who presented at 30 weeks with the incidental finding of fetal SVT. Prolonged monitoring showed non-sustained fetal SVT, and no medical intervention was initiated. Case 2 is a 26-year-old G3P1102 who presented with sustained fetal SVT at 26 weeks. Treatment with digoxin was initiated with eventual resolution of the tachycardia. Case 3 is a 26-year-old G1P0 who presented at 28 weeks with sustained fetal SVT resulting in fetal hydrops. Multiple medication regimens were used in an attempt to control the rhythm.

Conclusion: Fetal SVT can result in serious fetal complications and may require varying treatments depending on the presentation.

Introduction

The fetal heart first develops during the third week of gestation, and its conduction system matures by 16 weeks [1]. Fetal tachycardia is defined as a heart rate faster than 160 beats per minute (BPM) [2]. Common causes of fetal tachycardia include infection, hypoxemia, maternal hyperthyroidism, and tachyarrhythmia. The suspicion for a tachyarrhythmia increases when the fetal heart rate is over 220 BPM. Seventy to 75% of tachyarrhythmias are supraventricular in origin, with sinus and ventricular tachycardias being much rarer [3,4]. Sustained tachyarrhythmias, which are often defined as the presence of tachycardia for more than 50% of fetal monitoring time, may cause fetal heart failure, nonimmune hydrops, and/or Ballantyne's syndrome, all of which are complications that can lead to fetal death. They are also an important cause of premature deliveries and perinatal morbidity [4]. Therefore, identification and appropriate management of these cases is important in an attempt to prevent these adverse outcomes.

We describe three different cases of fetal tachyarrhythmia and the management required in each case.

Case One

TB was a 25-year-old African-American G2P1001 who presented at 30 w 6 d to a community hospital with complaints of preterm contractions, at which time fetal tachycardia was incidentally identified. The patient was admitted for further evaluation with workup including a maternal EKG that showed a first-degree heart block with a PR interval of 212 milliseconds; metabolic panel and blood counts were unremarkable. Fetal ultrasound showed an LGA fetus at greater than the 90th percentile with polyhydramnios (amniotic fluid index of 28 cm), with no evidence of hydrops or cardiac abnormalities. Twenty-four hours of fetal monitoring was noted for non-sustained tachycardia (less than 4 hours); no medications were

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started and patient was discharged with plans for weekly MFM follow up in conjunction with continued care per her primary OBGYN.

Patient was delivered at 36 w 1 d after a fetal monitoring was non-reassuring. She delivered a live born female infant weighing 3400 g via uncomplicated repeat cesarean section. The neonate was awarded Apgars of 9 and 9 at 1 and 5 minutes, respectively. Umbilical artery pH was 7.24 with a base deficit of 3.5 mmol/L and the umbilical vein pH was 7.32 with a base deficit of 2.3 mmol/L. The baby was admitted to the NICU due to prematurity and history of fetal SVT. EKG on day of life 1 showed normal sinus rhythm. Forty hours of cardiac monitoring post-delivery showed no cardiac arrhythmia. Neonate was discharged on day of life 2 with plans for outpatient follow up with cardiology.

Case Two

JS was a 26-year-old Caucasian G3P1102 who presented for an anatomical survey at 26 w 0 d that noted no structural abnormalities, but the fetus was found have a tachyarrhythmia. Patient was admitted to the hospital for further monitoring. Maternal workup was overall unremarkable with a normal comprehensive metabolic panel, thyroid studies, and EKG. Maternal drug screen was positive for benzodiazepines, opiates, oxycodone, and tetrahydrocannabinol. External fetal monitoring could not be interpreted due to the tachyarrhythmia, so BPPs were performed to monitor fetal well-being. A fetal echo was obtained, which confirmed the diagnosis of supraventricular tachycardia. Pediatric cardiology and electrophysiology were consulted, and patient was started on digoxin with loading dose of 500 mcg every 12 hours until a goal trough level of 0.5-2 ng/mol was reached.

Due to childcare and social issues that prohibited inpatient treatment, the patient was managed via frequent outpatient and triage visits, each of which included testing of her digoxin levels, EKGs and either external fetal monitoring or biophysical profile evaluations. At 27 w 1 d, improvement in the tachyarrhythmia was noted with intermittent instead of sustained SVT, but the digoxin dose was increased to 375 mcg BID to further control the rhythm. Two days later a maternal EKG showed changes consistent with maternal first-degree atrioventricular block. A digoxin trough at that time was 1.3 ng/ml (goal 1-2 ng/ml). The patient was admitted to the hospital the next day, at which time the digoxin was discontinued per recommendations by adult and neonatal electrophysiology. Maternal telemetry was continued until the patient's EKG normalized. Digoxin 125 mcg BID was initiated before patient discharge.

A follow up ultrasound at 28 w 0 d showed signs of hydrops including a pericardial effusion, trace fetal ascites, and scalp edema. The fetal heart rhythm appeared consistent with bigeminy at the time. The patient was given antenatal corticosteroids for enhancement of fetal lung maturation, but she again declined admission to the hospital. Ultrasound evaluation the next day showed intermittent periods of trigeminy with transient fetal bradycardia and worsening fetal ascites. Digoxin was increased to 250 mcg BID. She underwent another fetal echo at 28 w 2 d that revealed atrial bigeminy, blocked premature atrial contractions, and a trace pericardial effusion, but neither SVT nor hydrops were present.

For the remainder of the pregnancy, serial fetal echocardiograms showed intermittent, nonsustained SVT, and biophysical profiles BPP remained reassuring. No further medication changes were made. The patient delivered via a scheduled repeat cesarean at 36 weeks due to history of a prior classical hysterotomy. She delivered a live born female infant weighing 2700 g; Apgars of 9 and 9 at 1 and 5 minutes, respectively; umbilical artery pH 7.31 with base deficit 1.7 mmol/L and venous pH 7.37 with base deficit 1.6 mmol/L. The neonate was in sinus rhythm at delivery and was transferred to the NICU for monitoring. An echocardiogram was normal, but an EKG on day of life one showed Wolff Parkinson White. Neonate was started on propranolol 1 mg/kg every 6 hours. The neonate was monitored for 72 hours after initiation of propranolol before being discharged with plans for outpatient follow up.

Case Three

RD was a 26-year-old Caucasian G1P0 who presented at 28 w 6 d with fetal tachycardia identified during a growth ultrasound. The fetus was noted to be appropriately grown at the 75th percentile, but a persistent heart rate in the 250 s was appreciated without evidence of hydrops fetalis. Complete blood counts, comprehensive metabolic panel, TSH, and a maternal EKG were normal. Given the persistent tachycardia, the patient was started on digoxin with a loading dose of 500 mcg followed by 250 mcg every 6 hours for 2 doses. She was then started on a weight-based maintenance dose of 7.5 mcg/kg/day. She was admitted for continuous fetal monitoring to assess for response to treatment, with plans for daily ultrasounds to assess fetal well-being and monitor for development of hydrops. Daily EKGs were obtained to monitor for maternal toxicity.

Digoxin was administered for 24 hours without evidence of fetal response. On hospital day 2, treatment was changed to flecainide 100 mg every 8 hours due to difficulty in increasing dose of digoxin. Flecainide was administered for 36 hours with no fetal response, so adult and pediatric electrophysiology was consulted. Per their recommendations, on hospital day 4 digoxin was added back to the treatment regimen at 250 mcg daily. Continuous telemetry, daily EKGs and daily labs were measured to monitor for maternal toxicity. Fetal ultrasound at that time showed a heart rate in the 220-240 s with new findings of hydrops including a

pericardial effusion, a pleural effusion, and ascites). Antenatal corticosteroids were administered given the progression of concerning fetal findings.

A fetal echocardiogram was obtained on hospital day 5 that confirmed sustained fetal SVT without evidence of structural cardiac abnormalities. Due to changes on the maternal EKG indicative of flecainide toxicity (widening of QRS complex), flecainide was discontinued and amiodarone was started at 600 mg every 8 hours; digoxin was decreased to 125 mcg daily. The amiodarone dose was increased to 800 mg every 8 hours on hospital day 6 due to continued fetal SVT.

Evidence of a fetal response was noted on hospital day 10. The electrophysiology consultant recommended discontinuation of digoxin and initiation of an amiodarone taper (400 mg BID for 1 week, 400 mg daily for 4 weeks, then 200 mg daily till delivery). Inpatient monitoring was continued for 2 more days with normal fetal heart rate tracings noted. A repeat fetal echo was likewise within normal limits. The patient was then discharged to home on hospital day 12.

Follow up ultrasound showed improvement in fetal hydrops at 31 w 2 d, and complete resolution at 35 w 2 d. Twice weekly NSTs continued to be reassuring. The patient underwent an uncomplicated primary cesarean delivery for breech presentation at 39 w 4 d. The patient delivered a vigorous male neonate weighing 3780 g with Apgars of 8 and 9 at 1 and 5 minutes, respectively. Cord gases were not obtained. Maternal amiodarone was discontinued after delivery. Neonate EKG initially showed a normal sinus rhythm, but the neonate was admitted to the NICU for continuous telemetry. A follow up EKG on day of life four was consistent with Wolff Parkinson White, and the infant was started on weight-based propranolol. A fetal echo showed mild ascending aortic dilation but was otherwise normal. Follow up with cardiology showed at 3 weeks of life revealed an infant in normal sinus rhythm in the setting of well-tolerated propranolol therapy.

Discussion

Fetal arrhythmias are detected in 1-2% of pregnancies and are usually identified during routine obstetrical visits or ultrasound. The majority of fetal arrhythmias is of little clinical significance and result in favorable perinatal outcomes. Supraventricular tachyarrhythmias, however, can result in fetal cardiac failure, fetal hydrops and/or fetal demise. Management of fetal arrhythmias is complicated as most means of fetal assessment and treatment are indirect. As external fetal monitoring often cannot trace the high heart rates associated with fetal tachyarrhythmias, ultrasound and echocardiography remain the mainstays of evaluation. However, while these modalities can provide correlates to electrical activity in the heart based on contractile patterns, they cannot directly evaluate it. Other noninvasive techniques such as fetal magneto cardiography and fetal electrocardiography can better evaluate the fetal electrophysiology, but these techniques may not be widely available [1,5,6]. Fetal echocardiography in particular is useful in clarifying the atrial-ventricular relationship and attempting to define the type of tachyarrhythmia [7,8].

Correct identification of the type of tachyarrhythmia is important as it may alter plans for intervention. Supraventricular tachycardia is the most common subtype of tachyarrhythmias, occurring in the setting of an accessory pathway, an atrial ectopic focus, or atrial flutter. Less common subtypes include junctional ectopic tachycardias (JET), which can be associated with SSA antibodies, and ventricular tachycardias, which can occur due to a range of conditions such as unstable heart block, long QT syndrome, ventricular aneurysms, and other cardiac conditions [4,9].

The goal of treatment of tachyarrhythmia is to restore the normal fetal heart rate and prevent or reverse fetal heart failure and/or nonimmune hydrops. Gestational age plays a role in determining the optimal treatment strategy. Treatment is generally reserved for those fetuses at highest risk of developing heart failure, specifically those fetuses with sustained tachycardia and an earlier gestational age at presentation. Other factors such as the heart rate itself and the exact mechanism of tachycardia have not been shown to be predictive of hydrops [3,6,10,11]; however, it is important to note that rapid SVT can deteriorate into atrial fibrillation, which can have serious consequences. For those fetuses with non-sustained tachycardia and no evidence of heart failure or hydrops, no medical treatment is indicated, but close monitoring for conversion to a sustained tachycardia is recommended. For the fetus that is term or late-preterm, delivery is usually recommended as the risks of treatment often outweigh that of delivery [1,6,7,9,12].

For the fetus with sustained tachycardia, previously defined by Cuneo as persistent tachycardia for greater than 12 hours in a 24-hour period, or with evidence of heart failure, medical treatment is recommended if the risk of prematurity outweigh the risks of therapy [12]. There is no consensus on the choice of antiarrhythmic therapy, but a number of commonly used protocols exist. Digoxin is often used as a first line agent as it has been well studied and has an acceptable safety profile. However, it is important to note that digoxin can worsen certain tachyarrhythmias [8]. Digoxin slows conduction through the AV node, and thus can slow ventricular response with atrial fibrillation. However, in patient with a tachyarrhythmia with WPW, digoxin may cause paradoxical worsening of tachyarrhythmia by causing preferential conduction over the faster accessory pathway over the slowed AV nodal pathway [8,13].

Other first or second line therapies include flecainide and sotalol. Amiodarone is generally used in refractory

cases as it has more risk of toxicity, including hypothyroidism and fetal goiter [6-8]. The American Heart Association published a consensus statement that outlines recommendations for generally acceptable agents and dosing protocols [7]. Medication failure can occur due to maternal toxicity, increased volume of distribution in pregnancy, and decreased transplacental transfer of drugs in the setting of hydrops. In these cases, directed fetal therapy via an intramuscular or vascular route can be considered depending on the gestational age.

The cases presented in this paper represent a spectrum of presentations and management of fetal tachycardia. The first case with intermittent SVT required no medical intervention, with no evidence of arrhythmia postnatally. The second case was complicated by maternal social factors, which made fetal monitoring and medication dosing challenging. However, digoxin was successfully used to decrease the amount of time the fetal heart was in SVT, leading to resolution of hydrops and a late-preterm delivery. The management of the third case resulted in complete resolution of the SVT and hydrops, but required trials of multiple regimens, including digoxin alone, flecainide alone, a combination of digoxin and flecainide, and finally amiodarone. Medication changes were instituted due to logistical issues in obtaining correct dosing, lack of fetal response to single agents, and evidence of maternal toxicity with flecainide.

Interestingly, the two fetuses with sustained tachycardia were postnatally diagnosed with a Wolff Parkinson White pattern. This is a condition in which one or more accessory pathways exist between the atria and ventricles that bypass the normal conduction pathway through the AV node. As noted before, an accessory pathway is the most common cause of fetal SVT. This is reported to be present in 1 to 4 out of 1000 live births [5]. Early diagnosis is important as there is a 1.3-1.6% risk of ventricular fibrillation or sudden cardiac death in the first two decades of life [14]. Although the infants in cases 2 and 3 did not demonstrate any tachyarrhythmias after birth, both were treated upon diagnosis with a rate controlling medication and were doing well without arrhythmias at follow up.

Our case reports show that management of fetal SVT can be challenging but that with successful multidisciplinary treatment, good fetal and neonatal outcomes can be obtained.

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