CASE REPORTS

Successful Digoxin Therapy of Fetal Supraventricular Tachycardia

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INTRODUCTION

Irregularities of fetal cardiac rhythm often lead to levels of anxiety among parents and treating physicians. Fetal supraventricular tachycardia (SVT) is a rare but most commonly encountered fetal cardiac arrhythmia in pregnancy that may be associated with cardiac failure, hydrops, and fetal death. If no underlying cardiac defects are present, medical management has been successful.[1] Digoxin has been successfully used to treat fetal supraventricular tachycardia.[2] When therapy with digoxin fails, alternative therapies may be used with equivocal success. In this report, successful fetal therapy for fetal SVT with maternally administered digoxin is presented. The rationale of this therapy and a review of pertinent literature are also presented.

CASE REPORT

26-year-old Gravida 3 Abortion 2 lady at 33 weeks of gestation was referred to fetal medicine unit of our department because of fetal tachycardia noted at her routine antenatal visit. She gave history of loss of fetal movements of one day duration. Real-time ultrasound assessment revealed a singleton intrauterine pregnancy. The anatomical survey including cardiac evaluation was found to be within normal limits. The 4-chamber view of the heart was normal. Most important the finding on M-mode echocardiographic was supraventricular tachycardia with a rate of 240 beats per minute. There was evidence of mild fetal ascites. In her obstetrical history she was married for five years and had two spontaneous abortions at 8 weeks and 11 weeks of gestation. During workup for previous abortions in her first antenatal visit at 6 weeks of gestation she was found to be case of protein C deficiency and was put on low dose asprin and low molecular weight heparin (LMWH). She was on regular antenatal visit. There was no maternal or family history of cardiac disease, and the patient did not consume excessive amounts of caffeine. There was no history of smoking or use of any illicit drugs. The patient was admitted for administration of antiarrhythmic therapy. She was started on a regimen of digoxin, with a loading dose of 0.5 mg followed by 0.25 mg every 12 hours. This was followed by resolution of the tachyarrhythmia and ascites 24 hours of starting digoxin therapy. From day 3 onwards dose of digoxin was reduced to 0.25 mg per day, and there was no recurrence of tachyarrhythmia. At 36 weeks 5 days of gestation there was recurrence of supraventricular tachycardia with fetal heart of 210-240 beats per minute. Emergency cesarean section was done and the patient delivered a 2838 g male infant with APGARs of 8 and 9 at 1 and 5 minutes, respectively. After a 7 days period of neonatal cardiac monitoring (including echocardiography) the patient and her baby were discharged in stable condition. The neonate is healthy four weeks after delivery with no evidence of cardiac rhythm disturbance.

DISCUSSION

Fetal tachycardia, first recognized in 1930 by Hyman et al, is a condition occurring in approximately 0.4-0.6% of all pregnancies. A subset of these cases with more sustained periods of tachycardia is clinically relevant. Fetal supraventricular tachycardia is a rare but most commonly encountered fetal cardiac arrhythmia in pregnancy that may be associated with adverse perinatal outcome.[3] Fetal supraventricular tachycardia is rhythm disturbance characterized by sustained fetal heart rate between 220 and 260 beats per minute. Excessive Caffeine, smoking, illicit drugs, fetal cardiac malformation and extracardiac malformation like diaphragmatic hernia may contribute to frequent fetal premature atrial contractions which may progress to unrelenting tachyarrhythmia. There are three forms of abnormal conduction defects which may result in

supraventricular tachycardia. The first form is that of atrioventricular nodal re-entrant tachycardia, in which two pathways with differing conduction velocities and refractory periods are located within the atrioventricular node. An extrasystole generates an electrical wavefront of depolarization that arrives at the atrioventricular node. The "fast tract" pathway within the atrioventricular node is refractory to the arriving wave of depolarization; the "slow tract" has recovered sufficiently from its prior depolarization to depolarize again with the arrival of the extrasystolic wavefront. However, the His-Purkinje system is still refractory from the prior "normal" depolarization. At precisely the right time, the "fast tract" within the atrioventricular node becomes repolarized and accepts the wavefront from the "slow tract" and depolarizes in a retrograde fashion, re-entering atrial tissue. Depolarization occurs through the fastest conduction pathway available, and so this "circular movement" predominates over the slower impulses generated by the sinoatrial node. In atrioventricular re-entrant tachycardia, the same "circular movement" is established, but the "fast tract" limb of the pathway occurs outside of the atrioventricular node. This pathway directly connects the atria and ventricular myocardium and is known as the Wolff-Parkinson-White syndrome. The final mechanism for supraventricular tachycardia is that of automatic atrial tachycardia resulting from a discreet pacemaker within the atrium outside of the sinoatrial node. Atrial flutter and fibrillation, characterized by rates of 400-460 beats per minute in the fetus, result from "circular pathways" within the atria themselves rather than in atrioventricular nodal or atria-ventricular connecting pathways.

The diagnosis of supraventricular tachycardia is made by using M-mode echocardiography, which may demonstrate paroxysms of atrial tachycardia in the range of 220-260 beats per minute, often following an extrasystole. The management of the fetus with a normal anatomical survey and supra-ventricular tachycardia is dependent upon the gestational age of fetus at the time of diagnosis, and the presence or absence of hydrops fetalis. In the nonhydropic fetus in which fetal lung maturity can be demonstrated (usually > 34 weeks of gestation) delivery with evaluation of the neonate should be considered. At earlier gestational ages, treatment ideally should be directed at the exact underlying electrophysiological abnormality accountable for the arrhythmia. Unfortunately, this is difficult to define for the fetus. Therefore, pharmacologic agents are prescribed on the basis of the arrhythmia itself, rather than on the precise knowledge of the location of the abnormal conduction pathways. For example, if digoxin therapy results in atrioventricular nodal block without resolution of the arrhythmia, then atrioventricular nodal re-entrant tachycardia and atrioventricular re-entrant tachycardia have been ruled out in an indirect fashion. Given the above limits of diagnosis, digoxin usually remains the drug of first choice for the treatment of supraventricular tachycardia. Digoxin at therapeutic levels terminates "circular movements" within re-entrant circuits by prolonging the refractory phase so that the aberrant wave of excitation reaches depolarized tissue. There is debate as to the effective levels of digoxin reaching the cardiac conduction system of the hydropic fetus but larger series of patients have demonstrated resolution of SVT in a significant number of these cases.[4] Second line medications such as sotalol hydrochloride, depress atrioventricular nodal conduction, produce an increase in the duration of the action potential and lengthen the effective and absolute refractory periods.[5] Flecainide, another antiarrhythmic agent which depresses conduction throughout the myocardium and prolongs the refractory period has also been used for treatment of fetal SVT. Its use is mainly concentrated on fetal SVT complicated by hydrops. Amiodarone which prolongs the repolarization has gained much popularity recently. However adverse effects of Amidrone are of concern (mainly neonatal hypothyroidism) and hence should be used as second line treatment.[6] Transplacental therapy should be the mode of therapy in nonhydropic fetuses and first choice in hydropic fetuses. However, when conversion to sinus rhythm is not achieved with several maternally administered antiarrhythmic drugs, one may also opt for direct fetal therapy. In the international literature, several modes of administration, intraumbilical, intra-amniotic, intraperitoneal, intramuscular and intracardiac have been described. An intraumbilical injection allows direct access to the fetal circulation and thereby the potential for a quick response to therapy, a characteristic also observed with intracardiac injections. However, both of these invasive measures carry significant risk to the fetus. Intraperitoneal, intra-amniotic and intramuscular injections (preferably in the buttock of the fetus), pose less risk to the fetus and provide a more sustained release of the medication. If one chooses to opt for direct fetal therapy, one must bear in mind that the antiarrhythmic drug will probably distribute to the maternal compartment, unless this compartment is primed with the drug. Therefore, direct fetal therapy should always be administered as an adjunct to maternal therapy. The direct fetal treatment approach should only be used in cases of fetal tachycardia complicated by hydrops with resistance to transplacental multidrug therapy.[7]

The diagnosis, careful assessment and management of a fetal arrhythmia may lead to a successful outcome. The complexity of the problems experienced may warrant early referral to a tertiary center where the overall management of the mother, fetus and neonate, may be undertaken.

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