Atrial Fibrillation with Rapid Ventricular Response in Pregnancy

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SUMMARY

Objective: To report a case of atrial fibrillation with rapid ventricular response occurring during pregnancy.

Case Report: A 35-year-old woman, gravida 3, para 1, abortus 1, with a history of persistent supraventricular arrhythmia, presented at 22 weeks' gestation. After adenosine administration, electrocardiography revealed atrial fibrillation with rapid ventricular response. The episode was complicated by hemodynamic instability and was refractory to verapamil. Sinus rhythm was restored after synchronized electrical cardioversion under sedation. Sotalol (80 mg) was given for arrhythmia and to control heart rate. The patient experienced a second episode of supraventricular arrhythmia at 26 weeks' gestation, which was also reversed after cardioversion. She delivered a healthy male baby at 38 weeks' gestation via scheduled cesarean section.

Conclusion: Arrhythmias with underlying heart disease can result in serious hemodynamic deterioration. Electrical cardioversion is well-tolerated and effective in pregnant women and should not be withheld if clinically indicated. [*Taiwan J Obstet Gynecol* 2008;47(3):327–329]

Key Words: atrial fibrillation, cardioversion, paroxysmal supraventricular tachycardia, pregnancy, rapid ventricular response

Introduction

The incidence of arrhythmias may increase during pregnancy owing to the hyperdynamic state. Most healthy pregnant women with transient arrhythmias have a good prognosis and do not require antiarrhythmic treatment. However, arrhythmias linked with underlying heart disease, particularly congenital heart disease, can result in serious hemodynamic deterioration. All antiarrhythmic agents can potentially cause adverse effects in both mother and fetus during pregnancy. As medical and surgical treatment for heart disease improves, obstetricians may encounter more pregnant women with congenital or rheumatic heart disease, so being both alert to and familiar with the issues relating to pregnant women with arrhythmias is important.

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Here, we present a case of a woman whose pregnancy was complicated by atrial fibrillation (AF) with rapid ventricular response (RVR) and hemodynamic instability, who was successfully treated with electrical cardioversion and sotalol.

Case Report

A 35-year-old woman, gravida 3, para 1, abortus 1, with a 5-year history of arrhythmia without need for medication, presented at 22 weeks' gestation. During this pregnancy, she had experienced frequent palpitation attacks that were spontaneously relieved by rest. This was followed by persistent palpitations and chest discomfort. She went to the emergency room of the West Garden Hospital, where a complete electrocardiogram (EKG) showed supraventricular arrhythmia (SVT), with a ventricular rate of 185 beats per minute (bpm). Laboratory data including complete blood count, renal function, liver function, electrolytes and cardiac enzymes were all within normal limits. The patient failed to respond to the rapid administration of three intravenous doses

of 12 mg of adenosine. Verapamil 2.5 mg was later given twice, but also without success. She was then transferred to our emergency room. A complete EKG showed SVT with a heart rate (HR) of 180 bpm. Adenosine 12 mg rapid intravenous push was prescribed, after which her HR fell from 186 bpm to 138 bpm, and her EKG showed AF with RVR. Unfortunately, the patient's HR quickly rose again to 182 bpm. Two doses of intravenous verapamil 2.5 mg were prescribed, but with short-term responses. Amiodarone was avoided on account of its potential adverse effect on fetal thyroid function. Because of persistent arrhythmia (SVT, AF with RVR) refractory to medication, external electrical cardioversion was indicated. A synchronized direct current (DC) shock (100 J) was performed under sedation. Following cardioversion, the maternal HR returned to sinus rhythm and the fetal heartbeats were stable. However, SVT with HR 180–190 bpm and blood pressure (BP) 91/60 mmHg recurred again 2 hours later. A second electrical cardioversion (synchronized 100 J) was performed. The maternal HR returned to sinus rhythm without fetal heartbeat deceleration. Two-dimensional echocardiography revealed moderate mitral regurgitation, moderate to severe tricuspid regurgitation, dilated right and left atria and normal left ventricular size with good left ventricular contractility. A third SVT attack occurred again 2 hours later but again returned to sinus rhythm after a third electrical cardioversion (synchronized 50 J). Sotalol 80 mg twice daily was prescribed, and the patient was admitted to the intensive cardiac care unit for further observation. One further episode of SVT occurred in the intensive cardiac care unit and was returned to normal sinus rhythm (80–90 bpm) after a fourth DC shock (synchronized 50 J) under propofol (class B) sedation. After this, the patient's condition remained stable.

Follow-up laboratory data showed anemia (hemoglobin fell from 11.8 g/dL to 7.7 g/dL), electrolyte imbalance, and hypoalbuminemia. No evidence of internal bleeding or hemolysis was noted. Packed red blood cells 1,000 mL were transfused to correct the anemia and to reduce the cardiac load. Thyroid function tests and antinuclear antibodies were all within normal limits. There were no further episodes of arrhythmia, and the patient was discharged in a stable condition 6 days later. Oral sotalol 80 mg twice daily was maintained after discharge.

EKG follow-up 1 week later in the outpatient department showed normal sinus rhythm with a rate of 63 bpm. The patient's BP was 120/60 mmHg. Unfortunately, however, at 26 weeks' gestation, palpitations returned while sleeping and persisted for more than 2 hours. She came to our emergency room again where her HR was 180 bpm and BP was 76/42 mmHg. EKG showed SVT. Oral sotalol 80 mg was given. Electrical cardioversion (synchronized 20 J) restored normal sinus rhythm. A fetal monitor revealed a reassuring fetal heartbeat. After observation for 6 hours in the emergency department, with no more SVT attacks, she was discharged. No further episodes of SVT occurred under sotalol treatment. Owing to worries that SVT induced by labor pains might cause maternal hemodynamic compromise and fetal distress, a scheduled cesarean section was performed smoothly at 38 weeks' gestation. A male baby weighing 3,006 g was delivered with Apgar scores of 9 at 1 minute and 9 at 5 minutes. The mother's postpartum course was uneventful, and she was scheduled to receive further management in the cardiologic outpatient department.

Discussion

Arrhythmias can occur *de novo* or can be exacerbated by pregnancy. Potential risk factors in pregnancy that can promote arrhythmogenesis include the hyperdynamic state, the altered hormonal milieu, and underlying heart disease [1]. Normal pregnancy is characterized by increased cardiac output, reduced systemic vascular resistance, and a modest decline in mean BP. These changes are associated with a 10–15 bpm increase in HR. SVTs include atrial tachycardia, atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reentrant tachycardia (AVRT), atrial flutter, and AF.

The most commonly encountered SVT in both healthy gravid and nongravid patients is AVNRT, which occurs in those with and without structural heart disease [2]. It is uncommon for AVNRT to cause any significant problems for the healthy pregnant woman or the fetus. In contrast, AVNRT occurring in the presence of heart disease may produce hemodynamic instability because of the rapidity of the tachycardia [3]. AVRT caused by accessory pathways associated with some forms of congenital heart disease is the second most common form of SVT, and can result in serious hemodynamic deterioration. AF is also rare in healthy pregnant women, but is encountered frequently in pregnant women with underlying heart disease or thyrotoxicosis. It may have serious hemodynamic consequences in patients with heart failure due to systolic or diastolic dysfunction, congenital heart disease, or a rapidly conducting antegrade accessory pathway [4].

Management of arrhythmias in pregnancy includes the use of pharmacologic and non-pharmacologic therapies, such as cardioversion and radiofrequency ablation. All antiarrhythmic agents have the potential to cause adverse effects in both the mother and the fetus. Adenosine is indicated for the termination of acute

AVNRT and AVRT, and its efficacy is >90%, without obvious adverse effects. Adenosine is also recommended for unknown types of SVT, because it can assist in their differential diagnosis [5–8].

In our patient, the type of SVT was initially difficult to determine. Adenosine was the first choice of treatment, because AVNRT and AVRT are the most commonly encountered types of paroxysmal supraventricular tachycardia. After challenged with adenosine, the HR declined and a complete EKG showed AF with RVR. According to American College of Cardiology/American Heart Association/European Society of Cardiology guidelines, the class I recommendation for management of AF in pregnancy is to control the rate of ventricular response with digoxin, a beta-blocker, or a calcium channel blocker. Additionally, direct current cardioversion is indicated if the patient is hemodynamically unstable because of AF [9]. Verapamil (a calcium channel blocker) is effective for AVNRT and AF or AF with RVR, with no reported embryopathy or adverse fetal effects [10,11], but our patient was refractory to verapamil and persistent SVT with hemodynamic instability was noted. Digoxin was not used in the emergency room on account of its narrow therapeutic range and its ineffectiveness in controlling ventricular rate during acute episodes. Amiodarone is a highly effective drug for treating maternal and fetal ventricular and SVTs, including those utilizing a bypass tract, but it should be used cautiously in pregnancy (class D). It is highly protein- and lipid-bound, resulting in significant bodily storage, with a half-life of 26 days to 6 months. Furthermore, each 200-mg tablet contains 75 mg of iodine, and serious adverse effects such as neonatal hypothyroidism have been reported [12,13]. Our patient refused amiodarone. Electrical cardioversion was, therefore, performed under sedation with propofol, which was chosen for its rapid onset, short duration and safety in pregnancy (class B). The HR returned to sinus rhythm after synchronized DC shock. Electrical cardioversion is well tolerated and effective in pregnant women and should not be withheld if clinically indicated [7].

Because AF is rare in pregnancy unless associated with underlying heart disease or thyrotoxicosis, cardiac echo and thyroid function tests were performed in our patient. Two-dimensional echocardiography revealed moderate mitral regurgitation, moderate to severe tricuspid regurgitation, and dilated right and left atria. Dilated atria caused by mitral and tricuspid regurgitation can cause the myocardial fibers to stretch, so resulting in abnormal electrical activity. Sotalol is a class III antiarrhythmic agent that blocks potassium channels, prolongs action potential duration and lengthens QT intervals. It is also a noncardiac selective beta-adrenergic blocker and possesses 30% of the beta-blocking activity of propranolol. Sotalol demonstrates both antiarrhythmic and rate controlling effects. Because the patient was suffering from acute paroxysmal AF which could be restored to normal sinus rhythm, anti-thrombotic therapy was not required to prevent embolic events.

Taking into account the stress of labor pains, hemodynamic changes after delivery and uncertainty during the course of labor, a cesarean section was performed smoothly at 38 weeks' gestation. This was a successful experience in managing a pregnant women with frequent paroxysmal supraventricular tachycardia. External cardioversion is safe and recommended if first-line pharmacologic treatment fails and the patient remains hemodynamically unstable.

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