Brief Report

Supraventricular Tachycardia Resistant to Treatment in a Pregnant Woman

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Supraventricular tachycardia is relatively common in pregnancy, and is related to the normal physiologic changes of the cardiovascular system in the gravid state. When the arrhythmia is secondary to an ectopic atrial focus of automaticity, it can be particularly difficult to convert to a normal sinus rhythm with medications or DC cardioversion. A case of supraventricular tachycardia complicating a normal pregnancy and resistant to treatment is presented, with particular attention devoted to flecainide and propranolol, agents ultimately effective in this patient.

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Cardiovascular physiology undergoes tremendous change during pregnancy.[1] Dysrhythmias are far from rare in the gravid patient and include most disorders of rhythm seen in nongravid patients.[2] Of the dysrhythmias seen during pregnancy, supraventricular tachycardias frequently occur, and paroxysmal supraventricular tachycardia (PSVT) is often a complication of the third trimester.[3]

Supraventricular tachycardias are divided into two major groups: those with ectopic or automatic foci and those depending on delayed conduction and reentry. It is now generally agreed that most PSVT seen in adults is due to easily terminated reentry.[4] Unlike PSVT, ectopic supraventricular tachycardia occurs rarely and is characteristically persistent and refractory to treatment.[5]

Supraventricular tachycardias are usually well tolerated in the pregnant patient without underlying cardiac disease and usually resolve spontaneously. However, both PSVT and ectopic supraventricular tachycardias have been associated with fulminant pulmonary edema in the setting of organic or valvular heart disease.

Several cases of supraventricular tachycardia during pregnancy have been reported in the medical literature, ranging from those easily controlled or converted to normal rhythms either pharmacologically or electrically[6,7] to those more difficult to control or absolutely resistant to intervention.[8] Even in the more resistant cases, the absence of underlying cardiac pathology favors an uncomplicated delivery, often with spontaneous resolution of the dysrhythmia postpartum.

The following case report describes a pregnant patient with ectopic atrial tachycardia, initially resistant to pharmacologic and electrical cardioversion but eventually controlled after trials of several medications.

Case Presentation

A 33-year-old woman, G4, P0, A3, with a history of palpitations (which spontaneously resolved and for which she had never seen a physician), went to the emergency department at approximately 25 weeks' gestation with a complaint of rapid heartbeat and mild shortness of breath. The patient had developed a rapid heart rate the previous evening and had tolerated it throughout the night before seeking medical attention the next morning.

The patient's heart rate was noted to be 175 to 215 beats per minute with fetal heart tones noted to be 140 to 160 beats per minute. Her blood pressure was stable at 124/72 mm Hg and her respiratory rate was 18 breaths per minute. Physical examination revealed a gravid white woman in mild distress without dyspnea. Lung fields were clear, there was no jugular vein distension, the cardiac apex was not displaced, there were no heaves or thrills, and auscultation revealed no abnormalities. No central or peripheral cyanosis was noted. An ECG demonstrated a regular, narrow complex tachycardia with

Figure 1. Supraventricular tachycardia at rate of 170 beats per minute.

Figure 2. Heart rate response to oral propranolol while receiving flecainide therapy.

uniform P waves and no delta waves at a rate of 170 beats per minute (Figure 1).

Forty percent oxygen was provided with a mask. Conversion to normal sinus rhythm was attempted with ice packs, vagal maneuvers, and carotid massage, to no avail. Intravenous (IV) access was obtained, and the patient received digoxin 0.25 mg IV without effect. Subsequently, she received adenosine 6 mg IV, resulting in a decrease in heart rate from the 200 range to the low 70s accompanied by severe lightheadedness, nausea, and a sense of dread (with stable blood pressure) before the heart rate returned to the 180 to 200 range. She then received two additional boluses of digoxin 0.25 mg IV without effect.

The patient was transferred to the intensive care unit and was given two doses of propranolol 1 mg IV; there was no effect on her heart rate, but there was a slight decrease in her systolic blood pressure. After demonstrating no change in atrial rate, she received two boluses of verapamil 2.5 mg IV, resulting in a transient decrease in the atrial rate to approximately 100 beats per minute before it returned to 190. She tolerated her dysrhythmia throughout the night with stable blood pressures and respiratory rates and a stable fetal heart rate in the 140 to 160 range. The next morning, she received a 1-g loading dose of procainamide and was started on a 3 mg/min infusion, with no effect on her supraventricular rhythm. Procainamide and N-acetylprocainamide levels were obtained, and subsequently the procainamide drip was increased to 4 mg/min to obtain adequate procainamide levels. The patient was then given three separate 5-mg IV boluses of metoprolol without effect. DC cardioversion was attempted with anteroposterior paddles at 50 J and then 150 J without change in rhythm. Laboratory findings were unremarkable: her complete blood count, thyroid function, blood glucose, and O'Sullivan test results were all within normal limits. Anticardiolipin antibodies were negative at a titer of 1:64, and antinuclear antibody was positive at a titer of 1:80.

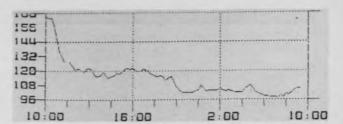On the 3rd hospital day, procainamide was discontinued and the patient was started on flecainide, 100 mg by mouth (PO) every 12 hours. After the sixth dose of flecainide, propranolol 20 mg PO every 6 hours was added. This resulted in a gradual decrease in the atrial rate (Figure 2). The propranolol was increased to 30 mg PO every 6 hours, and the flecainide was decreased to 50 mg PO every 12 hours and then discontinued on the 9th hospital day, with heart rates noted in the 100 to 110 range.

The patient was discharged on the 11th hospital day with her heart rate in the 100 to 110 range and otherwise stable vital signs. Propranolol 30 mg PO every 6 hours was prescribed, and she continued taking this medication during the remainder of her pregnancy. Fetal surveillance, with a weekly biophysical profile, was continued, and there were no abnormal findings. At term, the patient had a vaginal delivery with vacuum extraction. Her heart rate spontaneously decreased to 70 to 80 beats per minute about 1 hour before delivery of a healthy female infant. In the postpartum period, the patient complained of palpitations on two occasions, but her pulse rate was never noted to be greater than 100 beats per minute. For this reason, her propranolol was tapered over the next 3 weeks. She has done well since discontinuation of the medication and has had no further symptoms or cardiac arrhythmias.

Discussion

Although no electrophysiologic studies were performed and no prior electrocardiogram was available for comparison, the consistently distinct P-wave morphology, normal PR interval, failure to respond to cardioversion, and duration of the rhythm argued for an automatic or ectopic focus. Patients with ectopic atrial tachycardias are difficult to convert, and are frequently refractory to treatment with sedation, vagal stimulation, or administration of quinidine, procainamide, or digoxin.[5,9] Several antiarrhythmic agents were used in an attempt to control our patient's ectopic atrial tachycardia. Digoxin and verapamil are frequently successful in breaking reentry circuits but, as demonstrated in this patient, may fail to alter rhythm. Propranolol is effective in controlling both re-

entry and automatic atrial tachycardias[10] but was unable to be initially used in our patient because of her markedly lowered systolic pressure. Concerns about causing placental insufficiency prevented increasing the dose.

After failing to suppress or convert our patient's supraventricular tachycardia with trials of digoxin, adenosine, verapamil, procainamide, metoprolol, and cardioversion, flecainide and propranolol were employed.

Flecainide acetate has local anesthetic activity and belongs to the membrane stabilizing (class I) group of antiarrhythmic agents. It slows depolarization of the cardiac cell, but unlike most other antiarrhythmic agents, it has little effect on the duration of the action potential and so it is classified further into subclass C.[11-13]

Flecainide produces a dose-related decrease in intracardiac conduction in all parts of the heart (prolongation of PR and QRS interval), with the greatest effect on the His-Purkinje system.[11] It appears to be more effective than quinidine in suppressing premature ventricular contractions (PVCs) and nonsustained ventricular tachycardia, but it can aggravate existing arrhythmias and precipitate new ones, especially in patients with underlying heart disease and sustained ventricular tachycardia.[11-14] It can also aggravate congestive heart failure.[11] In both ventricular tachycardias and atrioventricular reentrant tachycardias, flecainide frequently reduces the tachycardia in patients in whom it does not completely abolish the dysrhythmia.[13]

Flecainide is well absorbed after oral administration. The plasma half-life averages 20 hours, which generally permits administration of the drug every 12 hours.[12,13] Steady-state levels are approached in 3 to 5 days. Once at steady state, no accumulation occurs during continued therapy unless the patient is renally impaired. Flecainide elimination depends on renal function.[11]

The efficacy of flecainide depends on the patient's underlying cardiac disease and type of arrhythmia.[13] In patients with high-frequency ectopic depolarizations and episodes of nonsustained ventricular tachycardia, flecainide has produced more than 80% suppression of arrhythmia and virtual abolition of complex forms in approximately 90% of patients (while generally preserving left ventricular performance). Doses of 100 to 200 mg twice daily achieved this effect in 75% of such patients.[13]

The provocation of serious ventricular arrhythmias by flecainide has been described.[11-13] The incidence approaches 20% in patients with severe ventricular dysfunction and a previous history of sustained ventricular tachycardia who are taking more than 400 mg of the drug daily; moreover, in about 10%, the proarrhythmic events were fatal.[11,13] Patients with less severe dysrhythmias (chronic PVCs and nonsustained ventricular tachycardia) have had a 3% rate; 0.1% were fatal.[11] First degree,

second degree, and third degree A-V block have also occurred with flecainide. Transient dizziness and visual difficulties are fairly common but can be managed with reductions in the dose. Unlike procainamide, quinidine, and disopyramide, flecainide rarely produces gastrointestinal effects.[11,13]

Flecainide is a category C drug for use in pregnancy. In two recent reports, flecainide 100 to 150 mg twice daily was given to a 24-year-old woman, G4, P1, A3, at 35 weeks' gestation for treatment of Wolff-Parkinson-White syndrome.[15] In the other report, a 23-year-old woman was being treated for bursts of ventricular tachycardia and polymorphous ventricular premature complexes. She was given flecainide 100 mg twice daily and sotalol 80 mg twice daily, and became pregnant during therapy.[16] Both patients gave birth to normal babies with no congenital anomalies or changes in the neonatal electrocardiograms or hemodynamics.[15,16]

Propranolol is a β-adrenergic blocking agent that competes with β-adrenergic agonists for available β-receptor sites. It inhibits both β_1-receptors (located chiefly in cardiac muscle) and β_2-receptors (located chiefly in the bronchial and vascular musculature). It inhibits the chronotropic, inotropic, and vasodilator responses to β-adrenergic stimulation. Clinical responses to beta blockade include slowed sinus heart rate, depressed A-V conduction, decreased cardiac output at rest and with exercise, and reduced systolic and diastolic blood pressure at rest and with exercise.[17]

Propranolol, a category C drug, has been extensively used during pregnancy and there have been many favorable reports on its effects. Some reports, however, have associated the use of propranolol with intrauterine growth retardation, bradycardia, apnea, hypoglycemia, and hyperbilirubinemia in the newborn. The incidence of these complications is low, and the use of propranolol during pregnancy may be considered safe.[18,19]

Conclusions

Supraventricular tachycardias in pregnancy are relatively common and are likely related to the changes in cardiovascular physiology that occur in this state. Supraventricular dysrhythmias may be divided into two groups: the more common type whose mechanism is based on delayed conduction and reentry, often presenting as PSVT; and those whose mechanism is automatic and ectopic. The latter are less frequent and present as persistent atrial tachycardias. Ectopic atrial tachycardias are notoriously resistant to treatment, and in this case the dysrhythmia was controlled with propranolol and flecainide initially, then propranolol alone.

Hemodynamic stability is essential to a healthy pregnancy and is the goal of aggressive treatment of these dysrhythmias. Medications commonly used in nonpregnant patients, and indeed DC cardioversion when necessary, must be employed with the understanding that the risk to the pregnancy of cardiovascular instability may outweigh the risk of these modalities. Our case illustrates the acute and long-term management of ectopic atrial tachycardia in pregnancy, the difficulty that may be encountered in controlling this type of dysrhythmia, and in this case the spontaneous resolution of the problem at time of delivery.

Acknowledgment

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