A Case of Supraventricular Tachycardia Associated with Wolff-Parkinson-White Syndrome and Pregnancy

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ABSTRACT

A 25-year-old pregnant woman was admitted with frequent episodes of supraventricular tachycardia associated with Wolf-Parkinson-White syndrome. She was treated acutely with adenosine therapy during induction of labor and post-partum. Generally, pharmacologic treatment should be undertaken only for symptomatic arrhythmias or in hemodynamically compromised patients. Adenosine is the first choice for acute treatment of supraventricular tachycardia in pregnancy; several other options exist, but all have the potential for negative side effects for mother and fetus. Direct-current cardioversion is acceptable in all stages of pregnancy.

CASE REPORT

A 25-year-old woman at 39 weeks gestation was transferred to our institution from an outside hospital. Prior to transfer, the patient had developed supraventricular tachycardia (SVT) with a heart rate of approximately 200 beats per minute (bpm). Vagal maneuvers had no effect until she stood up to go to the bathroom, at which time she converted spontaneously to sinus rhythm. She was started on oral labetalol to control her heart rate. Two hours later the patient had a second episode of SVT at 220 bpm, and when vagal maneuvers failed she was given 6 mg of intravenous adenosine resulting in conversion of the SVT to sinus rhythm. She had 2 more episodes of SVT that were treated with intravenous adenosine before transfer to our facility.

The patient's past medical history was significant for obesity and a history of Wolff-Parkinson-White (WPW) syndrome diagnosed in childhood. She had one previous pregnancy with an uncomplicated course. The patient reported approximately 5 yearly episodes of palpitations that lasted seconds to minutes and resolved spontaneously. Her family history was positive for maternal WPW and asthma. The only medications she had been using were prenatal vitamins.

Blood chemistries were remarkable for mildly elevated liver function tests (alkaline phosphatase 197, AST 66, ALT 82) and magnesium of 1.7. Two electrocardiograms (ECGs) from outside our facility were available (Figure 1). The first ECG showed SVT with narrow QRS complexes (measuring 0.08 seconds) and a heart rate of 196 bpm. The second ECG (12 lead) showed sinus rhythm with a heart rate of 80 bpm, PR interval of 0.12 secs, and QRS duration of 0.08 secs. The electrical axis was intermediate. There were positive delta waves in I, aVL and V2-V6; the ST segments were normal. An echocardiogram was essentially normal with normal left ventricle size and systolic function and an ejection fraction of 58%. Color flow imaging revealed mild mitral and tricuspid regurgitation.

A physical examination showed a pulse of 80/min and blood pressure of 141/89 mmHg. Her cardiovascular exam revealed regular rate and grade 2/6 systolic ejection murmur at apex with no gallops or rubs. An examination of the lungs and abdomen was unremarkable. She had trivial edema in her lower extremities bilaterally. The fetal heart tones were in the 130s with moderate variability by continuous external fetal monitoring.

Overall, these findings were consistent with a pregnant female with a known history of WPW syndrome and recurrent, narrow complex SVT probably worsened by the physiologic changes of pregnancy.

Induction of labor with Pitocin and artificial rupture of membranes allowed the patient to progress to delivery of a normal infant who did not require resuscitation. During the immediate postpartum period, the patient had 2 more episodes of SVT, which resolved after intravenous administration of adenosine. She was seen in our cardiology clinic after dis-

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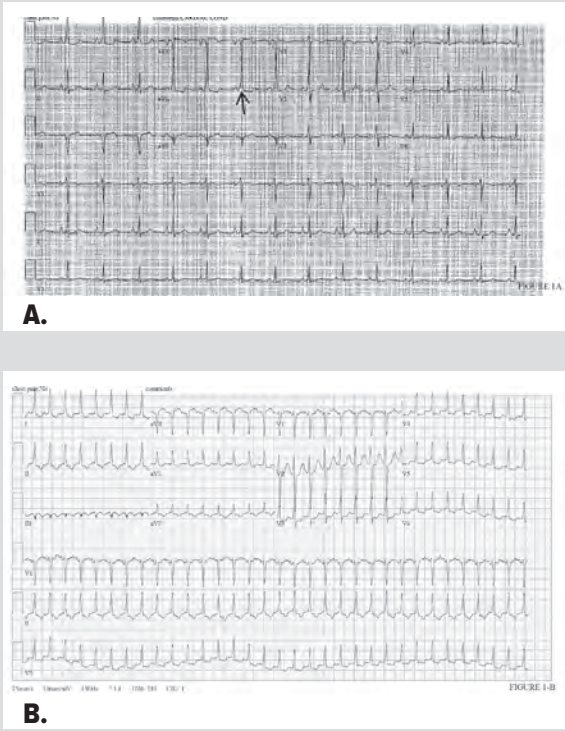
Figure 1. 12 Lead ECG



A. Shows baseline rhythm and pre-excitation. Arrows point to "delta waves" seen between the P wave and onset of QRS complex.
B. Shows SVT with narrow QRS complex tachycardia, QRS duration 0.08 secs.

Figure 2. A Narrow QRS Complex Tachycardia (also known as "Orthodromic reentrant atrioventricular tachycardia")

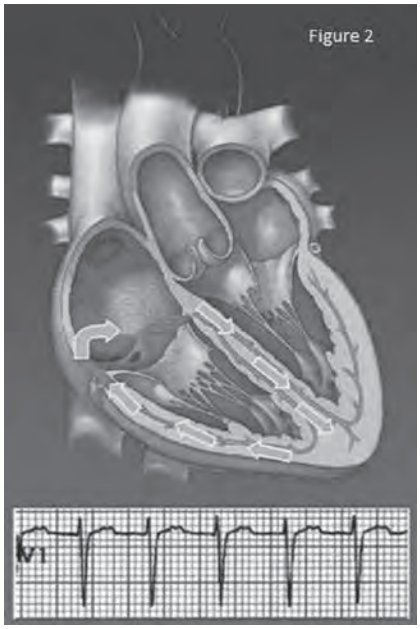


Figure 2. A Narrow QRS Complex Tachycardia (also known as "Orthodromic reentrant atrioventricular tachycardia") Arrows demonstrate anterograde conduction of the impulse from the AV node to the ventricle and then retrograde to the atrium via the accessory pathway (right-sided accessory pathway).

charge from the hospital to discuss various therapies for long-term treatment of her tachyarrhythmia associated with WPW syndrome. A post-discharge, 24-hour Holter monitor showed no evidence of SVT, and both mother and newborn infant were doing well otherwise.

DISCUSSION

Palpitations in pregnancy commonly are due to premature atrial or ventricular contractions or caused by sinus tachycardia.[1] In patients with WPW syndrome, atrioventricular reciprocating tachycardia can lead to hemodynamic compromise that needs immediate treatment.[2,3] Treatment of such patients can be challenging.

Several reports have described successful nonpharmacologic (eg, carotid sinus massage, Valsalva maneuver) and pharmacological treatments of supraventricular arrhythmias in pregnant patients.[4-6] It is recommended that nonpharmacological maneuvers be tried first before embarking on pharmacological treatments. As a general rule, all antiarrhythmics should be regarded as potentially toxic to the fetus and, as such, should be avoided if possible during the first trimester of pregnancy. The American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology have published recommendations summarizing evidence and expert opinion for classifying indications (Tables 1 and 2).[7]

The drugs used more commonly to acutely terminate these arrhythmias include adenosine, beta-blockers, and calcium channel blocker.[6-12] Adenosine is considered the drug of choice, given its short half-life. In general, beta blockers with beta-1 properties are preferred because, theoretically, they interfere less with peripheral vasodilatation and uterine relaxation.[7] Calcium channel blockers have been used, but due to the risk of causing maternal hypotension and uterine relaxation, they generally are used with caution.[7] Agents of second choice include intravenous procainamide.[6,13] Elective and emergent direct-current cardioversion in all stages of pregnancy have been performed safely and should always be considered when needed.[14,15]

Caution: The above-mentioned drugs can be used in WPW patients with narrow QRS complex tachycardia (orthodromic atrioventricular reentrant tachycardia) (Figure 2). However, they are not recommended for patients with wide QRS complex tachycardia (antidromic atrioventricular reentrant tachycardia) due to the potential of causing selective electrical conduction over the bypass tract (accessory pathway) from atria to ventricles, thus causing complex arrhythmias and even death

Table 1. Level of Evidence and Recommendations by the American College of Cardiology, American Heart Association Task Force, and the European Society of Cardiology

Recommendations for classifying indications (summarizing evidence and expert opinion)	
Class 1	Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment • Class IIa: The weight of evidence or opinion is in favor of the procedure or treatment • Class IIb: Usefulness/efficacy is less well established by evidence or opinion
Class III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful
Level of Evidence	
Level A	Derived from multiple randomized clinical trials
Level B	Data are on the basis of a limited number of randomized trials, non-randomized studies, or observational registries
Level C	Primary basis for the recommendation was expert consensus

Table 2. Recommendations for Treatment Strategies for Supraventricular Tachycardia (SVT) During Pregnancy[a]

Recommendation	Classification	Level of Evidence
Treatment Strategy : Acute conversion of PSVT		
Vagal maneuver	I	C
Adenosine	I	C
DC cardioversion	I	C
Metoprolol, propranolol	IIa	C
Verapamil	IIb	C
Treatment Strategy: Prophylactic therapy		
Digoxin	I	C
Metoprololb	I	B
Propranololb	IIa	B
Sotalol,[b] flecainide	IIa	C
Quinidine, propafenone, verapamil	IIb	C
Procainamide	IIb	B
Catheter ablation	IIb	C
Atenolol	III	B
Amiodarone	III	C

Abbreviations: DC, direct current; PSVT, paroxysmal supraventricular tachycardia; SVT, supraventricular tachycardia.

The order in which treatment recommendations appear in this table within each class of recommendation does not necessarily reflect a preferred sequence of administration. Please refer to text for details. For pertinent drug dosing information, please refer to the ACC/AHA/ESC Guidelines on the Management of Patients with Atrial Fibrillation.

[a]Adapted from the Journal of the American College of Cardiology.[7] Used by by permission.

[b]Beta-blocking agents should not be taken in the first trimester, if possible.

when atrial fibrillation or atrial flutter develop.

The ECG is helpful in diagnosing and understanding the underlying arrhythmias in patients with WPW syndrome for atrioventricular reentrant tachycardia (AVRT). If the tachycardia has a narrow QRS complex of ≤ 0.08 secs (orthodromic AVRT), the antegrade limb is the pathway that conducts the impulse to the ventricle via the AV node/his purkinje system. In this scenario, the delta wave seen during sinus rhythm is lost since anterograde conduction is not via the accessory pathway; ie, ventricle is not pre-excited (Figure 2). If the tachycardia has a wide QRS complex of ≥ 0.12 secs (antidromic AVRT), the antegrade limb is usually the accessory pathway. An antidromic AVRT may be associated with a wide QRS complex in the presence of a pre-existing or rate-related functional bundle branch block.

Chronic or prophylactic therapy for SVTs during pregnancy is challenging and the general recommendations are to use the lowest dose of the safest drug available (Tables 1, 2, 3). Several reports have addressed the use of anti-arrhythmic agents in pregnancy.[16-22] Since 1975, the US Food and Drug Administration (FDA) has assigned risk factors to all drugs available in the United States.[7] Specific information on the fetal and neonatal risks of maternal drug ingestion during pregnancy and lactation also are available from several resources in the pharmacological literature.

Catheter ablation is the procedure of choice in selected patients for drug refractory, poorly tolerated SVT in pregnancy.[7,23] Because of the complex nature of the procedure and the potential for inducing life-threatening arrhythmias, this should be done only in tertiary care centers where advanced fetal heart monitoring and other expertise is readily available for the patient and fetus.

Caution: Agents with AV nodal specific activity (beta blockers, calcium blockers, and Digoxin) are second-line drugs for the chronic suppression of orthodromic atrioventricular reentrant tachycardia (OAVRT) in patients with WPW syndrome. In addition, these AV nodal blocking agents should not be given to patients with WPW syndrome who have documented atrial fibrillation or flutter in addition to OAVRT.

CONCLUSION

Tachyarrhythmia in pregnancy in association with WPW is considered serious and should be evaluated because of potential life-threatening consequences to both mother and fetus. In such patients, close monitoring should occur to prevent maternal and fetal morbidity and mortality. Patients with mild symptoms and normal hearts need reassurance; treatment with antiarrhythmics is reserved for intolerable symptoms.[6-7] The

Table 3. Antiarrhythmic Drugs in Pregnancy[24]

Drug	Pregnancy	Breastfeeding
Adenosine	No evidence of increased risk of teratogenesis or increased risk of adverse fetal/neonatal effects	No information. Because of very short half-life, it is unlikely to have any adverse effects on the neonate.
Amiodarone	Has been associated with serious adverse effects. Congenital goiter/hypothyroidism and hyperthyroidism can occur after in utero exposure. Other potential risks include prolonged QT interval in neonates.	Not recommended because of potential risk of hypothyroidism in neonate.
Beta blockers	No evidence of increased risk of teratogenesis, but some (particularly atenolol) may impair fetal growth when used for a prolonged duration in the 2nd and 3rd trimesters. Use only in the 3rd trimester is associated with reduced placental weight. Newborns of women taking these drugs near delivery are at risk of bradycardia, hypoglycemia, and other symptoms of beta-blockade. Among this class of drugs, atenolol appears to have the most unfavorable effect on birth weight.	AAP considers these agents compatible with breastfeeding, but newborns should be observed for signs of beta-blockade. Atenolol is a weak base that will accumulate in milk. Accumulation is enhanced by its water-soluble, low protein binding, little or no hepatic metabolism, and renal excretion properties. Because it has been associated with beta-blocking effects and cyanosis in nursing infants, it is best avoided during breast feeding.
Digoxin	No evidence of increased risk of teratogenesis or increased risk of adverse fetal/neonatal effects.	AAP considers digoxin compatible with breastfeeding.
Flecainide	Developmental toxicity has been noted in animals, but limited information on human risk from early pregnancy exposure. This risk appears to be low when used for refractory fetal arrhythmia. It may be the treatment of choice for tachycardia in hydropic fetuses.	AAP considers flecainide compatible with breastfeeding.
Procainamide	No evidence of increased risk of teratogenesis or increased risk of adverse fetal/neonatal effects.	AAP classifies procainamide as compatible with breastfeeding. However, the long-term effects of exposure in the nursing infant are unknown, particularly with respect to potential drug toxicity (eg, development of antinuclear antibodies and lupus-like syndrome).
Quinidine	No evidence of increased risk of teratogenesis. In therapeutic doses, the oxytocic properties of quinidine have been rarely observed, but high doses can produce this effect and may result in preterm labor or abortion.	AAP considers quinidine compatible with breast feeding.
Sotalol	Sotalol, which has both beta blocker and type III antiarrhythmic properties, is not teratogenic, and its use has not been associated with fetal growth restriction. Its use near birth has been associated with newborn bradycardia. It may prolong the QT interval on the ECG and potentially induce *Torsades de Pointe*.	Sotalol is concentrated in breast milk, with milk levels several-fold higher than those in maternal plasma, so close monitoring for bradycardia, hypotension, respiratory distress, and hypoglycemia is advised.
Verapamil	No evidence of increased risk of teratogenesis .	AAP considers verapamil compatible with breastfeeding.

Abbreviation: AAP, American Academy of Pediatrics.

goal of treatment is to terminate complex arrhythmias, prevent recurrence, and control ventricular rate. Careful consideration should be given to the choice of antiarrhythmic based on individual patient characteristics, correct identification of the arrhythmia, and properties of the medication.[3] Adenosine appears to be safe for acute termination of narrow QRS complex tachycardia in pregnancy and probably is the best initial acute treatment, especially if nonpharmacologic maneuvers have failed. Direct current cardioversion is acceptable in all stages of pregnancy.

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REFERENCES

1. Shotan A, Ostrzega E, Mehra A, Johnson JV, Elakam U. Incidence of arrhythmias in normal pregnancy and relation to palpitations, dizziness, and syncope. *Am J Cardiol.* 1997;79(8):1061-1064.

2. Perez-Silva A, Merino JL. Tachyarrhythmias and pregnancy. European Society of Cardiology. E-Journal of Cardiology., E-pub Vol. 9; 31. May 20, 2011. http://www.escardio.org/communities/councils/ccp/e-journal/volume9/Pages/Tachyarrhythmias-Pregnancy-Perez-Silva-A-Merino-JL.aspx. Accessed July 19, 2012.

3. Gleicher N, Meller J, Sandler RZ, Sullum S. Wolff-Parkinson-White syndrome in pregnancy. *Obstet & Gynecol.* 1981; 58(6):748-752.

4. Ghosh N, Luk A, Derzko C, Dorian P, Chow CM. The acute treatment of maternal supraventricular tachycardias during pregnancy: a review of the literature. *J Obstet Gynaecol Can.* 2011; 33(1):17-23.

5. Kounis NG, Zavras GM, Papadaki PJ, Soufras GD, Kitrou MP, Poulos EA. Pregnancy-induced increase of supraventricular arrhythmias in Wolff-Parkinson-White syndrome. *Clin Cardiol.* 1995;18(3):137-140.

6. Cox JL, Gardner MJ. Treatment of cardiac arrhythmias during pregnancy. *Prog Cardiovasc Dis.* 1993;36(2):137-178.

7. Blomström-Lundqvist C, Scheinman MM, Aliot EM et al. ACC/AHA/ESC Guidelines for the management of patients with supraventricular arrhythmias—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines and the European Society of Cardiology Committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Hearth Rhythm Society. *J Am Coll Cardiol.* 2003;42(8):1493-1531.

8. Belardinelli L, Linden J, Berne RM. The cardiac effects of adenosine. *Prog Cardiovasc Dis.* 1989;32(1):73-97.

9. Rotmensch HH, Elkayan U, Frishman W. Antiarrhythmic drug therapy during pregnancy. *Ann Intern Med.* 1983;98(4):487-497.

10. Leffler S, Johnson DR. Adenosine use in pregnancy: lack of effect on fetal heart rate. *Am J Emerg Med.* 1992;10(6):548-549.

11. Harrison JK, Greenfield RA, Wharton JM. Acute termination of supraventricular tachycardia by adenosine during pregnancy. *Am Heart J.* 1992;123(5):1386-1388.

12. McGovern B, Garan H, Ruskin JN. Precipitation of cardiac arrest by verapamil in patients with Wolff-Parkinson-White syndrome. *Ann Intern Med.* 1986;104(6):791-794.

13. Josephson ME, Caracta AR, Ricciutti, MA, Lau SH, Damato AN. Electrophysiologic properties of procainamide in man. *Am J Cardiol.* 1974;33(5):596-603.

14. Barnes EJ, Eben F, Patterson D. Direct current cardioversion during pregnancy should be performed with facilities available for fetal monitoring and emergency caesarean section. *BJOG.* 2002;109(12):1406-1407.

15. Schroeder JS, Harrison, DC. Repeated cardioversion during pregnancy. Treatment of refractory paroxysmal tachycardia during 3 successive pregnancies. *Am J Cardiol.* 1971;27(4):445-446.

16. Kunze KP, Schluter M, Kuck KH. Sotalol in patients with Wolff-Parkinson-White syndrome. *Circulation.* 1987;75(5):1050-1057.

17. Kappenberger LJ, Fromer MA, Steinbrunn W, Shenassa M. Efficacy of amiodarone in the Wolff-Parkinson-White syndrome with rapid ventricular response via accessory pathway during atrial fibrillation. *Am J Cardiol.* 1984;54(3):330-335.

18. Ludmer PL, McGowan NE, Antman EM, Friedman PL. Efficacy of propafenone in Wolff-Parkinson-White syndrome: electrophysiologic findings and long-term follow-up. *J Am Coll Cardiol.* 1987;9(6):1357-1363.

19. Wellens HJ, Lie KL, Bär FW et al. Effect of amiodarone in the Wolff-Parkinson-White syndrome. *Am J Cardiol.* 1976;38(2):189-194.

20. Feld GK, Nademanee K, Stevenson W, Weiss J, Klitzner T, Singh BN. Clinical and electrophysiologic effects of amiodarone in patients with atrial fibrillation complicating the Wolff-Parkinson-White syndrome. *Am Heart J.* 1988;115(1 Pt 1):102-107.

21. Page RL. Treatment of arrhythmias during pregnancy. *Am Heart J.* 1995; 130(4):871-876.

22. Mitani, GM, Steinberg I, Lien EJ, et all. The pharmacokinetics of anti-arrhythmic agents in pregnancy. *Clin Pharmacokinet.* 1987; 12(4):253-291.

23. Szumowski L, Szufladowicz E, Orczykowski M, et al. Ablation of severe drug-resistant tachyarrhythmia during pregnancy. *J Cardiovasc Electrophysiol.* 2010; 21(8):877-882.

24. Briggs GC, Freeman RK, Yoffe SJ. *Drugs in pregnancy and lactation.* 8th Ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008.

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