The Acute Treatment of Maternal Supraventricular Tachycardias During Pregnancy: A Review of the Literature

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Abstract

Objective: Since evidence-based guidelines for the treatment of acute supraventricular tachyarrhythmia (SVT) in pregnancy are not available, our objective was to document published reports and immediate outcomes in this patient population.

Data sources: A search of the literature was performed using Medline, Embase, CINAHL, American College of Physicians Journal Club, Database of Abstracts of Reviews of Effects, and Cochrance Central Register of Controlled Trials, using key word searching and citations in the English language literature from January 1950 to March 2010, on the subject of SVT.

Study selection/data extraction: We reviewed 38 studies (case-controlled cohort studies, case series, and case reports) using the key words "supraventricular tachycardia," "paroxysmal tachycardia," and "atrial tachycardia," combined with "pregnancy" or "pregnancy complications."

Conclusion: No randomized controlled trials have addressed the acute treatment of SVT in pregnancy. If non-invasive manoeuvres fail, adenosine should be the first-line agent for treatment if needed during the second and third trimester. There is a paucity of data on management of SVT in the first trimester.

Résumé

Objectif : Puisque nous ne disposons pas de lignes directrices factuelles sur la prise en charge de la tachyarythmie supraventriculaire aiguë (TSV) pendant la grossesse, notre objectif était de documenter les signalements publiés et les issues immédiates au sein de cette population de patientes.

Sources de données : Une analyse documentaire a été menée dans Medline, Embase, CINAHL, le *American College of Physicians Journal Club*, la *Database of Abstracts of Reviews of Effects* et le *Cochrance Central Register of Controlled Trials*, au moyen de mots clés et de citations dans la littérature de langue anglaise, publiée entre janvier 1950 et mars 2010, portant sur la tachycardie supraventriculaire pendant la grossesse.

Sélection d'étude / extraction de données : Nous avons analysé 38 études (études de cohorte cas-témoins, séries de cas et exposés de cas) au moyen des mots clés « *supraventricular tachycardia* », « *paroxysmal tachycardia* » et « *atrial tachycardia* », conjointement avec les termes « *pregnancy* » ou « *pregnancy complications* ».

Conclusion : Aucun essai comparatif randomisé ne s'est penché sur la prise en charge de la TSV aiguë pendant la grossesse. Lorsque les manœuvres non effractives échouent, l'adénosine devrait constituer l'agent de première intention pour le traitement, lorsqu'il s'avère requis, pendant le deuxième et le troisième trimestre. Les données sur la prise en charge de la TSV au cours du premier trimestre sont rares.

Key Words: Tachycardia, supraventricular, pregnancy, pregnancy complications

Competing Interests: None declared

Received on April 17, 2010

Accepted on September 15, 2010

J Obstet Gynaecol Can 2011;33(1):17–23

INTRODUCTION

Paroxysmal supraventricular tachycardia refers to intermittent pathologic tachycardia excluding the subtypes of atrial fibrillation and flutter, as well as multifocal atrial tachycardia. Its reported incidence is 35 per 100 000 person-years in the general population.[1] The main mechanism for the development of SVT is via re-entry, most commonly atrioventricular nodal re-entrant tachycardia (in 60% of cases) and atrioventricular re-entrant tachycardia (in 30%). Other subtypes include atrial tachycardia, sino-atrial nodal re-entrant tachycardia, and junctional ectopic tachycardia.[2]

In women with a history of SVT, episodes of SVT occur with increased frequency during pregnancy, especially in those with underlying congenital or structural heart disease.[3,4] Proposed mechanisms include increased

circulating levels of catecholamines during pregnancy, increased adrenergic receptor sensitivity, and increased maternal effective circulating volume causing atrial stretch.[5] Although manoeuvres to control SVT such as carotid sinus massage and Valsalva manoeuvre are well tolerated during pregnancy,[6] aggressive management strategies such as electrical cardioversion must be applied in cases of maternal instability.[7] We conducted a review of the literature to evaluate and summarize the variety of treatment options available for the acute treatment of SVT during pregnancy. It should be noted that the usual decisions regarding management and criteria for the urgent treatment of SVT may apply differently in the pregnant woman, because of both the physiologic changes in pregnancy and the risk of placental and fetal compromise, even when the mother appears stable.

METHODS

We conducted a search of Medline from January 1950 to March 2010 (week 2), of Embase between 1980 and 2010 (week 11), CINAHL from 1982 to March 21, 2010, and American College of Physicians Journal Club, Database of Abstracts of Reviews of Effects, and Cochrane Central Register of Controlled Trials databases, using the search terms "tachycardia, supraventricular," "tachycardia, paroxysmal," and "tachycardia, atrial" in combination with the terms "pregnancy" and "pregnancy complications." Additional studies were found by using reference lists from identified studies.

Thirty-eight English language studies were selected, consisting of case reports, case series, and cohort studies. Each selected article described the acute management of SVT in pregnant women (within 72 hours of onset of symptoms), as well as the immediate maternal and fetal outcomes following treatment. We excluded studies that included discussion of the treatment of fetal arrhythmias and studies that excluded immediate outcomes. Data including the number of patients, gestational age of patients, interventions selected, and maternal and fetal outcomes were summarized. The treatment in each case was determined to be successful or unsuccessful, with the former defined as restoration of normal sinus rhythm with no adverse outcomes. The latter was defined as an inability to convert the arrhythmia or the occurrence of an adverse event. Rate control was defined as a heart rate of < 100 beats per minute, and adverse outcomes included maternal bradycardia, hypotension, dyspnea, uterine contractions, or death. Fetal adverse outcomes were bradycardia and death.

ABBREVIATIONS

ACC/AHA/ESC	American College of Cardiology, American Heart Association Task Force, and European Society of Cardiology
FDA	United States Food and Drug Administration
NSR	normal sinus rhythm
SVT	supraventricular tachycardia

RESULTS

The outcomes of all reported arrhythmic events (n = 138), including restoration of NSR or rate control and adverse outcomes, were organized by intervention and are shown in Table 1. The recommendations by the American College of Cardiology, American Heart Association Task Force, and the European Society of Cardiology have been summarized in Tables 2 to 4.

DISCUSSION

The results indicate that SVT during pregnancy presents a challenging clinical problem, in which multiple agents can be used for successful cardioversion with few adverse events. No randomized controlled trials have been published to date; the recommendations published by the ACC/AHA/ESC are solely based on expert consensus.[46] Thus, a review of the published literature is integral to evaluation of the recommendations of the task force.

Our review of the literature, from 1950 to March 2010, has shown that adenosine is the agent most commonly used in management, with conversion to NSR in 84% of cases of acute SVT after non-pharmacological manoeuvres have failed. Although adverse outcomes have been reported in both mother and fetus (8% and 6% respectively, see Table 1), only one case of SVT led to both maternal and fetal demise. In the latter case a larger dose (36 mg IV) was used than in the other studies,[19] and the outcome may be explained by adenosine's pharmacokinetic and pharmacodynamic properties. During pregnancy, the concentration of adenosine deaminase (the enzyme responsible for its degradation into inactive metabolites) decreases due to the expansion of the maternal effective circulating volume. This allows successful cardioversion at standard doses (6 to 12 mg IV).[19] Adenosine's safety has also been well described in terms of fetal effects. Animal studies have documented its efficacy and safety, because its short half-life (10 seconds) renders it unlikely to reach the fetal circulation.[21] The results found in our study are consistent with the ACC/AHA/ESC recommendations. In the second and third trimesters, the recommendation from

Table 1. Summary of interventions used to treat maternal supraventricular tachycardia in pregnancy

Intervention	Cases (n)	Trimester	Doses	Restoration NSR /rate control, n (%)	Maternal adverse outcomes, n (%)	Fetal adverse outcome, n (%)	Successful, without adverse outcome, n (%)
Adenosine[8-26]	58 (63 arrhythmic episodes)	First (3); Second (12); Third (32); Unknown (11)	6–36 mg IV	53 (84)	5 (8)	4 (6)	48 (63)
Amiodarone[23,27-28]	3	Second (2); Third (1)	800 mg IV	2 (67)	1 (33)	0 (0)	2 (67)
Beta blockers[4,10, 23, 26-27, 29-33]	13	First (1); Second (5); Third (5); Unknown (2)	–	5 (38)	4 (30)	1 (7)	5 (38)
Digoxin[10,16,26-27,29,34,35]	7	First (1); Second (4); Third (2)	0.375–0.5 mg PO, 0.25-1 IV	2 (29)	1 (14)	0 (0)	2 (29)
Diltiazem[13, 23]	2	Second (2)	–	1 (50)	1 (50)	0 (0)	1 (50)
Disopyramide[29,31-32,36,37]	5	Second (1); Third (4)	300 mg PO; 150 mg IV	1 (20)	4 (80)	0 (0)	1 (20)
Electrical cardioversion[4,10,23,26-27,29-31,35,37-42]	18	First (1); Second (5); Third (8); Unknown (4)	50–400 joules (mono and biphasic)	12 (67)	1 (6)	1 (6)	11 (61)
Flecainide[26-27,37]	3	Second (1); Third (2)	100 mg PO	2 (67)	0 (0)	0 (0)	2 (67)
Procainamide[4,26,30,41]	4	First (1); Third (2), Unknown (1)	–	2 (50)	0 (0)	0 (0)	2 (50)
Quinidine[32,33,35]	4	First (1); Third (3)	–	1 (25)	0 (0)	0 (0)	1 (25)
Verapamil[10,16-17,21,23,26-27,33,37,40,43-45]	16	Second (5); Third (9); Unknown (2)	2–10 mg IV	7 (44)	2 (13)	0 (0)	7 (44)
Total	133 (138 events)			89 (64)	19 (14)	6 (4)	82 (59)

the ACC/AHA/ESC is to use IV adenosine as first-line treatment if vagal manoeuvres are not effective.[46] Standard doses (6 to 12 mg IV) should be used in the acute setting.

The use of beta blockers or calcium channel blockers has been reported in fewer cases than adenosine, with a lower conversion rate (38% for beta blockers, 44% for diltiazem, and 50% for verapamil). Hypotension is the most commonly reported adverse outcome (7 cases in our study), with no resultant episodes of fetal hypoperfusion.

We noted the use of a variety of beta blockers in our review; in general, beta blockers are deemed safe for use in pregnancy, but particular agents are preferred. Some data suggest beta-1 selective agents are preferred to non-selective agents because beta-2 agents cause uterine relaxation and peripheral vasodilatation, although other reports have not found a significant difference.[47,48] Acebutolol and pindolol are classified by the FDA as category B drugs (see Table 2), and are considered first-line beta blockers for use in pregnancy. Metoprolol and propranolol (FDA category C) have both been

found to be safe and effective for use in pregnancy as well.[48] Chronic use of atenolol should be avoided, as long-term use has been associated with intrauterine growth restriction.[48] The ACC/AHA/ESC guidelines recommend the use of IV propranolol or metoprolol if adenosine fails. As acebutalol and pindolol were not used in the cases we reviewed, it is difficult to compare their efficacy with metoprolol and propranolol. Of interest, although sotalol, a non-selective beta blocker and a class III anti-arrhythmic, is considered safe for use in pregnancy, its efficacy for conversion of SVT has not been well established. In our reviewed cases, only one patient received sotalol as treatment, and conversion was unsuccessful. We recommend the use of propranolol, metoprolol, acebutalol, or pindolol in the acute setting of SVT if both vagal manoeuvres and administration of adenosine fail. Calcium channel blockers, including diltiazem and verapamil, may be used despite their association with maternal hypotension (13% and 50% of cases, respectively). As both are classified as FDA category C drugs,[48] the ACC/AHA/ESC guidelines recommend the use of verapamil if beta blocker therapy does not convert the arrhythmia.[46] Given that more cases of verapamil use have been reported than diltiazem use (16 vs. 2), with fewer cases of hypotension with use of verapamil, our findings support their recommendations.

If medical intervention fails in a hemodynamically stable patient or in an unstable patient, direct current cardioversion can be used in all three trimesters.[46,47] Our review found that cardioversion was successful in restoring NSR in 61% of patients, with one reported case of hypertonic uterine contractions, fetal distress, and fetal bradycardia as a direct result of the procedure. Unfortunately, the energy delivered was not documented.[10] Close fetal monitoring is imperative during cardioversion and a low energy level (50 J) should be administered initially.

The use of other agents including amiodarone, digoxin, flecainide, and procainamide has been reported in very few cases (Table 1). As amiodarone use in the first trimester has been associated with intrauterine growth restriction, and its chronic use during pregnancy as prophylactic antiarrhythmic therapy can cause congenital goitre, hypothyroidism or hyperthyroidism, and fetal QT-prolongation,[49] its long-term use should be avoided. The use of amiodarone in acute cases of SVT is limited to only three reported cases. Amiodarone, digoxin, flecainide, and procainamide should therefore not be used for acute conversion of SVT as experience with them in this setting is lacking, and other agents are readily available.

Disopyramide (a class Ia antiarrhythmic) has been found to promote uterine contractions.[50] This observation was seen in our review, with one reported case of severe postpartum hemorrhage that was thought to be due to the use of disopyramide in the acute setting.[36] Although not observed in this review, quinidine can also induce uterine contractions.[47] Given these potential adverse effects and the availability of many other safer agents, empiric use of these two drugs is not recommended.

This review provides detailed information regarding agents and approaches available to treat acute episodes of SVT in the pregnant woman. Nonetheless, it is recognized that the degree of comfort and experience among obstetricians in treating maternal SVT is likely variable. Although maternal SVT is rarely life-threatening, early involvement of a cardiologist once SVT has been documented is recommended, so that any associated conditions that may be present and that may alter the prognosis of the arrhythmia (including previously undetected structural heart disease) can be addressed. Furthermore, cardiology consultation is required for accurate diagnosis of the arrhythmia. When a pregnant woman with SVT presents de novo to her obstetrician, monitoring of the patient, including assessment of vital signs, acquisition of a 12-lead electrocardiogram, establishment of intravenous access, and frequent assessment of maternal symptoms and fetal stability are important. Adequate management may require urgent transfer to a monitored area such as the emergency department. In the majority of cases, patients will be hemodynamically stable and there will be sufficient time to request the urgent consultation of a cardiologist or other expert. Close collaboration between the cardiologist and the obstetrician is important throughout the pregnancy and puerperium to develop care strategies for potential recurrences of SVT.

In cases where the patient is hemodynamically unstable due to SVT and urgent expert consultation is not available, advanced cardiovascular life support algorithms modified to address the physiological changes of pregnancy should be implemented. These should include positioning the patient in the left lateral position, administering 100% oxygen, and establishing intravenous access. However, details regarding the management of a pregnant woman in such a scenario are beyond the scope of this review and readers should refer to appropriate detailed sources.[51]

As only seven cases of SVT in our patients (5%) were reported to occur in the first trimester, it is difficult to recommend the use of a specific agent until evidence

Table 2. United States Food and Drug Administration classification scheme for medication use in pregnancy[46]

FDA category	Implication	Evidence for drug safety
A	No evidence of risk	Adequate, well-controlled trials in pregnant women have not shown a risk to the mother or fetus in any trimester
B	Risk remote, but possible	Adequate, well-controlled trials in pregnant women have not shown increased fetal risk despite adverse findings in animals, or there are no adequate human studies, and animal studies show no fetal adverse outcomes
C	Risk cannot be ruled out	Adequate, well-controlled human studies are lacking, and animal studies are either lacking or have shown a risk to the fetus
D	Evidence of risk	Studies in humans or investigational or post-marketing surveillance have shown fetal risk
X	Contraindicated in pregnancy	Studies in animals or humans have shown clear evidence of fetal abnormalities or risk that outweighs possible benefit

Table 3. Level of evidence and recommendations by the American College of Cardiology, American Heart Association Task Force, and the European Society of Cardiology[46]

Recommendations for classifying indications (summarizing evidence and expert opinion)	
Class I	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 bases of a limited number of randomized trials, non-randomized studies, or observational registries
Level C	Primary basis for the recommendation was expert consensus

of safety during this key time of organogenesis is available. Unfortunately, the drug safety and efficacy data summarized in this review are essentially limited to second and third trimester use. Although no adverse fetal events were reported when these agents were used in the first trimester, the fetal risk from these drugs cannot be determined because of the small number of case reports available.

All of the data acquired were from case reports and cases series, as there were no randomized clinical trials from which data could be obtained. Furthermore, there was no standardized drug dosing or electric current delivery (direct current cardioversion), and thus clear recommendations with respect to drug or energy doses cannot be made.

This review focuses only on the acute management of SVT, and does not address either arrhythmia prophylaxis or the long-term adverse effects of such prophylaxis.

CONCLUSION

The acute management of SVT in pregnancy remains a difficult clinical conundrum as the available data are limited to observational studies and case reports. Treatment remains a unique challenge, as a clinical decision must be tackled with appropriate consideration of both maternal and fetal factors. Monitoring of both mother and fetus should be continued during acute treatment, and in a stable patient non-invasive manoeuvres should first be attempted. It is recommended that adenosine should be used first, followed by specific beta-blockers, and verapamil should be used in the second and third trimesters if other measures fail. In an unstable patient, direct current cardioversion should

Table 4. Summary of treatment strategies for the acute treatment of SVT during pregnancy

Treatment	Classification[46]	Level of evidence[46]	FDA category	Adverse effects	Safety during lactation
Adenosine	1	C	C	None in second and third trimester, little evidence in first trimester	Safe, as short half-life
Beta blocker					
Acebutalol	–	–	B	Hypotension, bradycardia, and tachypnea	Use with caution
Atenolol	–	–	D	Do not use, as associated with IUGR	Use with caution
Metoprolol	IIa	C	C	–	Safe
Pindolol	–	–	B	–	Safe
Propranolol	IIa	C	C	–	Safe
Sotalol	–	–	B	Bradycardia, hypotension, IUGR, prematurity	Enters breast milk
Calcium-channel blocker	–	– C	C	Teratogenic effects in animal studies	Enters breast milk
Diltiazem	IIb			Crosses the placenta, may have tocolytic effects	Enters breast milk
Verapamil					
DC cardioversion	I	C	–	–	–

IUGR: intrauterine growth restriction

be used initially, with delivery of the lowest energy level (50 J). Optimal evidence-based management of maternal SVT in pregnancy, especially in the first trimester, is limited by the paucity of reported cases.

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