CASE REPORT

Refractory Supraventricular Tachycardia during Pregnancy

B.W.P Habaragamuwa MBBS, MD*, Saroja Jayasinghe MBBS, MD, FRCA**, Lakshman Kariyawasam MBBS, MS, MRCOG***, V.D. Joseph MBBS, MD

*Senior Registrar, **Consultant Anaesthetist,
Dept of Anesthesiology & Intensive Care, De Soysa Maternity Hospital, Colombo (Sri Lanka).
***Consultant Obstetrician and Gynecologist, De Soysa Maternity Hospital, Colombo (Sri Lanka).
**** Senior Registrar, Dept of Cardiology, The National Hospital, Colombo (Sri Lanka).

Correspondence: B.W.P Habaragamuwa MBBS, MD, Dept of Anesthesiology & Intensive Care, De Soysa Maternity Hospital, Colombo (Sri Lanka). E-mail budhi190@yahoo.com/ Tel 0094776364612.

ABSTRACT

We present a case report of a 34 year old pregnant mother who presented in her 37th week of gestation with acute onset palpitation and shortness of breath. Examination and investigations revealed that she had supraventricular tachycardia (SVT). She continued to have SVT despite active treatment and termination of pregnancy and died four days after admission.

Key words: Supraventricular tachycardia, Cardioversion, Dysrrhythmia

INTRODUCTION

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

The following case report describes a pregnant patient with supraventricular tachycardia which continued into postpartum period despite active pharmacological intervention and repeated attempts at electrical cardioversion, eventually resulting in her death.

CASE REPORT

A previously healthy 34 year old, 70kg mother presented in her 37th week of gestation to the maternity hospital complaining of shortness of breath and palpitations for the last four hours. She was in her fourth pregnancy with two living issues and one miscarriage. She had mild chest pain [2] years ago for which she was investigated and found to have a thickened mitral valve and mild mitral regurgitation. On examination, she was dyspnoeic with a pulse rate of 180 beats per minute and blood pressure of 100/60mmHg; and had clear lung bases on auscultation. Patient was rushed to the intensive care unit (ICU) and maternal pulse rate, NIBP, SpO_2 and temperature as well as fetal monitoring was started. Patient was kept propped up and 60% oxygen administered via a venturi mask.

An electrocardiogram (ECG) revealed

narrow complex tachycardia and a tentative diagnosis of supraventricular tachycardia (SVT) was made. She did not respond to carotid sinus massage and was treated with intravenous verapamil. Adenosine was not available at that time. Her heart rate remained between 180-190 beats per minute despite three doses of IV verapamil 2.5mg each. Our cardiologist was also involved in the patient's management. Adenosine was started but SVT didn't revert completely despite IV use of adenosine in 6 mg, 12mg and 12mg doses. Therefore, intravenous amiodarone 150 mg bolus over 20 minutes was given via external jugular line and oral amiodarone 200mg 8 hourly and oral metoprolol 25mg 8 hourly were started. Despite all above medications she continued to have SVT at a rate of 180-190 beats per minute, but her blood pressure maintained at 100/60mmHg. Temporary pace maker (TPM) was inserted through the femoral vein to over pace the heart but with no response. Transthoracic 2D echocardiogram revealed thickened mitral valve and mild mitral regurgitation. As her condition failed to settle, emergency caesarian section was planned to deliver the baby. General anaesthesia was crash induced with thiopentone sodium 250mg and suxamethonium 120mg. Intravenous lignocaine 1mg/kg was used to blunt the intubation and laryngoscopy response. A 2.8 kg healthy baby was delivered. Intravenous oxytocin 5mg bolus slowly followed by 20mg infusion was started to augment the uterine contraction. Atropine 0.6mg and neostigmine 2.5mg were used for the reversal of muscle relaxant. Intravenous lignocaine 1mg/kg was repeated at extubation. Mother had an uneventful recovery from anaesthesia and surgery except for the heart rate of 180-190 beats per minute. She had slight improvement of her shortness of breath after delivery, though she continued to suffer from tachycardia. Intravenous morphine 1mg per hour infusion was given for pain relief and subcutaneous enoxaparin 40mg was started for thromboprophylaxis. After few hours of delivery, her dyspnoea got worsened and systolic blood pressure dropped to 80mmHg. Synchronized electrical cardioversion (100J) was done twice under sedation and it was followed by intravenous amiodarone 300mg bolus and 600mg over next 24hours. Despite the treatment, she again became very dyspnoeic and hypotensive. Invasive ventilation was started and infusions of dobutamine, adrenaline and noradrenaline were started to support the cardiovascular system. Emergency transthoracic 2D echocardiogram showed an ejection fraction of 30-35% and a pericardial effusion. As her blood pressure was unstable despite inotropic support, intra-aortic balloon pump was inserted. Emergency pericardiocentesis was done and nearly 300ml of blood stained fluid removed to relieve the diastolic dysfunction of the heart due to pericardial effusion. Emergency sternotomy was decided to evacuate the pericardial effusion and to exclude right ventricular perforation by the tip of the TPM as she continued to have haemodynamic instability. Bilateral pleural effusion was noted other than small pericardial effusion during sternotomy. She was monitored with ECG, invasive blood pressure, central venous pressure and temperature. She deteriorated further and died about 36 hours after sternotomy.

Post mortem examination revealed only the thickened mitral valve.

DISCUSSION

The incidence and severity of supraventricular arrhythmias may increase during pregnancy.[1] Although the reasons for this observation are unclear, some explanations have been proposed: increased awareness, haemodynamic, hormonal, autonomic and emotional changes related to pregnancy, which may include an increase in plasma catecholamine concentrations and adrenergic receptor sensitivity, atrial stretch and increased end-diastolic volumes due to intravascular volume expansion.[2] A cardiac lesion also may predispose to SVT as with this patient with thickened mitral valve and trivial mitral regurgitation.

Though there was no maternal death directly attributable to SVT according to the Confidential Enquiry into Maternal and Child Health (CEMACH) 2003-2005 report, SVT may have contributed to some of the maternal deaths from cardiac disease.[3]

Treatment of SVT in pregnancy may also affect the fetus. For this reason, pharmacological

treatment is best reserved for those with sever symptoms, haemodynamic changes or sustained arrhythmias.[2] Non pharmacological treatment including vagal manoeuvres such as carotid massage, Valsalva manoeuvre and facial ice immersion are well tolerated and aid in diagnosis. Carotid massage was tried in this patient with no effect. There were all the reasons to initiate pharmacological treatment in this patient as she was dyspnoeic and had sustained SVT despite vagal manoeuvres.

Verapamil acts by blocking atrioventricular (AV) node in SVT and is recommended in acute SVT. Although verapamil administered during the third trimester is generally safe and not teratogenic, caution must be exercised, because some reports showed that verapamil used for fetal SVT may result in fetal AV block and bradycardia, reduced contractility of the uterus and hypotension.[2,4] Intravenous verapamil 7.5mg was used in this case without any effect.

Numerous case reports, and a retrospective study, suggest that adenosine is safe and effective.[5] Adenosine may cross the placenta only in small quantities, because of its brief plasma half life. An important advantage compared with verapamil is that adenosine doesn't affect fetal haemodynamics while verapamil may reduce fetal blood pressure.[6] Adenosine was used second to verapamil as it was not available immediately. But SVT of this case continued despite three doses of adenosine (6mg, 12mg and 12mg).

Amiodarone crosses the placenta less easily than other antiarrhythmic drugs, but it has been associated with a significant incidence of fetal hypothyroidism, hyperthyroidism, goiter and growth retardation.[7] Most investigators recommend its use only in life threatening cases where other therapies have failed and also to use it in the lowest possible doses. Amiodarone was used as a third line drug in this patient and intravenous dose was confined to 150mg bolus. As her SVT was resistant to all of the above therapies, oral amiodarone 200mg 8hourly and oral metoprolol 25mg 8 hourly were started. Beta blockers have been used extensively in pregnancy, to treat maternal hypertension and cardiac problems, and are generally well tolerated.8 Metoprolol dose had to be restricted to 25mg 8 hourly in this case due to low blood pressure of the patient.

Digoxin has been used in all stages of gestation for maternal and fetal indications without causing harm.[2] Digoxin was not used in this case.

Overdrive atrial pacing also been used successfully in SVT during pregnancy.[2] It was also tried prior to the emergency caesarean section in this case with no response.

Emergency caesarian section was decided as it was felt the safest course of action for the fetus. It was also thought that the delivery of the baby might terminate the SVT if it was related to pregnancy. Patient's haemodynamic parameters were maintained throughout anaesthesia. Intravenous lignocaine was used to blunt the untoward intubation, laryngoscopy and extubation responses. As glycopyrrolate was not available, atropine 0.6mg was used with neostigmine for reversal of muscle relaxants.

As she was unstable despite treatment after few hours of caesarean section, electrical cardioversion was attempted twice with minimal success. Though not used in this case, synchronized electrical cardioversion has been used successfully and safely during pregnancy.[9,2] The risk of inducing fetal arrhythmia is small because the electrical current reaching the fetus is insignificant. Furthermore, the mammalian fetus has a high fibrillation threshold.[2] However, because transient fetal arrhythmia has been reported, it has been suggested that the procedure preferably be performed with fetal rhythm monitoring.

Despite all above treatment strategies SVT continued in our patient and her haemodynamic instability failed to respond to full doses of inotropes, vesopressors and even the use of intra-aortic balloon pump and ultimately the patient could not be saved.

REFERENCES

1. Tawam M, Levine J, Mendelson M, Goldberger J, Dyer A, Kadish A. Effect of pregnancy on paroxysmal supraventricular tachycardia.

American Journal of Cardiology 1993; 72:838-840.

2. Tan HL, Lie KI. Treatment of tachyarrhythmias during pregnancy and lactation. European Heart Journal 2001; 22:458-464.
3. Saving Mother's Life: Report on Confidential Enquiry into Maternal and Child Health in United Kingdom 2003-2005.
4. Klein V, Repke JT. Supraventricular tachycardia in pregnancy: Cardioversion with verapamil. Obstetric and Gynecology 1984; 63:169-185
5. Elkayam U, Goodwin TM. Adenosine therapy for supraventricular tachycardia during pregnancy. American Journal of Cardiology 1995; 75:521-523.
6. Mason BA, Ricci-Goodman J, Koos BJ. Adenosine in the treatment of maternal paroxysmal supraventricular tachycardia. Obstetric and Gynecology 1992; 80:478-480.
7. Magee LA, Downar E, Sermer M, Boulton BC, Allen AC, Koren G. Pregnancy outcome after gestational exposure to amiodarone in Canada. American Journal of Obstetric and Gynecology 1995; 172:1307-1311.
8. Frishman WH, Chesner M. Beta adrenergic blockers in pregnancy. American Heart Journal 1988; 115:147-152.
9. Pahlow B, Geisler AK, Davis GH. Management of acute paroxysmal supraventricular tachycardia in pregnancy. Journal of the American Osteopathic Association 2000; 91(1):51-51.

★★★

The Code of Ethical Behavior for Patients

1. Do not expect your doctor to share your discomfort.
Involvement with the patient's suffering might cause him to lose valuable scientific objectivity.

2. Be cheerful at all times.
Your doctor leads a busy and trying life and requires all the gentleness and reassurance he can get.

3. Try to suffer from the disease for which you are being treated.
Remember that your doctor has a professional reputation to uphold.

4. Do not complain if the treatment fails to bring relief.
You must believe that your doctor has achieved a deep insight into the true nature of your illness, which transcends any mere permanent disability you may have experienced.

5. Never ask your doctor to explain what he is doing or why he is doing it.
It is presumptuous to assume that such profound matters could be explained in terms that you would understand.

6. Submit to novel experimental treatment readily.
Though the surgery may not benefit you directly, the resulting research paper will surely be of widespread interest.

7. Pay your medical bills promptly and willingly.
You should consider it a privilege to contribute, however modestly, to the well-being of physicians and other humanitarians.

8. Do not suffer from ailments that you cannot afford.
It is sheer arrogance to contract illnesses that are beyond your means.

9. Never reveal any of the shortcomings that have come to light in the course of treatment by your doctor.
The patient-doctor relationship is a privileged one, and you have a sacred duty to protect him from exposure.

10. Never die while in your doctor's presence or under his direct care.
This will only cause him needless inconvenience and embarrassment.