Case Report

SUPRAVENTRICULAR TACHYCARDIA DURING REGIONAL ANAESTHESIA (SPINAL ANAESTHESIA)

Tahmina Banu[1], Wahiuddin Mahmood[2]

SUMMARY:

Supraventricular tachycardia, though not very common may develop in any patient under spinal anaesthesia. A 50 years old lady admitted at square hospital through the emergency unit with left loin pain and fever with chill. She was already diagnosed as a case of left ureteric stone and was scheduled for Ureteorenoscopic Intracorporeal Pneumatic Lithotripsy (URS-ICPL) under spinal anaesthesia. During anaesthesia, she developed

supraventricular tachycardia of unknown origin. Ultrashort-acting (3-blocker was given slowly. As there was no improvement, intravenous propranolol was given slowly, which resulted in a conversion to sinus rhythm. This paper discussed methods of cardio version for patients with supraventricular tachycardia in such clinical settings.

Key words: *Spinal anaesthesia, SVT.*

INTRODUCTION:

Supraventricular tachycardia (SVT) is a rapid rhythm of the heart in which the origin of the electrical signal is higher, the atrial or the AV nodal. This is in contrast to the deadlier ventricular tachycardia, in which rapid rhythms that originate from the ventricle of the heart i.e. below the atria and AV node. SVT can develop suddenly and may go away without treatment. It lasts for a few minutes to 1-2 days. Heart rate varies between 140-250/min. Diagnosis of SVT is confirmed by ECG. i) Absence of P wave (hidden by QRS) ii) Narrow QRS complex (less than 0.6 sec), iii) Short regular PR interval. Abnormally short PR interval can be seen with either low atrial rhythm or pre-excitation phenomenon. Pre-excitation usually refers to early depolarization of the ventricles by an abnormal conduction pathway from the atria to ventricles. The most common form of pre-excitation is due to the presence of an accessory pathway (bundle of kent) that connects one atria with one of the ventricles. The Wolff-Parkinson-White (WPW) syndrome is often applied to ventricular pre-excitation associated with tachyarrythmia. Pre-excitation occurs approximately in 0.3% of the general population[s]. Supraventricular tachycardia is one of the commonest tachyarythmias during anaesthesia particularly during spinal anaesthesia. Thus all patients with history of SVT should be evaluated preoperatively by a cardiologist for possible electrophysiological studies, for curative radio frequency ablation of the bypass tract or the need for perioperative drug therapy. However, patient with only occasional asymptomatic tachyarythmia generally do not require investigations or prophylactic drug therapy. Those with frequent episodes of tachyarythmia associated with significant symptoms require drug therapy and close evaluations.

CASE REPORT:

Sakina Begum, a female aged fifty years was admitted to square hospital on September 16, 2008 through the emergency department with the complaints of

- High grade fever with chill
- Left loin pain
- Burning sensation during micturation for few days

The patient was a known case of left ureteric stone. She was planned for URS and ICPL on September 18, 2008. Her co-morbidity was diabetes mellitus, controlled with diet. Other parameters were normal except ECG, which showed sinus tachycadia (Heart-rate 105/min). Spinal anaesthesia was given at L3-L4 space with 0.5% bupivacaine (2.5 ml)

with fentanyl 20 μg. Continuous monitoring of oxygen saturation, blood pressure at 5 minutes

1. Associate Consultant, Department of Anaesthesia, Square Hospital Limited.
2. Consultant, Department of Anaesthesia, Square Hospital Limited.

interval and ECG tracing was going on. The procedure was uneventful throughout the operation except pulse-rate of the patient varied from 120 to 130 per minute. Blood pressure was normal, no vasopressor was needed at all.

As the stone was high up in the left ureter, it was pushed to the left kidney instead of removal. Thick pass was drained out of the left kidney on normal saline wash. A D-J stent with bichannel foley's catheter was kept in for free drainage. The whole procedure took about one hour. During the process of transfer from the OT table, the patient suddenly developed severe shivering. 25 mg pethidine was given intravenously. Oxygen saturation fell down and oxygen was started with nasal prong immediately. But oxygen saturation was not improving, then 100% oxygen was given through bain circuit. As shivering continued, 25 mg pethidine IV was repeated after 10 minutes. Her heart-rate was increasing and went up to 166/min. As the patient developed supraventricular tachycardia, carotid massage was started. As there was no improvement, patient was treated with Dormicum 2 mg I-V. Ultrashort-acting (3 blocker esmolol 30 mg I-V was given slowly. It caused little improvement, 1.0 mg propranolol was given slowly I-V. As the cause of shivering could not be diagnosed and assuming allergic reaction 100 mg hydrocortisone IV was also given. Arterial blood gas analysi~as done, base excess was -10 meq/litre. Sodi-bi-carb was given slowly. 500 mg meropenem was given I-V slowly to prevent septicaemia. After improvement of oxygen saturation and decrease of heart-rate (about 140/min), the patient was transferred to post-operative ward. No additional treatment was needed except continuation of oxygen support. The patient's condition was uneventful in the post-operative ward, and she was transferred to cabin in the next morning.

DISCUSSION:

The incidence of WPW syndrome in asymptomatic individuals has been reported to be between 0.1 and 2.5 per 1000[2]. Regional anaesthesia may result in increased arythmogenecity by causing a sudden decrease in atrial filling pressure due to sympathetic blockade[3]. Historically pharmacological interventions includes digoxin, a-agonist, example: methoxamine and phenylephrine, b antagoniss example propranolol, esmolol, calcium channel blocker example: verapamil and finally the rapid acting vasodilator adenosine[4,5,6].

Under anaesthesia where a 12 lead ECG is not available, diagnosis of exact cause of arrthmia is difficult, particularly, when there was no previous diagnosis such as in this case. Certain SVT's such as atrial flutter and other atrial tachyarythmia may be difficult to manage pharmacologically. Calcium channel blocker ma$_y$ not only be ineffective, but also may precipitate prolonged hypotension in this setting 'g'[9]. In recent years, adenosine has become the agent of choice for pharmacological conversion of SVT. It has very rapid onset of action of less than 1 minute with very short duration. In our case, as esmolol was readily available we treated the patient with esmolol first.

An a-agonist such as phenylephrine has in isolated cases been shown to be helpful for slowing the heart rate of patient with SVT under spinal and general anaesthesia 3,10,> > Esmolol may be effective as propanolol in converting the SVT and is without the vasodilatory effects of the adenosine and verapamil.

Propranolol nonselectively block (3l and (32 receptors. It slows atrioventricular conduction and stabilizes myocardial membranes. It is very effective in slowing the ventricular response to SVT [2]. So in this case when esmolol failed to convert sinus rhythm, then we switched on to another option i.e. long acting b blocker, propanolol. Slow intravenous injection of I mg propranolol converted the SVT to sinus rhythm. The patient was completely recovered from SVT with any additional drug therapy and was transferred to postoperative ward with oxygen support only.

DC cardioversion may be another choice [13,14,15] although extreme caution and airway protection would be required in the administration of additional sedation.

Anaesthetic vigilance and prompt intervention even with esmolol and propranolol saved a life, which is a lesson for all in day to day aneasthetic practice.

REFERENCES:

1. Morgan GE, Mikhail MS, Murray MJ: Clinical Anesthesiology, 4th ed. McGraw-Hill, 2006;
2. Averill KH, Fosmoe RJ, Lamb LE. Electrocardiographic findings in 67,375

asymptomatic patients, IV. Wolff-Parkinson-White syndrome. Am J Cardiol 1960; 6: 108-129.

3. Van Zijl DHS, Dyer RA, Scott Miller RN James MFM. Supraventricular tachycardia during spinal anaesthesia for caesarian section. Int J Obstet Anesth 2001; 10: 202-205.
4. Sprague DH, Mandel SD. Paroxysmal supraventricular tachycardia during anesthesia. Anesthesiology 1977; 46: 75-77.
5. Mason BA, Ricci-Goodman J, Koos BJ. Adenosine in the treatment of maternal paroxysmal supraventricular tachycardia. Obstet Gynecol 1992; 80: 478-480.
6. Afridi I, Moise KJ, Rokey R. Termination of supraventricular tachycardia with adenosine in a pregnant women with Wolff-Parkinson-White syndrome. Obstet Gynecol 1992; 80: 481-483.
7. Chow T, Galvin J, McGovern C. Antiarrythmic drug therapy in pregnancy and lactation. Am J Cardiol 1998; 82: 581-621.
8. Ferguson JE, Siukola LV, Albright GA. Use of verapamil for Paroxysmal supraventricular tachycardia during epidural anesthesia for caesarian section. Am J Perinatol 1988; 5: 128-130.
9. Treakle K, Kostic B, Hulkower S. Supraventricular tachycardia resistant to treatment in a pregnant woman. The J Fam Pract 1992; 35: 581-584.
10. Lawrenson JB, Okreglicki AM, Scott Miller RN. Cardiovascular collapse due to intravenous verapamil in two patients with persistent atrial tachycardia. S Afr Med J 1995; 85: 1236-1238.
11. Gajraj NM, Wallac DH, Pace NA. Supraventricular tachycardia in a parturient under spinal anesthesia. Reg Anesth 1993; 18: 261-263.
12. Jacobson I, Tumquist K, Masley S. Wolff-Parkinson-White syndrome: Termination of supraventricular tachycardia with phenylephrine. Anaesthesia 1985; 40: 657-660.
13. Stickles BJ. Idiosyncratic supraventricular tachycardia after epidural anesthesia. J Nurse Midwifery 1993; 38: 42-44.
14. Klepper I. Cardioversion in late pregnancy. The anesthetic management of a case of Wolff Parkinson-White syndrome. Anesthesia 1981: 36. 611- 616.
15. Joglar J A, Page R L. Treatmemt of cardiac arrythmias during pregnancy: safty considerations. Drug Saf 1999; 20: 85-94.