Int. Med J Vol. 5 No 1 June 2006

Streptokinase after Spinal Anaesthesia – A Case Report

M. R. A. Hadi, M. D., M.N.Basri, M.D., O Ariff, M.D.,
Departments of Anesthesiology and Critical Care, International Islamic University Malaysia and Tengku Ampuan Afzan Hospital, Kuantan, Pahang, Malaysia.

Key Words: Streptokinase; spinal anaesthesia, spinal epidural haematoma.

Introduction:

The use of thrombolytic agent e.g. streptokinase is indicated in patients with early acute ST elevation myocardial infarction (MI) (if there are no contraindications) is becoming increasingly routine [1]. Its use is however significantly limited by bleeding complications. Spinal epidural haematoma (SEH) is haemorrhage in the spinal epidural space after spinal anaesthesia. SEH may be acute or chronic, spontaneous, posttraumatic, or iatrogenic but its occurrence appears to be particularly associated with acquired coagulopathy from medications and disease states. Patients usually present with acute axial spine pain and evolving focal neurological deficits [2]. With increasing number of available anticoagulants and patient receiving them, anaesthesiologists today have to face the challenge of balancing between risks and benefits of regional anesthesia in patients under such medications. The treatment of this condition involves the principles of conservative follow-up directed by an improving examination and an understanding of the pathophysiology of coagulopathy-induced spontaneous epidural bleeds. When the diagnosis is accomplished rapidly, surgical decompression can result in full functional recovery [1].

CASE REPORT

A 73-year-old, 60-kg man with indirect right inguinal hernia was admitted for an elective laparoscopic herniotomy under spinal anaesthesia. He was otherwise healthy with preoperative laboratory data, chest X-ray and electrocardiogram (ECG) were within normal limits. The premedication consisted of tablet midazolam 7.5 mg at night and 7.5 mg orally 2 hours before induction of anesthesia. Intraoperative monitoring included an ECG (lead II), pulse oximetry and urine output measurement. Spinal anesthesia was induced with patient in sitting position at the level L3/L4 intervertebrae space using *Spinocan* needle size 25G. A total of 2.6cc of 0.5% marcaine and 25 mg fentanyl were administered into subarachnoid space, following which he was positioned supine to facilitate surgical exposure. The anesthetic and surgical courses were uneventful for the initial 30 minutes until the patient complained of sharp stabbing pain at the surgical site. It

was followed by a sudden rise in blood pressure (from 120 mm Hg to 190 mm Hg systolic and 120 mm Hg to 110 mm Hg diastolic) and heart rate (from 80 bpm to 140 bpm), but the oxygen saturation was maintained at 98% to 99%. As a result, the spinal anaesthesia was converted to a general anesthesia technique in order to proceed with the operation. The operation was uneventful and completed after about 2 hours. At the end of the operation, all the anesthetics gases were discontinued and lungs were ventilated with 100% oxygen. The reversal process was uneventful but the patient became desaturated (SpO2 from 99% to 85%) and EtCO2 increased (from 38 mmHg to 67mmHg) after 10 minutes. There was neither history of sudden onset of chest pain nor abnormal ECG to account for these changes. The patient was then reintubated and sent to intensive care unit (ICU) for assisted ventilation and close monitoring. After five hours of assisted ventilation, he was weaned off from ventilator and extubated following achievement of extubation criteria. Approximately an hour later, he suddenly became tachycardia and hypotensive with ST elevation on 12 leads ECG. He however did not complain of any chest pain. Anterior myocardial infarction was diagnosed, cardiac enzymes were sent and he was started on thrombolytic therapy with streptokinase. However, he subsequently developed hypotension and thus dobutamine support was instituted. Since the patient had already been given subarachnoid (spinal) block, a full dose of streptokinase can be dangerous to the patient which might cause spinal haematoma, secondary to bleeding tendencies. Therefore, in view of 6 hours post spinal anaesthesia, a half dose of streptokinase 750,000 units was given to this patient after appropriate discussion between physicians and anaesthetists. He was also started on aspirin 300 mg stat and 150 mg orally once daily. 2 hours post streptokinase therapy, the ST changes resolved but minimal bleeding was noted from the CVP site. There was no back pain or lower extremity neurological deficits to suggest spinal cord compression secondary to spinal epidural haematoma. He also developed a few episodes of atrial fibrillation and supraventricular tachycardia but resolved with digoxin and amiodarone. There was no further streptokinase given. He was discharged five days later from ICU though requiring dobutamine support until 13-post operative day in ward.

DISCUSSION

Treatment with reperfusion therapies are associated with increased short- and medium term survival after infarction[4]. Nevertheless, the greatest risk of thrombolytic therapy is hemorrhage. Streptokinase (plasminogen activators) 1.5 million U infused in 30 to 60 minutes, converts single chain plasminogen to double chain plasminogen, which has fibrinolytic activity. Thrombolytic therapy is most effective in the first few minutes and hours after the onset of MI. During the acute phase of Q-wave MI, thrombolytic

drugs reduce hospital mortality between 30 and 50%. It does not require concomitant heparin therapy. Greatest benefit occurs within 3 hours, but effectiveness up to 12 hours has been demonstrated [6].

The risk of spinal epidural hematoma is low if single puncture was done provided no vessel was injured during the procedure but it is considerable if multiple puncture is performed and large needle is used. Traumatic insertion of the spinal needle is an independent risk factor for epidural hematoma formation [8].The risk of spinal epidural hematoma after spinal anaesthesia without concomitant antithrombotic medication is 1:200 000 [5,9]. However, with the use of anticoagulant or thrombolytic therapy, the frequency of SEH formation is increased [8]. The risk is also higher if a patient has low platelet (£ 50,000) or history of bleeding tendency. SEH, eventhough rare, is a potentially devastating complication [3]. The common presentation always involves severe axial spine (neck or back) pain with or without neurological deficit [2]. Others symptoms include muscle weakness, back pain, sensory deficit and urinary retention [3]. Pain can be aggravated by straining or Valsalva maneuver (eg. coughing). Progressive neurological deficit if occur can take hours or days to resolve.

In this case, an acute MI was confirmed by serial ECG recording. As the patient had spinal anaesthesia, half dose of streptokinase was given after extensive discussion between anaesthetists and physicians to avoid possibility of a spinal epidural haematoma formation and at the same time to reverse the thrombus formation in the coronary artery. This approach was successful where the patient showed improvement in the ST segment without any evidence of spinal epidural haematoma.

Hence, in managing such a case, a high index of suspicious among physicians and anaesthetists in-charged regarding this rare hemorrhagic complication following thrombolytic therapy is the utmost important, particularly in elderly patients [7]. A definitive diagnosis of spinal epidural haematoma can be made based on imaging studies such as magnetic resonance imaging (MRI) which may provide information about the precise location and severity of spinal cord compression. In case if SEH occur, urgent surgical decompression is generally warranted to preserve neurological function [1]. The outcome of surgery appears to depend on the time between the onset of neurological deficits, the extent of haematoma, the severity of neurological deficit and surgery[3]. The operative approach to the haematoma is based on the location of SEH and its extent[2]. In cases where the deficit is minimal or resolving especially after 12 hours, a conservative follow up approach with magnetic resonance imaging may be warranted.

CONCLUSIONS:

Spinal epidural hemorrhage is a rare complication of thrombolytic therapy. It is advisable to use lower gauge (e.g. 27G) spinal needle in our practice of spinal anaesthesia in order to avoid this complication. In a situation where thrombolytic therapy is really indicated after regional anaesthesia, administration of half the usual dose of streptokinase may be warranted, however it must be accompanied by close observation of any neurological deficit which may result from a spinal epidural haematoma formation.

REFERENCE

1. Connolly, E. Sander Jr MD; (1996) Winfree, Christopher J. MD; McCormick, Paul C. MD. Management of Spinal Epidural Hematoma After Tissue Plasminogen Activator: *A Case Report. Spine.* July; 21(14):1694-1698.
2. Binder, Devin K.; Sonne, D. Christian. (2004) Spinal Epidural Hematoma. *Neurosurgery Quarterly.* March; 14(1):51-59.
3. David A. Rosen, Denzil W. Hawkinberry, Kathleen R. Rosen, Robert A. Gustafson, Jeffery P. Hogg, Lynn M. Broadman,. (2004). An epidural haematoma in an adolescent patient after cardiac surgery. *Anesth Analg*.; 98: 966-9.
4. French JK, Hyde TA, Patel H, Amos DJ, McLaughlin SC, Webber BJ, (1999) White HD. Survival 12 years after randomization to streptokinase: the influence of thrombolysis in myocardial infarction flow at three to four weeks. *J Am Coll Cardiol.* Jul; 34(1): 62-9.
5. Jens W. Krombach, Oguzhan Dagtekin, Sandra Kampe. (2004)Regional anesthesia and anticoagulation. *Current Opinion in Anaesthesiology.*; 17: 427-433
6. Nicolas Danchin, Edoardo De Benedetti and Philip Urban. (1992)Acute Myocardial Infarction. *Lancet*.; Mar 28; 339(8796):753-70.
7. Ozgocmen S, Yoldas T, Kocakoc E, Ozkurt-Zengin F, Ardicoglu O. (1988) Spinal epidural haematoma associated with streptokinase treatment for myocardial infarction. *Can South Med J.* Sep; 81(9):1202-3.
8. Scott A. Lang, Calgary Alberta, Thomas Grau, Heidelberg. (2003) Spinal epidural haematoma and epidural analgesia. *Canadian Journal of Anesthesia*; 50(4):420-426.
9. Tyagi, A. and Battacharya, A. (2003) Central neuraxial blocks and anticoagulation: a review of current trends. *European Journal of Anaesthesiology*.; 20(3): 254.