Journal of Cardiovascular Disease Research 4 (2013) 236–238

Contents lists available at ScienceDirect

Journal of Cardiovascular Disease Research

journal homepage: www.elsevier.com/locate/jcdr

Short communication

Spinal epidural hematoma – A rare and debilitating complication of thrombolytic therapy

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ARTICLE INFO

Article history:
Received 25 August 2013
Accepted 10 January 2014
Available online 19 February 2014

Keywords:
LV aneurysm
Spinal epidural haematoma
Thrombolysis
STEMI
Morbidity

ABSTRACT

Thrombolytic therapy directed to the achievement of early reperfusion in cases with acute ST elevation myocardial infarction can have significant complications which can be due to bleeding or in the form of allergic reactions. Sometimes these complications can cause mortality or significant and incapacitating morbidity which may at times surpass the risk possessed by the disease itself.

We are reporting an interesting case of 63-year-old male, who presented to us with acute anterior wall myocardial infarction and developed acute onset paralysis following intravenous administration of streptokinase and heparin. MRI spine revealed spinal epidural hematoma. Patient was advised urgent surgical evacuation of hematoma, but opted for conservative management. Patient had significant residual neurological deficits at follow-up. In conclusion, spinal epidural hematoma is a rare complication following thrombolysis for acute ST elevation myocardial infarction. Though rare, high index of suspicion is required by physicians, as prompt treatment may lead to complete recovery, which otherwise can lead to debilitating neurological sequel.

1. Introduction

Acute coronary syndrome (ACS) is one of the most common causes of morbidity and mortality in the middle aged population and amongst the diverse array of presentations, the maximum mortality and morbidity is seen in cases with ST elevation MI. Prompt reperfusion therapy causing restoration of blood supply to the jeopardized myocardium is the cornerstone of the therapy and can significantly reduce mortality. In the age of primary percutaneous intervention which forms the best and time tested modality for early and successful reperfusion, the role of thrombolytic therapy to restore perfusion is on the decline. Still in majority of the parts of the world, thrombolytic therapy is the best and often the only form of therapy which is easily accessible to most patients. Thrombolytic therapy has its known side effects, the most important of which are related to bleeding. Sometimes these complications may even be fatal. About 1–2% of patients treated with thrombolytic therapy develop intracranial bleeding which is often fatal. Rarely a patient may develop a spinal bleed which can be debilitating and can incapacitate a patient for his entire lifetime.

2. Case report

A 63-year-old male presented to the emergency department with retrosternal chest pain associated with breathlessness and profuse sweating. An acute anterior wall ST elevation myocardial infarction was diagnosed. Risk factors for coronary artery disease were type-2 diabetes, hypertension, and smoking. The patient had presented within 3 h of the onset of pain and there were no contraindications to thrombolytic therapy. The blood pressure at admission was 116/84 mm Hg. The patient was also not affording for a percutaneous intervention and so the patient underwent thrombolytic therapy with intravenous streptokinase injection, 1.5 million international units, given over a period of 1 h. Subcutaneous low molecular weight heparin (enoxaparin) was administered 6 h after thrombolysis at a dose of 1 mg/kg body weight given twice daily. Approximately, 24 h following thrombolysis patient complained of acute onset weakness of both lower limbs and inability to pass urine. There was no history of backache, headache, or vomiting.

On examination, pulse was 80 per minute and all peripheral pulses were well felt. Blood pressure was 120/80 mm of Hg. Per abdomen examination, revealed distension of the urinary bladder. Neurological examination, revealed paraplegia, absent deep tendon reflexes in both lower limbs and graded sensory loss below D10 sensory level. A clinical diagnosis of acute paraplegia at D10 spinal level with bladder and bowel involvement was made.

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0975-3583/$ – see front matter
http://dx.doi.org/10.1016/j.jcdr.2014.01.005

Differential diagnosis of anterior spinal artery embolism, spinal epidural hematoma or a saddle embolism of distal aorta was considered. Since peripheral pulses were well felt, saddle embolism of aorta was excluded. Anterior spinal artery infarction due to an embolus was less likely as there is usually sparing of proprioception in anterior spinal artery syndrome, while our patient had loss of all sensations in a graded manner below D10.

On investigating the patient, all routine bleeding and coagulation parameters after the occurrence of the event including PT/INR and aPTT were within normal range. Routine measurements of all the bleeding parameters are not done as a protocol in our institution for all cases. The bleeding time was prolonged secondary to the antiplatelet therapy given to the patient far the management of ACS. 2D echocardiography showed regional wall motion abnormalities pertaining to LAD territory with mild left ventricular dysfunction but didn't show any evidence of LV clot. MRI spine showed epidural collection located posterior to the spinal cord extending from D8 to L2 spinal level (Fig. 1) with significant compression at D12–L1 level (Fig. 2) suggestive of acute spinal epidural hematoma with cord compression at D12–L1 level. Immediately following the confirmation of epidural hematoma, heparin and clopidogrel were withheld and patient was advised for urgent surgical evacuation of hematoma. Urgent neurosurgical reference was taken. But patient opted for conservative management on account of his age and the high risk involved in the surgery. Following this, patient was treated with intravenous dexamethasone 4 mg every six hourly. Patient was discharged after stabilization of cardiac condition and referred to neurosurgical centre for further management.

3. Discussion

Spontaneous spinal epidural hematomas occur relatively rarely in comparison with traumatic spinal epidural hematomas. Blood dyscrasias, platelet dysfunction, anticoagulation therapy or thrombolysis have been held responsible.[1–4] Only few cases of spinal epidural hematoma following thrombolysis for acute myocardial infarction have been reported in the literature.[5–14] In eight cases the thrombolytic agent was alteplase and streptokinase was used only in one case.[7] Ours is the second case report of spinal epidural hematoma following thrombolysis with streptokinase for acute ST elevation myocardial infarction.

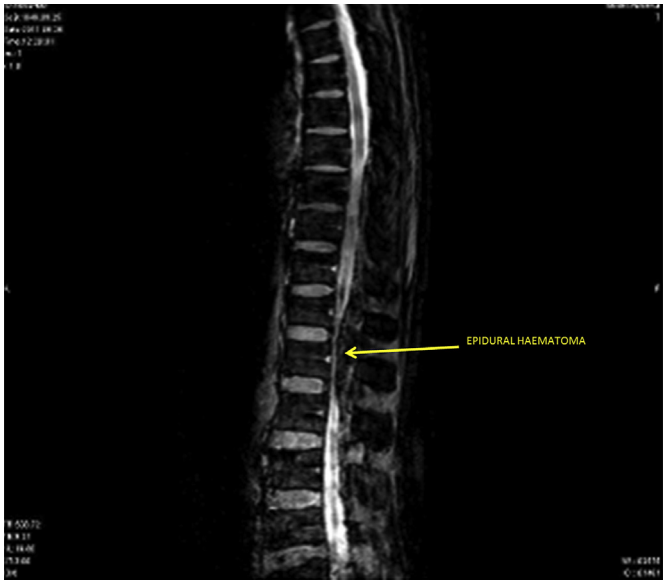

Fig. 1. Sagittal T2 weighted MRI image. This shows a spinal epidural hematoma which is hypointense on T2 weighted MRI image.

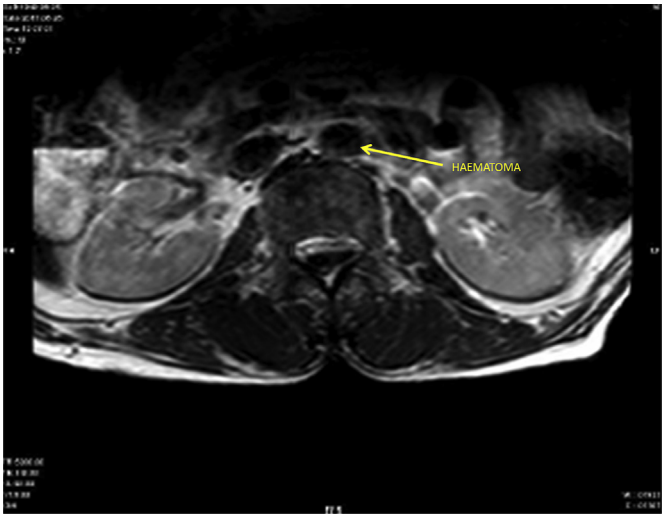

Fig. 2. T2 weighted axial MRI image. This shows a spinal epidural hematoma at the level between D12-L1 thoracic spine with cord compression.

Although, thrombolysis for acute ST elevation myocardial infarction within 6 h from the onset of symptomatic ischemia is a proven evidence based approach to salvage myocardial tissue, restore LV function and reduce mortality, risk of bleeding is the only limiting factor.[15,16] It varies from <5% to 40% in reported series. The most detrimental bleeding occurs in brain with a frequency of 0–2% but with an associated mortality of as high as 60%.[15] Epidural hematoma is a rare complication of thrombolysis. In our case, it is difficult to determine the precise contribution of each agent, i.e. streptokinase and heparin in the causation of the hematoma. Analyzing data on patients who have sustained an intracranial hemorrhage after receiving intravenous rTPA, it can be considered that a combined effect of the thrombolytic agent and heparin may be suggested by the relatively low frequencies of intra-cerebral hemorrhage (0.08%) with the use of placebo-plus-heparin anticoagulation alone, compared to a frequency of 0.27% of intracerebral hemorrhages in patients receiving alteplase-plus-heparin.

Whatever be the cause of epidural hematoma, the importance of recognizing this rare but debilitating complication in time, cannot be overstated. Continuing anticoagulation in absence of suspicion of this condition may be fatal and treatment delay may leave the patient with permanent neurological sequel. Source of bleeding is usually spinal veins. In case of suspicion, MRI spine should be considered as the first diagnostic tool.[16] Contrast MRI shows peripheral enhancement of hematoma, which facilitates clear preoperative localization. Immediate interruption of thrombolytic or anticoagulant therapy or correction of coagulation abnormality is mandatory. Standard therapy which is sometimes recommended for myeloprotection corticosteroid but the role of this is uncertain. Although rapid diagnosis and operation offer the best chances of neurological recuperation, late decompression may be followed by complete recovery.[17]

4. Conclusion

Thrombolytic therapy is the most efficacious modality to achieve reperfusion in STEMI in cases where primary percutaneous intervention is not available. Thrombolytic therapy has its own

spectrum of side effects which needs to be borne in mind while deciding on the optimal strategy of reperfusion.

Neurological sequel in the form of spinal bleeding can be a rare but potentially debilitating complication of thrombolytic therapy.

Conflicts of interest

All authors have none to declare.

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