Spinal Epidural Hematoma Following Tissue Plasminogen Activator and Heparinization for Acute Myocardial Infarction

Kuei-Chuan Chan,1 MD, Der-Jinn Wu,1 PhD, Kwo-Chang Ueng,1 MD, Cheng-Sheng Lin,1 MD, Chin-Feng Tsai,1 MD, Kwo-Shuen Chen,1 MD, Ming-Cheng Lin,1 MD, Kao-Lun Wang,1 MD, and Chung-Sheng Lin,1 PhD

SUMMARY

The case of a 43-year-old Taiwanese man who presented with spinal epidural hematoma following intravenous administration of recombinant tissue plasminogen activator (rTPA) and heparin therapy for acute myocardial infarction (AMI) is reported. Upper back pain and progressive neurological dysfunction ensued, secondary to spinal epidural hematoma with spinal cord compression. The patient did not recover neurological function postsurgically, possibly because the operation was delayed. In conclusion, cardiologists should be alert to this rare, severe complication of rTPA and should perform early laminectomy (≤ 36 hours for those with complete deficit and ≤ 48 hours for those with incomplete deficit) if possible. (Jpn Heart J 2002; 43: 417-421)

Key words: Spinal epidural hematoma, Recombinant tissue plasminogen activator, Acute myocardial infarction

THROMBOLYTIC therapy, in combination with heparin and aspirin, reestablishes coronary blood flow, reduces infarct size, improves left ventricular function, and reduces mortality following acute myocardial infarction (AMI). However, its use is limited by bleeding complications, especially intracranial hemorrhage. Spinal epidural hematoma associated with recombinant tissue plasminogen activator (rTPA) is very rare. We report here a case of spontaneous spinal epidural hematoma after combined rTPA and heparin therapy for AMI.

CASE REPORT

A 43-year-old Taiwanese man was admitted with acute inferior myocardial infarction. His coronary artery disease risk factors were smoking and uncontrolled hypertension. We administered thrombolytic therapy with recombinant
tissue plasminogen activator (r-TPA) (total dose was 65 mg) and heparin (a 5000 unit bolus injection and 800 units per hour). About 30 minutes later, gum bleeding developed so heparin infusion was stopped. He experienced recurrent chest pain 18 hours later and underwent successful rescue percutaneous transluminal coronary angioplasty (PTCA) of the distal part of the right coronary artery by the transfemoral approach. We gave him a bolus injection of 10,000 units of heparin before PTCA and 0.6 mL of low molecular weight heparin (Fraxiparin) was injected subcutaneously thereafter. He complained of upper back pain, and his blood pressure dropped to 80/50 mmHg 6 hours after the PTCA. No evidence of myocardial ischemia was found and his general condition recovered after fluid supply and analgesic medication. Unfortunately, he experienced urine retention, paraplegia, and numbness below the nipples about 43 hours after thrombolytic therapy. Magnetic resonance imaging (MRI) revealed an epidural hematoma from the level of the sixth cervical spine (C6) to the fifth thoracic spine (T5) with compression of the spinal cord. Surgery was not conducted until 7 days later due to reluctance on the part of his family. The patient's neurologic deficits persisted despite the surgery.

Figure 1. Noncontrast T1-weighted sagittal magnetic resonance imaging of the cervical and thoracic spines shows epidural hematoma in front of the spinal cord that is isointense to the spinal cord from the sixth cervical spine to the fifth thoracic spine (arrows).
Figure 2. Noncontrast T2-weighted sagittal magnetic resonance imaging of the cervical and thoracic spines shows epidural hematoma in front of the spinal cord that is hyperintense to the spinal cord from the sixth cervical spine to the fifth thoracic spine (arrows).

Figure 3. Noncontrast T2-weighted axial MRI at the level between 3rd and 4th thoracic spine (T3-T4) shows epidural hematoma in the anterior aspect of the spinal canal that is hyperintense to the spinal cord with cord compression (arrow).
DISCUSSION

The Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) trial established that concurrent use of intravenous rTPA and intravenous heparin was the most effective thrombolytic therapy available, hence, the widespread use of this combination. However, the incidence of bleeding complications (usually minor and in the vascular access sites) using rTPA ranges from 15 to 33%. The most serious complication is intracranial hemorrhage, with a 53% mortality rate. A recent study of patients treated with tPA for AMI found the incidence of intracranial hemorrhage to be 0.95%. The plasma half-life of r-TPA is 2 to 7 minutes, however, the fibrinolytic activity may last for up to 24 hours after discontinuation of the therapy.

In the past 12 years, only 8 cases of spinal epidural hematoma following administration of tPA for AMI and pulmonary embolism have been reported. The most common presenting symptom in spinal epidural hematoma is neck or back pain, with or without radicular radiation, and neurologic dysfunction begins simultaneously or follows. Potential risk factors include marked elevation of partial thromboplastin time (>100 seconds) and fibrin split products, as well as possibly the combined use of heparin and thrombolytic agents, and recent epidural anesthesia administration. Heparin, low-molecular-weight heparin, and other anticoagulants may also pose an independent risk for epidural hematoma. Unlike intracranial hemorrhage, bleeding in the spinal epidural space usually arises from the epidural veins and this fact may account for the silent interval between back pain and neurologic deterioration. Incomplete preoperative sensorimotor deficit correlates highly with favorable outcomes, and recovery is significantly better when decompression is performed in ≤36 hours in patients with complete sensorimotor loss and in ≤48 hours in patients with incomplete sensorimotor deficit. Postoperative improvement in motor function can be anticipated in 95% of patients with incomplete sensorimotor lesions, 87% with incomplete sensory but complete motor deficits, and 45% with complete sensorimotor loss. Complete sensorimotor recovery is expected in 42%, 26%, and 11% of patients in these groups, respectively. However, surgery within 6 months of acute myocardial infarction carries a substantial risk of further cardiac morbidity and mortality. A determination of "cardiac risk" is essential prior to considering surgical intervention. In addition, an intravenous bolus of 30 mg/kg of methylprednisolone over 15 minutes within 8 hours of spinal cord injury followed by a 45-minute pause, and then followed by 5.4 mg/kg/hr for 23 hours may be beneficial.

In conclusion, spinal epidural hemorrhage following administration of thrombolytic agents is exceedingly rare. The sudden onset of back pain or neck
pain and neurologic deficits should alert the clinician to the possibility of spinal hematoma with cord compression. Early surgical decompression (≤36 hours for those with complete deficit and ≤48 hours for those with incomplete deficit) offers the best therapy for the majority of cases.

REFERENCES