Spontaneous Remission of Epidural Hematoma Following Continuous Epidural Anesthesia

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Spinal epidural hematoma (SEH) is a relatively rare event caused by vascular malformations, trauma, epidural anesthesia, coagulation disorders, anticoagulant therapy and spontaneous bleeding[1–4] without prompt recognition and immediate surgical decompression[5]. SEH usually produces a permanent neurological deficit. Spontaneous recovery from SEH has rarely been reported[2,6–8]. We report here a case of SEH following continuous epidural anesthesia with spontaneous recovery over a short time period.

Case Report

A 76-year-old woman with a history of untreated rheumatoid arthritis in her right knee joint underwent a subtotal gastrectomy for early gastric cancer. Laboratory data showed a normal blood count: a platelet count of 235,000; prothrombin time of 10.4 sec; activated partial prothrombin time of 34.1 sec; fibrinogen at 284 mg/dl; and a bleeding time of 1 min. Other preoperative screening examinations were normal except for the ECG which revealed an incomplete right bundle branch block. A thoracic spinal x-ray showed no rheumatic or other degenerative changes.

After several trials an epidural catheter was inserted preoperatively in the Th8–9 intervertebral space with a 18 G Tuohy needle using paramedian approach, for intra- and postoperative pain management. At the time of insertion, slight bleeding was noticed but soon disappeared. The test dose of 3 ml of 1% mepivacaine resulted in no untoward effects. As premedication she was given diazepam (5 mg, P.O.), ranitidine hy-

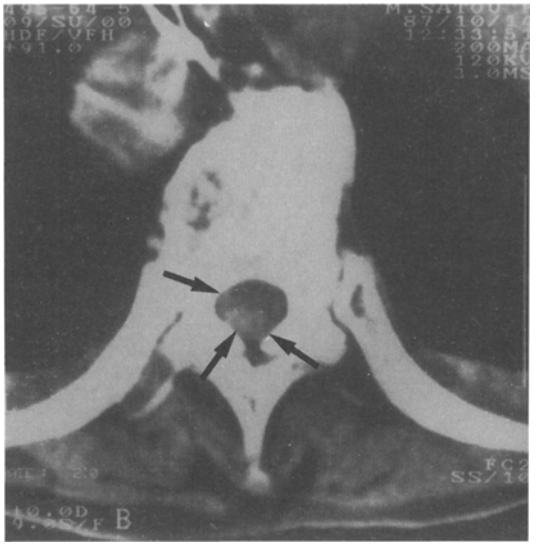

Fig. 1. CT scan of the thoracic spine at the level of Th10. A high density mass (arrow) compresses the spinal cord anteriorly.

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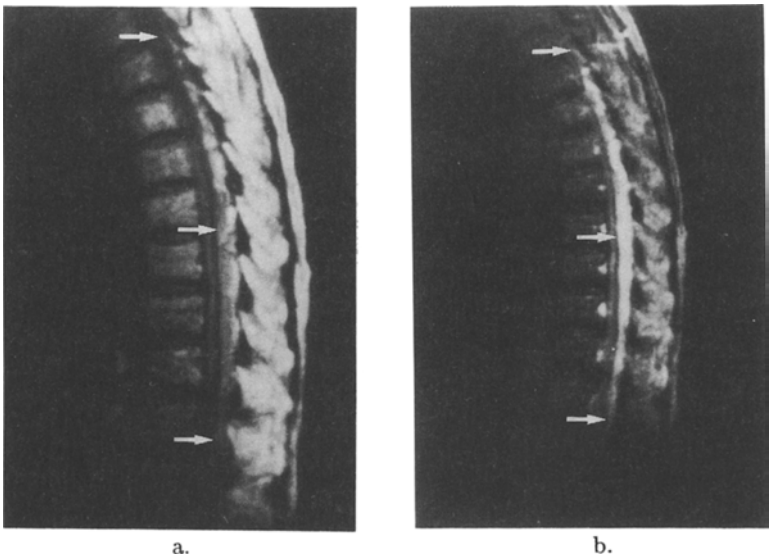

a. b.

Fig. 2. MRI of the thoracic spine 2 hr after the onset of the neurological disorder.

a: T1-weighted image of the sagittal view.

b: T2-weighted image of the sagittal view demonstrates the dorsally located high intensitymass (arrow), over segments Th4 to Th12, compressing the spinal cord.

drochloride (H_2 receptor antagonist, 50 mg, I.M.), and atropine sulphate (0.5 mg, I.M.). Anesthesia was induced with thiopental (5 $mg \cdot kg^{-1}$) and succinylcholine chloride (1.5 $mg \cdot kg^{-1}$), and maintained with 20 ml of 1% mepivacaine for the epidural block, in 60% nitrous oxide and 40% oxygen and pancuronium bromide (initial dose; 0.1 $mg \cdot kg^{-1}$, maintenance; 0.05 $mg \cdot kg^{-1} \cdot hr^{-1}$). After a temporary fall in blood pressure caused by the epidural block, there were no remarkable hemodynamic changes and she recovered from anesthesia uneventfully. The epidural catheter was left in place to control postoperative pain with a continuous infusion of buprenorphine (initial bolus dose; 0.08 mg with 10 ml of saline at 1 hr before the end of the operation, continuous infusion; 2 $\mu g \cdot hr^{-1}$). In neither the operating room nor the recovery room, were any neurological abnormalities noticed.

On the second day following the operation, a surgeon removed the epidural catheter. Twenty minutes after removal, the patient began to complain of severe back pain. One hour later, her legs were totally paralyzed, flaccid, and areflexic, and all sensory modalities were completely lost below the Th10 dermatome on both sides. An immediate computed tomography (CT) scan of the thoracic spine revealed the presence of a high density mass compressing the thoracic spinal cord anteriorly (fig. 1). To identify the extent of the compressed lesion and edema of the spinal cord more clearly for prognosis, magnetic resonance imaging (MRI) of the thoracic spine was performed 2 hr after the onset of this neurological disorder. Just before MRI, paralysis of the lower extremities began to gradually resolve. The MRI revealed a lentiform lesion in the posterior epidural space extending from Th4 to Th12. The MR signal characteristics on the T1- (fig. 2a) and T2-weighted images (fig. 2b) were consistent with acute spinal epidural hematoma (SEH). Four hours later, total resolution of paralysis and sensory disturbance was recovered. Therefore the patient

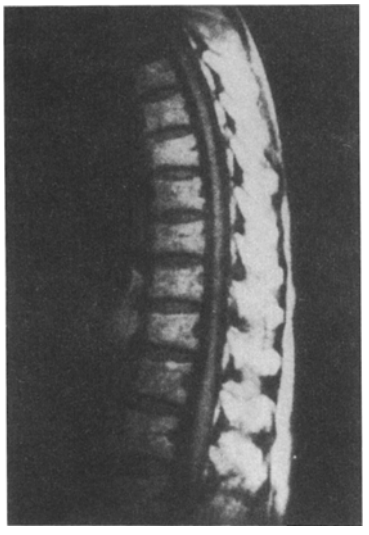

Fig. 3. Repeated T1-weighted MRI performed 1 month later shows the disappearance of the hematoma.

was treated conservatively, with thyrotropin releasing hormone (TRH, 2 mg·day^{-1}; I.V.) and methylprednisolone (1 g·day^{-1}; I.V.) for 7 days for protection from swelling around the hematoma. There were no subsequent signs of aggravation of symptoms and MRI 1 month later showed no SEH (fig. 3).

Discussion

There have been some reports of SEH following epidural anesthesia, but most patients were given anticoagulant therapy or had coagulopathy[2,9–11]. However, no incidence of peridural hematoma leading to spinal cord compression was reported in patients who received placement of epidural catheters with anticoagulant therapy[12,13]. Other SEH etiologies reported included vascular malformation, laminar stenosis, trauma, pregnancy and hypertension[4,14–16]. SEH may also occur spontaneously; most instances were of unknown etiology. SEH is believed to originate in the rich venous plexus of the epidural space. The most common area involved is the thoracic spine because the epidural space is more prominent and the spinal cord occupies a greater percentage of the spinal canal in the thoracic region than in any other region[5]. As epidural veins, which communicate freely with the intraabdominal and retroperitoneal venous structures, have no valves and are thin-walled, it has been postulated that a sudden brief increase of transmitted intrathoracic or intra-abdominal pressure may be the cause of spontaneous epidural bleeding[2]. Blood leakage from the epidural space is prevented by the elevated underlying epidural pressure, probably due to distended extradural veins sealing off the intervertebral foramina. Especially in geriatric patients, narrow paravertebral space also prevents extravasation of blood[2]. With our patient, removing the epidural catheter may have dislodged a small clot formed at the time of catheter insertion or it may have caused a new trauma and formed the hematoma. In addition to the above mentioned pathophysiology, our patient received a continuous infusion of buprenorphine. It was a small volume, but might have been enough to cause a relatively high basal extradural pressure and a rapid increase of extradural pressure in spite of the small size of the hematoma.

The medical management of patients with acute SEH consists of administering steroids and diuretics, and maintaining spinal cord perfusion pressure. As the release of endorphins in the spinal injury exacerbates posttraumatic ischemia by reducing spinal cord blood flow indirectly, TRH, a partial opiate receptor antagonist, is used in the therapy of acute spinal cord injury[17]. Unlike other opiate antagonists, TRH does not cause increased pain.

We found reports of spontaneous remitted epidural hematoma over a relatively short time period in only 5 cases[3,6–8]. Although early detection of epidural hematoma and laminectomy give a better neurological outcome[5], our case had no need for surgical decompression because of its rapid recovery, like the 5 cases mentioned above. Recovery from the neurological deficit in a relatively short time may be due to the dissipation of the liquified hematoma in the extradural space or into the paravertebral space.

Epidural hematoma has been diagnosed by myelography or by computed tomography (CT) with intrathecal contrast material[18]. However, these methods which use contrast materials have a risk of worsening neurological deficits[19]. Magnetic resonance imaging (MRI) of the spine is capable of detecting areas of hemorrhage or cord edema that are not diagnosed with other imaging techniques. And, the ability to obtain different sections in sagittal, axial or coronal planes without having to move the patient is advantageous for preventing the growth of the hematoma. Therefore MRI is of great use in investigating hematoma leisons in detail, and it is furthermore, noninvasive[20].

After epidural anesthesia, the patient should be checked at frequent intervals for return of sensation and motion, prolonged numbness or paralysis, paresthesia and back pain. Spontaneous recovery, as in our patient, is very rare, so neurologic consultation is essential as soon as any signs or symptoms are detected. The definitive diagnosis should be established by MRI if possible. If the MRI shows a space occupying lesion and there is a progressive loss of recovery of spinal cord functions, emergency decompressive laminectomy is imperative. Furthermore, it is important that both anesthesiologists and surgeons be aware that this complication may follow epidural anesthesia.

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