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• Case Report •

Delayed Posttraumatic Spinal Epidural Hematoma: Importance of Early Surgical Treatment for Neurologic Deficits

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Delayed posttraumatic spinal epidural hematoma is an extremely rare disease, and it remains a challenge for surgical teams of trauma centers. Magnetic resonance imaging is an essential tool for early diagnosis, and emergent evacuation of the hematoma is the best choice of treatment. We report the case of a 33-year old man with posttraumatic epidural hematoma in the thoracic spine (T10 and T11 levels), who developed an abrupt-onset paraplegia 5 days after the trauma. [J Trauma Inj 2016; 29: 176-179]

Key Words: Trauma, Spinal cord, Paraplegia

I. Introduction

Spinal epidural hematoma (SEH) is a relatively rare clinical entity that causes spinal cord compression, leading to neurological deficits. SEH is caused by several mechanisms, including coagulopathy, trauma, vascular lesions, and spontaneous causes.(1) Posttraumatic SEH is an uncommon entity, with a reported incidence of less than 1% to 1.7% of all spinal injuries.(2) Rapid surgical evacuation of the hematoma is one of the most important treatment options. Here, we report a case of posttraumatic SEH in the thoracic spine in a patient who developed delayed neurological deficits.

II. Case Report

A 33-year-old man with a negative medical history and experiencing severe back pain was transported to our emergency department. The day before presentation, he had a fall due to a slip that caused his back to forcefully strike a large rock. His body mass index was measured to be 30.8 (170 cm height and 89 kg body weight), which was considered obese. On arrival, he showed an alert mental status and stable vital signs. There was no abnormality in his neurological examination. Initial laboratory findings showed normal ranges of measured parameters, except for an aspartate aminotransferase level of 190 U/L and an alanine aminotransferase level of 172 U/L. Chest, abdomen, and spinal computed tomography (CT) scans were performed to evaluate the internal organs. The chest and abdomen CT scans revealed multiple rib fractures from the left seventh to the eleventh rib with left minimal pneumothorax, left scapular fracture, and spleen and liver contusion without a hemoperitoneum. A spinal CT scan found multiple spinous process fractures from T6 to T12,

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superior end-plate fractures of T10 and T11, and right transverse process fractures from the L1 to L4 levels (Fig. 1).

On meticulous evaluations, no serious lesions that needed urgent surgeries were found; thus, we decided to perform conservative treatments, including bed rest, pain control, and bronchial toilet. Atelectasis of the right lung became aggravated, and the right diaphragm became more elevated after 2 days of hospitalization. Pulmonary physiotherapy and bronchoscopy were actively carried out to prevent pulmonary complications. Because there were no neurological abnormalities, we carefully allowed the patient to walk around the bedside for active lung care with ambulation. On the 5th day of hospitalization, the atelectasis of the right lung had almost

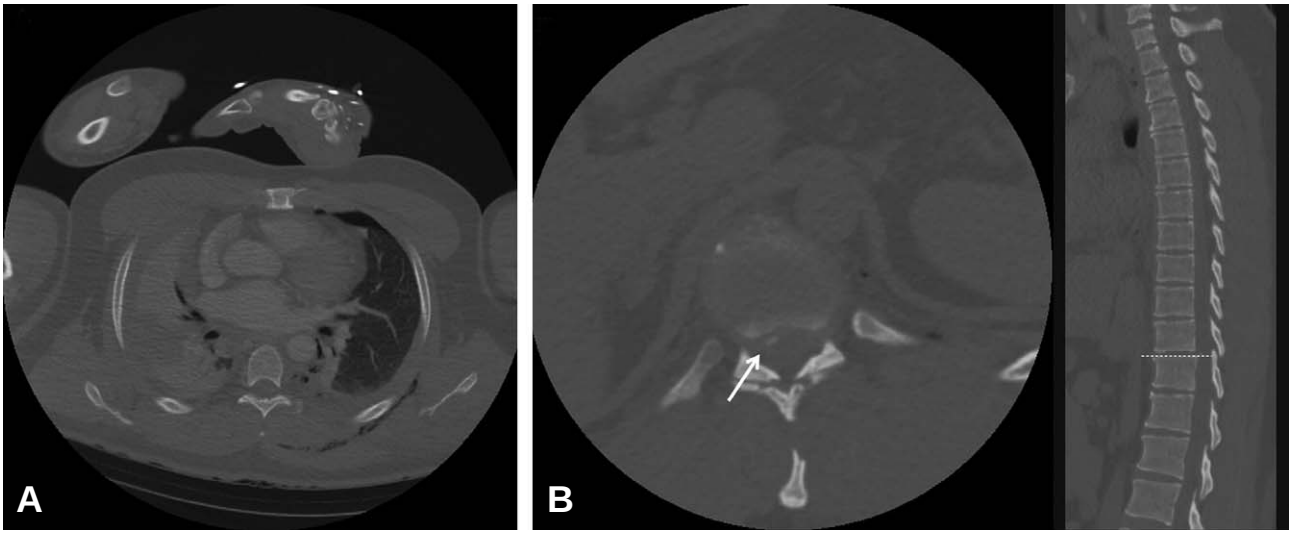

Fig. 1. (**A**) Chest computed tomographic scan showing multiple rib fractures, left minimal pneumothorax, and right hemothorax with lung contusion. (**B**) Spinal computed tomographic scan showing multiple spinous process fractures and superior end-plate fractures at the T11 level (arrow and dashed line).

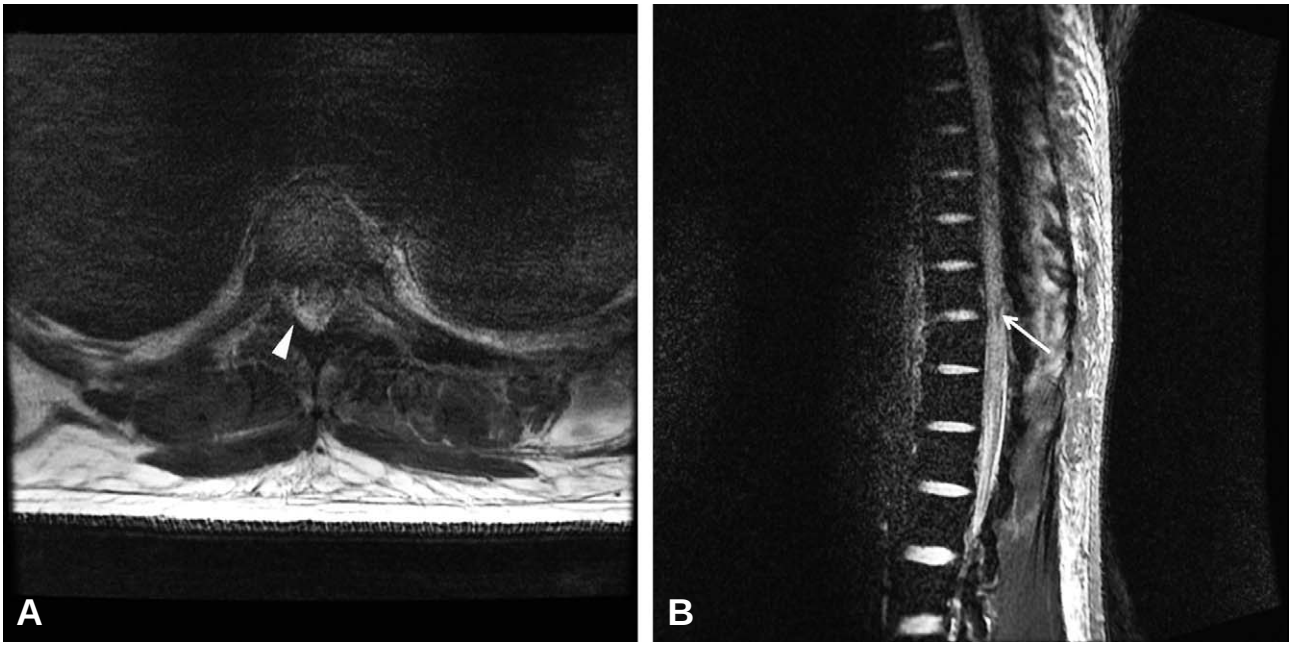

Fig. 2. Axial (**A**) and sagittal (**B**) spinal magnetic resonance images showing dark-signal-intensity lesions (arrowhead in A and arrow in B), suggesting intrathecal hemorrhage at the T10 and T11 levels.

subsided and the general condition of the patient was improving. That night, however, he complained of abrupt paralysis in both lower extremities. Physical examination showed bilateral lower-limb paralysis, sensory loss, and the absence of both anal sphincter tone and bulbocavernosus reflex. Spinal CT scan and magnetic resonance imaging (MRI) were immediately performed. We found that the spinal cord was compressed by a hematoma at the T10-T11 levels. The MRI findings also revealed lesions with dark signal intensity, which suggested intrathecal hemorrhage at the T10-T11 levels (Fig. 2). Finally, delayed epidural hematoma at the T10-T11 levels was diagnosed. The patient underwent emergent decompression at the T10 level and evacuation of the epidural hematoma. The definite bleeding focus could not be found in the epidural space, and the hematoma was not large enough to compress the cord directly. Additionally, we performed partial laminectomy at the T9 lower margin and the T11 upper margin. The neurological deficits of the patient were not improved postoperatively. Unfortunately, 10 days later, he has not gained sensory and motor deficits below the T12 level, with no anal sphincter reflex. The patient and his family strongly wanted to receive rehabilitation treatment at another specialized hospital; thus, he was transferred to another hospital on postoperative day 10.

III. Discussion

SEH is an uncommon disease entity. A traumatic cause of SEH is rarer than spontaneous causes. The incidence of traumatic SEH is usually known to be 1% to 1.7% of all spinal injuries.(2) Several vertebral diseases including spondylosis, rheumatoid arthritis, Paget's disease, and ankylosing spondylitis have been considered as risk factors for posttraumatic SEH.(3,4) It is difficult to identify the causes of bleeding related to SEH accurately. However, hemorrhage from the valveless venous plexus in the epidural space has been the most accepted cause. Rupture of the venous plexus could originate from a sudden pressure change in the epidural space after a blunt trauma.(2,5) In addition, minor trauma without vertebral fracture could be a cause of traumatic SEH because of tearing of the epidural vein derived from acute vertebral disc herniation or disruption. Traumatic SEHs more frequently occur in young men than in old women.(2) Hematomas are more commonly located in the cervical spine than in the thoracic spine; however, thoracic spinal hematoma is usually more symptomatic even with a small amount of hematoma because the thoracic spinal canal is narrower than the cervical or lumbar spine.(4) The clinical manifestations of SEH are generally acute painful episodes and progressively aggravating neurological deficits such as sensory, motor, and sphincter dysfunction at the corresponding level of cord compression. The symptoms are generally present immediately after the trauma; however, the duration between the onset of symptoms and the time of trauma may vary from minutes to months.(6,7)

In this case, the patient was 33-year-old young man who experienced severe back pain without any neurological deficits on the day of the trauma. Paraplegia developed abruptly 5 days after the trauma, unlike other reported cases in which delayed neurological symptoms developed progressively 2 or 3 weeks after the trauma event.(6,7) Although it could not be determined whether the bleeding associated with SEH originated from the spinal artery or valveless venous plexus, we could assume that the epidural hemorrhage occurred suddenly on day 5 after trauma in our patient. The patient had severe atelectasis in the right lower lobe; however, his spinal end-plate fractures were relatively mild and he had no neurological symptoms. Therefore, we advised the patient to perform bedside exercises rather than complete bed rest. This early ambulation improved his lung atelectasis; however, it could also be a determining cause of SEH. We suggest that early ambulation should be carefully decided according to the individual characteristics of trauma patients, as early ambulation in patients with vertebral fracture or spinal cord injury remains controversial. We also cautiously suggest it would be safer to conduct closed neurologic examination and imaging study before deciding an ambulation.

The treatment of choice is still early surgical decompression, although some SEH patients with minimal neurological symptoms could be treated conservatively (e.g., steroid treatment). Lawton et al. report-

ed that early surgical intervention also correlated with outcomes and the neurological deficits improved in 87% of patients who underwent surgical decompression. Patients taken to surgery within 12 h had better neurological outcomes than those whose surgery was delayed beyond 12 h.(1) In addition, thoracic spinal canal is generally known to be narrower. Thoracic part of vertebral column is more complex combining narrow spinal canal and critical vascular supply.(8)

In our case, the patient developed sudden onset paraplegia at midnight. He also had claustrophobia, which led to repeated spinal CT and MRI attempts until radiologic images were successfully obtained. Surgical decompression was performed 14 h after the occurrence of neurdlogical deficits. Unfortunately, surgery did not improve those neurological deficits.

In conclusion, SEH is an uncommon spinal lesion and delayed SEH can occur a few days after a trauma. Emergent surgical decompression could improve some neurological deficits; however, if surgery is delayed, neurological sequelae may persist for a long time.

REFERENCES

1) Lawton MT, Porter RW, Heiserman JE, Jacobowitz R, Sonntag VK, Dickman CA. Surgical management of spinal epidural hematoma: relationship between surgical timing and neurological outcome. Journal of neurosurgery 1995; 83: 1-7.
2) Foo D, Rossier AB. Post-traumatic spinal epidural hematoma. Neurosurgery 1982; 11: 25-32.
3) Lefranc F, David P, Brotchi J, De Witte O. Traumatic epidural hematoma of the cervical spine: magnetic resonance imaging diagnosis and spontaneous resolution: case report. Neurosurgery 1999; 44: 408-10; discussion 10-1.
4) Cuenca PJ, Tulley EB, Devita D, Stone A. Delayed traumatic spinal epidural hematoma with spontaneous resolution of symptoms. J Emerg Med 2004; 27: 37-41.
5) Beatty RM, Winston KR. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg 1984; 61: 143-8.
6) Hsieh CT, Chiang YH, Tang CT, Sun JM, Ju DT. Delayed traumatic thoracic spinal epidural hematoma: a case report and literature review. Am J Emerg Med 2007; 25: 69-71.
7) Rodrigues LM, Abreu F, Fujiki EN, Milani C. Delayed traumatic spinal epidural hematoma with neurological deficits. Einstein (Sao Paulo) 2010; 8: 477-9.
8) Kaur K, Singh R, Prasath V, Magu S, Tanwar M. Computed tomographic-based morphometric study of thoracic spine and its relevance to anaesthetic and spinal surgical procedures. J Clin Orthop Trauma 2016; 7: 101-8.