

Spinal Epidural Hematoma Following Epidural Anesthesia Managed Safely Without Surgery: A Case Report

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Abstract

Spinal Epidural Hematoma (SEH) is known to occur as a complication of invasive spinal procedures, such as epidural anesthesia, and can cause dramatic neurologic deficits if not diagnosed and treated immediately. The present report describes the case of a gynecologic patient who presented with weakness and numbness of both lower limbs soon after an epidural test bolus was injected. Urgent magnetic resonance imaging of the thoracic spine demonstrated an epidural hematoma at T12/L1 with slight spinal cord compression. The patient demonstrated significant improvement in neurologic deficits within a short time. Therefore, general anesthesia was induced, and the scheduled operation was performed uneventfully. After the operation, the patient had no detectable neurologic abnormality, and repeat imaging showed almost complete resolution of the hematoma. Although urgent decompression is the treatment of choice for SEH, conservative management may be indicated if the patient demonstrates rapidly improving neurologic deficits.

Keywords: Spinal Epidural Hematoma (SEH); Conservative treatment

Case Report

The patient, a 27-year-old woman (height, 168 cm; weight, 51.4 kg), had an insignificant medical history with no recent trauma or illness, and she was not taking any medication. Laboratory values revealed that platelet counts, prothrombin time, and activated partial thromboplastin time were normal. No visible signs of coagulopathy were present. She was scheduled for a vaginal radical trachelectomy to treat early-stage cervical cancer. With the patient in the left lateral position, a 19-Ga epidural catheter (Flex Tip plus[®]; Epidural Catheter #KM-05500-J, Arrow International, Reading, PA, U.S.A.) was easily placed at the T11/12 level, prior to induction of general anesthesia. However, soon after placement of the epidural catheter, she experienced slightly reduced sensation in both lower extremities (Frankel Grade [1] D). A test dose injection of 1% lidocaine (3 mL) with 1/100,000 adrenaline, following negative aspiration for blood or Cerebral Spinal Fluid (CSF), induced loss of voluntary movement in both lower extremities and a clear sensitivity to a pinprick at the T6 level (Frankel Grade B). The patient was neither distressed nor drowsy and had no papillary changes or ventilator difficulty, and her vital signs were stable. Intrathecal catheter placement was suspected, despite CSF being aspirated through the catheter. Because the ferromagnetic catheter, which was composed of a combination of medical grade polyurethane and inner stainless steel coil wire, might interact with the magnetic field during subsequent Magnetic Resonance Imaging (MRI), the epidural catheter was removed (Figure 1 and 2).

When the catheter was removed, there was no resistance to catheter removal or bleeding from the site of insertion. Urgent MRI of the thoracic spine demonstrated a mass in the epidural space at T12/L1, with spinal cord compression. The contents of the epidural space were isointense with focal hyperintensities on both T1- and T2-weighted images. The MRI characteristics were consistent with the early stage of an epidural hematoma, but the patient's rapidly improving neurologic deficit prompted conservative management, under consultation with a neurosurgeon. The patient continued to demonstrate dramatically improved neurologic symptoms and was soon able to move without difficulty (Frankel Grade E). Laboratory values also revealed normal platelet counts, prothrombin time, and activated partial thromboplastin time. No visible signs of coagulopathy were present. The patient did not complain of even slight back pain; therefore, general anesthesia

was induced, approximately 2 h after her urgent departure from the operating theater, and the operation proceeded without further complications.

The patient's postoperative course was uneventful, without symptoms of a Post-Dural-Puncture Headache (PDPH) or other neurological deficits. There were no bowel or bladder disorders, and no complaints of back pain.

The patient was discharged 10 days later after demonstrating complete neurological recovery and a nearly complete resolution of the epidural hematoma on MRI findings.

Discussion

The patient in the current report demonstrated temporary neurological symptoms subsequent to the placement of an epidural catheter. An MRI scan revealed the presence of SEH with spinal cord compression soon after the procedure. As there was a slight numbness in both lower extremities during epidural catheterization, her symptoms were attributed to spinal cord compression due to the SEH. Retrospectively, we should have carefully evaluated her slight numbness prior to a test dose injection; this may have led to a differential diagnosis, such as subdural injection or atypical spinal anesthesia. Accidental subdural injection or atypical spinal anesthesia may have also been the cause of the temporary neurologic deficit reported by the patient.

Over the last 30 years, there have been numerous reports of accidental subdural injections, some supported by radiographic evidence [2,3], and atypical spinal anesthesia [4]. The subdural space is a potential cavity between the dura and arachnoid mater, recognized by anatomists and radiologists [5]. The space runs from the lower

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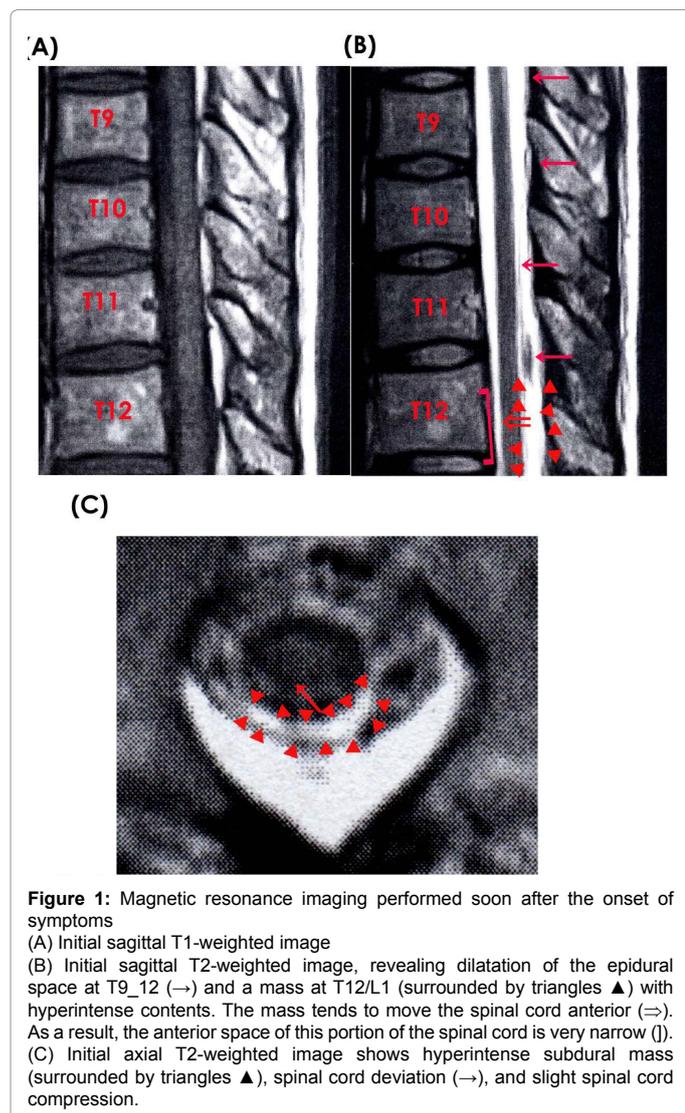


Figure 1: Magnetic resonance imaging performed soon after the onset of symptoms

(A) Initial sagittal T1-weighted image
 (B) Initial sagittal T2-weighted image, revealing dilatation of the epidural space at T9_12 (↔) and a mass at T12/L1 (surrounded by triangles ▲) with hyperintense contents. The mass tends to move the spinal cord anterior (→). As a result, the anterior space of this portion of the spinal cord is very narrow (|).
 (C) Initial axial T2-weighted image shows hyperintense subdural mass (surrounded by triangles ▲), spinal cord deviation (↔), and slight spinal cord compression.

border of the second sacral vertebra into the cranial cavity, but does not communicate with the subarachnoid space [5]. However, Collier [6] suggested that there was a slight risk of an extensive block, possibly following rupture of the remaining layers of the dura or the arachnoid, produced by trauma from needle or catheter insertion and subsequent injection, leading to diversion of the intradural solution into the subarachnoid spaces (atypical spinal block, or a high spinal block). A clinical feature of a subdural injection, which may not be diagnosed on clinical grounds, is the slow onset of a high sensory block, usually 10-35 min after an apparently uneventful epidural block insertion, or even after injection of the test dose alone [2]. For atypical spinal anesthesia, the mean reported time required for total spinal block is 10 min [4]. Therefore, our patient's temporary neurological deficits were not almost certainly due to a subdural or atypical spinal block.

SEH, as a complication of epidural anesthesia, is a well-recognized [7] condition constituting a neurological emergency and requiring urgent investigation and treatment. Indeed, the therapeutic outcome for SEH patients depends on the length of the delay between diagnosis and surgical decompression [8]. MRI scans are useful for conclusively establishing the diagnosis [9]. The diagnosis of SEH in our patient was also confirmed by MRI, but the MRI findings alone cannot dictate the management of this condition nor predict its outcome [10]. A

review of the relevant literature [11] shows that the most important predictor of treatment outcome is a rapidly improving neurologic deficit during the SEH patient's preoperative period. A retrospective study by Lawton et al. [12] demonstrated that the average interval from onset of initial symptoms to maximum neurological deficits in SHE patients was 13 hours, and that patients who were operated on within 12 hours had better neurological outcomes than patients with identical preoperative Frankel grades whose surgery was delayed beyond 12 hours. Formal indicators for conservative treatment of this condition do not exist. However, absence of bleeding and abnormal laboratory data, with continued improvement of the neurological deficits, justifies conservative treatment of the patient within the 12-h period from symptom onset, in consultation with a neurosurgeon. However, if neurologic deterioration occurs or if the improvement plateaus at an unacceptable level, surgical evacuation of the hematoma is indicated.

In conclusion, patients with SEHs should be rapidly evaluated by MRI because spinal compression tends to develop afterwards. Early recognition, accurate diagnosis, and appropriate treatment can result in significant neurologic improvement and good outcomes. Severity of neurologic impairment has the greatest impact on management and outcome. Non-operative treatment may be successful in cases with minimal neurologic deficits, despite spinal cord compression revealed by MRI.

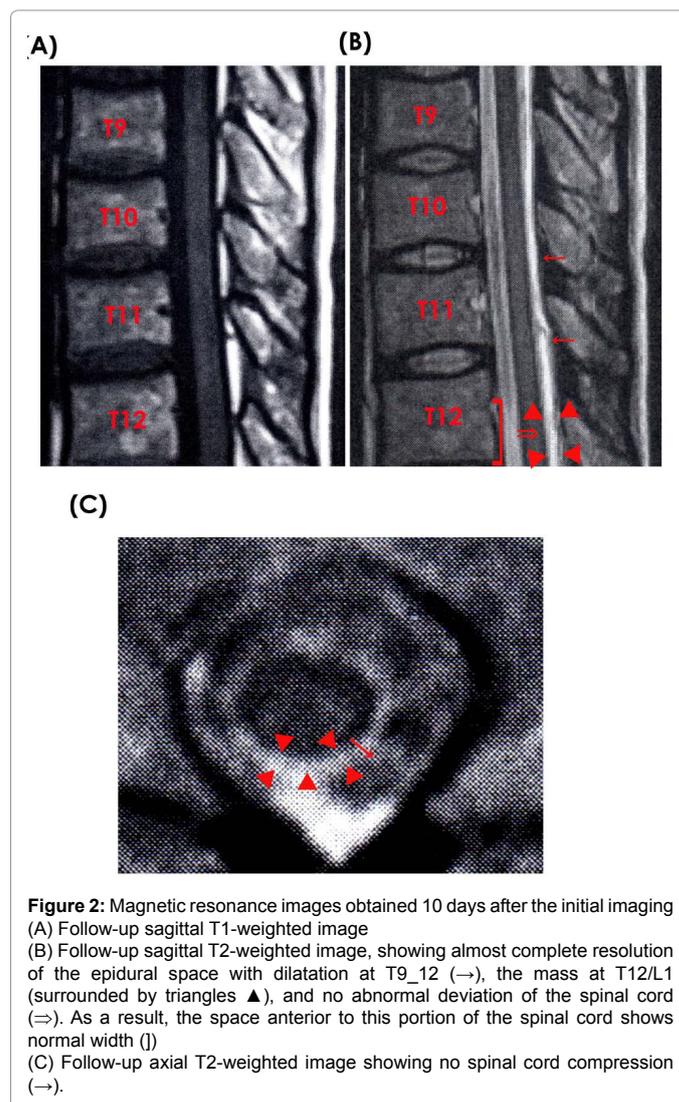


Figure 2: Magnetic resonance images obtained 10 days after the initial imaging
 (A) Follow-up sagittal T1-weighted image
 (B) Follow-up sagittal T2-weighted image, showing almost complete resolution of the epidural space with dilatation at T9_12 (↔), the mass at T12/L1 (surrounded by triangles ▲), and no abnormal deviation of the spinal cord (→). As a result, the space anterior to this portion of the spinal cord shows normal width (|)
 (C) Follow-up axial T2-weighted image showing no spinal cord compression (↔).

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