CASE REPORT

Spinal Subdural Hematoma Mimicking Epidural Lipomatosis

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We report a case of spinal subdural hematoma in the lumbar spine of a 75-year-old woman. Magnetic resonance imaging showed lesions of homogeneous high intensity in the spinal canal on both T1- and T2-weighted images, findings closely resembling those for epidural lipomatosis. Identification of 2-layered signal intensity surrounding the cauda equina on axial images is the key for accurate diagnosis.

Keywords: epidural lipomatosis, MRI, spinal subdural hematoma

Introduction

Spinal subdural hematoma is a rare condition that can cause progressive spinal cord compression syndrome. We encountered a case of lumbar subdural hematoma in which findings on magnetic resonance (MR) imaging mimicked those of spinal epidural lipomatosis.

Case Report

We evaluated a 75-year-old woman with a 10-day history of low back pain and weakness of the left lower extremity after she had done a stretching exercise. She had a history of Wegener’s granulomatosis and had been treated with corticosteroid for one year. MR imaging showed long and segmented homogeneous high signal intensity in the spinal canal from the L2 to S3 level on both T1- and T2-weighted images, and compression of the cauda equina was noted (Fig. 1). Initially, we interpreted the findings as spinal epidural lipomatosis, but careful observation of MR axial T2-weighted images revealed a double layer of signal intensity surrounding the cauda equina (Fig. 2). In the sagittal images, we observed a difference in signal intensity between the lesion and epidural fat in the lower sacral level of the spinal canal, which led us to suspect lumbar subdural hematoma. Follow-up MR imaging performed 3 weeks after symptom onset showed a decrease in the signal and volume of the posterior compartment that had shown high intensity, and we noted a structure of high signal intensity on fat-suppressed T1-weighted sagittal images in the subdural space and confirmed diagnosis of lumbar subdural hematoma (Fig. 3).

Because the hematoma volume decreased on MR imaging and neurological status had shown gradual

Fig. 1. Magnetic resonance (MR) imaging taken 10 days after symptom onset. On both sagittal T1- (A) and T2-weighted (B) images, long and segmented homogeneous high signal intensity is observed in the spinal canal from the L2 to S3 level. Compression of the cauda equina is noted. Careful observation revealed signal difference between the lesion and fat in the spinal canal of the lower sacral level.

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Fig. 2. Magnetic resonance (MR) imaging taken 10 days after symptom onset. On T2-weighted axial image, 2-layered signal intensity surrounding the cauda equine is noted. Signal intensity of the inner layer is slightly high and thought to represent subacute subdural hematoma (large arrows), whereas intensity of the outer layer is very high and interpreted as epidural fat (short arrows).

Fig. 3. Magnetic resonance (MR) imaging taken 3 weeks after symptom onset. (A) The previously noted area of high signal intensity in the anterior compartment of the spinal canal on T1-weighted sagittal images has nearly disappeared. A decrease in the high signal intensity and volume of the posterior compartment is also seen. (B) Fat-suppressed T1-weighted sagittal images demonstrate a structure with high signal intensity in the subdural space (arrows).

improvement, conservative treatment was maintained. The patient was discharged 2 weeks after admission with significant clinical improvement.

Discussion

Spinal subdural hematoma is a rare entity, comprising about 3% of all spinal hematomas, and far less frequent than spinal epidural hematoma or subarachnoid hemorrhage.1 No gender predominance is seen, although prevalence is slightly higher in the fifth and sixth decades of life.2 Spinal subdural hematoma is most commonly associated with bleeding disorders, including patient response to anticoagulant therapy. It can also result from intradural tumor or vascular malformation, iatrogenic causes, such as from lumbar puncture or spinal drainage, or infrequently arise as an idiopathic case.1,2 Spinal subdural hematoma usually presents with signs of compression of the spinal cord or cauda equina, often with acute onset preceded by back pain or radicular pain.3 Such hematomas are most often located in the thoracolumbar or lumbar segment and posteriorly rather than anteriorly or circumferentially.2,3 On computed tomography and MR imaging, it tends to demonstrate a crescent rather than biconvex shape that resembles the appearance of intracranial subdural hematoma.3 Partial or even total recovery can be expected following surgical intervention comprising clot evacuation and laminectomy in the acute phase. Conservative treatment is recommended if early recovery is observed, neurological deficits are mild, the degree of extension precludes surgical treatment, or coagulopathy is present.2–4

Spinal hematoma can occur in either the epidural or subdural space.5 Subdural hematoma is diagnosed if the dura mater is identified between the hematoma and epidural fat.3,6 Previous reports of subdural hematoma have also shown differences in signal intensity between the hematoma and epidural fat.3 Braun and associates described signal changes in spinal subdural hematoma on MR imaging according to the time course.4 They reported that in the late subacute phase (one to 2 weeks after the initial event), hematoma shows hyperintensity on T1- and T2-weighted images, which reflects extracellular methemoglobin after lysis of red blood cells. Our patient underwent initial MR imaging 10 days after onset, which showed the hematoma as areas of high signal intensity on both T1- and T2-weighted images, findings consistent with previous descriptions.3,6,7 Braun’s group also pointed out that fat-suppressed T1-weighted imaging in the subacute phase is helpful in differentiating subdural
hematoma from epidural fat. Diagnosis of our patient might have been easier if fat-suppressed sequences had been performed at the initial visit.

Spinal epidural lipomatosis is defined as an accumulation of normal unencapsulated fat in the extradural space. Epidural lipomatosis is known to occur among steroid users and patients who are obese, have other idiopathic causes, and sometimes cause neurological symptoms.

In this case, findings from the initial MR imaging mimicked those of epidural lipomatosis because of the close similarity between the signal intensity of subacute-phase hematoma and lipid. The hematoma also existed circumferentially and demonstrated a homogeneous appearance, which is unusual in subdural hematoma. Our patient’s history of corticosteroid therapy and relatively mild symptoms also contributed to the initial confusion.

Guilfoyle and colleagues reported a case of spontaneous spinal subdural and subarachnoid hematoma in a patient with history of Wegener’s granulomatosis and proposed the effect of granulomatosis on the spinal meninges as a possible cause of spinal intradural hematoma. Although the etiology of our case remains unknown, Wegener’s granulomatosis could contribute to inducing spinal subdural hematoma.

In conclusion, we encountered a case of subacute-phase spinal subdural hematoma that mimicked spinal epidural lipomatosis on MR imaging. Although rare, spinal subdural hematoma should be considered among the differential diagnoses for progressive spinal compression syndrome. Close observation of both axial and sagittal images and selection of appropriate imaging sequences are essential for accurate interpretation of spinal MR imaging.

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