CASE REPORT



Infected epidural hematoma of the lumbar spine associated with invasive pneumococcal disease

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ABSTRACT

Spinal epidural abscess (SEA) and spinal epidural hematoma (SEH) are neurologic emergencies with distinct etiologies and treatment. Despite similarities on magnetic resonance imaging (MRI), their differentiation is usually possible with meticulous history taking and neurologic examinations. We report an unusual case of SEA that developed from preceding SEH, posing a diagnostic challenge to physicians. A 65-year-old diabetic man suddenly experienced back pain and weakness of both legs when he lifted heavy luggage. He was afebrile, and his laboratory tests were mostly unremarkable. Spinal MRI consisting of T1-weighted, T2-weighted, and fat-suppressed T2-weighted images revealed an epidural mass over the L2-L4 spinous process. He was diagnosed with SEH based on his symptoms and MRI findings, and was treated conservatively using steroid pulse therapy. Despite initial improvement, he suddenly developed into septic shock and coma on the 10th hospital day, and died shortly thereafter. An autopsy revealed massive pus accumulation in the lumbar epidural space and brain, and a postmortem diagnosis of infected SEH associated with invasive pneumococcal disease was established. Serial MRI studies, including diffusion-weighted and/or gadolinium-enhanced T1-weighted images are warranted in patients with a presumed diagnosis of SEH receiving steroid therapy to detect such infectious transformation.

Key words: Invasive pneumococcal disease, magnetic resonance imaging, spinal epidural abscess, spinal epidural hematoma

Introduction

Spinal epidural abscess (SEA), once considered a rare infection, is currently encountered more commonly because of both heightened awareness among physicians and widespread availability of magnetic resonance imaging (MRI).^[1,2] In the majority of cases, SEA is either a consequence of spondylodiskitis or a complication of medical procedures such as epidural catheter placement, and spontaneous SEA is relatively rare.^[1-4] Spinal epidural hematoma (SEH) is another neurologic emergency that should be differentiated from SEA. Although these diseases may be similar radiographically, differentiation is usually possible if a meticulous history is

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Dr. Joji Inamasu, Department of Neurosurgery, Fujita Health University Hospital, 1-98 Kutsukake, Toyoake 458-0831, Japan. E-mail: inamasu@fujita-hu.ac.jp obtained and the differences in the clinical manifestation of each disease are taken into consideration.^[3,4] Here we report a rare case of SEA, which developed from preceding SEH after invasive pneumococcal disease, posing a diagnostic challenge to physicians.

Case Report

A 65-year-old man with long-standing diabetes suddenly experienced low back pain and mild weakness of both legs when he lifted heavy baggage from the ground. He thought he had back strain and tried to alleviate the pain using aspirin. However, the pain worsened with time, and he visited our emergency department 4 days after the initial event. He was alert and oriented, and his vital signs including body temperature were within the normal range. Laboratory data showed a normal white blood cell count and moderately elevated serum C-reactive protein level (6.4 mg/dL). Neurologic examination revealed diminished plantar reflexes and numbness and mild weakness of the lower legs that was rated as 3/5 on manual motor testing. Although the back pain was aggravated by dorsiflexion, no tenderness was noted on percussion. MRI of the lumbar spine revealed an epidural mass over the L2-L4 spinous process, which was observed as a mildly hyper-intense signal on the T1-weighted image (T1WI), an iso-intense signal on the T2WI, and a mildly hyper-intense signal on the fat-suppressed T2WI [Figure 1a-c]. Furthermore, mildly hyper-intense signals were noted in the posterior ligament complex of the L1 and L2 on the fat-suppressed T2WI [Figure 1c]. Both vertebral bodies and intervertebral disks appeared to be intact except mild degenerative changes. Taking the clinical history and neurologic findings into consideration, a diagnosis of SEH was made by emergency physicians and the MRI findings were considered compatible with SEH by radiologists.

Because neural compression by the epidural mass seemed to be minimal on axial images of the MRI [Figure 1d and e], the patient underwent conservative management including bed rest and steroid pulse therapy: A 1000 mg intravenous bolus of methylprednisolone was administered on the 1st hospital day, followed by a 500 mg bolus on the second and 3rd hospital day. His back pain was relieved; the neurologic findings improved substantially after treatment, and he underwent a rehabilitation program. On the 10th hospital day, he complained of recurrence of the back pain, and within 6 h of his complaint, he became obtunded without manifesting fever. He subsequently developed into coma and severe hypotension refractory to vasopressors, and died on the 12th hospital day because of multi-organ failure. An autopsy was granted, which revealed massive pus accumulation and hemorrhage both in the lumbar epidural space and brain [Figure 2a and b]. Immunohistochemistry of the spinal cord section revealed many round cells positive for pneumococcal anti-gen [Figure 2c], establishing a postmortem diagnosis of fulminant pneumococcal meningoencephalitis that evolved from SEH.

Discussion

Clinical presentation, MRI and autopsy findings in this case strongly suggest the diagnosis of infected SEH, although the possibility that the epidural mass had been a pneumococcal abscess from the beginning cannot totally be denied. The symptom, strain-induced low back pain developing in an elderly man taking an anti-platelet mediation, is typical of SEH: Both strain-induced abdominal/venous pressure elevation and drug-induced bleeding diathesis are well-known precipitating factors.^[5] In addition, the presence of a mildly hyper-intense epidural mass on the T1WI [Figure 1a] and mild hyper-intensity in the posterior ligament complex on the fat-suppressed T2WI [Figure 1c] indicated that the hyper-intense signals represented aging blood components.

Transformation of SEH into SEA is very rare. In the brain, however, transformation of an epidural or subdural hematoma

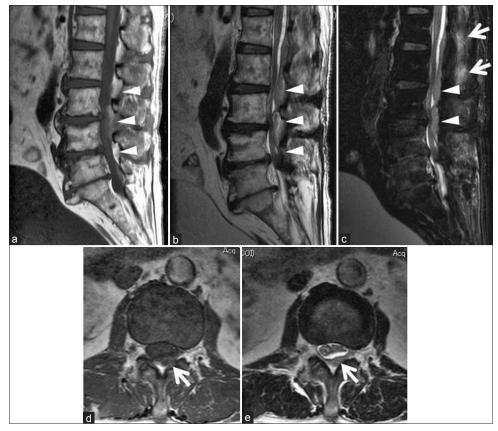


Figure 1: An epidural mass was observed as a mildly hyper-intense signal on the T1-weighted (a), iso-intensity signal on the T2-weighted (b), and a mildly hyper-intense signal on the fat-suppressed T2-weighted image (c, arrowheads). Furthermore, mildly hyper-intense signals in the posterior ligament complex on the fat-suppressed T2-weighted image indicated the presence of aging blood (c, arrows). On axial view (L2–L3 disk level), a mixed intensity signal on the T1-weighted (d) and an iso-intensity signal on the T2-weighted image (e) was shown to compress the dura (arrows)

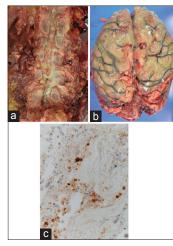


Figure 2: (a and b) Macroscopic photographs of the autopsy specimen. Both the spinal column (a) and brain (b) were covered with thick pus. (c) An immunohistochemical photomicrograph showing a cluster of round cells positive for pneumococcal anti-gen, establishing a diagnosis of pneumococcal meningoencephalitis (Original magnification: ×100)

into an abscess has been reported in the literature.^[6,7] The most common causative organism of SEA is *Staphylococcus aureus*,^[2] and SEAs due to *Streptococcus pneumoniae* are uncommon, with approximately 50 reported cases in the literature.^[8] Pneumococcal SEA is mostly a secondary event following pneumococcal meningitis; the bacteria propagate from the cerebrospinal fluid to the epidural space after penetrating the dura.^[9,10] Therefore, the mode of infection propagation in this case was different from that in the other reported cases of pneumococcal SEA.^[9,10] An autopsy failed to reveal the primary focus of the pneumococcal infection, and it remains unknown how the bacteria entered into the spinal epidural space.

Spinal epidural hematoma and SEA may differ radiographically in that the former exhibits an iso-to hyper-intense signal, whereas the latter exhibits an iso-to hypo-intense signal on T1WI.^[11] However, there may be substantial overlap because the signal intensities of SEH are influenced by age and biochemical evolution of the hematoma, and initial mix-ups between these diseases have been reported.^[12,13] The addition of diffusion-weighted image (DWI) and/or gadolinium-enhanced T1WI to the routine spinal MRI sequences may allow early detection of SEA.^[14,15] In the majority of noniatrogenic SEA cases, inflammation spreads from the adjacent spondylodiskitis:[1-3] therefore, an abscess is usually located in the anterior half of the spinal canal. This case is unique in that an abscess developed in the posterior half of the spinal canal without involvement of the vertebral body or disk, which also suggests the development of SEA from preceding SEH.^[3,5] The radiographic (i.e. MRI) differences to facilitate differentiation between SEH and SEA are summarized in Table 1.^[1-3,11,14-17]

Establishing early diagnosis of SEA may be particularly challenging when the pertinent clinical findings are ambiguous. The absence of fever in this case may have been attributable to

Table 1: Radiographic differences between spinal epidural hematoma and epidural abscess^[1-3,11,14-17]

	Spinal epidural hematoma	Spinal epidural abscess
TıWI	lsointense (acute): Mildly hyperintense (subacute)	Hypo-isointense
Gadolinium enhancement on T1WI	Uncommon	Almost always
T2WI	Mildly hyperintense (acute): Isointense (subacute)	Hyperintense
DWI	Mildly hyperintense	Hyperintense
Vertebral body/disk involvement	Rare	Frequent
Location of an mass (ant. vs. post.)	Posterior>anterior	Anterior>posterior
Spinal level (cervical vs. thoracolumbar)	Cervical>thoracolumbar	Thoracolumbar>cervical

 $\mathsf{DWI}-\mathsf{Diffusion}\text{-weighted image}; \mathsf{T}_2\mathsf{WI}-\mathsf{T}_2\text{-weighted image}; \mathsf{T}_2\mathsf{WI}-\mathsf{T}_2\text{-weighted image}$

an impaired immune response due to long-standing diabetes and also to the use of steroid pulse therapy. Steroid pulse therapy has occasionally been used either as an adjunct to surgery^[18] or as a stand-alone treatment^[19] in SEH patients. Considering its potentially adverse effects such as the negative impact on the immune functions, steroid therapy for SEH should be used more judiciously in patients with medical comorbidities. Serial MRI studies including DWI and/ or gadolinium-enhanced T1WI may particularly be warranted in patients with a presumed diagnosis of SEH undergoing conservative management because the intraoperative verification of SEH is not feasible in such cases. Alternatively, image-guided percutaneous drainage may be a less invasive measure which may have a diagnostic/therapeutic value in the management of patients with an epidural mass, particularly of those with a lesion in the lumbar spine.^[20]

Conclusion

A rare case of SEH which transformed into abscess after invasive pneumococcal infection was reported. Although steroid pulse therapy may have a role in the treatment of SEH, it should be used judiciously in patients with medical comorbidities. Periodic radiographic survey by repeated MRI including DWI is warranted in patients with the presumptive diagnosis of SEH, who is treated conservatively.

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