Lumbosacral Subdural Hematoma

—Case Report—

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Abstract

An 18-year-old man presented with a rare spinal subdural hematoma (SSH) manifesting as headache, which developed following diving into the sea the day before. Computed tomography of the head revealed no abnormality. His headache persisted followed by neck stiffness and mild lumbago. He came to see us 6 days after the first visit. He underwent lumbar puncture to eliminate the possibility of meningitis. The cerebrospinal fluid showed xanthochromia. Cerebral angiography showed no abnormality. Magnetic resonance imaging of the spine revealed lumbosacral subdural hematoma. The hematoma and his symptoms regressed spontaneously during several weeks of conservative treatment. A sharp increase in intraabdominal and/or intrathoracic pressures following diving may have caused the bleeding. Headache may be an initial symptom associated with subarachnoid hemorrhage concomitant with SSH. Delayed progression of lumbosacral SSH must be considered if the patient complains of lumbago following minor trauma.

Key words: spinal subdural hematoma, trauma, subarachnoid hemorrhage

Introduction

Spinal subdural hematoma (SSH) is a rare condition which is often related to blood dyscrasia, anticoagulation, arteriovenous malformation (AVM), lumbar puncture, and trauma.²⁰⁾ Spontaneous cases have been also reported.^{1,7,8,10,11,17,21)} The cause of the bleeding is not well understood. Lumbar SSH usually presents with cauda equina syndrome or lumbago.^{2,5,9,11,14,16,17,22)} We report a case of lumbosacral subdural hematoma with subarachnoid hemorrhage (SAH) following a dive into the sea, which manifested only as headache at the beginning and caused lumbago a few days later.

Case Report

A 18-year-old man visited our unit with headache, which had persisted since he jumped into the sea the day before. He reported that he jumped headfirst

into the sea from a height of 2 m and, after he had jumped in, he had fainted away, but soon regained consciousness. Abdominal and head bruising were denied. Computed tomography of the head showed no abnormality. He visited our unit 6 days later again with complaints of persistent headache followed by neck stiffness and mild lumbago. Neurological examination revealed no abnormalities and no scars on his head and waist. Lumbar puncture was performed with a 23-gauge spinal needle at the L3–4 levels. Cerebrospinal fluid (CSF) examination revealed xanthochromia with opening pressure of 150 mmH₂O, 95 white blood cells/mm³, with 37 polymorphonuclear cells and 58 mononuclear cells, and glucose and protein levels of 44 and 31 mg/100 ml, respectively.

Routine laboratory analysis, including cell count and coagulation screening, revealed no abnormalities. Cerebral angiography showed no cerebral aneurysm, AVM, or dural arteriovenous fistula.

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Fig. 1 Sagittal T₁-weighted (left) and T₂-weighted (center) magnetic resonance (MR) images on admission showing an isointense mass (arrowheads) in the subdural space extending from the L-5 to S-2 levels. T₁-weighted MR image with gadolinium (right) showing no abnormal staining. Note the inter-spinous enhancement showing the scar following lumbar puncture at L3-4 levels.

Fig. 2 Sagittal T₁-weighted (left) and T₂-weighted (center) magnetic resonance (MR) images 5 months after the onset showing no residual hematoma. T₁-weighted MR image with gadolinium (right) showing no abnormal staining.

Magnetic resonance (MR) imaging showed an isointense mass on both sagittal T₁-weighted and T₂-weighted images in the subdural space extending from L-5 to S-2 (Fig. 1). These findings were compatible with subdural hematoma. T₁-weighted MR imaging with gadolinium showed no findings of AVM and no tumor-like staining. His headache disappeared 1 day after admission, and his lumbago was gradually reduced and finally disappeared 2 weeks later. MR imaging showed the hematoma had almost disappeared spontaneously 1 month later and showed no hematoma and no abnormalities 5 months after the onset (Fig. 2).

Discussion

The pathogenesis of the bleeding in SSH is controversial, because the spinal subdural space is almost avascular. One theory is that the SSH might

originate in the subarachnoid space, dissect the arachnoid membrane, and spread into the subdural space.^{3,12)} The blood could be dissipated by the CSF faster in the subarachnoid space than in the subdural space. However, only one report has described bleeding from the subarachnoid space into the subdural space, namely from the radicular vein, as confirmed at the autopsy of a patient who had undergone lumbar puncture.¹²⁾ Nevertheless, a subarachnoid origin of the bleeding cannot be eliminated. Many authors continue to support this theory in cases of simultaneous SSH and SAH.^{7,11,21)}

One theory has been proposed to account for the dynamics of bleeding.¹⁹⁾ Sudden and sharp increases in abdominal and thoracic pressures are transmitted intravascularly along the lateral spinal arteries and veins through the intervertebral foramina to the intraspinal segments of the vessels.¹⁹⁾ Such a rapid increase in intravascular pressure cannot be

neutralized by a simultaneous increase in spinal fluid pressure because of the shielding effect of the spinal column and ligaments. This great disparity between the intravascular and extravascular pressures in the spinal canal then results in rupture of spinal vessels. As our patient jumped into the sea, his abdominal and/or thoracic pressure could have increased to cause the bleeding.

Most lumbar SSH cases manifest as back pain or cauda equina compression.^{2,5,9,11,14,16,17,22)} In many cases, these symptoms are preceded by events such as minor trauma. However, delayed onset was also reported. One case of SSH manifested as lumbago 36 hours after the trauma.¹⁵⁾ This slow onset was probably due to venous origin of the hematoma. SSH following lumbar puncture resulted in lumbago and cauda equina compression 72 hours later.⁹⁾ The postulated origin of the bleeding was radicular vessels associated with the nerve roots. A transient increase in intrathoracic and intraabdominal pressure was involved in the delayed progression of the bleeding. In our patient, lumbago developed a few days after the initial event. This delay can be explained by the speculation that the bleeding originated from the veins and accumulation of a considerable volume in the spinal canal required some time. Alternatively, the lumbago had already developed before the lumbar puncture and the hematoma was present at a different spinal level from the puncture. The lumbar puncture was not considered to be related to the hematoma.

Another symptom of the SSH is, interestingly, meningeal irritation sign, such as headache or neck stiffness. Ten of 58 patients with SSH had headache and stiffness of the neck among the first symptoms.²⁰⁾ Two patients with SSH in the lumbar region, in addition to our patient, presented with headache as an initial symptom.^{8,21)} This headache was considered to originate from the spinal SAH, which coexists with the SSH.

MR imaging is the examination of choice to identify SSH. SSH is often found in a ventral location, but can occur posteriorly, laterally, and sometimes circumferentially.¹⁸⁾ The indications for the surgical intervention depend on distinguishing subdural hematoma from epidural hematoma. Epidural hematoma has a convex shape on sagittal and axial MR imaging, whereas subdural hematoma appears as concave on sagittal and irregular on axial MR imaging.^{8,10)} Subdural hematomas are located within the thecal sac, so are separate from the adjacent extradural fat.¹⁸⁾ In the lumbar region, the arachnoid membrane and subarachnoid components surrounded by subdural hematoma can appear as an inverted "Mercedes star sign."⁵⁾

The characteristic MR imaging findings for SSH are thought to be identical with those for brain hematomas.^{4,8-10,18)} The age of the hematoma is important to identify, because SSH can occur several hours or days later than the first event. In our case, the initial MR imaging was taken 1 week after the initial event. The SSH was isointense to the cord on T₁-weighted and T₂-weighted images. The hematoma was considered to be in the acute stage, namely 1-3 days, which corresponded to the onset of the lumbago. Our patient first presented with headache related to SAH followed by delayed onset of lumbago. This delay may have reflected the slow progression of the lumbosacral subdural hematoma.

SSH is generally regarded as a surgical emergency, and requires surgical decompression with variable neurological recovery. Recent reports have pointed out the possibility of spontaneous resolution of the SSH.^{2,6,7,9,11,13,15,17)} Conservative treatment can be recommended for patients with stable neurological status.

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