

Case Report

Idiopathic spontaneous spinal subarachnoid hemorrhage

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Introduction: Spontaneous spinal subarachnoid hemorrhage (SAH) occurs in less than 1% of all cases of SAH, and idiopathic spontaneous spinal SAH is even more rare.

Methods: A 48-year-old man presented with a sudden onset of low back pain without any neurological deficit. There was no history of either trauma or the use of anticoagulants. Magnetic resonance (MR) imaging of the lumbar spine revealed an intradural linear mass from T12 to L3, ventral to the conus medullaris and cauda equina. The mass was of iso-signal intensity in T2-weighted images, of high signal intensity in T1-weighted images, and was not enhanced following injection of gadolinium. Lumbar tapping revealed bloody cerebrospinal fluid, confirming SAH. At 1 month after the onset of symptom, his pain subsided spontaneously and no vascular abnormality was found by spinal angiography. Follow-up MR imaging confirmed the complete resolution of the SAH.

Conclusion: We report a case of idiopathic spontaneous spinal SAH at the lumbar level in a 48-year-old man.

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Introduction

Spinal subarachnoid hemorrhage (SAH) is rare, and occurs in 0.05–1.5% of all cases of SAH in the literature.^{1–3} Spinal SAHs could be caused by trauma (often caused by lumbar puncture), vascular lesions (arteriovenous malformations, arteriovenous fistula, aneurysm), neoplastic lesions, coagulopathy, periarteritis nodosa, systemic lupus erythematosus, hypertension, coarctation of the aorta, or Behcet's disease.⁴ Spontaneous SAH of unknown pathogenesis was first reported in 1937,⁵ and it remains difficult to rule out the underlying causes without selective spinal angiography, computed tomography (CT), magnetic resonance (MR) imaging, or autopsy. We report upon a case of spontaneous spinal SAH of unknown origin.

Case report

A 48-year-old man experienced discomfort in the sacral area after swimming. A few hours later, a severe plucking pain developed in the S1 sensory dermatome. There was no history of a prior headache, back pain, trauma, or the use of anticoagulant drugs. He visited a

local hospital and received conservative management. Persistent pain, despite conservative care for 4 days, led to an MR imaging study of the lumbar spine, which revealed a linear high signal intensity mass, ventral to the spinal cord, on both T1-weighted images and T2-weighted images from T12 to L3 (Figure 1a,b). No clue suggestive of vascular malformation was evident. At 8 days after the onset of the severe pain, he was referred to our hospital for further evaluation and treatment.

On admission, he showed paresthesia with radiating pain in the S1 sensory dermatome and no weakness. On follow-up MR images, the lesion showed iso-signal intensity on T2-weighted images and high-signal intensity in T1-weighted images (Figure 2). The lesion did not enhance. However, the extent and thickness of the lesion had diminished. An incidental hepatic hemangioma was also detected. Lumbar tapping revealed bloody cerebrospinal fluid. Since he had no neurological deficit except pain, conservative management was chosen. Hematologist and rheumatologists found no evidence of other systemic diseases such as coagulopathy, periarteritis nodosa, systemic lupus erythematosus and Behcet's disease.

A follow-up MR imaging study revealed complete resolution of the SAH 1 month later. No vascular

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Figure 1 Initial MRI. A linear high-signal intensity mass, located in subarachnoid space, was showed from T12 to L3 on T2-weighted (a) and T1-weighted (b) sagittal image. These findings are compatible with spinal SAH



Figure 2 Follow-up MRI. The signal of the lesion changed to iso-signal intensity on the T2-weighted image and to high-signal intensity on the T1-weighted image, and the lesion became thinner, 1 month later

abnormality was detected by selective spinal angiography. He returned to his normal life.

Discussion

We report a case of SAH of unknown origin, located in the thoracolumbar spine, without neurological deficit. To the authors' knowledge, only 11 cases of spinal SAH without any apparent source of bleeding have been reported in the literatures.⁶⁻⁹

Intracranial SAH can cause spinal SAH. However, our patient did not complain of headache, nausea and vomiting without the neck stiffness. Considering the clinical pictures, we decided that intracranial imaging and four-vessel angiography were not necessary. In other reported cases without intracranial symptoms, intracranial imaging was not performed too.^{4,6,9}

Spinal SAH presents clinically as sudden back pain or headache, acute sciatic pain, paraparesis, sensory disturbance or sphincter disturbance. The most frequent causes of spinal SAH include trauma (50% of cases), vascular malformations, and spinal tumor.¹⁰ With the development of safe and accurate spinal angiographic techniques, the incidence of spinal SAH of unknown origin has decreased. The spontaneous occurrence of SAH of unknown origin is extremely rare.⁴ MR imaging

plays an important role in delineating the extent of the hemorrhage and in disclosing the relationship between the hemorrhage and spinal cord.^{4,9,11} Selective spinal angiography is necessary to rule out underlying vascular diseases, even though sometimes a spinal MR imaging study may suggest the presence of vascular abnormality by signal voids.

Our patient was successfully treated without operative decompression, contrary to many reports recommending operative decompression for spinal SAHs.^{4,9–11} Komiyama *et al*⁴ classified spinal SAHs as dorsal or ventral. They postulated that the ventral type usually showed no neurological deficit and ran a benign course, so that surgical decompression may be unnecessary. Our patient had a ventral spinal SAH without neurological deficit.

Komiyama *et al*⁴ explained elegantly the reason why surgical decompression is not necessary in ventral spinal SAH. When bleeding occurs in the spinal subarachnoid space, cerebrospinal fluid could dilute a subarachnoid hematoma. Furthermore, defibrination by pulsation of the spinal cord may reduce the likelihood of the formation of subarachnoid hematoma.

However, the location of the hematoma does not seem to be the only factor along decision. In the literatures, although the hematoma was located in the dorsal and ventral, patients recovered without surgical decompression.^{4,12} Accordingly, if bleeding cannot produce a hematoma in the subarachnoid space, the spinal cord is not compressed by a mass effect. In this situation, no neurological deficit develops. So the decision for surgical decompression in spinal SAH is dependent on neither the location of the hemorrhage nor hematoma, but the neurological status of the patients.

Conclusion

We report a case of idiopathic spontaneous spinal SAH at lumbar level in a 48-year-old man. Surgical decompression of spinal SAH seemed not to be dependent on

the location of the hemorrhage or hematoma, but the neurological status of the patients.

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