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Case Report

Spontaneous spinal subdural hematoma with spontaneous resolution

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Study design: Single patient case report.
Objectives: To present and discuss a case of spontaneous spinal subdural hematoma that was not associated with coagulation abnormality.
Setting: Seoul National University Hospital, Seoul, Korea.
Methods: Clinical and radiological magnetic resonance imaging follow-up of the patient between August 1997 and December 1998.
Results: The hematoma was initially extensive, ranging from T5 to L3. However, it resolved spontaneously with the subsequent development of an adhesive arachnoid cyst.
Conclusion: Even though urgent surgical decompression is necessary in most cases of spinal subdural hematoma, conservative management could be an option in cases with rapid resolution of the neurological deficit.
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Keywords: spinal subdural hematoma; hematoma; spontaneous

Introduction

Spinal subdural hematoma (SSH) is a rare cause of spinal cord compression. It may follow major or minor trauma, spinal surgery, or lumbar puncture.[1] Spontaneous spinal subdural hematoma is even more rare and may occur in patients who have coagulation defects or who are receiving anticoagulants. At least 12 cases of spontaneous SSH with no associated coagulation problems are reported in the literature.[2,3] Spinal cord compression by SSH is known to be a neurosurgical emergency.[4] Although the surgical treatment by decompressive laminectomy and drainage of hematoma is mandatory in most cases of SSH,[2,4] some cases of spontaneous resolution of SSH have been reported:[3,5–7] two of these cases were of spontaneous onset.[3,6] We report another case of SSH of spontaneous onset and spontaneous resolution without coagulation problems.

Case report

A 49-year-old woman presented with a sudden severe mid-dorsal pain extending to suboccipital region and with paraparesis. The patient had no previous history of trauma or bleeding diathesis. Neurological examination revealed motor strengths of grade 4+/5 in both lower extremities, hypesthesia below the T7 dermatome and neck stiffness. Deep tendon reflexes remained within normal limits, and no pathologic reflexes were present. She complained of difficulty in initiating voiding, pain and paraparesis. The brain computed tomography was normal. Lumbar puncture was performed to rule out the possibility of subarachnoid hemorrhage, and revealed a bloody cerebrospinal fluid. Cerebral angiography and aortography disclosed no abnormalities. Magnetic resonance (MR) imaging 10 days after the back pain began, revealed a ventral subdural hematoma extending from the level of T5 down to L3 with compression of the spinal cord (Figure 1). The lesion was hyperintense on both the T1-weighted and T2-weighted images.

The patient was transferred to the Department of Neurosurgery at Seoul National University Hospital for further evaluation and management. At this time neurological examination revealed meningeal irritation signs only, without paraparesis or hypesthesia. Routine laboratory analyses, including complete blood counts and coagulation screen, were normal. Since the recovery from the previous neurological deficits was complete, conservative management was chosen over the surgical evacuation of the hematoma, despite the extensiveness of the lesion.

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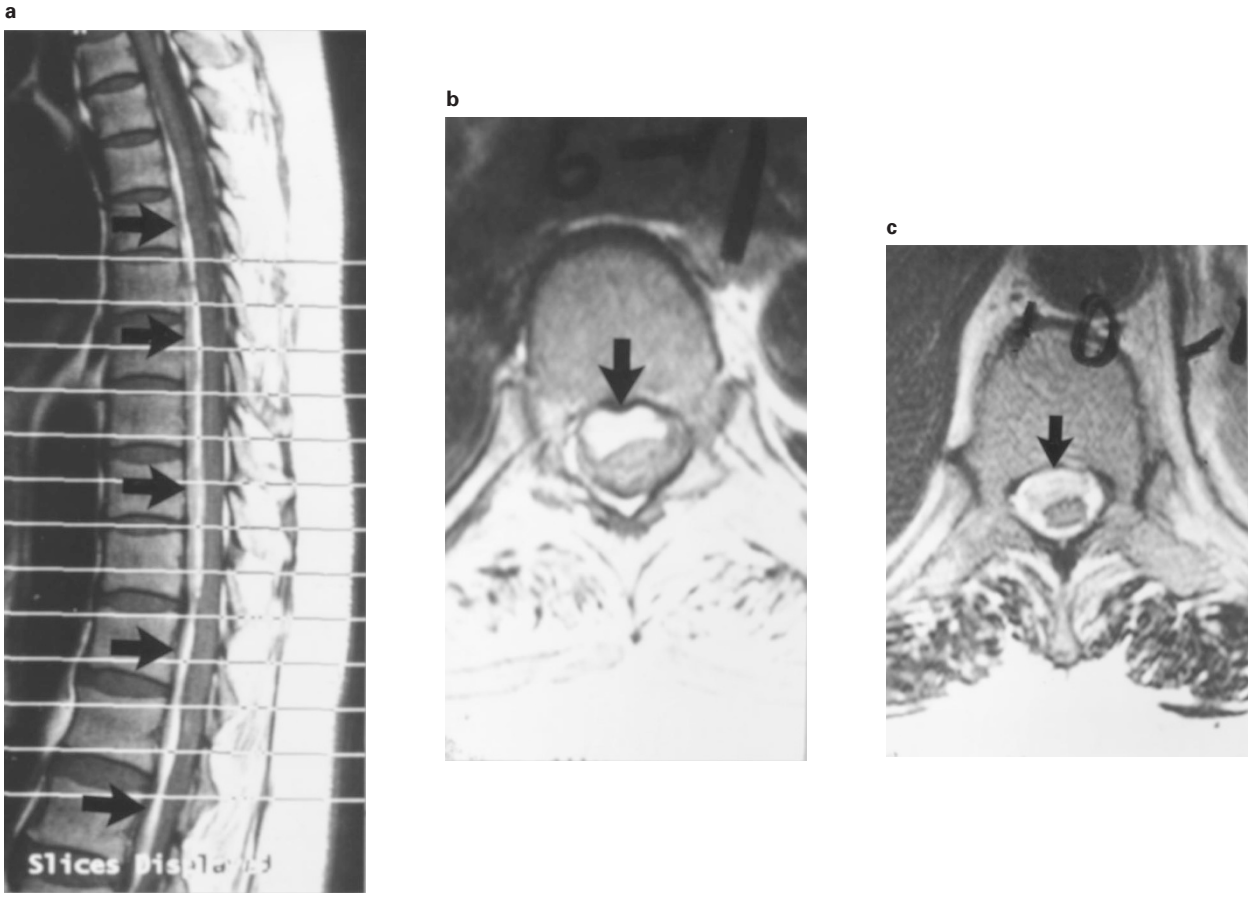

Figure 1 **(a)** MR imaging taken 10 days after the onset of symptoms shows subdural hematoma extending from T5 to L3 (arrows). (**b**,**c**) It is revealed as high signal intensity (arrow) on T1-(**b**) and T2-(**c**) weighted axial images, displacing the thoracic cord posteriorly at the level of T6-7 and T10-11

Magnetic resonance imaging taken 6 weeks after the onset of symptoms revealed a residual hematoma at the level of T7 to T9 (Figure 2) and after 4 months, showed a complete resolution of the hematoma, though an adhesive arachnoid cyst was noted at the previous hematoma site (Figure 3). Magnetic resonance imaging taken after 16 months showed no change of arachnoid cyst (not shown). The neurological condition of the patient remained stable, with decreased back pain.

Discussion

The etiology of spontaneous SSH without coagulopathy is still obscure. The spinal subdural space is a potential avascular space: thus, the bleeding might have come from an extradural or subarachnoid source. The latter seems more probable since arachnoid is more brittle and penetrable than dura mater. Also, more cases of combined subarachnoid hemorrhage (SAH) and subdural hematoma (SDH) have been reported,[6,8] and only one case of combined spontaneous spinal epidural hematoma and SDH[9] has been reported.

The theory of 'isolated SDH' seems to be an acceptable explanation [1,10] for this case. According to this theory, our case presented with a transitional state of combined SAH (proved by lumbar puncture) and SDH, a state between pure SAH and pure SDH. In 1955 Rader[11] proposed a possible mechanism of spontaneous SAH: 'a forgotten effort or minor trauma increases both the intrathoracic pressure and the intraluminal pressure of the vessels traversing the subarachnoid space. When the cerebrospinal fluid pressure momentarily lags behind the intravascular pressure, the vessel ruptures resulting in SAH.' Through the transitional state, isolated SDH might remain after a more rapid resolution of SAH. Mavroudakis *et al*[8] thought the direction of the movement of hematoma might be reversed, namely from subdural to subarachnoid space, and suggested this as the mechanism of the spontaneous regression of SSH in their case. Although the exact mechanism of the development and resolution of spontaneous SDH is unknown, our case is unusual in that the hematoma was extensive and an adhesive arachnoid cyst developed at the expense of resolving the hematoma. Similarly Langmyre *et al*[4] reported a

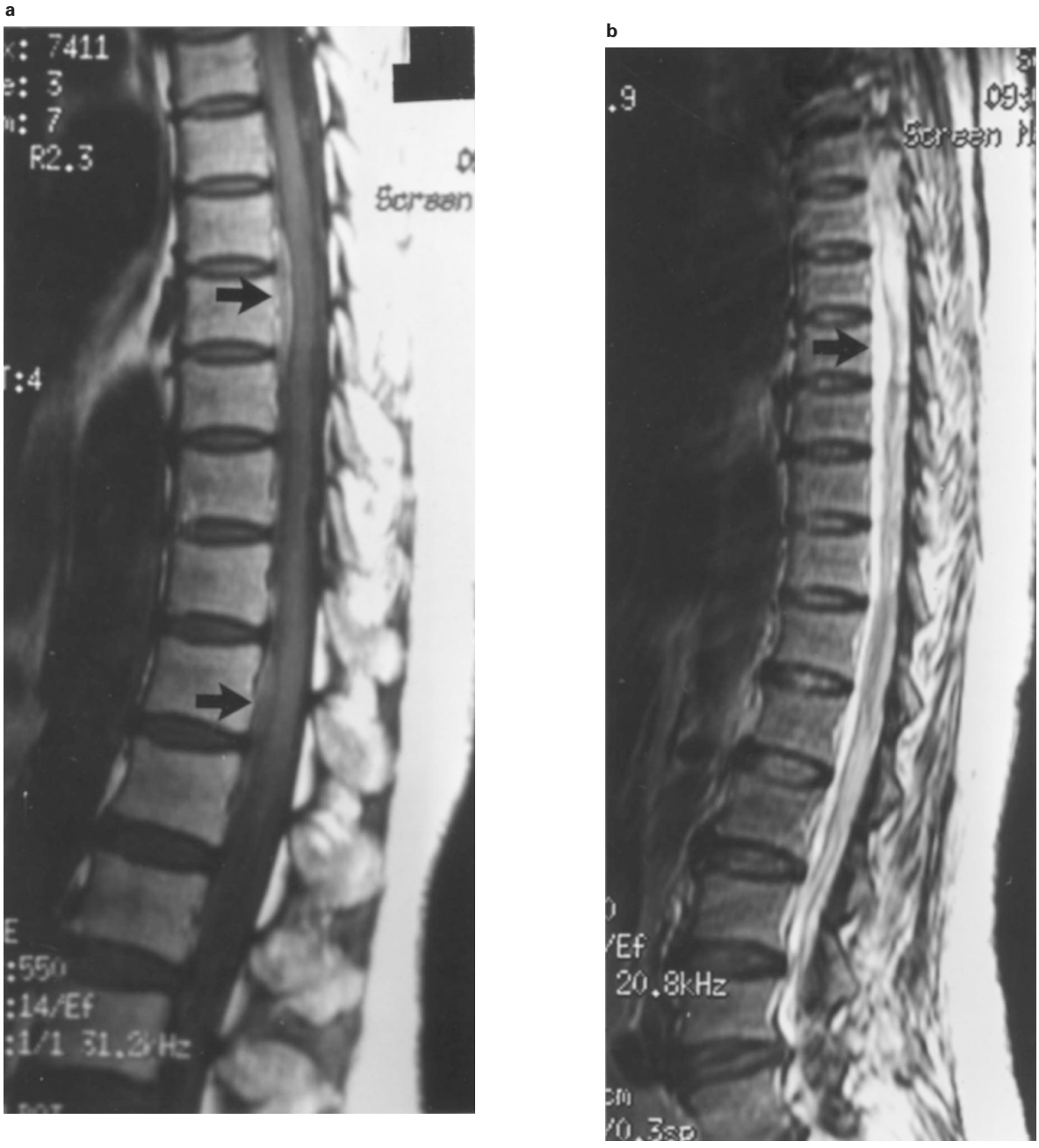

Figure 2 **(a)** Most of the hematoma resolved 6 weeks after the onset of symptoms. Some residual hematoma is shown in T7–T9 level and T12–L1 (arrows). (**b**) It is revealed as high signal intensity on T2-weighted image (arrow). On T1 weighted image the signal density of the central portion of the hematoma was decreased, with the peripheral rim of high signal intensity

patient with chronic SDH who developed arachnoid adhesions.

According to the theory of 'isolated SDH', the signal intensities of SDH combined with SAH are likely to correspond to acute to subacute hemorrhage, and those of isolated SDH, to subacute to chronic hematoma with MR imaging. The patient in our case showed the lesion, which was hyperintense on both the T1-weighted and T2-weighted MR images taken 10 days after onset of the symptoms. This feature corresponds to the subacute stage of hematoma and combination with SAH seems to be feasible.

There are three treatment options for SSH: (1) conservative management, (2) percutaneous drainage, or (3) surgery. In cases showing acute deterioration and severe neurological deficit, emergency surgical decompression is the best option. Conservative management can be an option in cases with mild

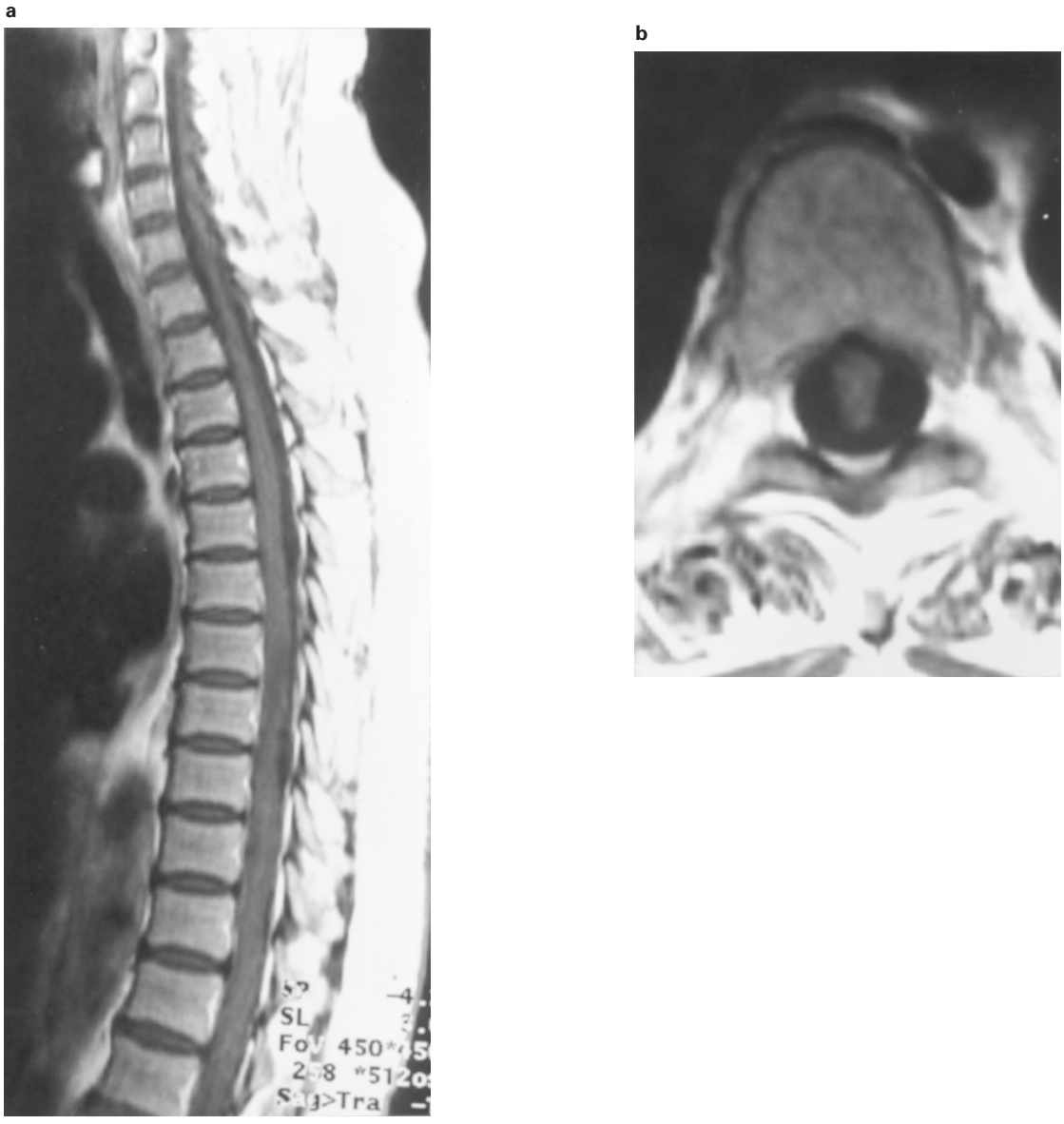

Figure 3 **(a)** The hematoma is completely resorbed 4 months later. (**b**) The anteroposterior length of the spinal cord is longer than transverse width, due to the bilateral leptomeningeal cyst, between T7 and T10. There was no enhancing portion in the cyst (not shown)

neurological deficits and progressive improvement in the early period. In some patients with stable deficit, with dorsally located hematoma of moderate extent in lumbosacral area, and with no bleeding diathesis, percutaneous drainage may be the option.[12] In our case conservative management was chosen since the patient's recovery from the previous neurological deficits was complete without any deterioration. Although the complete resolution of hematoma took rather a long time due to its extensiveness in the beginning and the adhesive arachnoid cyst was seen on the follow-up MR imaging, conservative therapy was successful in our case. Further follow-up is necessary since the remained adhesive arachnoid cyst might become symptomatic and may require an additional treatment.[4]

Conclusion

Even though emergent surgical decompression is necessary in most cases of spinal subdural hematoma, conservative management could be an option in cases with rapid resolution of the neurological deficits.

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