

Case Report

Spinal Subdural Hematoma Associated with Intracranial Subdural Hematoma

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The simultaneous occurrence of an intracranial and a spinal subdural hematoma (SDH) is rare. We describe a case of cranial SDH with a simultaneous spinal SDH. An 82-year-old woman visited the emergency room because of drowsiness and not being able to walk 6 weeks after falling down. A neurological examination showed a drowsy mentality. Brain computed tomography showed bilateral chronic SDH with an acute component. The patient underwent an emergency burr-hole trephination and hematoma removal. She exhibited good recovery after the operation. On the fourth postoperative day, she complained of low-back pain radiating to both lower limbs, and subjective weakness of the lower limbs. Spine magnetic resonance imaging revealed a thoracolumbosacral SDH. A follow-up spinal magnetic resonance imaging study that was performed 16 days later showed a significant decrease in the size of the spinal SDH. We discuss the pathogenesis of this simultaneous occurrence of spinal and cranial SDH.

Key Words : Brain · Spine · Trauma · Subdural hematoma.

INTRODUCTION

Spinal subdural hematoma (SDH) is usually attributed to trauma, blood dyscrasia, anticoagulation, spinal puncture, cranial surgery, and vascular malformation⁷. More than 100 cases of spinal SDH have been reported, and magnetic resonance imaging (MRI) has increased the incidence of detection¹⁹. However, the simultaneous occurrence of an intracranial and a spinal SDH is rare^{5,7}. In addition, the etiology of the simultaneous occurrence of a cranial and a spinal SDH is unclear.

We describe a case of cranial SDH with a simultaneous spinal SDH, and discuss the pathogenesis of this condition.

CASE REPORT

An 82-year-old woman slipped and fell on her back and occiput. She was unconscious for a few seconds and complained of a moderate headache that resolved after 3 days. She had bruises in the scalp and back areas. Four weeks later, the patient had a falling episode and complained of a tingling sensation in her right leg and lumbago. The patient underwent medical treatment for pain. Fifteen days after the second trauma, she was unable to walk and was referred to our hospital for assessment.

She had a history of alprazolam (Pfizer Pharmaceuticals Korea, Seoul, Korea), trazodone (Myung In Pharm, Seoul, Korea), and venlafaxine medication for depression, but no history of anticoagulant or antiplatelet medication. And she had no history of blood dyscrasia and coagulopathy. Physical examination demonstrated an ill-looking appearance. Neurological examination showed a drowsy mentality; however, lateralizing signs were not present. Laboratory data regarding coagulation were normal. Her activated partial thromboplastin time was 33 s and her prothrombin time was 12.7 s (international normalized ratio, 0.99). Brain computed tomography showed bilateral chronic

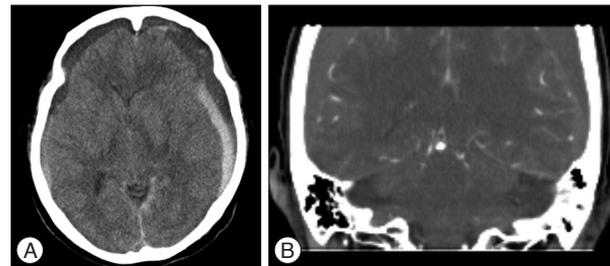


Fig. 1. Brain computed tomography demonstrates bilateral chronic subdural hematomas with an acute component located in both frontotemporo-parietal areas. No hematoma was found in the posterior fossa or tentorium. A : Axial image. B : Coronal image.

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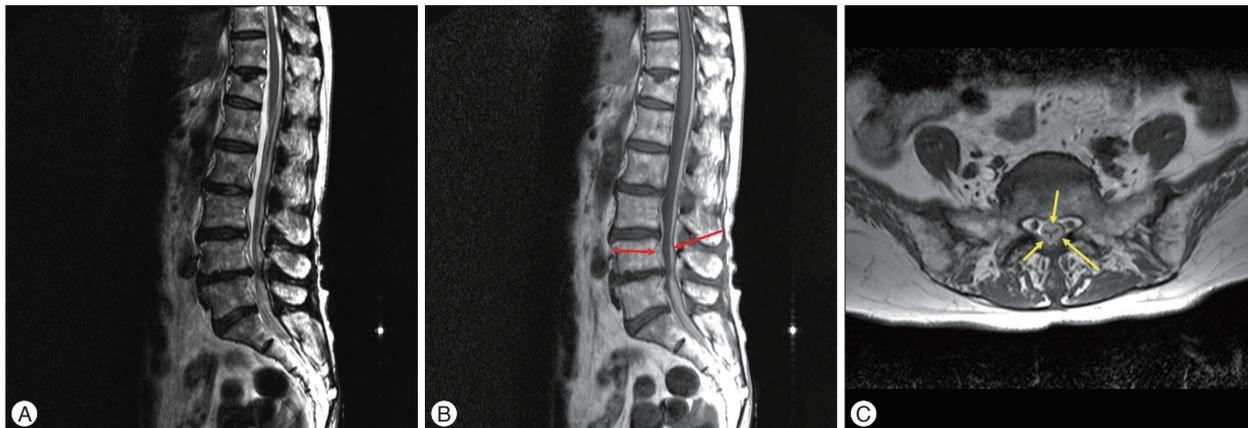


Fig. 2. Magnetic resonance images obtained on the fourth postoperative day showing a spinal subdural hematoma. T2- and T1-weighted images show the high signal intensity of the lesion that was typical of extracellular methemoglobin, which is suggestive of late subacute or early chronic hematoma. The hematoma is separated from the posterior epidural fat by the dura mater and is located in the ventral and dorsal subdural space (red arrow). Moreover, this hematoma determines the mass effect on the nerve root of the cauda equina, leading to a “3-branch star” appearance (yellow arrow). A : T2-weighted sagittal image. B : T1-weighted sagittal image. C : T2-weighted axial image.



Fig. 3. T1-weighted magnetic resonance imaging performed 20 days postoperatively shows a decreased spinal subdural hematoma.

SDH with an acute component (Fig. 1). The patient underwent an emergency burr-hole trephination and hematoma removal under general anesthesia. The postoperative course was uneventful, and the patient showed an improved mentality. She had no history of lumbar puncture at perioperative period. On the fourth postoperative day, she exhibited a clear mentality; however, she complained of low-back pain radiating to both lower limbs and had subjective weakness of the lower limbs. Thoracic and lumbar spine X-ray showed no fracture. Spine MRI revealed a thoracolumbosacral SDH (Fig. 2). Because the spinal SDH remained well tolerated by the patient, with progressive resolution of the pain, conservative treatment was chosen. A follow-up spinal MRI study that was performed 16 days later showed a significant decrease in the size of the spinal SDH and in the associated mass effect (Fig. 3). The patient was dis-

charged the following day without any neurological deficit.

DISCUSSION

SDH of the lumbar spine is rare, but has been described in patients who have undergone lumbar puncture, spinal or epidural anesthesia, and other invasive procedures¹⁵⁻¹⁷. It has also been reported as a spontaneous occurrence in patients treated with anticoagulation or antiaggregation therapy^{6,10}. There are reports of SDH in the lumbar spine after blunt trauma to this area³ as well as reports of delayed spinal SDH¹³.

The etiology of the simultaneous occurrence of cranial and spinal SDH has not been elucidated. There are two suggested mechanisms underlying the simultaneous occurrence of cranial and spinal SDH : one is migration from the cranial to the spinal compartment, and the other is a traumatic spinal SDH.

Intracranial migration of an acute SDH has been documented in some cases^{4,18}. An acute SDH showed gradual sedimentation on the cerebellar tentorial surface and skull base. A spinal SDH with an acute cranial SDH could result from this phenomenon. However, whether this mechanism is also applicable to chronic cranial hematoma remains unclear. A chronic SDH has outer and inner membranes, in contrast to an acute SDH. The contents of a chronic SDH are unlikely to move freely in the subdural space¹⁹; however, the MRI included in the report by Morishige et al.¹¹ may support this theory. These hematomas were sequenced from the cranial to the sacral level and were thought to have the same origin.

Yamaguchi et al.¹⁹ reported that the signal intensity and the changes in the spinal hematoma were similar to those of the cranial lesion, suggesting that both hematomas had the same origin. A thin hematoma was present in the retrocerebellar space, suggesting a migrating hematoma in the posterior fossa¹⁹. Enlargement of a hematoma caused by rebleeding in a chronic cranial SDH might rupture the membranes, leading to

the redistribution of the hematoma to the spine, most likely as a result of gravity.

Moscovici et al.¹²⁾ reported the case of a rare symptomatic spinal SDH leading to cauda equina syndrome that was diagnosed 3 days after complete resolution of a cerebral SDH in an elderly patient who sustained mild head trauma with no evidence of spinal injury. Those authors¹²⁾ proposed that the penetration of cerebrospinal fluid into the subdural space after a trauma-induced arachnoid tear may facilitate SDH migration via a dilutional and “water hammer”-like effect.

In our case, we found no hematoma in the posterior fossa or tentorium (Fig. 1). However, this finding does not exclude the possibility of hematoma migration from the cranial to the spinal compartment.

A second potential mechanism to explain the spinal SDH observed in this patient is a trauma-induced effect. The pathogenesis of traumatic spinal SDH remains unclear. The only vessels of substantial size are the radiculomedullary artery and its corresponding vein, which pierce the dural sac, often above the L3 nerve root. Rader¹⁴⁾ postulated that a sudden increase in abdominal and thoracic pressure could raise the pressure in the spinal vessels as they cross the subdural and subarachnoid spaces. If the pressure of the cerebrospinal fluid cannot immediately neutralize this force, rupture of the vessels could ensue¹⁴⁾. Another theory is that the spinal SDH originates within the subarachnoid space and subsequently dissects into the subdural space¹⁹⁾. To date, neither of these theories have been proven. However, it should be remembered that minor trauma may result in a spinal SDH, although clinical presentation might be delayed²⁾.

Chen et al.²⁾ reported the delayed onset of a spinal SDH. This patient had an acute intracranial epidural hematoma and recovered well after a craniotomy, but presented with cauda equina syndrome because of spinal SDH 2 weeks after the accident. This patient showed no intracranial SDH. We believe that the spinal SDH reported by Chen et al. developed as a result of trauma.

Although the prevalence of spinal SDH associated with a chronic cranial SDH is uncertain, many previous cases exhibited minor clinical signs and were consequently missed without confirmative imaging diagnosis. Currently, MRI is the main investigative tool, and allows the prompt diagnosis of spinal SDH; hence, the recognition of spinal SDH will probably increase in the near future because of the extensive use of MRI⁶⁾.

In our case, we identified paraspinal muscle trauma, but did not know the spine status after the first and second trauma, especially regarding the presence of spinal subdural hematoma. Therefore we cannot confirm traumatic origin of spinal SDH in our case.

We believe that two possible mechanisms of spinal SDH formation underlay the simultaneous occurrence of cranial and spinal SDHs in our case. One mechanism is the migration of a cranial SDH to the spinal subdural space. The other is trauma

to the lumbosacral area resulting in a delayed spinal SDH. In this specific case, we were not able to identify the precise mechanism that underlies the simultaneous occurrence of cranial and spinal SDH.

CONCLUSION

We propose that delayed simultaneous spinal and cranial SDH may develop after head and back injury. Patients treated for a traumatic cranial SDH who develop late-onset neurological deterioration attributable to any region of the spine should be evaluated for spinal SDH.

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