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Case Report

Acute Cervical Spinal Subdural Hematoma Not Related to Head Injury

Hee Yul Kim, M.D., Chang Il Ju, M.D., Seok Won Kim, M.D.
Department of Neurosurgery, College of Medicine, Chosun University, Gwangju, Korea

We report an extremely rare case of traumatic cervical spinal subdural hematoma not related to intracranial injury. There has been no report on traumatic cervical spinal subdrual hematoma not related to intracranial injury. A 27-year-old female patient was admitted to our emergency room due to severe neck pain and right arm motor weakness after car collision. On admission, she presented with complete monoplegia and hypoesthesia of right arm. Magnetic resonance imaging (MRI) revealed subdural hematoma compressing spinal cord. Lumbar cerebrospinal fluid (CSF) analysis revealed 210,000 red blood cells/mm^3. She was managed conservatively by administrations of steroid pulse therapy and CSF drainage. Her muscle power of right arm improved to a Grade III 16 days after admission. Follow-up MRI taken 16th days after admission revealed almost complete resolution of the hematoma. Here, the authors report a traumatic cervical spinal SDH not associated with intracranial injury.

KEY WORDS : Spinal subdural hematoma · Cervical spine · Monoplegia.

INTRODUCTION

Traumatic spinal subdrual hematoma (SDH) is a rare clinical entity and it has known to be related to intracranial injury[1]. Moreover, most of them are thoracic or lumbar SDH and cervical spinal SDH is extremely rare[10]. Padres et al. reported the only case of cervical SDH, but it was associated with subarachnoid and intraventricular hemorrhage. Here, we report a rare case of traumatic cervical SDH, not related to brain injury.

CASE REPORT

A 27-year-old female patient with severe neck pain and motor weakness after a car stroke was referred to emergency room. As her left shoulder struck the ground, she had left clavicle fracture. There was no previous history of anticoagulant therapy or hematological coagulopathy, and she had been in good health. On physical examination, she was alert and fully oriented. No neurologic abnormalities were found in the cranial nerves and cerebellar system. However, sensory testing of right arm demonstrated loss of touch, pin-prick and vibration sense. Motor examination revealed complete right upper extremity monoplegia (Grade 0) with normal motor functions in remaining extremities. Brain computed tomography (CT) scans didn't reveal any abnormal findings. Her coagulation profiles including platelet count, prothrombin time, and partial thromboplastin time were within normal range. Cervical plain X-ray films and CT scans showed no abnormality. Immediate magnetic resonance imaging (MRI) of the cervical spine revealed a well-defined mass in the right ventrolateral aspect, C1 to C5 levels. The T1-weighted MR images showed the mass as a homogeneous isointensity lesion, and the T2-weighted MR images as a mixed signal intensity lesion. There was no signal void area, suggesting no abnormal vessels in the mass (Fig. 1). Based on these MR findings, an acute cervical spinal SDH at the right side of ventral C2-C6 level was diagnosed. For differential diagnosis from other conditions such as cervical epidural hematoma, lumbar spinal puncture was performed (Fig. 2). Lumbar cerebrospinal fluid (CSF) analysis revealed 210,000 red blood cells/mm^3 and 367 white blood cells/mm^3. With the diagnosis of subdural hematoma, intravenous fluid and steroid was started with 1

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• Address for reprints : Seok Won Kim, M.D.
Department of Neurosurgery, School of Medicine, Chosun University,
588 Seoseok-dong, Dong-gu, Gwangju 501-717, Korea
Tel : +82-62-220-3126, Fax : +82-62-227-4575
E-mail : ns64902@hanmail.net

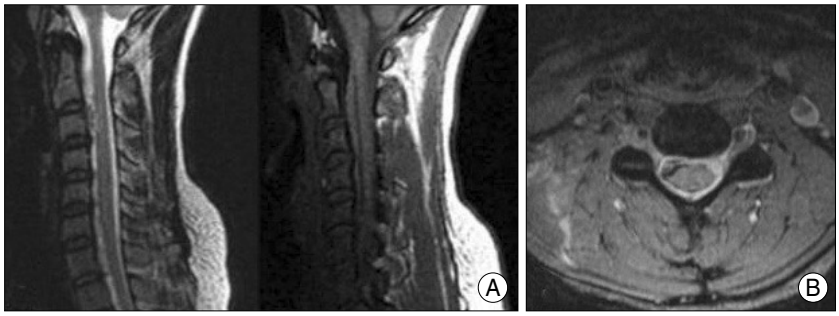

Fig. 1. Magnetic resonance imaging of cervical spine. A : T1 & T2 weighted sagittal image shows a long segment of iso signal intensity lesion form C1 to C5. B : T2 weighted axial image reveals the lesion compressing the spinal cord in the right ventral side.

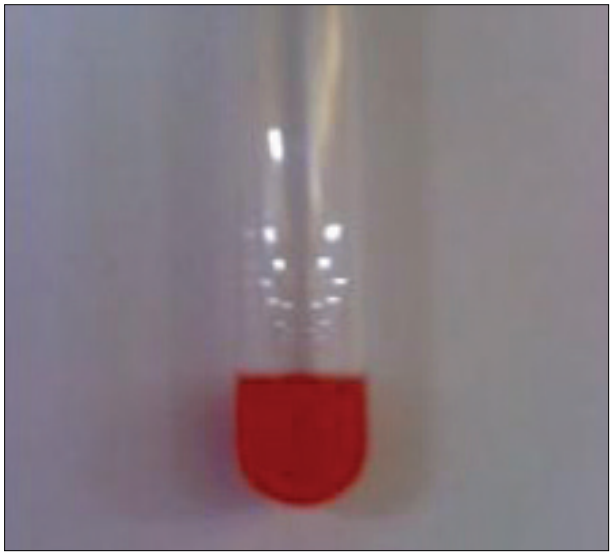

Fig. 2. Hemorrhagic cerebrospinal fluid collected in a test tube.

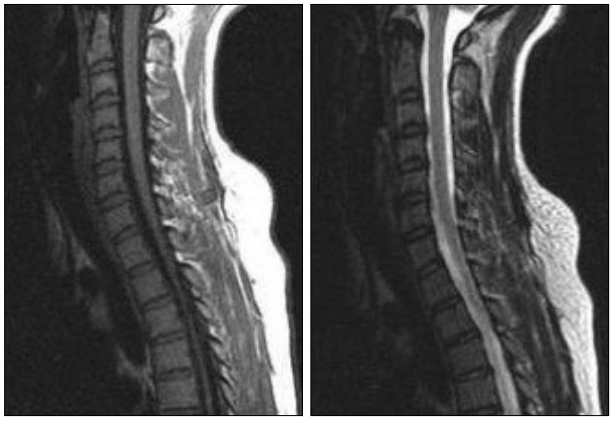

Fig. 3. Follow-up MRI on 6th day posttrauma show complete resolution of the hematoma.

g of methylprednisolone for 3 days per course plus the intravenous administration of mannitol in an attempt to reduce the intramedullary edema. Immediately after the completion of the first course, an improvement in the range of motion (ROM) was recognized and therefore the second course was consecutively administered with a 3-day interval. MR images on the 16th day admission revealed that the spinal SDH had resolved almost completely (Fig. 3). Examination of the CSF acquired from repeated lumbar puncture showed only 5 red blood cells/mm^3. The sensory disturbances gradually improved and complete recovery was confirmed in about 3 weeks. By 16 days after admission, her muscle power of right arm recovered to Grade III and she was referred to Rehabilitation Department for her rehabilitation.

DISCUSSION

Spinal SDH is a very rare disease entity that is usually associated with intracranial injury or underlying hematological disorders[12]. Although there is no evidence of any potential cause and the hematoma is regarded as being spontaneous[7], the majority of author have reported that hemorrhagical disorder, anticoagulant therapy, lumbar puncture or traumatic brain injury predispose spinal SDH[1,3,5]. Traumatic spinal SDH is extremely rare, and so far, only 13 cases have been described in English literature. Among them, we found out that, if we exclude three patients before the era of CT scan, 6 out of the 10 cases also had a concurrent intracranial hemorrhage, whereas the remain 4 did not (Table 1)[4,6,8,9]. Hung et al.[5] hypothesized that a rise of intracranial pressure may also increase shearing force between spinal subdural and subarachnoid spaces so that the inner dura may tear and bleed. However, this cannot explain the exact mechanism of traumatic SDH not accompanying brain injury. Rader proposed that sudden and sharp increases in abdominal and thoracic pressures elevate intravascular pressure in the spinal subdrual space, to the extent that a momentary disparity between intravascular and extravascular pressures could result in the tear of a spinal vessels[11]. Unlike the epidural space and intracranial anatomy of the dura, the spinal subdural space does not contain major blood vessels or bridging veins, rather the vasculature involves only a delicate network of vessels along the lateral margins of the dura. Moreover, relative larger and more vascular epidural fatty tissues are present and the spinal dura is not adherent to the bone. So, spinal epidural hematoma occurs more frequently than spinal SDH.

Spinal SDH can rapidly compromise the spinal cord or cauda equina, which result in paralysis or sensory loss in areas associated with dermatomes. Therefore, prompt diag-

Table 1. Demographical and clinical characteristics of previously reported traumatic spinal subdural hematoma before the era of CT scan

Case (Year)	Sex/Age	Level	Head injury	Surgery	Outcome
Paredes (1981)	M/9	Cervical	O	O	Quadriplegic
Juvone (1994)	M/63	Thoracic	X	X	Good
Shimada (1996)	M/68	Lumbar	O	O	Good
Lee (1996)	M/15	Lumbosacral	O	X	Good
Chen (2001)	M/31	Lumbar	O	X	Good
Hung (2002)	M/12	Lumbosacral	O	X	Good
Bortolotti (2004)	F/23	Lumbosacral	O	O	Good
Morris (2004)	F/26	Lumbosacral	X	X	Good
Mashiko (2006)	M/18	Lumbosacral	X	X	Good
Cho (2009)	M/43	Lumbosacral	X	X	Good

nosis and identification of the extent of the hematoma are essential. MRI is considered the best imaging study for the diagnosis and follow-up of patients with spinal SDH[2]. However, differentiation of epidural and subdural spinal hematoma may be difficult in some cases. Epidural hematoma appears as a lentiform, biconvex, and clearly outlined mass with tapering superior and inferior margins on sagittal MR images. The dura mater can occasionally be seen as a curvilineal low signal structure separating the hematoma from the spinal cord. In the axial scan, the subdural location is clear when the hematoma extends within the dura mater. Since spinal epidural hematoma spreads widely in the epidural space, the shape of the dura mater is often irregular. A subdrual hematoma has a semicircular appearance on axial scans, whereas an epidural hematoma tend to be more convex. Subdural hematoma may also show a convex appearance, but usually has more crescenic appearance on axial images[1,3]. In our case, despite these differential findings, the differentiation between a subdural and epidural hematoma was difficult. Therefore, lumbar tapping was helpful to prove the exact location of hematoma. It is widely accepted that prompt surgical evacuation should be performed before permanent irreversible damage to the spinal cord occurs when severe neurological symptoms or clinical worsening are observed. In our case, however, surgical treatment was not initially considered due to the risk of iatrogenic spinal cord injury because the subdural hematoma was on the ventrolateral side of the cervical spinal cord.

CONCLUSION

Until now, traumatic cervical SDH not related to an intracranial injury has not been reported. The present case is unique for its cervical location and lumbar tapping as a useful diagnostic tool to diagnose spinal SDH.

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