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Spinal Chronic Subdural Hematoma Cured by Lumbar Drainage: A Case Report and Literature Review

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Abstract

Chronic spinal subdural hematoma is an extremely rare condition. We recently encountered a case of symptomatic thoracolumbar chronic spinal subdural hematoma in an older patient caused by a fall. The patient was a man in his 80s with a history of cerebral infarction, who was receiving oral antiplatelet therapy. He was hospitalized for conservative treatment for a brain contusion and mild acute subdural hematoma, and was discharged home after 6 days. However, 9 days after the injury, the patient developed back pain, weakness in both lower limbs, and urinary incontinence and was brought to our hospital. A computed tomography scan on admission displayed a high-density area in the thoracolumbar spinal canal, and magnetic resonance imaging 2 weeks after the fall displayed a spinal subdural hematoma from 8th thoracic to sacral 2nd, with a hyperintensity signal on T1weighted image and T2 weighted image and partial low intensity on T2* imaging. On day 22 after the injury, lumbar drainage was performed, and a motor-oil-like hematoma was aspirated. A total of 330 mL of hematoma content was drained for 3 days. Immediately after treatment, the patient's back pain and lower limb weakness improved, and imaging confirmed the disappearance of the spinal subdural hematoma. Most reported cases to date of chronic spinal subdural hematoma were treated with invasive laminectomy for hematoma removal. In the present case, the authors suspected this condition from the late subacute stage of onset and were able to cure the patient with minimum invasive lumbar drainage after diagnosis of liquefaction of the hematoma by magnetic resonance imaging.

Keywords: neurotrauma in older patients, spinal chronic subdural hematoma, minimally invasive treatment, MRI, spinal drainage

Introduction

With the aging of society, there has been a marked increase in the number of cases of trauma in older adults, which are mainly falls. Typical examples of trauma caused by falls in older adults owing to frail conditions include subdural hematoma (SDH) caused by head trauma, femoral neck fractures, and spinal compression fractures caused by falling on one's buttocks. When such injuries occur, they should be examined and treated by a specialist at each site of trauma, however, it is important to note that these traumatic conditions may occur alone or in combination.

We recently encountered an older male patient who was diagnosed with a cerebral contusion and thin SDH at his previous hospital and who presented with paraparesis after being transferred to our hospital. Spinal magnetic resonance imaging (MRI) was performed, and findings indicative of a SDH in the thoracolumbar region were observed.

In this paper, we report a case of a patient who was diagnosed and treated for spinal SDH together with a review of the literature.

Case Report

A man in his 80s fell while walking and bruised his

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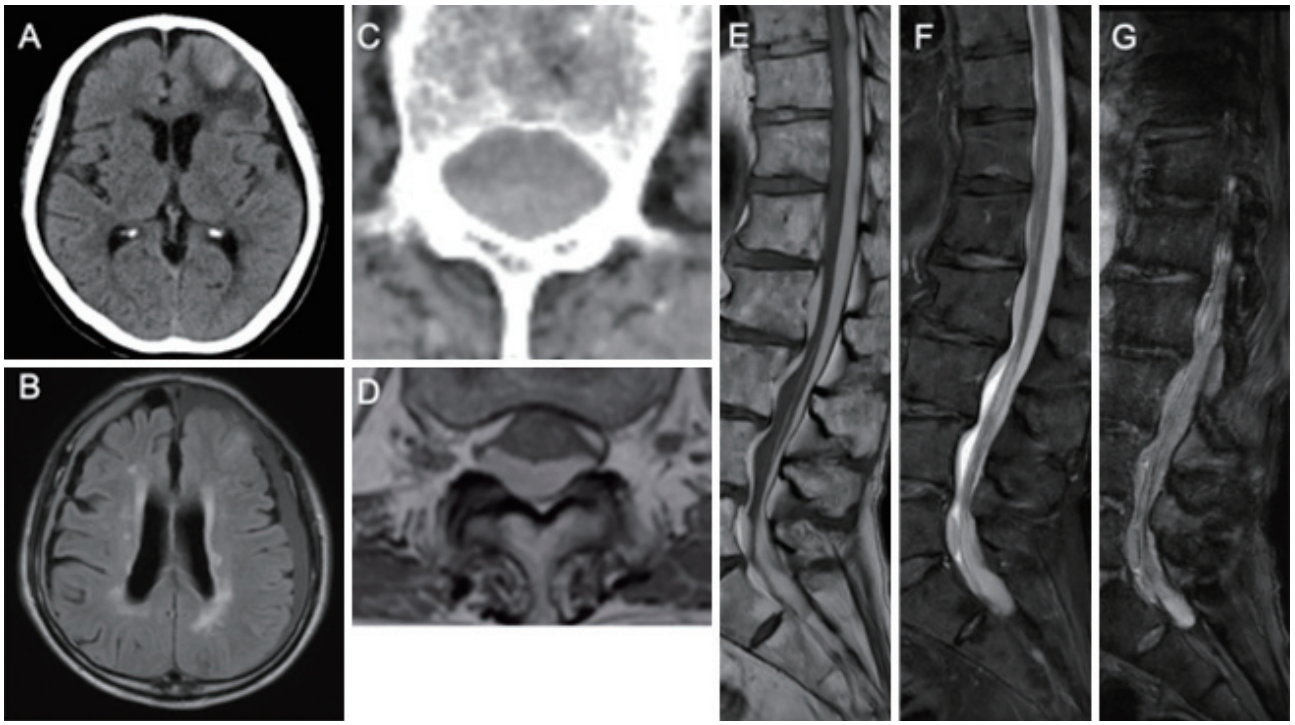

Fig. 1 Pretreatment neuroradiological findings.
A) A head CT scan of the patient on admission displayed a recovery process from a brain contusion in the left frontal lobe and a tendency toward enlargement of the bilateral subarachnoid spaces.
B) Head MRI FLAIR image on admission displayed a thin bilateral chronic subdural hematoma with left-side predominance.
C) A chest CT scan on admission displayed a hematoma on the dorsal spinal cord within the thoracic spinal canal.
D) Thoracic spinal MRI horizontal section T1WI displayed a subdural hematoma with hyperintensity that compressed the spinal cord anteriorly.
E-G) Sagittal MRI image from the lower thoracic spine to the lumbar spine. (E: T1WI, F: T2WI, G: T2* image)
A subdural hematoma extending from Th8 to S2, primarily posterior to the spinal cord, with some extension into the anterior aspect. Both T1WI and T2WI displayed hyperintensity, and a hemosiderin signal was observed on the T2* image.
CT: computed tomography: MRI: magnetic resonance imaging: T1WI: T1-weighted imaging: T2WI: T2-weighted imaging

head. He visited the neurosurgery department of a university hospital and was admitted and treated conservatively with a diagnosis of cerebral contusion, traumatic subarachnoid hemorrhage and thin acute SDH in the frontal lobe. He had a medical history of cerebral thrombosis diagnosed 3 years previously, and was treated with cilostazol. He was a very active individual and took walks of about 10 km as his normal daily routine. Conservative therapy was successful, and the patient was discharged home after 6 days of hospitalization. However, from the 9th day after the injury, he gradually developed back pain, numbness and weakness in both lower limbs, and also urinary incontinence. After being examined at his previous hospital, he was judged to be experiencing the effects of disuse owing to hospitalization, and he was referred to our hospital for treatment.

The next day, when he visited our hospital, he had difficulty walking owing to significant muscle weakness in both lower limbs and complained of urinary incontinence. The right lower extremities were manual muscle test (MMT) score 2, and the left lower extremities were MMT 3, with more than 6/10ths of sensory insensitivity in areas below the Th10 level. His Japanese Orthopedic Association (JOA) score[1)] was 3 out of 29, which included, back pain:1, leg pain:1, walking ability:1, straight leg raising:1, sensory: 0, muscle strength: 0, activities of daily living: 5, and bladder function: -6.

A head computed tomography (CT) scan performed on admission displayed signs of cerebral contusion in the left frontal lobe and thin subarachnoid space enlargement in the bilateral frontal lobes, but treatment was considered to be unnecessary (Fig. 1A and B). In addition, a chest CT scan routinely performed on admission displayed a slight high-density area on the posterior aspect of the spinal cord within the spinal canal (Fig. 1C). Thoracolumbar spine MRI performed 15 days after the head trauma displayed intradural signal abnormalities from the Th 8 to S 2 level in the ventral and dorsal spinal cord and cauda equina nerves. MRI displayed a slightly high signal on T1-weighted imaging (T1WI), a high signal on T2WI, and a

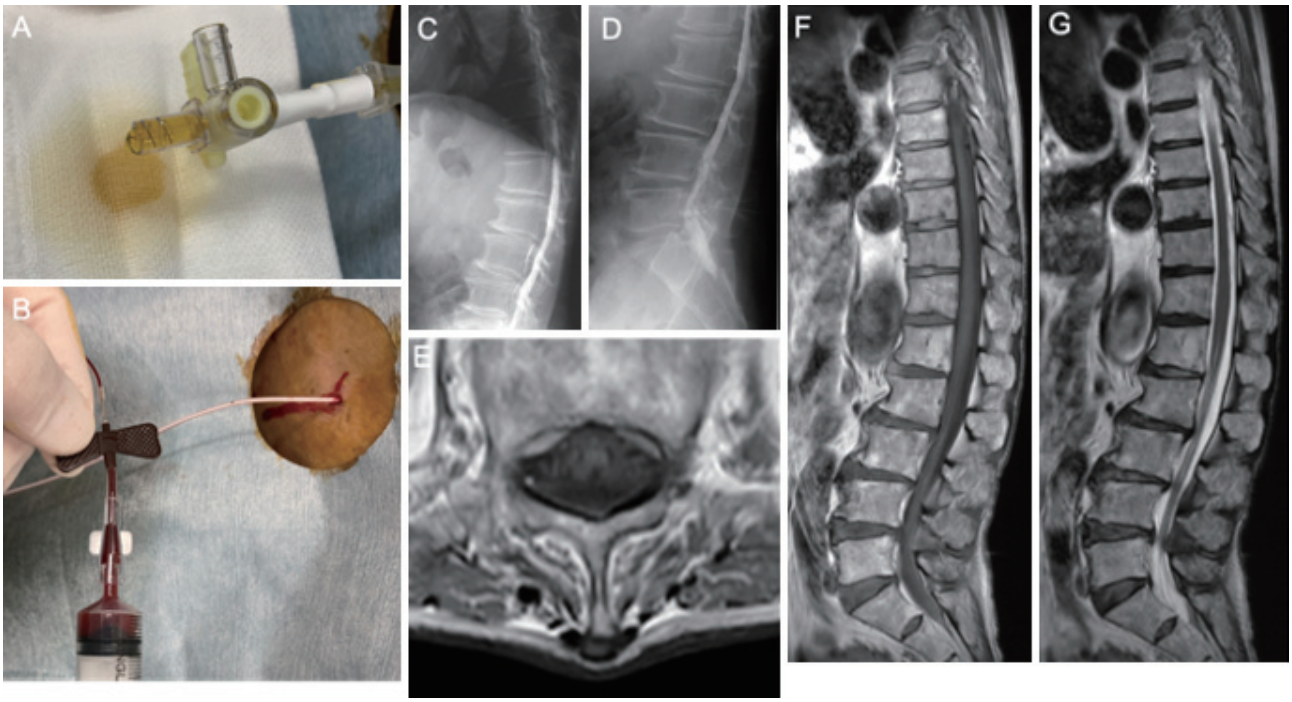

Fig. 2 Findings during the treatment procedure and post-treatment imaging findings.
A) Lumbar puncture under fundoscopic guidance from between L2-3 resulted in the flowing out of xantho-chromic fluid.
B) A spinal drainage tube was then inserted caudally into the hematoma cavity, and a thick, motor oil-like hematoma was aspirated.
C, D) Contrast media was injected through the drainage tube and the space was found to be completely isolated from the subarachnoid space.
E-G) MRI images of the thoracolumbar spine after 4 days of treatment displayed complete resolution of the subdural hematoma. (E, F: T1WI, G: T2WI)
MRI: magnetic resonance imaging: T1WI: T1-weighted imaging: T2WI: T2-weighted imaging

low signal on T2 star imaging suggesting hemosiderin on the dorsal surface of the region with an abnormal signal (Fig. 1D-G). Head MRI performed the day after this examination displayed a low signal on T1WI and a high signal on T2WI in the subarachnoid space of the bilateral frontal lobes, with a signal difference from the thoracolumbar region (Fig. 1B, D and E). No abnormal signals suggestive of obvious vascular malformation were displayed on 3 dimensional CT angiography of the thoracolumbar region.

Treatment

We hypothesized that this abnormal signal in the thoracolumbar region was a late subacute hematoma, based on the elapsed time since the fall (2 weeks), and the characteristics of the signal intensity on MRI. We hence expected the hematoma to be fluid, similar to intracranial chronic SDH, and considered the possibility that it could be cured by spinal drainage alone.

A total of 22 days after the fall, a spinal tap was performed between L2-3 under fluoroscopic guidance. The initial pressure was a 10-cm water column, resulting in the flowing out of xantho-chromic fluid (Fig. 2A). A 10-cm long spinal drainage tube was then inserted caudally, and a relatively dark-colored fluid hematoma was aspirated (Fig. 2B). When 7 mL of water-soluble contrast medium (OMNIPAQUE 240) was subsequently injected into the hematoma cavity through the spinal drainage tube, only the hematoma cavity, which was distinct from the spinal fluid cavity, was visualized, suggesting an encapsulated chronic SDH (Fig. 2C and D). After the procedure, bed rest was indicated, and spinal drainage fixation pressure was started at a height of 10 cm (based on the midaxillary line), with a target drainage volume of 100 mL per day. The spinal drain was removed after a total of 330 mL of hematogenous outflow over 3 days, and the patient's back pain was reduced by about a half.

Post-treatment clinical course

Rehabilitation was started early after treatment, and the function of both lower limbs gradually improved. He became able to smoothly stand on both legs and his numbness and paresthesia in the lower extremities improved. His JOA score[1)] recovered to 16 points, but his urinary function did not show sufficient improvement. This was subsequently found to be owing to the effects of prostate hypertrophy and irritable bladder disease on urological ex-

amination. Subsequent urological prescriptions led to a gradual improvement in urinary dysfunction. Thoracolumbar spine MRI after treatment displayed a complete disappearance of the abnormal signal seen before treatment (Fig. 2E-G). In addition, a brain CT scan also confirmed the disappearance of enlargement of the subarachnoid space in the bilateral frontal lobes. The patient was transferred to a convalescent rehabilitation hospital for further improvement of lower limb function.

Discussion

Chronic intracranial SDH is one of the most frequently observed diseases of head trauma in older adults. However, it is extremely rare for neurosurgeons to encounter a patient with chronic spinal SDH. Once a diagnosis of chronic intracranial SDH is made, there is often a lack of consideration of lumbar spine trauma as a complex trauma. However, when, as in the present case, symptoms of the lower extremities are observed that cannot be explained by intracranial disease alone, it is important to consider the possibility of spinal SDH.

To date, about 30 cases of spinal SDH have been reported, most of them were complicated by intracranial SDH.[2,3] Therefore, many authors have postulated that intracranial SDH is caused by gravity-induced descent of the hematoma as a mechanism for the development of spinal SDH.[2,3] However, as in the case reported here, there were many questions about the gravity descent theory, such as the difference in the timing of onset of symptoms, the difference in MRI signals, and most importantly, the lack of continuity between the intracranial and spinal SDH. To confirm the veracity of this theory, Kokubo et al.[4] prospectively performed sequential spinal cord MRI in 168 patients with intracranial SDH beginning in the first postoperative week. They found that only 2 (1.2%) of the 168 patients developed spinal SDH over time.[4] The 2 cases were both men, aged 70 and 83 years, the former with a history of hypertension and diabetes, and the latter with prostate cancer and myelodysplastic syndrome, and multiple head and lower back traumas. Upon examination of the trauma sites from these 168 patients, 75 were not clear, and of the 70 patients with single head trauma, not a single case of spinal SDH occurred.[4] From these findings, the authors suspected that even if intracranial and spinal SDH coexist, they are by no means a single pathology, but rather multiple traumas of the head and lumbar region, thus expressing their negative opinion regarding the gravity descent theory.

In most previous reports of cases of spinal SDH, the patients were treated by removal of SDH by invasive laminectomy under general anesthesia.[2,3] Of course, in cases of rapid progression of myelopathy caused by compression of the hematoma, a timely spinal decompression operation is considered necessary. However, in cases of slowly progressive, mild to moderate myelopathy, such as the present case, it is possible that, as with intracranial chronic SDHs, minimally invasive local anesthetic drainage procedures may resolve the problem after waiting for encapsulation and liquefaction of the hematoma. The decisive factor is the degree of liquefaction of the SDH, which, as with intracranial hematomas, may depend on the time since injury, and requires evaluation of the hematoma signal on CT and MRI. This patient was treated for the hematoma 22 days after the apparent fall trauma, and the hematoma signal on MRI was high on T1WI and also high on T2WI, suggesting that the patient was in the late subacute stage when the hematoma contents are being converted to free methemoglobin. Therefore, although not completely, we considered that most of the hematoma contents were liquefied, and we hence first attempted lumbar subdural drainage under local anesthesia. The resulting hematoma was a liquefied motor oil-like hematoma, and intraoperative hematoma cavity visualization using contrast medium displayed an isolated hematoma cavity. After 330 mL of hematoma mixed with cerebrospinal fluid was drained in three days, the clinical symptoms in this case improved dramatically and the draining tube was removed. The spinal MRI performed immediately afterward showed complete resolution of the SDH. The early introduction of physical treatment in our present case rapidly improved the patient's lower limb function.

To our knowledge, only 4 cases of minimally invasive treatment of spinal SDH have been reported previously (Table 1).[5-8] All of the previous 4 patients presented with relatively short-range hematoma with symptoms of radiculopathy, mainly lumbar pain, and were treated with 1 or 2 spinal taps. All showed a high signal on T1WI and a high signal on T2WI MRI, suggesting subacute liquefaction, and a small hematoma volume.[4-8] These procedures resulted in complete resolution of the SDH on MRI, and the course of symptoms was described as being excellent.[4-7] In the case presented here, the hematoma volume was clearly larger than in the other 4 cases, so the authors chose to perform spinal drainage, resulting in a 3-day drainage period.

As most falls that occur in older adults result in compound traumas, the occurrence of spinal SDH should always be considered in clinical practice. If the symptoms are closely examined and it is determined that spinal symptoms cannot be explained by intracranial SDH, this is an indication for treatment. Patient conditions, the clinical symptoms, the timing of the injury, the assessment of liquefaction on MRI images, and the amount of hematoma should be considered to determine a treatment plan. Furthermore, we would like to stress that it may be favorable to first try minimally invasive procedures that neurosurgeons are accustomed to, such as spinal tap and drainage, in the late subacute phase of spinal SDH.

Table 1 Spinal subdural hematoma treated by spinal tap

Case no.	Author	Age (years), sex	Presence of intracranial SDH	Location of spinal SDH	Interval between trauma and treatment	Presenting symptom	Signals on MRI	Treatment for spinal SDH	Post-treatment MRI findings	Clinical prognosis after procedure
1	Morishige M, 2007	54, male	(+)	Posterior fossa to S2	N/A	Lumbago	T1 hyper intensity, T2 hyper intensity	Lumbar tap twice	Improved	Improved
2	Cho DC, 2009	43, male	(-)	L5 to S2	1 month	Paresthesia in L5 to S1, severe leg pain	T1 hyper intensity, T2 hyper intensity	Lumbar tap	Complete disappearance of the hematoma	Improved
3	Ichinose D, 2018	40, male	(+)	L2 to S1	1 month	Lower abdominal pain, lumbago	T1 hyper intensity, T2 iso intensity	Lumbar tap	Complete disappearance of the hematoma	Improved
4	Kim DG, 2019	67, male	(+)	L4 to S1	3 weeks	Paraparesis, mild lumbago	T1 hyper intensity, T2 hyper intensity	Lumbar tap	Resolved to chronic status	Improved
5	Akimoto J, 2023	80, male	(+)	Th8 to S2	22 days	Paraparesis, hyperesthesia below Th10, urninary incontinence	T1 hyper intensity, T2 hyper intensity	Lumbar drainage for 3 days	Complete disappearance of the hematoma	Improved

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Ethics Approval

All procedures performed on this case were in accordance with the standards of the Ethics Committee of Kohsei Chuo General Hospital, and with the 1964 Declaration of Helsinki and its later amendments.

Informed Consent

Informed consent was obtained from the patient and his family for this case study.

Conflicts of Interest Disclosure

There are no conflicts of interest.

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