Concomitant Intracranial and Lumbar Chronic Subdural Hematoma Treated by Fluoroscopic Guided Lumbar Puncture: A Case Report and Literature Review

Daisuke ICHINOSE,¹ Satoru TOCHIGI,¹ Toshihide TANAKA,¹ Tomoya SUZUKI,¹ Jun TAKEI,¹ Keisuke HATANO,¹ Ikki KAJIWARA,¹ Fumiaki MARUYAMA,¹ Hiroki SAKAMOTO,¹ Yuzuru HASEGAWA,¹ Satoshi TANI,² and Yuichi MURAYAMA²

¹Department of Neurosurgery, Jikei University Kashiwa Hospital, Kashiwa, Chiba, Japan; ²Department of Neurosurgery, Jikei University School of Medicine, Tokyo, Japan

Abstract

A 40-year-old man presented with a severe headache, lower back pain, and lower abdominal pain 1 month after a head injury caused by falling. Computed tomography (CT) of the head demonstrated bilateral chronic subdural hematoma (CSDH) with a significant amount in the left frontoparietal region. At the same time, magnetic resonance imaging (MRI) of the lumbar spine also revealed CSDH from L2 to S1 level. A simple drainage for the intracranial CSDH on the left side was performed. Postoperatively, the headache was improved; however, the lower back and abdominal pain persisted. Aspiration of the liquefied spinal subdural hematoma was performed by a lumbar puncture under fluoroscopic guidance. The clinical symptoms were dramatically improved postoperatively. Concomitant intracranial and spinal CSDH is considerably rare so only 23 cases including the present case have been reported in the literature so far. The etiology and therapeutic strategy were discussed with a review of the literature. Therapeutic strategy is not established for these two concomitant lesions. Conservative follow-up was chosen for 14 cases, resulting in a favorable clinical outcome. Although surgical evacuation of lumbosacral CSDH was performed in seven cases, an alteration of cerebrospinal fluid (CSF) pressure following spinal surgery should be reminded because of the intracranial lesion. Since CSDH is well liquefied in both intracranial and spinal lesion, a less invasive approach is recommended not only for an intracranial lesion but also for spinal lesion. Fluoroscopic-guided lumbar puncture for lumbosacral CSDH following burr hole surgery for intracranial CSDH could be a recommended strategy.

Key words: spinal subdural hematoma, chronic subdural hematoma, spinal puncture, low back pain

Introduction

In general, spinal chronic subdural hematoma (CSDH) is uncommon, and the mechanism for its formation remains unclear. A variety of hypothesis have been proposed, such as migration of the hematoma from intracranial lesion due to gravity, spinal subarachnoid hemorrhage dissecting into subdural space which remains and changes into CSDH.¹,²) In addition, the coexistence of intracranial and spinal CSDH is extremely rare, and pathogenesis has not been elucidated as well.

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Here, we report a case of concomitant intracranial and spinal CSDH treated by burr hole surgery for intracranial lesion followed by a percutaneous lumbar puncture of the hematoma under fluoroscopic guidance. We also discuss the clinical features, etiology, and therapeutic strategy for concurrent CSDH.

**Case Report**

A 40-year-old physician who had experienced a head injury caused by falling on his forehead 1 month ago presented to our institute with progressive headaches and lumbago accompanied by lower abdominal pain. Computed tomography (CT) revealed bilateral CSDH with significant amount in the left frontoparietal area with a mild midline shift (Fig. 1). In addition, lumbosacral magnetic resonance imaging (MRI) demonstrated spinal CSDH extending from L2 to S1 (Figs. 2A and 2B). He had no significant past medical history. Laboratory data eliminated the possibility of thrombocytopenia and coagulopathy.

Burr hole surgery for the left intracranial lesion was performed with a closed drainage. Postoperatively, his headache subsided; however, he was still suffering from persistent severe lumbago and abdominal pain. Because of the severe symptoms, which was unbearable and subsequent CT indicating the hematoma was a liquefied CSDH (Figs. 2C and 2D), percutaneous lumbar puncture under fluoroscopic guidance was performed 2 days after burr hole craniotomy. About 15 ml of dark-brownish serous and well-liquefied blood was aspirated (Figs. 3A and 3B).

No cerebrospinal fluid (CSF) was aspirated at all during the procedure. The clinical symptoms were dramatically improved after the lumbar puncture. The hematoma was significantly reduced on MRI postoperatively. There was no residual hematoma a week after the procedure (Figs. 4A and 4B). Hematoma or other abnormal findings were not observed in the cervical and thoracic spine. Postoperative course was uneventful without recurrence a year after operations.

![Fig. 1 Preoperative initial computed tomography (CT) 1 month after a head injury reveals the bilateral chronic subdural hematoma with significant mass effect on the left side.](image1)

![Fig. 2 Preoperative magnetic resonance imaging (MRI) showing the hematoma extending from L2 to S1 appearing high intense on T1-weighted sagittal imaging (A) and isointense on T2-weighted axial imaging with a “three-branch star” appearance (B). Subsequent sagittal (C) and axial (D) computed tomography (CT) demonstrating an isodense chronic subdural hematoma extending from L2 to S1.](image2)
Discussion

In general, hematoma in the spinal canal is usually located in the epidural space with or without trauma or hematological abnormality, and spinal CSDH is considerably rare. Although intracranial CSDH is common, concomitant intracranial and lumbar CSDH is extremely rare. Spinal subdural space does not contain any major blood vessels or bridging veins that act as a source for a spinal CSDH, which is different from the intracranial circumstances. Occult spinal CSDH might coexist among patients with intracranial CSDH; however, lumbar MRI is not performed routinely after burr hole surgery especially in patients without clinical symptoms or neurological deficits regarding a spinal disorders, such as severe lumbago, radicular pain, paraparesis, or urinary dysfunction.

The etiology of concomitant intracranial and spinal CSDH was discussed as follows: one is migration of the hematoma from the intracranial space and another one is an accidental simultaneous occurrence of hemorrhage in the intracranial and spinal subdural space by multiple injuries to head and lumbar area. Intracranial acute subdural hematoma and ruptured CSDH caused by enlargement could migrate to the spinal compartment as a result of gravity because of the anatomical continuity of the subdural space between the cranium and spine and also the low resistance of the dura-arachnoid interface filled with amorphous material.

An elevated intracranial pressure, CSF inflows into the subdural space following trauma-induced arachnoid tear, or low intracranial pressure resulting from ventriculoperitoneal shunt can facilitate hematoma migration into a remote area. In some cases, hematoma could be detected in the posterior fossa and all through the spine, suggesting migration from the intracranial lesion. The similarity of the signal intensity and the changes in the spinal CSDH and those of intracranial lesion also suggested that both hematomas had the same origin. On the other hand, the major mechanism for the concomitant intracranial and spinal CSDH might be double trauma because of its low incidence among the patients with intracranial CSDH. Kokubo et al. reviewed lumbar MRI obtained in consecutive 168 patients with intracranial CSDH treated by burr hole irrigation. They found only two patients (1.2%) who revealed concomitant intracranial and lumbar CSDH and both of them hit their head and lumbar...
### Table 1 Summary of reported cases of concomitant chronic intracranial and spinal subdural hematomas

| Case no. | Age | Sex | Location of intracranial CSDH | Location of spinal CSDH | Sequence of detected lesion | Trauma/interval between trauma and diagnosis | Other predisposing factor | Presenting symptoms | Treatment for intracranial SDH | Treatment for spinal SDH | Author |
|----------|-----|-----|-------------------------------|--------------------------|-----------------------------|-------------------------------------------|--------------------------|----------------------|-------------------------------|----------------------------|---------|
| 1        | 54  | M   | Bil                           | L1–S2                    | Intracranial                | 3 weeks                                   | –                        | Headache, lumbago       | Conservative follow-up         | Surgical evacuation           | Leber, 1997[27] |
| 2        | 54  | M   | Bil                           | T12-S2                   | Intracranial                | 2 weeks                                   | –                        | Headache, lumbago, bilateral S1 & S2 radiculopathy | Conservative follow-up         | Surgical decompression        | Tillich, 1999[28] |
| 3        | 59  | M   | Bil + posterior fossa         | Th11-S1                  | Simultaneous                | –                                        | Anti-platelet therapy   | Lumbago, numbness & motor weakness in both legs | Conservative follow-up         | Conservative follow-up        | Yamaguchi, 2005[6] |
| 4        | 54  | M   | Lt + posterior fossa          | C1–S2                    | Simultaneous                | –                                        | –                        | Headache, lumbago       | Burr hole drainage           | Lumbar puncture              | Morishige, 2007[11] |
| 5        | 12  | M   | Posterior fossa               | C1–S3                    | Simultaneous                | –                                        | Aplastic anemia          | Headache, lumbago       | Conservative follow-up        | Conservative follow-up        | Jain, 2008[7]   |
| 6        | 65  | F   | Lt                            | Th12–S1                  | Simultaneous                | 5 weeks                                   | –                        | Gait disturbance, pain in buttocks and posterior aspect of thighs | Burr hole irrigation         | Conservative follow-up        | Nakajima, 2009[8] |
| 7        | 35  | F   | Lt                            | L3–S1                    | simultaneous                | –                                        | –                        | Headache, dizziness, lumbago, paraparesis | Burr hole irrigation         | Laminectomy with removal      | Yang, 2009[18]  |
| 8        | 66  | M   | Bil                           | L1–S1                    | spinal                      | –                                        | –                        | paraparesis, severe leg pain | Surgery                   | Conservative follow-up        | Nagashima, 2010[9]  |
| 9        | 60  | M   | Bil                           | L3–S2                    | intracranial                | –                                        | –                        | headache, lumbago, radiating leg pain | Surgery                   | Conservative follow-up        | Kim K, 2010[10] |
| 10       | 24  | F   | Bil                           | L4–S2                    | simultaneous                | 1 month                                   | –                        | posture headache, lumbago, transient sensory disturbance of the right extremities, aphasia | Conservative follow-up        | Conservative follow-up        | Kim K, 2010[10] |
| 11       | 47  | M   | Bil                           | L3–S1                    | Intracranial                | 2 months                                  | Anti-platelet therapy   | Lumbago                | Burr hole surgery (Lt then Rt) | Conservative follow-up        | Hagihara, 2010[31] |
| Case no. | Age | Sex | Location of intracranial CSDH | Location of spinal CSDH | Sequence of detected lesion | Trauma/interval between trauma and diagnosis | Other predisposing factor | Presenting symptoms | Treatment for intracranial SDH | Treatment for spinal SDH | Author |
|---------|-----|-----|-------------------------------|-------------------------|-----------------------------|---------------------------------------------|--------------------------|---------------------|-----------------------------|--------------------------|--------|
| 12      | 73  | M   | Bil                          | L3–S2                   | Simultaneous                | −                                          | −                        | Bilateral sciatica, confusion, Lt. hemiparesis | Craniotomy               | Conservative follow-up    | Jibu K, 2012²² |
| 13      | 67  | F   | Lt                           | L4–S1                   | Simultaneous                | −                                          | Anti-platelet therapy | Headache, back pain, radiating leg pain, motor weakness in lower limbs | Burr hole drainage       | Conservative follow-up    | Wang, 2012²³ |
| 14      | 39  | F   | L1–S2                        | Spinal                  | −                           | −                                          | −                        | Lumbago, radiating leg pain, headache | Burr hole irrigation | Conservative follow-up    | Moon, 2013²⁴ |
| 15      | 70  | M   | Bil                          | L4–S1                   | Simultaneous                | −                                          | −                        | Back pain, pain in both legs | Drainage                | Conservative follow-up    | Lin, 2014²⁵ |
| 16      | 83  | M   | Bil                          | L5–S1                   | Intracranial                | −                                          | Myelodysplastic synd. | Asymptomatic | Burr hole irrigation | Conservative follow-up | Kokobo, 2014²⁶ |
| 17      | 70  | M   | Bil                          | S1                      | Intracranial                | −                                          | −                        | Asymptomatic | Burr hole irrigation | Conservative follow-up | Kim MS, 2015²⁷ |
| 18      | 45  | M   | Bil                          | L4–S3                   | Spinal                      | −                                          | −                        | Saddle pain & dysuresia | Conservative follow-up | Surgical evacuation | Cui, 2015²⁸ |
| 19      | 82  | F   | Bil                          | L3–4                    | Intracranial                | 4 weeks                                    | −                        | Lumbago, right leg tingling sensation | Burr hole irrigation | Conservative follow-up    | Kwon, 2015²⁹ |
| 20      | 57  | M   | Lt                           | L2–S1                   | Spinal                      | 2 weeks                                    | −                        | Lumbago, radicular pain & weakness in both legs | Burr hole irrigation | L3–L5 hemilaminectomy | Kwon, 2015³⁰ |
| 21      | 58  | M   | Rt + posterior fossa         | Th1–S1                  | Intracranial                | 2 months                                   | −                        | Headache, Lt hemiparesis, lumbago, Lt. lower limb weakness | Burr hole drainage | L5 hemilaminectomy, evacuation | Matsumoto, 2016³¹ |
| 22      | 67  | M   | Bil                          | L4–S1                   | Intracranial                | 2 weeks                                    | −                        | Headache, nausea, neck pain, saddle anesthesia | Bilateral craniotomy | L5 laminectomy, evacuation | Kanamaru, 2016³² |
| 23      | 40  | M   | Bil                          | L2–S1                   | Simultaneous                | 1 month                                    | −                        | Headache, lower abdominal pain, lumbago | Burr hole irrigation | Lumbar puncture | present case |

Bil: bilateral, CSDH: chronic subdural hematoma, F: female, Lt: left, M: male, Rt: right.
area at the time of injury. In the present case, the patient did not have direct trauma to the lumbar area. We speculated that he had developed acute subdural hematoma at the time of injury. Despite the fact that he suffered a worsening headache, he continued his daily activities. Thus, his continuous upright positioning may have prompted migration of the acute hematoma toward the lumbar region. However, the cervical and thoracic spine MRI did not reveal the residue of passing hematoma.

The majority of cases of concomitant intracranial and spinal CSDH showed a favorable outcome following conservative management. Based on our review of the literature (Fig. 1), conservative following-up was chosen for spinal CSDH in 14 patients (60.9%) in the present series of 23 patients with concomitant intracranial and spinal CSDH. Probably due to mainly composed of liquefied hematoma, spinal CSDH does not always have a mass effect and may resolve spontaneously; therefore, conservative management can be recommended for patients with or minimal neurological deficit and/or poor general condition. In spite of the possibility of spontaneous remission of spinal CSDH, prompt decompression by surgery is required in cases with severe symptoms and neurological deterioration caused by raising CSF pressure resulting in the spinal cord or nerve root compression.1,17–23 Seven patients (30.4%) with lumbosacral CSDH have been treated by open surgery. According to the intraoperative findings, the hematoma was well liquefied and evacuated easily by suction.21,23 On the other hand, CSF also flowed out during their operation because of the absence of a visible outer membrane, which would have caused recollection of the intracranial hematoma due to CSF hypotension.

Unexpectedly, a lumbar puncture was attempted in only one case besides the present case in the series of 23 patients1) Levy et al. reported that a fluoroscopically guided lumbar puncture led to an immediate resolution of clinical symptoms caused by spinal CSDH.26 This simple technique could be beneficial for several reasons. First, it provides anatomically precise puncture site. Second, the hematoma could be evacuated through a spinal needle because it is well liquefied. Third, the puncture could avoid CSF hypotension, which might exacerbate the recollection of the intracranial CSDH. Last, there is no need for general anesthesia or laminectomy.

When the neurological deficits such as severe radicular pain caused by compressing the nerve roots are rapidly progressive, prompt decompression should be considered. Based on the intraoperative findings revealing the liquefied hematoma as reported previously including the present case, we believe that simple lumbar puncture under fluoroscopic guidance could be an appropriate treatment to improve clinical symptoms of patients with concomitant intracranial and spinal CSDH.

Conflicts of Interest Disclosure

None of the authors have any conflicts of interest to declare.

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Address reprint requests to: Satoru Tochigi, MD, Department of Neurosurgery, Jikei University Kashiwa Hospital 163-1 Kashiwa-shita, Kashiwa, Chiba 277-8567, Japan. e-mail: tochigi@jikei.ac.jp