Concomitant Intracranial Chronic Subdural Hematoma and Spinal Subdural Hematoma: A Case Report and Literature Review

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Key words

- Chronic subdural hematoma
- Headache
- Lumbago
- Magnetic resonance imaging
- Spinal subdural hematoma

Abbreviations and Acronyms

CSDH: chronic subdural hematoma CT: computed tomography MRI: magnetic resonance imaging SDH: subdural hematoma

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Citation: World Neurosurg. (2016). http://dx.doi.org/10.1016/j.wneu.2016.03.020

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

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INTRODUCTION

Intracranial chronic subdural hematoma (CSDH) is a common pathology in elderly individuals after minor head injury. On the other hand, spinal subdural hematoma (SDH) is rare, and the pathogenesis remains unclear.¹⁻³ Several cases of concomitant intracranial CSDH and spinal SDH have recently been reported.⁴⁺²² In general, the coexistence of these conditions is extremely rare, and the etiology has not been elucidated. Migration of the hematoma intracranially to a spinal site or coincidence of both intracranial and spinal CSDHs have been suggested as etiologies.^{9,11,12,18,21}

Here we describe a case of concomitant CSDH and spinal SDH in which the etiology was considered to involve migration of the hematoma intracranially to a spinal site. We reviewed reported cases of concomitant intracranial CSDH and spinal SDH and discuss the features, etiology, and treatment strategy. BACKGROUND: Concomitant intracranial chronic subdural hematoma (CSDH) and spinal subdural hematoma (SDH) are rare, and the etiology has yet to be elucidated. However, migration of the hematoma intracranially to a spinal site or coincidence of both intracranial and spinal CSDHs have been proposed as etiologies. We report a case of concomitant intracranial CSDH and spinal SDH in which spinal hematoma might have migrated from the cranial lesion.

CASE DESCRIPTION: A previously healthy 58-year-old man with previous trauma to the occiput and lumbar spine suffered from headache, lumbago, and left hemiparesis. Head computed tomography revealed right-sided intracranial CSDH, and he underwent single burr-hole craniotomy. Although clinical symptoms tended to improve, left lower-limb weakness and lumbago remained. Spinal magnetic resonance imaging (MRI) 3 days after craniotomy revealed SDH extending from T1-S1. Because conservative therapy had not improved clinical symptoms, hematoma evacuation was performed via a left L5 hemilaminectomy 1 week after craniotomy. The patient showed complete recovery immediately postoperatively.

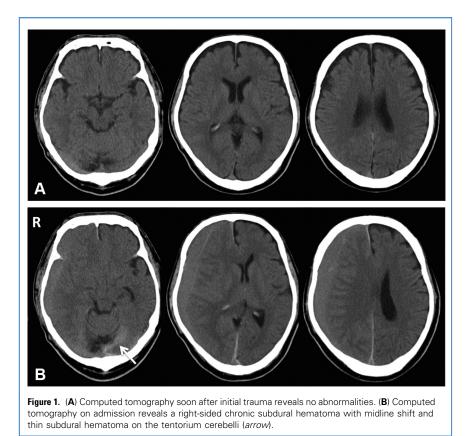
CONCLUSIONS: We reviewed the cases of 22 patients with concomitant intracranial CSDH and spinal SDH to discuss the features, etiology, and treatment strategy. Although surgical intervention was mainly selected for intracranial CSDH, conservative observation was mainly selected for spinal SDH. Outcomes were good in all patients. We created a new classification of spinal SDH shape using sagittal MRI. This classification indicates that cases with both ventral and dorsal SDH tend to require surgical intervention. This classification may help in deciding treatment strategies.

CASE REPORT

History and Examination

A previously healthy 58-year-old man presented to our hospital after experiencing trauma to the occiput and lumbar spine in a fall from the driver's seat of a truck. He was alert without neurologic deficits, and computed tomography (CT) of the head revealed no traumatic lesions (Figure 1A). Two months later, he suffered from headache, lumbago, and left hemiparesis. He had no history of using anticoagulant or antiplatelet medications. Laboratory examinations eliminated the possibility of coagulopathy-related disorders. Neurologic examination revealed disorientation with mild left hemiparesis (manual muscle test score, 4/5). Head CT revealed rightsided CSDH with a midline shift and thin

SDH on the tentorium cerebelli (Figure 1B). Clinical symptoms were attributed to CSDH and treated surgically by single burr-hole craniotomy and close drainage. Although disorientation, weakness in the left upper limb, and headache improved postoperatively, mild weakness in the left lower limb and lumbago remained. The straight leg-raising test was 60° on lying down, and deep tendon reflexes were decreased without sensory, bladder, or rectal disturbance. Spinal magnetic resonance imaging (MRI) 3 days after craniotomy revealed SDH extending from TI-SI (Figures 2A, 2B, and 3B). Axial MRI revealed the hematoma was dominant on the left side (Figures 2C, 2D, and 3C). Thin SDHs were also detected on the tentorium cerebelli and in the posterior fossa (Figure 3A).



Operation and Postoperative Course

Although the patient was managed conservatively, clinical symptoms remained. He underwent hematoma evacuation via a left L5 hemilaminectomy 1 week after craniotomy. The dural sac was pulseless and darkly discolored (Figure 4A). When the dura was opened, dark liquid blood was evacuated (Figure 4B). The arachnoid seemed normal without typical membranes as CSDH and subarachnoid fluid pulsation were noted after removal of the SDH (Figure 4C). The patient showed immediately complete recovery postoperatively. Head CT and spinal MRI at 3 months postoperatively revealed no recurrent SDH (Figure 5).

LITERATURE REVIEW

The PubMed search engine of the National Library of Medicine and National Institutes of Health was used to conduct an Internet literature search of MEDLINE. We reviewed 22 cases of concomitant intracranial CSDH and spinal SDH, comprising previous reports and our own case, to discuss the clinical features, etiologies, and treatment strategies.⁴⁻²²

Analysis of Literature Review

Results of our analysis are summarized in **Table 1**. Patients ranged in age from 10 to 83 years (mean, 52.7 years) and were predominantly men (17 men and 5 women). The predisposing event was mainly trauma (n = 12; 54%).^{4,6-11,15,16,19} In 7 patients (31%),^{5,12-15,21,22} the SDH arose spontaneously, and ventriculoperitoneal shunt placement was performed in 3 patients (13%).^{17,18,20} In posttraumatic patients, 8 patients sustained trauma to both the head and back.

Of the 22 cases, 5 patients (23%) had a bleeding tendency, 2 presented with blood dyscrasia, and 3 were on medications known to affect bleeding.

Clinical Features

Frequent presenting symptoms were lumbago (80%) and headache (47%). Neurologic deficits caused by spinal SDH, such as leg pain (40%) and paraparesis (31%), were more frequent than those caused by intracranial CSDH, such as hemiparesis (10%) and aphasia (5%). Nine patients suffered from spinal symptoms alone.^{5,8,10,12,15-17,20,21}

Diagnosis and Radiologic Features

In terms of the diagnosis, intracranial CSDH and SDH were detected at the same time in 10 patients.^{5,6,9,12,14,15,17,20-22} In 8 patients, intracranial CSDH was detected prior to spinal SDH.^{4,7,11,16,18,19} In 4 patients, spinal SDH was detected prior to intracranial CSDH.^{8,10,13,15} The interval between detection of intracranial CSDH and spinal SDH ranged from 1 day to 2 months, with most cases identified within 1 week (median, 4 days).

As for laterality of the intracranial CSDH, 14 cases were bilateral^{5-7,9,11,12,15,17-21} and 8 cases were unilateral (left side: n = 7; right side: n = 1).^{4,8,10,13,14,16,22} Spinal SDH was diagnosed by MRI in all except I case. In that remaining case, spinal SDH was diagnosed from spinal CT and myelogram. Almost all cases of spinal SDH were located below the thoracic spine, with the thickest part in the lower lumbar or sacral spine.

Treatment

For intracranial CSDH, irrigation by single-burr craniotomy was performed in 14 patients,^{4,7-10,12-14,16,22} whereas 8 cases were treated conservatively.^{5,7,11,17-21} On the other hand, for spinal SDH, surgical treatment was performed in only 7 patients: 6 patients underwent hematoma evacuation via laminectomy, and 1 patient underwent drainage of the SDH via lumbar puncture.^{10,11,14,17,19,22} Conservative observation was performed in 15 patients because the clinical symptoms were mild.^{4,5,7,9,12,13,16,18,20,21} Outcomes were good in all patients.

Etiology

In 14 patients, migration of the SDH from the cranial to spinal compartment was proposed as the etiology.^{4-6,10,12,14,15,17,18,20-22} On the other hand, coincidental intracranial and spinal CSDH caused by trauma was proposed as the etiology in 6 patients.^{8,9,11,16,19}

DISCUSSION

Although intracranial CSDH is common, concomitant intracranial CSDH and spinal

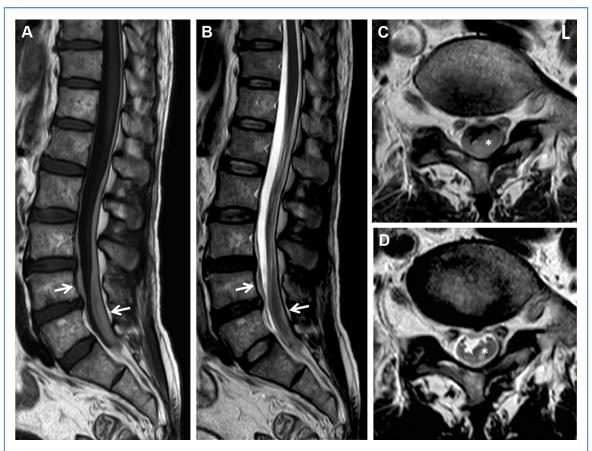


Figure 2. Lumbar sagittal T1- (A) and T2-weighted (B) imaging reveal hematoma (*arrows*) in the ventral and dorsal spinal at the T9-S1 levels. Axial T1- (C) and T2-weighted (D) imaging at the L5

level reveals subdural hematoma (asterisks). The hematoma is left-side dominant.

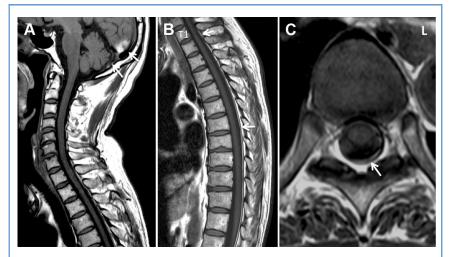


Figure 3. (A) Cervical sagittal T1-weighted magnetic resonance imaging reveals thin subdural hematoma (*arrows*) on the cerebelli and in the posterior fossa. (B) Thoracic sagittal T1-weighted imaging reveal continuity of hematoma (*arrows*) below the T1 level. (C) Axial T1-weighted imaging at the T5 level reveals a thin subdural hematoma (*arrow*).

SDH appear extremely rare in general. However, Kokubo et al.⁹ reported that 1.2% of intracranial CSDH patients showed concomitant spinal SDH. In that report, spinal MRI was performed for screening, and the patients were asymptomatic. Silent coexistence of these lesions may therefore be more than previously imagined. Reported symptomatic cases have usually exhibited clinical symptoms caused by spinal lesions, such as lumbago, paraparesis, and leg pain, in addition to those caused by cranial lesions, and diagnosis of both lesions was made at the same time. In cases with a time lag between diagnoses, continuity or deterioration of clinical symptoms suggested the need for additional imaging studies. In our case, the patient had a history of head trauma and suffered from cranial symptoms, such as headache and left hemiparesis, in addition to lumbago.

CONCOMITANT INTRACRANIAL AND SPINAL SUBDURAL HEMATOMA

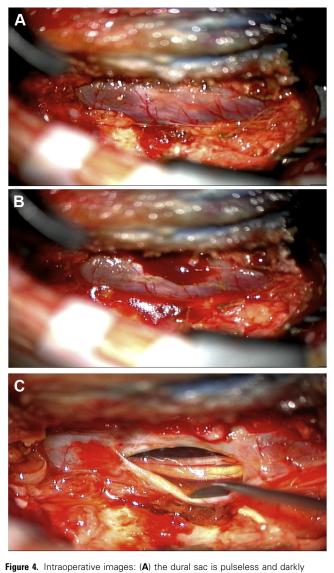


Figure 4. Intraoperative images: (A) the dural sac is pulseless and darkly discolored; (B) when the dura is opened, dark liquid blood is evacuated and (C) the arachnoid seems normal without typical membranes and subarachnoid fluid pulsations are noted.

We suspected and diagnosed intracranial CSDH. Initially, we considered the lumbago as a chronic disease. After treatment of the cranial lesion, the patient continued to show lumbago and right lower-limb weakness. We then suspected a spinal lesion and diagnosed spinal SDH. Concomitant spinal SDH should therefore be suspected in patients with intracranial CSDH showing lumbago and neurologic symptoms in the lower limbs.

Whether coexisting spinal SDH should be considered as CSDH remains in

question. Hematomas in the spinal canal are most commonly epidural, and spinal SDHs are reported less frequently.^{1-3,23} Spinal SDHs are usually related to anticoagulation, blood dyscrasia, lumbar puncture, trauma, spinal anesthesia, or vascular malformation.^{1-3,23} Spontaneous cases have also been reported.³ Spinal SDHs are usually classified by chronology or cause as follows: acute spinal SDH, subacute spinal SDH, and spinal CSDH.¹ Among these, spinal CSDH is extremely rare and represents only 6% of spinal

SDHs.^{1,19,23} Similar to intracranial CSDH, spinal CSDH has typical membranes and a liquefied hematoma, and minor trauma and spontaneous occurrence are the most frequently cited causes.^{1,23,24} Spinal CSDH most often occurs in patients between 50 and 70 years old.²⁴ The lumbar and thoracolumbar regions are the most common sites.²⁴ In traumatic spinal CSDH, a fluctuating course of weakness, spasticity, and bowel and bladder dysfunction caused by spinal cord compression can occur over 2 weeks to years after trauma.^I The pathogenesis of spinal CSDH remains unclear and is not considered identical to that of intracranial CSDH.^{1,3,24} Unlike the cranial subdural space, the spinal subdural space lacks bridging veins as an origin for SDH.^{1,3} Spinal CSDH has been hypothesized to originate from the subarachnoid space. Vinters et al.25 suggested that spinal subarachnoid hemorrhage might be the primary lesion that eventually dissected into the subdural space. Although the subarachnoid blood is diluted and absorbed, the subdural clot remains and changes into CSDH with the membrane. In the cases of concomitant intracranial CSDH and spinal SDH, another question arises as to which method to use when determining whether coexisting spinal SDH represents CSDH. Because MRI cannot clearly distinguish between acute and chronic spinal SDH, unlike cranial lesions,²¹ the answer must be obtained from intraoperative findings. In other words, proof of the existence of a typical membrane is needed to investigate coexisting whether spinal SDH represents CSDH. In this case, because the operative findings did not demonstrate the existence of typical membranes, we diagnosed the spinal SDH as distinct from CSDH.

The etiology of concomitant intracranial CSDH and spinal SDH has not been elucidated. Two mechanisms have been suggested: migration from the cranial to the spinal compartment and accidental coincidence of both intracranial and spinal CSDHs.^{9,11,12,18,21} In the reported cases, migration of SDHs has been a major finding in the reported cases. In those cases, MRI revealed that SDHs moved sequentially from the cranial to the sacral level.^{4-6,10,12,14,15,17,18,20-22} In the same way,

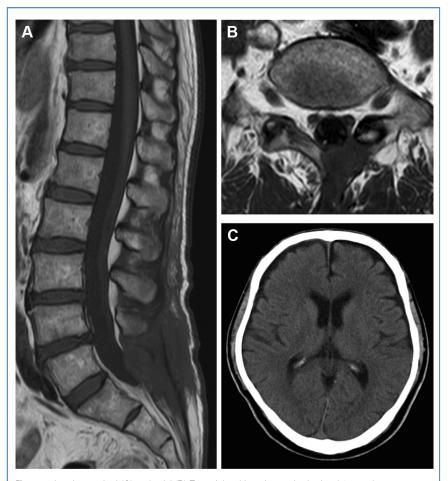


Figure 5. Lumbar sagittal (A) and axial (B) T1-weighted imaging at the L5 level 3 months postoperatively reveals no recurrence of subdural hematoma. (C) Computed tomography 3 months postoperatively reveals no recurrence of chronic subdural hematoma.

migration of intracranial acute SDH from the cranial to the spinal compartment has been documented in some cases.^{26,27} In these cases, spinal SDH was detected after spontaneous resolution of intracranial acute SDH. From a postmortem study, continuity of the subdural compartment between the cranium and spine has been proven by air injection.²⁸ The mechanism of migration from the cranial to the spinal component is unclear. Both low and high intracranial pressure have been proposed as predisposing factors.²¹ An elevated intracranial pressure might displace the SDH toward the skull base or spinal canal.²⁶ On the other hand, low intracranial pressure resulting from ventriculoperitoneal shunt can result in dissection between the dura and arachnoid layer, readily causing migration

of the hematoma.^{18,20} As a result, intracranial SDH may distribute to the spinal subdural space, particularly the lumbar region under the influence of gravity. The same intensity of SDHs on MRI supports the migration theory.²¹ whether a hematoma However, surrounded by outer and inner membranes can move freely in the subdural space remains unclear. Yamaguchi et al.²¹ answered the question by suggesting that the growing hematoma might have torn the membrane. In fact, Hirakawa et al.29 reported a case in which the intracranial CSDH ruptured intracerebrally, forming an acute subcortical hematoma. Hence, a progressively growing hematoma can tear the inner membrane and leak hematoma into the subdural space. In our case, head CT on admission revealed thin SDH

on the tentorium cerebelli, indicating hematoma leakage. Moreover, MRI revealed continuity of SDH from the cranium to the spine and the same intensity of hematomas on T1-weighted images. The evacuated spinal SDH contained the same dark liquid as the intracranial CSDH. We therefore considered that the spinal hematoma might have migrated from the cranial lesion. That is, first, the progressively growing CSDH might have caused not only clinical symptoms such as headache and left hemiparesis, but also tear of the inner membrane with leakage of hematoma into the subdural space. Second, high intracranial pressure might have displaced the leaked hematoma to the tentorial notch, and the hematoma might have migrated into the posterior subdural space of the posterior fossa and the spine under the influence of gravity. As a result, the hematoma might have collected in the lumbar region forming the bottom of the subdural space, causing symptoms such as lumbago and left lower-limb weakness.

On the other hand, several authors have reported accidental coincidence of CSDHs.^{8,9,11,16,19} In those cases, the patients hit both their head and back, and minor trauma might have caused both intracranial and spinal CSDHs. Because sole spinal CSDH has rarely been reported, as previously described,^{1,23,24} there is a possibility that intracranial and spinal CSDHs could arise simultaneously. However, this diagnosis cannot be made from imaging studies and may need proof of the existence of a membrane during surgery. Leber et al.¹¹ and Tillich et al.¹⁹ reported accidental coincidence of both intracranial and spinal CSDHs based on surgical and histopathologic findings. In our case, although the patient hit the head and back, the etiology was not considered to represent coincidence of CSDHs because the operative findings did not demonstrate the existence of typical membranes.

Diagnosis of spinal SDHs is usually performed by MRI.³ We made our own classification of the shape of spinal SDHs on sagittal MRI as follows: 1) J-shaped, 2) U-shaped, 3) reverse J-shaped, 4) dorsal localized, and 5) ventral localized. Details of these shapes are shown in Figure 6.

					Dominant		Location	Tre	atment		
Author and Year	Age (years)/Sex	Predisposing Event	Lesion Detected First (time lag*)	Clinical Symptoms	Side of CSDH	Factors in Hemorrhage	and Type of Spinal SDH	Cranial	Spinal	Prognosis	Etiology
Silver et al. (1991) ¹⁸	14/F	VP shunt	Cranial (2 weeks)	Headache, lumbago, bil. leg pain	bil.	—	T12-L2/ND	obs	obs	Good	Migration
Wurm et al. (1996) ²⁰	16/M	VP shunt	Simultaneous	Lumbago, bil. leg pain, bil. leg sensory dist.	bil.	—	L3-S2/U	obs	obs	Good	Migration
Leber et al. (1997) ¹¹	54/M	Trauma (back and occiput)	Cranial (ND)	Headache, lumbago	bil.	—	L1-S2/J	obs	Surgery	Good	Coincidence
Tillich et al. (1999) ¹⁹	54/M	Trauma (back and occiput)	Cranial (2 weeks)	Headache, lumbago, bil. S1 and S2 radiculopathy	bil.	—	L1-S2/J	obs	Surgery	Good	Coincidence
Ohta et al. (2001) ¹⁷	10/F	VP shunt	Simultaneous	Lumbago, paraparesis	bil.	—	L3-S2/U	obs	Surgery	Good	Migration
Yamaguchi et al. (2005) ²¹	59/M	Spontaneous	Simultaneous	Lumbago, paraparesis bil. leg numbness	bil.	APM	T11-S1/J	obs	obs	Good	Migration
Morishige et al. (2007) ¹⁴	54/M	Spontaneous	Simultaneous	Headache, lumbago	Left	—	C1-S2/J	Irrigation	Puncture and drainage	Good	Migration
Kishen et al. (2009) ⁸ (head)	76/M (4 days)	Trauma	Spinal	bil. leg pain	Left	APM	L4-5/VL	Irrigation	obs	Good	Coincidence
Nakajima et al. (2009) ¹⁶	65/F	Trauma (back and occiput)	Cranial (2 weeks)	Gait dist., bil. leg pain	Left	—	T12-S1/RJ	Irrigation	obs	Good	Coincidence
Yang et al. (2009) ²²	35/M	Spontaneous	Simultaneous	Headache, dizziness, lumbago, paraparesis	Left	—	L3-S1/J	Irrigation	Surgery	Good	Migration
Hagihara et al. (2010) ⁴	47/M	Trauma (head)	Cranial (2 months)	Right hemiparesis, lumbago	Left	APM	L3-S1/DL	Irrigation	obs	Good	Migration
Kim et al. (2010) ⁶	24/M	Trauma (back and occiput)	Simultaneous	Headache, lumbago, right sensory dist., aphasia	bil.	-	L4-S2/J	obs	obs	Good	Migration
Nagashima et al. (2010) ¹⁵	66/M	Spontaneous	Spinal (2 days)	bil. leg pain, paraparesis, incontinence	bil.	-	L2-S1/DL	Irrigation	obs	Good	Migration
Nagashima et al. (2010) ¹⁵	60/M	Trauma (head)	Cranial (3 days)	Headache, lumbago	bil.	Low platelet	L5-S2/DL	Irrigation	obs	Good	Migration
Jibu et al. (2012) ⁵	73/M	Spontaneous	Simultaneous	bil. sciatic pain	bil.	_	L3-S2/DL	obs	obs	Good	Migration

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CONCOMITANT INTRACRANIAL AND SPINAL SUBDURAL HEMATOMA

Moon et al. (2013) ¹³	39/F	Spontaneous	Spinal (1 day)	Headache, lumbago, bil. leg pain	Left	I	L4-S2/U	Irrigation	obs	Good	ND
Kokubo et al. (2014) ⁹	83/F	Trauma (head and back)	Simultaneous	No symptoms caused by spinal SDH	bil.	SOM	L3-S1/DL	Irrigation	sqo	Good	Coincidence
Kokubo et al. (2014) ⁹	M/07	Trauma (head and back)	Simultaneous	No symptoms caused by spinal SDH	bil.	I	L4-S2/DL	Irrigation	sqo	Good	Coincidence
Lin et al. (2014) ¹²	M/07	Spontaneous	Simultaneous	Lumbago, paraparesis	bil.	I	L4-S1/DL	Irrigation	obs	Good	Migration
Kim et al. (2015) 7	82/M	Trauma (back and occiput)	Cranial (4 days)	Drowsy mentality, gait dist., lumbago, paraparesis	bil.	I	T10-S1/DL	Irrigation	sqo	Good	Migration or coincidence
Kwon et al. (2015) ^{†0}	57/M	Trauma	Spinal	Lumbago, bil. leg pain	Left	I	L5-S1/J	Irrigation	surgery	Good	Migration
Present case	58/M	Trauma (back and occiput)	Cranial (3 days)	Headache, lumbago, left hemiparesis	Right	I	T1-S1/J	Irrigation	surgery	Good	Migration
CSDH, chronic subdural hematoma: SDH, subdural hematoma: F, female: VP, ventriculoperitoneal; bil., bilateral; ND, not described; obs, observation; M, male; dist., disturbance; U, U-shaped; J, J-shaped; APM, antiplatelet medication; DL, dorsal localized; MDS, myelodysplastic syndrome; VL, ventral localized; RJ, reverse J-shaped. *Time lag between detection of intracranial CSDH and spinal SDH.	s; SDH, subdural :ic syndrome; VL, intracranial CSDH	hematoma; F, female; , ventral localized; RJ, H and spinal SDH.	VP, ventriculoperitoneal; , reverse J-shaped.	bil., bilateral; ND, not descri	ibed; obs, obse	rvation; M, male; d	ist., disturbance; U	, U-shaped; J,	J-shaped; APM, ant	iplatelet medic	ation; DL, dorsal

Among 21 patients, the lesion was J-shaped in 8 patients,^{6,10,11,14,19,21,22} U-shaped in 3 patients,^{13,17,20} reverse J-shaped in 1 patient,¹⁶ dorsal localized in 8 patients,^{4,5,7,9,12,15} and ventral localized in 1 patient.⁸ According to the classification, hematomas are mainly located in the dorsal portion of the spinal canal. In particular, the group in which migration is considered as the etiology shows this tendency. On the other hand, the group in which the etiologies are considered to be coincidence showed various hematoma shapes.

In many cases, irrigation via 1 burrhole craniotomy is chosen for intracranial CSDH.^{4,7-10,12-14,16,22} On the other hand, conservative therapy tends to be chosen for spinal SDH because the clinical symptoms are mild.4,5,7-9,12,13,16,18,20,21 In fact, resolution of spinal SDHs has been reported in many cases. Because spinal SDHs contain liquid blood and can move freely, particularly when the spinal SDHs migrate from the cranial lesion, absorption of the hematoma or a decrease in the mass effect may be likely. However, when symptoms are progressive, surgical interventions, such as laminectomy and lumbar puncture, are performed.^{10,11,14,17,19,22} According to our classification of hematoma shapes, the operative cases are divided into J- and U-shaped lesions in which the nerve roots of the cauda equina are compressed from both the ventral and dorsal aspects. In fact, of the 8 patients with J-shaped hematoma, 6 patients (75%) underwent surgical intervention, and of the 3 patients with U-shaped hematoma, 1 patient (33%) underwent surgical intervention. Our classification of hematoma shape may therefore help in deciding treatment strategies. In the present case with a Jshaped spinal SDH, because lumbago was severe and gait disturbance persisted because of right lower-limb weakness, we chose hematoma evacuation via laminectomy. As a result, clinical symptoms improved immediately. The prognosis for recovery is reported to be good if the clot is evacuated before severe compression leads to ischemic changes.¹⁴ Surgical should therefore intervention be performed if the clinical symptoms persist or deteriorate, especially in J- or U-shaped SDH.

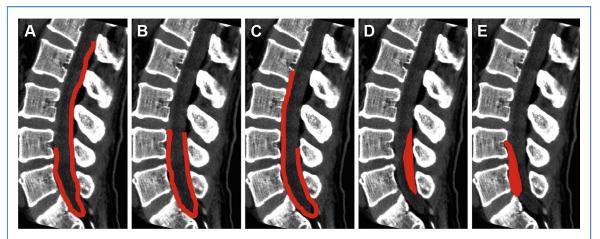


Figure 6. Classification of spinal subdural hematoma (SDH) in sagittal view. (**A**, **B**, and **C**) SDH is located in the ventral and dorsal spinal canal. (**A**) J-shaped: dorsal SDH is longer than ventral SDH. (**B**) U-shaped: dorsal SDH is the same length as

ventral SDH. (**C**) Reverse J-shaped: ventral SDH is longer than dorsal SDH. (**D**) dorsal localized: SDH is located in the dorsal spinal canal. (**E**) Ventral localized: SDH is located in the ventral spinal canal.

CONCLUSIONS

Coexistence of intracranial CSDH and spinal SDH is rare and should be suspected in patients with intracranial CSDH showing lumbago and neurologic symptoms in the lower limbs. For intracranial CSDH, irrigation via single burrhole craniotomy is chosen in many cases. Although conservative therapy tends to be chosen for spinal SDH because the clinical symptoms are mild, surgical intervention should be performed if clinical symptoms persist or deteriorate. Our suggested classification of the shape of spinal SDHs on sagittal MRI may help in deciding treatment strategies.

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Conflict of interest statement: The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received 1 January 2016; accepted 8 March 2016

Citation: World Neurosurg. (2016). http://dx.doi.org/10.1016/j.wneu.2016.03.020

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

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