

CASE REPORT

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Spinal Subdural Hematoma Associated With Traumatic Intracranial Interhemispheric Subdural Hematoma

—Case Report—

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Abstract

A 78-year-old female fell and hit the back of her head on the floor. Head computed tomography (CT) showed right acute interhemispheric subdural hematoma (ISDH). Her left hemiparesis worsened, so partial removal of ISDH was performed. The hemiparesis was improved, but leg monoparesis persisted. Lumbar magnetic resonance imaging showed spinal subdural hematoma (SSDH) at the S1-2 level. Nerve conduction velocity measurements at the knee joint to lower limb showed disappearance of the left peroneal nerve conduction wave, indicating that one of the causes of drop foot was common peroneal nerve palsy. With conservative therapy, her drop foot was gradually improved, then she recovered to walk with a stick and moved to a rehabilitation hospital. Lumbar MR imaging should be performed to rule out SSDH in a patient with posterior fossa subdural hematoma on initial head CT who develops leg palsy.

Key words: spinal subdural hematoma, intracranial interhemispheric hematoma, falx syndrome, trauma, drop foot

Introduction

Interhemispheric subdural hematoma (ISDH) is an uncommon complication of head trauma,^{1,2,17)} and accounts for approximately 6% of all traumatic subdural hematoma (SDH).^{2,17)} The male-to-female ratio in adult patients was 1.87:1.²⁾ The clinical characteristics include a high incidence of impact in the occipital or frontal regions, and a low incidence of skull fractures.²⁾ ISDH is usually unilateral and rarely bilateral. ISDH commonly develops anterior to the posterior half of the falx cerebri but may extend anteriorly and/or reach the subtemporo-occipital region, above the tentorium cerebelli.¹⁷⁾ The most distinctive clinical manifestations of ISDH are contralateral monoparesis of the leg, or hemiparesis if the leg is affected more than

the arm, which is known as falx syndrome.^{2,4,17)} Falx syndrome has been reported in approximately 30% of all patients with ISDH.¹⁴⁾ Spinal subdural hematoma (SSDH) concurrent with intracranial ISDHs is rare. Many patients with intracranial ISDHs are examined routinely only by head computed tomography (CT). Consequently, many SSDHs can be overlooked as a possible cause of leg weakness. We report a case of SSDH associated with intracranial ISDH.

Case Report

A 78-year-old female with osteoarthritis of the bilateral knees and currently receiving antiplatelet therapy fell and hit the back of her head on the floor. Two days later, she developed diffuse headache followed by repeated episodes of vomiting, and could not get out of bed for some hours.

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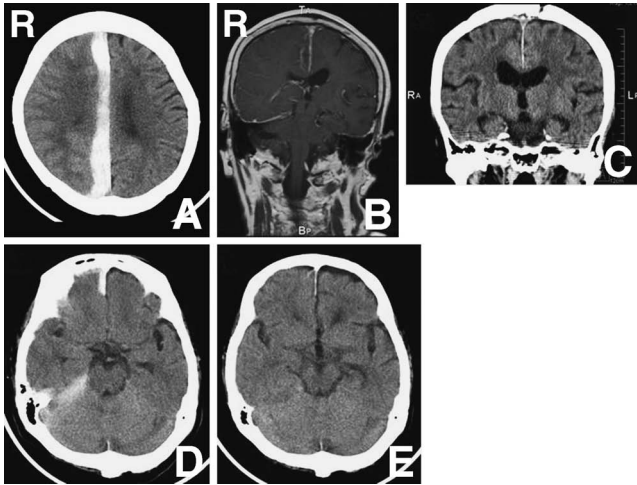


Fig. 1 Axial head computed tomography (CT) scans on admission showing a subdural hematoma in the right frontal and occipital interhemispheric fissure (A) and in the occipital fossa (D). Preoperative coronal T₁-weighted magnetic resonance image with gadolinium showing a subdural hematoma compressing the right motor cortex in the right frontal interhemispheric fissure (B). Postoperative coronal head CT scans showing disappearance of subdural hematoma in the right frontal interhemispheric fissure (C), as well as in the posterior fossa (E).

She was then transferred and admitted to our hospital. Physical examination revealed normal vital signs. She had normal pupils and was orientated. Slight left lower limb monoparesis was identified by the manual muscle test (MMT) 4+/5. Skull radiography identified no skull fractures. Head CT and brain T₁-weighted magnetic resonance (MR) imaging showed right ISDH extending along the full length of the interhemispheric fissure (Fig. 1A, B), and thin right frontal SDH in the posterior fossa (Fig. 1D). Five days after head trauma, she developed left partial seizures predominantly in the lower extremity, followed by left side hemiparesis (MMT upper extremity 4–/5, lower extremity 2/5). Lower extremity sensation was disturbed. Partial seizures were controlled with diazepam.

Six days after the head trauma, partial removal of the ISDH was performed through a parietal craniotomy. The hematoma within the right interhemispheric space was not liquid, but was easily removed with aspiration. In addition, no active bleeding from the bridging veins or other vessels was observed after removal of the hematoma. Postoperatively, her left hemiparesis and sensory disturbance were immediately improved. However, her right drop foot was persisted. Head CT, lumbar MR imaging, and nerve conduction velocity (NCV) measurements were performed after the operation. Head CT confirmed removal of the right ISDH, especially that compressing the



Fig. 2 Lumbar spinal magnetic resonance images showing subacute subdural hematoma at the S1-2 level after the removal of cranial subdural hematoma. Sagittal T₁-weighted image showing high signal intensity in the spinal subdural space (A). Sagittal T₂-weighted image showing high signal intensity in the spinal subdural space (B). Axial T₁-weighted image showing high signal intensity in the spinal subdural space at the S1-2 level (C). Sagittal T₁-weighted (D) and T₂-weighted images (E), and axial T₁-weighted image (F) at 2 months after head trauma showing the disappearance of spinal subdural hematoma.

right motor and premotor area (Fig. 1C), and no posterior fossa SDH was observed (Fig. 1E). Lumbar MR imaging showed SDH at the S1-2 level (Fig. 2A–C). NCV measurements from the knee joint to lower limb showed the disappearance of left common peroneal nerve conduction (Fig. 3B), indicating that one of the causes of her drop foot was peroneal nerve palsy. NCV was normal at the right knee joint (Fig. 3A). We thought that the severe osteoarthritis of the left knee and external rotation of the left knee for some hours had caused left peroneal nerve palsy resulting in drop foot state.

With conservative therapy, her drop foot gradually improved, she recovered to walk with a stick, and was moved to a rehabilitation hospital. NCV measurement from the knee joint to lower limb showed recovery of left common peroneal nerve conduction (Fig. 3D), NCV of the right knee joint was normal (Fig. 3C), and lumbar MR imaging showed disappearance of the SDH at the S1-2 level (Fig. 2D–F). This finding indicates that her drop foot was improved with resolution of the left common peroneal nerve palsy.

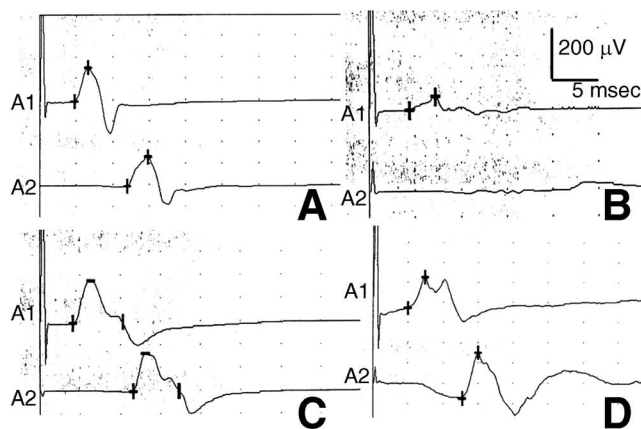


Fig. 3 Nerve conduction velocity (NCV) of the bilateral common peroneal nerves showing normal reaction in the right (A) and no reaction in left (B) associated with left drop foot. NCV showing normal reaction in both the right (C) and left (D) at 2 months after onset.

Discussion

Coagulopathy, anticoagulant therapy, and alcohol abuse have been recognized as predisposing factors for the development of ISDH.²⁾ Although the mechanism responsible for traumatic ISDH remains unclear, the generally accepted causative mechanism²⁾ for the development of ISDH is tearing of the fixed bridging veins between the medial cerebral cortex and the superior sagittal sinus. If a solid clot liquefies, it may migrate into the space over the convexity and behave like a chronic SDH.¹³⁾ In our case, antiplatelet therapy was the predisposing factor for developing of ISDH, but the anatomical findings responsible for ISDH remained unclear in this case.

ISDH is now considered to be a benign variant of acute SDH.^{2,14,17)} The mortality rate of only traumatic ISDH is 25% with further improvement in outcome.¹⁴⁾ Management of ISDH is controversial, with both conservative and surgical management advocated.^{1,2,4,13,14,17)} Conservative therapy is usually preferred if the patient's neurological condition is stable, or if any associated diseases contraindicate surgery. Surgical interventions ranging from parasagittal craniotomy and evacuation of hematoma in the acute phase to irrigation of SDH in the late phase have been tried. Removal of the clot has proved to be a viable option in the management of these patients, but some risk is due to the proximity of the superior sagittal sinus and bridging veins, so evacuating such hematomas through craniotomy without causing further trauma to the attached bridging veins can be difficult.

However, we believe that removal of the hematoma through craniotomy in the acute phase should be performed in patients with large acute SDH or rapidly deteriorating neurological status. In our case, removal of ISDH was necessary, because her left hemiparesis worsened after admission. Postoperatively, the left hemiparesis improved immediately. Though left drop foot persisted, this was considered to be caused mainly by peripheral nerve disturbance because of the absence of the NCV of the left peroneal nerve. Further studies are necessary to clarify whether surgery improves the outcome.

SSDH is also a rare condition like intracranial ISDH.^{3,5,6,8,9,12,16,18)} Some factors triggering or contributing

Table 1 Published cases of spinal subdural hematoma concurrent with traumatic intracranial subdural hematoma

Author (Year)	Age (yrs)	Sex	Posterior fossa lesion*	Cranial surgery	Spinal surgery	Recovery
Shimada et al. (1996) ¹⁶⁾	68	?	?	no	yes	full
Lee et al. (1996) ⁹⁾	15	male	no	no	yes	full
Leber et al. (1997) ⁸⁾	54	male	no	no	yes	full
Kirsch et al. (2000) ⁶⁾	42	male	yes	no	yes	poor
Hung et al. (2002) ⁵⁾	12	male	yes	no	no	full
Bortolotti et al. (2004) ³⁾	23	female	yes	no	yes	full
Sari et al. (2006) ¹⁵⁾	56	male	yes	no	yes	full
Wong et al. (2009) ¹⁸⁾	59	female	yes	no	no	full
Nagashima et al. (2010) ¹²⁾	73	male	yes	yes	no	full
Moscovici et al. (2011) ¹¹⁾	88	male	yes	no	yes	full
Present case	78	female	yes	yes	no	full

*Cranial subdural hematoma was detected in the posterior fossa.

to the development of SSDH have been reported.¹⁸⁾ In addition, migration from the cerebral subdural area to the spinal subdural space is a very likely cause of SSDH.^{3,5,11)} However, cases of SSDHs concurrent with traumatic intracranial SDHs are extremely rare, with only 11 reported cases^{3,5,6,8,9,11,12,15,16,18)} including our case (Table 1). In our case, slight posterior fossa SDH was observed on the initial head CT, but the hematoma disappeared with time. SSDH did not increase during this time. The relationship between posterior fossa SDH and SSDH is unclear, although posterior fossa SDH can be a cause of SSDH. This condition should be considered in patients with neurological signs derived from a brain lesion, to decide if surgery is indicated. Posterior fossa SDH was observed in most cases (Table 1), as well as in our case. However, the volume was so slight that whether the posterior fossa SDH caused the SSDH was unclear.

A meta-analysis⁷⁾ demonstrated that most patients (85.5%) with symptomatic SSDH underwent decompression surgery and that factors influencing surgical outcome were timing of surgical decompression of the spinal cord, preoperative status, rate of development of the clinical presentation, segmental localization, extension of SSDH, and age of the patient. However, spontaneous resolution of SSDH has also been reported.^{5,12,18)} In our case, complete resolution of the possibly asymptomatic SSDH was confirmed on MR imaging 2 months after onset. Severe osteoarthritis of the knee could present with drop foot caused by disturbance of the anterior tibial and peroneal compartments.¹⁰⁾ Therefore, we believe that decompression surgery for SSDH should be indicated only for patients with moderate or severe paraparesis, or deteriorating paraparesis.

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