Case Report

Cerebrospinal Fluid Leakage at the Lumbar Spine: A Unique Cause of Delayed Neurologic Deterioration in a Traumatic Acute Subdural Hematoma Patient

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Abstract

We present a rare case of traumatic acute subdural hematoma (SDH) in which intracranial hypotension (IC) secondary to cerebrospinal fluid (CSF) leakage at the lumbar spine caused delayed neurological deterioration. A 70-year-old male sustained a head injury after ground-level fall and was brought to our institution. A brain computed tomography (CT) showed a thin acute SDH with mild brain shift, and conservative management was initiated. He exhibited neurological deterioration on the 9th hospital day, however, and a brain CT showed a change in CT density and marked an increase in hematoma volume and brain shift. It was thought that conversion from acute to subacute SDH was associated with the deterioration, and emergency hematoma evacuation was performed. Despite the surgery, neither clinical nor radiographical improvement occurred. The lack of improvement pointed to the presence of underlying IC, and a CT myelography revealed the dural sleeve of the right L2 nerve root as the source of the CSF leakage. An epidural blood patch therapy was performed on the 12th hospital day to seal the CSF leakage. The postprocedural course was uneventful, and the patient was discharged free of symptoms on the 20th hospital day. Spinal CSF leakage should be considered as a cause of delayed neurological deterioration in patients with traumatic acute SDH which exhibits conversion to subacute SDH.

Keywords: *Acute subdural hematoma, cerebrospinal fluid leakage, intracranial hypotension, lumbar spine, subacute subdural hematoma*

Introduction

Prognosis of traumatic acute subdural hematoma (SDH) patients is determined mostly by hematoma volume, concomitant brain parenchymal damage, and patient age.[1-4] For patients who are neurologically intact and have only a small hematoma volume on imaging studies, conservative management is justified: acute SDH is absorbed gradually in such circumstances.[4] However, caution is required in cases which exhibit conversion of SDH from acute to subacute stage because delayed hematoma increase and neurological deterioration may occur.[5-8] Such deterioration can be managed with timely surgical intervention, that is, hematoma evacuation, and patient outcomes are mostly favorable.[5-8] We present a rare case of traumatic acute SDH in which intracranial hypotension (IC) secondary to cerebrospinal fluid (CSF) leakage at the lumbar spine was responsible for delayed neurological deterioration.

Case Report

A 70-year-old male with a history of a lacunar stroke sustained a ground-level fall and was brought to the emergency department. His medications did not include antiplatelets or anticoagulants. Although he remained fully conscious after the fall and was neurologically intact, a brain computed tomography (CT) was performed due to his complaint of a headache. The brain computed tomography (CT) showed no specific findings at the level of basal cistern [Figure 1a]. However, it showed a thin acute SDH of the right side with brain shift of < 5 mm [Figure 1b], and he was admitted to a neurosurgical ward for observation. There was no increase in the hematoma volume on the second CT performed 6 h after the initial CT. Conservative management was considered appropriate, and his headache subsided gradually. However, his headache recurred on the 7th hospital day, and on the 9th day, he became stuporous: he was E3V3M5 on the Glasgow Coma Scale.

How to cite this article: Mayanagi K, Nakatsukasa M, Inamasu J. Cerebrospinal fluid leakage at the lumbar spine: A unique cause of delayed neurologic deterioration in a traumatic acute subdural hematoma patient. Asian J Neurosurg 2019;14:219-22.

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Access this article online

Website: www.asianjns.org

DOI: 10.4103/ajns.AJNS_185_17

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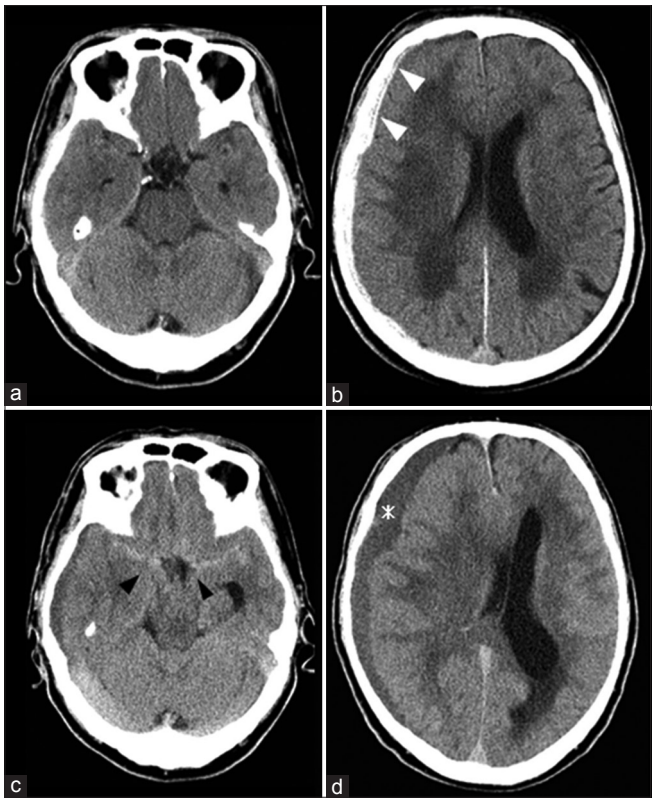

Figure 1: At the time of admission, brain computed tomography showed no specific findings at the level of the basal cistern (a). At the level of foramen semiovale, a small amount of acute subdural hematoma on the right side with minimal brain shift was noted (b, arrowheads). A computed tomography after neurologic deterioration showing a high density at the level of the basal cistern (c, arrowheads) and subacute subdural hematoma which increased markedly causing severe brain distortion (d, asterisk)

A brain CT revealed an isodensity SDH with a midline shift of >10 mm [Figure 1d]. Impending transtentorial herniation due to increased SDH was thought to be responsible for the deterioration, and a small right-sided craniotomy to evacuate the hematoma was performed emergently. No brain swelling was observed intraoperatively. Despite the surgery, he failed to improve neurologically, and postoperative CT showed that there was no improvement in the brain shift [Figure 2a]. Accumulation of intracranial air was also noted [Figure 2a, asterisk]. His consciousness level further deteriorated to E1V1M3 on the next day after surgery. Lack of neurological and radiographical improvement pointed to the presence of underlying IC. Review of the previous CT revealed a high density around the basal cistern, which had been overlooked [Figure 1c, arrowheads]. To prevent further deterioration, a volume of 40 mL saline was infused intrathecally through the spinal needle. The CSF pressure measured before saline infusion was 2 cm H_2O, confirming the diagnosis of IC. His consciousness level improved to E4M4V6 immediately after the saline infusion. A CT myelography revealed the dural sleeve of the right L2 nerve root as the source of CSF leakage [Figure 3a and b]. An epidural blood patch therapy was performed on the 12th hospital day to seal

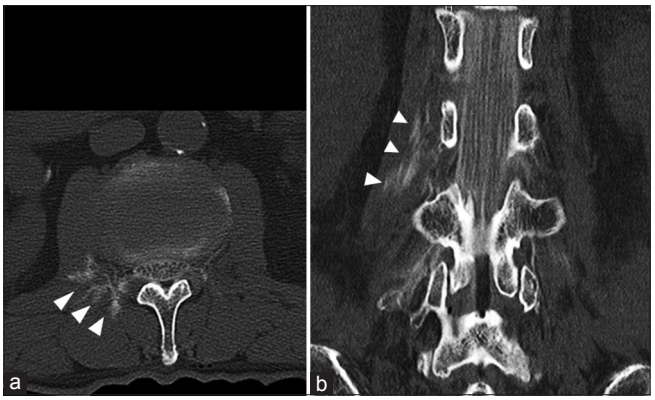

Figure 2: A postoperative computed tomography showing lack of improvement in brain shift despite the evacuation of hematoma (a). Accumulation of intracranial air was noted (asterisk). A computed tomography obtained a month after discharge showing reversal of brain shift (b)

the CSF leakage: a volume of 40 mL autologous blood was administered through an 18-G Tuohy needle inserted from the L3–L4 level. The postprocedural course was uneventful: he became fully conscious 2 days after the blood patch therapy and was discharged free of symptoms on the 20th hospital day. A brain CT performed 1 month after discharge showed a reversal of brain shift [Figure 2b]. He has not sustained recurrence of SDH for 6 months. Written consent from the patient to be enrolled and to have his data published was obtained. Publication of this case report was also approved by the Institutional Ethics Committee.

Discussion

Conservative management is appropriate for patients with traumatic acute SDH without neurological deficits and radiographical evidence of brain compression because acute SDH is absorbed or sequestered, and disappears eventually in most cases.[1-4] However, delayed neurological deterioration due to hematoma increase during transition from acute to subacute stage has been reported in the literature,[5-8] with varying degree of frequency: while Bajsarowicz reported that only 6.5% of acute SDH patients who received initial conservative management sustained delayed hematoma increase requiring hematoma evacuation,[6] the frequency was as high as 35% in a cohort reported by Kim *et al*.[7] The mechanism of hematoma increase during transition from acute to subacute stage has been attributed to invasion of CSF into hematoma driven by osmotic gradient generated by the fibrinolytic products in the hematoma.[5] Emergency hematoma evacuation was almost invariably effective in such circumstances.[5-8] In this context, the present case is unique in that IC was the cause of hematoma increase and neurological deterioration during transition from acute to subacute stage.

It has been reported that IC can be the underlying cause of SDH.[9-14] Inadvertent hematoma evacuation may not only be ineffective but also be harmful in such circumstances

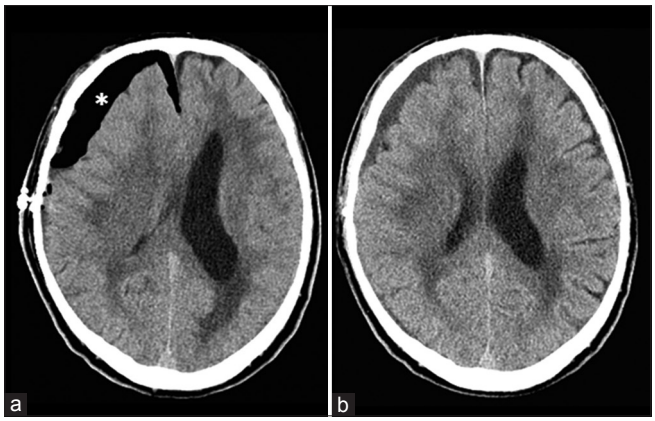

Figure 3: Computed tomography myelography showing the dural sleeve of the left L2 nerve root as the source of cerebrospinal fluid leakage (arrowheads: a, axial image; b, sagittal image)

Table 1: Summary of reported cases of intracranial hypotension with spinal dural tear at the root sleeve

Author	Year of publication	Spinal level
Pleasure *et al*.[15]	1998	Lumbar
Schievink and Jacques[16]	2003	Cervical
Watanabe *et al*.[17]	2009	Thoracic
Ohtonari *et al*.[18]	2009	Lumbar
Cheshire and Wharen[19]	2009	Cervical
Hasiloglu *et al*.[20]	2012	Thoracic/thoracic
Tontisirin *et al*.[21]	2018	Thoracic
Present case	2018	Thoracic

because further decrease in the intracranial pressure after hematoma evacuation may pull cortical bridging veins downward, resulting in venous wall laceration and acute SDH.[9-14] Interestingly, those cases have manifested almost invariably as chronic SDH from the beginning.[9-14] Spinal CSF leakage as the cause of conversion to subacute symptomatic SDH, as shown in the present case, has never been reported in the literature. The present case is educational to neurosurgeons in that spinal CSF leakage be differentiated as the cause of delayed hematoma increase and neurological deterioration in patients with traumatic acute SDH. In retrospect, earlier recognition of high density around the basal cisterns on preoperative CT [Figure 1c], which represents venous engorgement secondary to IC, may have led to diagnosis of IC. Although it remains unclear when and how the spinal CSF leakage developed in the present case, mechanical stress to the lumbar spine at the time of his fall to the ground may have created a tear in the spinal arachnoid membrane and overlying dura mater, resulting in IC. While dural tear may occur anywhere along the spinal axis, laceration, and subsequent CSF leakage at the nerve root sleeve may be relatively rare: PubMed search using the keywords IC and root sleeve yielded only seven previous cases [Table 1].[15-21] As shown in Figure 2b, reconstructed coronal view of the CT myelogram may particularly be useful to identify CSF leakage occurring at the root sleeve.

Conclusion

Recognition of IC as a possible cause of delayed hematoma increase is important in the treatment of traumatic acute SDH. Lack of clinical improvement or reversal of brain shift after hematoma evacuation in patients with subacute SDH should prompt an immediate search for CSF leakage at the spine.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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