Subacute spinal subdural hematoma after spontaneous resolution of cranial subdural hematoma: causal relationship or coincidence?

Case report

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The etiopathogenesis of traumatic spinal subdural hematoma (SSH) is uncertain. Unlike the supratentorial subdural space, no bridging veins traverse the spinal subdural space. The authors describe a case of subacute SSH that occurred after spontaneous resolution of traumatic intracranial SDH and suggest a causal relationship between the two.

A 23-year-old woman suffered an acute intracranial SDH after a snowboarding accident. There was no clinical or radiological evidence of spine injury. Conservative management of the supratentorial SDH resulted in spontaneous radiologically documented resolution with redistribution of blood in the subdural space.

Four days after the injury, the patient started noticing new onset of mild low-back pain. The pain progressively worsened. Magnetic resonance imaging of the lumbosacral spine 10 days after the original injury revealed a large L4–S2 SDH. Ten days after the original injury, bilateral L5–S1 laminotomy and drainage of the subacute spinal SDH were performed. The patient experienced immediate pain relief.

The authors hypothesize that in some cases spinal SDH may be related to redistribution of blood from the supratentorial subdural space.

KEY WORDS • spinal subdural hematoma • intracranial subdural hematoma • trauma

Spinal SDH is a rare condition usually related to blood dyscrasia, anticoagulation, lumbar puncture, and vascular malformations. In rare cases spinal SDH is the result of trauma. In a recent paper Hung and coworkers reviewed nine cases including data in one of their own. They noticed an association between spinal SDH and intracranial injury in five of the nine cases. No convincing explanation of such an association was established. We report a case of spontaneous resolution of traumatic intracranial SDH with subsequent subacute spinal SDH and propose an alternative theory of this association.

Case Report

History and Examination. This 23-year-old woman presented to an outside institution after a snowboarding accident. A CT scan of the head demonstrated a large acute SDH with midline shift (Fig. 1). Plain spinal radiographs revealed no abnormality and the patient did not complain of any back pain or tenderness. Because of her stable clinical condition (Glasgow Coma Scale Score 15), no immediate surgical treatment was undertaken. Instead she underwent follow-up head CT scanning 12 hours later; the studies revealed dramatic improvement with almost complete resolution of the convexity SDH and midline shift. This CT study also demonstrated layering of blood along the tentorium, much more than the original scan (Fig. 2). She was treated conservatively and discharged 3 days later.

Four days following the injury, she began noticing new onset mild low-back pain, which progressively worsened, and she started suffering pain radiating to both lower extremities as well as intermittent leg paresthesias. Lumbosacral MR imaging 10 days after the original injury revealed no evidence of soft-tissue injury. A large SDH, however, was demonstrated extending from L-4 to S-2 (Fig. 3).
toms, the hematoma was evacuated. After bilateral L5–S1 laminotomy and removal of the ligamentum flavum, no epidural blood was visualized. The dura mater appeared to have a dark blue discoloration, was tense, and pulseless. A small 3-mm midline incision was made through the dura by using a No. 11 blade with care to preserve the integrity of the underlying arachnoid. After opening the dura, subacute dark red/brownish blood under pressure spontaneously drained. Using a No. 4 Penfield dissector, the underlying arachnoid was gently compressed circumferentially around the dural opening, and with gentle microirrigation additional uncoagulated dark red blood was washed out. After decompressing the subdural space, the underlying arachnoid was visualized. There was no CSF leakage and no active site of bleeding in the subdural space. Through the intact transparent arachnoid, clear CSF could be visualized, and there was no macroscopic evidence of blood in the subarachnoid space.

**Postoperative Course.** Several hours after surgery, MR imaging revealed almost complete drainage of the SDH (Fig. 4). The patient experienced immediate relief of her pain and was discharged home 2 days after surgery.

**Discussion**

Traumatic spinal SHs are rare. Of 10 reported cases (including our own), six have been associated with intracranial injury. The genesis of traumatic spinal SDHs is difficult to explain in the absence of any violation of the dural layer. Unlike the intracranial subdural space, there are no bridging veins running in the spinal subdural space. Therefore, the true etiopathogenesis of spinal SDH remains obscure. The authors of several reports have described the spontaneous resolution of acute intracranial SDH. The phenomenon of spontaneous resolution of intracranial acute SDHs seems to be more common in children and young adults. It is hypothesized that the “elastic” young brain forces redistribution of the subdural blood. Spontaneous resolution of intracranial acute SDH has also been related to a dilutional effect exerted on the subdural blood by CSF when small arachnoidal tears occur. In our case, follow-up cranial CT scanning, performed 12 hours after the initial study, revealed remarkable reduction of the originally large SDH and redistribution of the blood along the tentorium (Fig. 2). Four days after the injury, our patient started complaining of mild low-back pain progressively increasing in severity over the ensuing week. Eventually the pain radiated to both lower extremities and she noticed bilateral leg paresthesias.

In view of the clinical history (absence of back pain immediately after the injury as well as delayed onset and progressive deterioration), the dramatic resolution of the acute intracranial SDH with redistribution of blood, and the intraoperative findings (subacute subdural blood, easily drained through a small dural incision), we hypothesize that the spinal SDH might have been related to progressive migration of the subdural blood to the most dependent areas of the lumbosacral region. As the subdural blood volumetrically expanded secondary to chronic

![Fig. 1. Postinjury noncontrast brain CT scans revealing SDH with marked midline shift.](image1)

![Fig. 2. Noncontrast brain CT scans obtained 12 hours after initial scans, demonstrating almost complete resolution of the convexity hematoma and layering of blood along the tentorium.](image2)

![Fig. 3. Neuroimages of the lumbosacral spine, acquired 10 days after the original injury. Left: Axial T₁-weighted MR image revealing a large SDH compressing the dural sac at L5–S1. Right: Sagittal T₁-weighted MR image demonstrating the large SDH extending from L-4 to S-2.](image3)

![Fig. 4. Postoperative neuroimages obtained several hours after drainage of the spinal SDH. Left: Axial T₂-weighted MR image demonstrating almost complete resolution of the hematoma at the same level as that in Fig. 3 left. Right: Sagittal T₂-weighted image confirming almost complete drainage.](image4)
changes, the patient began experiencing progressive low-back pain. This theory is supported by the electron microscopic observation of an anatomical continuity between the intracranial and spinal subdural space.\textsuperscript{14,18} It is possible that some cases of spinal SDH can be related to redistribution of intracranial blood to dependent regions such as the spinal subdural space.

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References


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