Spinal Subdural Hematoma Following Intracranial Aneurysm Surgery
—Four Case Reports—

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Abstract
Four patients presented with rare spinal subdural hematoma (SDH) occurring after intracranial aneurysm surgery and manifesting as postoperative back pain. Magnetic resonance imaging performed from 4 to 11 days after the operation showed acute or subacute thoracolumbar SDH. No patient had risk factors for bleeding at this site (e.g., lumbar puncture, coagulation abnormality). Overdrainage of the cerebrospinal fluid (CSF) was performed for brain retraction during the operation in all four cases. Computed tomography performed during the postoperative period showed a suspicious tentorial subdural hemorrhage in Case 1 and an interhemispheric subdural hemorrhage in Case 3. All four patients received conservative management and their lumbago improved. We hypothesize that CSF hypotension due to overdrainage of CSF and downward migration of intracranial SDH under the influence of gravity were involved in the formation of spinal SDH.

Key words: spinal subdural hematoma, migration, craniotomy

Introduction
Spinal subdural hematoma (SDH) is a rare condition, but the frequency is probably underestimated since reports have become more common since magnetic resonance (MR) imaging became available. Causative factors include lumbar puncture, surgery, trauma, bleeding diatheses, and vascular malformations. The small number of reports of spinal SDH after intracranial surgery have suggested diverse pathomechanisms including gravity-induced downward movement of blood from the cranial compartment, excessive decompression of the intracranial space during surgery, and reduced cerebrospinal fluid (CSF) pressure in drainage or shunt procedures.

We experienced four patients who suffered back pain due to spinal SDH after intracranial aneurysm surgery.

Case Reports

I. Patients and clinical characteristics
A total of 75 patients underwent aneurysm surgery (55 surgical and 20 endovascular) between January 2004 and December 2005. Four patients, one man and three women aged 34 to 60 years, presented with spinal SDH following intracranial aneurysm surgery. No patient had a history of recent significant trauma or perioperative lumbar puncture. Laboratory findings were normal without signs of inflammatory disease or coagulation disorder. The Hunt and Hess grade at admission was I in one patient, II in two, and 0 (unruptured aneurysm) in one (Case 3). Two aneurysms were located on the anterior communicating artery, one at the right middle cerebral artery bifurcation, and one at the right internal carotid artery bifurcation (Table 1). All four patients underwent aneurysm neck clipping through the pterional approach. In all four cases, overdrainage of the CSF was performed for brain retraction during the operation. Extraventricular drainage was performed in Cases 1 and 4. The patients started walking at 3 to 7 days after the operation, after which all patients complained of back and lower extremity pain.

II. Radiological characteristics and outcome
MR imaging performed at 4 to 11 days after operation showed acute or subacute spinal SDH at the thoracolumbar level. Computed tomography (CT)
Table 1  Summary of clinical and radiological features in four patients with spinal subdural hematoma (SDH) following intracranial aneurysm surgery

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)/Sex</th>
<th>Location of aneurysm</th>
<th>Hunt and Hess grade</th>
<th>Fisher grade</th>
<th>Time of operation (days)</th>
<th>Location of spinal SDH</th>
<th>MR imaging of spinal SDH</th>
<th>Time of MR imaging (days)</th>
<th>Follow-up period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50/M</td>
<td>AcomA</td>
<td>I</td>
<td>1</td>
<td>20</td>
<td>below T-11 level</td>
<td>T2 low, T1 iso</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>39/F</td>
<td>MCA bifurcation</td>
<td>II</td>
<td>2</td>
<td>1</td>
<td>L4–5 levels</td>
<td>T2 iso, T1 high</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>34/F</td>
<td>ICA bifurcation</td>
<td>0 no blood</td>
<td>3</td>
<td>2</td>
<td>below T-12 level</td>
<td>T2 high, T1 high</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>60/F</td>
<td>AcomA</td>
<td>II</td>
<td>3</td>
<td>1</td>
<td>S-1 level</td>
<td>T2 high, T1 high</td>
<td>10</td>
<td>1</td>
</tr>
</tbody>
</table>


Fig. 1 Case 3. Preoperative right carotid angiogram showing an unruptured aneurysm of the right internal carotid artery bifurcation (A), and left carotid angiogram showing a residual neck of the anterior communicating artery aneurysm (B).

Fig. 2 Case 3. Postoperative computed tomography scan showing a subdural hematoma in the frontal interhemispheric fissure.

performed during the postoperative period showed suspicious tentorial subdural hemorrhage in Case 1 and interhemispheric subdural hemorrhage in Case 3. All four patients underwent conservative treatment because the neurological signs were mild (only back and radiating pain). The follow-up period ranged from 1 month to 8 months (Table 1). At the last follow up, all patients were Glasgow Outcome Scale 5. All patients showed marked improvement of back pain and were discharged walking with intact bladder control.

III. Illustrative case (Case 3)

A 34-year-old woman with a history of neck clipping of a ruptured anterior communicating artery aneurysm 2 years before presented to our outpatient department with an unruptured aneurysm of the right internal carotid artery bifurcation. Neurological examination found no abnormality except for left eye blindness. She had no coagulation disorders. Cerebral angiography demonstrated a residual neck of the anterior communicating artery aneurysm and an unruptured small aneurysm of the right internal carotid artery bifurcation (Fig. 1).

She underwent reclipping of the residual neck of the anterior communicating artery aneurysm and clipping of the neck of the aneurysm of the right internal carotid artery bifurcation through a right pterional approach. No traumatic procedures were performed on the spinal canal during the operation or the postoperative period. CT obtained 1 hour after the operation demonstrated a SDH in the frontal
interhemispheric fissure (Fig. 2). She complained of lumbago persisting for 7 days. MR imaging of the lumbosacral spine obtained 11 days after the surgery showed a subacute-stage SDH below the T-12 level (Fig. 3). She underwent conservative treatment. Her neurological condition remained stable with decreased back pain. She had no back pain 1 month later.

**Discussion**

The pathogenesis of spinal SDH remains unclear, as the spinal subdural space lacks bridging veins. The only vessels of substantial size are the radiculomedullary artery and its corresponding vein, which pierce the dural sac, often above the L-3 nerve root. A sudden increase in abdominal and thoracic pressure may raise the pressure in the spinal vessels as they cross the subdural and subarachnoid spaces. If the pressure of the CSF cannot immediately neutralize this force, rupture of the vessels could ensue. Another theory is that spinal SDH originates within the subarachnoid space and subsequently dissects into the subdural space. Otherwise, spontaneous SDHs may be the isolated result of subarachnoid hemorrhage.

We hypothesize that spinal SDH might be related to progressive migration of the subdural blood to the most dependent areas of the lumbosacral region. This theory is supported by electron microscopic observation of an anatomical continuity between the intracranial and spinal space. Moreover, experiments showed that under normal conditions there is no evidence of the so-called 'subdural space.' When animals are subjected to subdural infusion of blood or bleeding takes place into the dural border cell layer, it splits without any particular predestined cleavage plane, although most often the cleavage passes close to the fibrous matter of the dura. The bleeding extends throughout the cerebral and spinal parts of the dural border cell layer and along the spinal nerve roots. We consider that the present cases of spinal SDHs represent an extension of intracranial subdural blood into the spinal subdural space.

Low CSF pressure because of overdrainage might also be important role in the migration of hematoma through the spinal canal. Overdrainage of CSF may lead to a collection of blood or CSF in the meninges. As the brain slowly shrinks with CSF drainage by extraventricular drainage or drainage during operation, tension is put on the dural border cell layer and on the points where the veins traverse the dura. This is because the structural continuation of the pia and arachnoid mater follow the brain, whereas the dura remains attached to the skull. Once again, the stress is concentrated on the dural border cell layer and is relieved when this layer splits open. Based on the morphological relationships between dural border cells and the walls of veins, shearing of the dural border cell layer may extend into the vein or a tear in the wall of a traversing vein may result in extravasated blood dissecting the dural border cell layer. This view is entirely consistent with models of subdural hemorrhage. In the presence of a preexisting subdural space, blood would certainly pool around the basilar aspect of the brain or even drain into the spinal meninges.

A patient presented with a symptomatic lumbar SDH, revealing a cranial SDH associated with a hemorrhagic cortical brain metastasis. The lumbar hematoma probably originated from the intracranial bleeding, which was substantiated by MR imaging observation of a thin hemorrhagic collection connecting the cranial and lumbar hematomas. A 4-year-old child presented with a clival and spinal SDH after a fall from a fourth-story window. The images supported the hypothesis that redistribution...
of the clival SDH to dependent areas in the spinal subdural space is a significant mechanism in the evolution of these lesions.

In all four cases in this report, overdrainage of CSF was performed for brain retraction during the operation. Spinal MR imaging in all four patients showed acute or subacute spinal SDH consistent with the time interval after the operation. There were no defects in hemostatic mechanisms, use of anticoagulants, history of spinal puncture or epidural block, intraspinal tumors, or spinal vascular anomalies. CT found suspicious tentorial subdural hemorrhage in Case 1 and interhemispheric subdural hemorrhage in Case 3 during the postoperative period. Therefore, we hypothesize that both CSF hypotension due to overdrainage of CSF and downward migration under the influence of gravity were involved in the formation of spinal SDH.\(^5\)

In our opinion, intraoperative or postoperative drainage with ensuing loss of CSF may be hazardous, especially during aneurysm surgery in patients with low grade or unruptured aneurysms.

Although the mechanism for the development of the spinal SDH was not definitely proven in the present cases, we would like to emphasize the possibility of blood spreading from the intracranial space to the spinal subdural space with CSF hypotension. After aneurysm surgery, the patient should be monitored neurologically for spinal findings as well as cranial signs. Consideration must be given to patients who show inappropriate signs of pain and muscle weakness not attributable to the direct surgical procedure. These patients must be evaluated urgently, with adequate neuroimaging including spinal MR imaging. We recommend that spinal SDH should be taken into consideration as a rare but potentially severe complication of intracranial aneurysm surgery.

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**References**


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