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## **Migrating Subdural Hematoma without Subarachnoid Hemorrhage in the Case of a Patient with a Ruptured Aneurysm in the Intracellular Anterior Communicating Artery**

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# Migrating Subdural Hematoma without Subarachnoid Hemorrhage in the Case of a Patient with a Ruptured Aneurysm in the Intracranial Anterior Communicating Artery

## CASE REPORT

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**SUMMARY:** Acute spontaneous subdural hematoma without the presence of a subarachnoid hemorrhage as a result of a ruptured aneurysm is rare. We present the case of a patient with an aneurysm of the intracranial anterior communicating artery that caused hemorrhage solely into the subdural space. The hemorrhage then migrated down the spinal canal. Our case is unique because all these 3 rare processes occurred in a single patient. Identification of the cause of this type of hemorrhage in a timely fashion is crucial to the management of such a patient.

**A**cute spontaneous subdural hematoma (SDH) without the presence of a subarachnoid hemorrhage (SAH) as a result of a ruptured aneurysm is a rare occurrence. Only 28 cases have been reported in the literature. There have been only 5 case reports of a migrating SDH to the spine from an intracranial subdural or SAH. In addition, there have been only 4 cases of aneurysms of the intracranial anterior communicating artery (AcomA) reported in the literature. We present a case of an aneurysm of the intracranial AcomA that ruptured solely into the subdural space. The hemorrhage then migrated down the spinal canal. This is the first reported case of all these unique features in a single patient.

### Case Report

The patient was a 47-year-old man with a past medical history of hypertension, which was controlled with medications. Three days before presentation, the patient experienced an acute onset of occipital headache and low back pain. The symptoms gradually worsened, and on the day of presentation to an outside hospital emergency department, the patient complained of nausea, vomiting, and diplopia. A CT scan demonstrated an acute SDH along the tentorium, cerebellopontine angle, and prepontine cisterns and in the region of the foramen magnum at the cervicomedullary junction. There was no evidence of a SAH. There was no history of recent or remote trauma. A lumbar puncture showed markedly elevated red blood cells but no xanthochromia. The patient was transferred to our institution for further work-up and management 7 days after the onset of his initial symptoms.

Neurologic examination was significant for a partial left sixth cranial nerve palsy. MR imaging of the brain demonstrated a subdural collection, most prominent along the posterior aspect of the clivus, and extending within the subdural space along the anterior aspect of the upper cervical spine (Fig 1). The collection was hyperintense on T1-weighted images (T1WI) and hypointense on T2-weighted images (T2WI), consistent with a subacute SDH. There was no SAH visualized on the MR imaging study. In addition, a saccular outpouching from the AcomA projecting inferiorly into the sella turcica suggestive

of an aneurysm was noted (Fig 1). There was no flow-related artifact seen, raising the possibility of a partially thrombosed aneurysm. Trace signal hyperintensity seen within the sella suggested the possibility of an intracranial hemorrhage secondary to a ruptured aneurysm of the intracranial AcomA.

A conventional angiogram demonstrated a 13 x 5 x 6-mm aneurysm extending inferiorly from the AcomA and projecting into the expected location of the sella turcica (Fig 2). The aneurysm was successfully coil-embolized with 7 GDC-10 Soft 2D SR detachable coils (Boston Scientific, Natick, Mass) of various sizes (Fig 2).

MR imaging of the entire spine, which was performed to evaluate the extent of the SDH, showed a large, extramedullary, circumferential collection of fluid in the cervical, thoracic, and lumbar spine, with signal intensity characteristics consistent with a SDH (Fig 3). The high signal intensity of the SDH persisted with fat-suppressed images, eliminating the remote possibility of epidural or subdural lipomatosis. The patient was discharged home neurologically intact 5 days after coiling of the aneurysm.

### Discussion

Acute spontaneous SDH without SAH secondary to rupture of an intracranial aneurysm is a rare entity. Of the 28 cases reported in the literature, 16 were aneurysms involving the internal carotid artery and posterior communicating artery, 4 were aneurysms of the middle cerebral artery, 4 were aneurysms of the distal anterior cerebral artery, 3 were aneurysms of the AcomA, and 1 was a bifurcation aneurysm of the internal carotid artery.<sup>1-7</sup> Several mechanisms have been proposed to explain the occurrence of SDH after aneurysmal rupture. An aneurysm may become adherent to local arachnoid granulation as a result of previous minor hemorrhage. After aneurysmal rupture, an arachnoid tear may occur, resulting in bleeding directly into the subdural space.<sup>2,3</sup> A second mechanism may be the result of hemorrhage under high pressure, leading to pia-arachnoid rupture and extravasation of blood into the subdural space.<sup>3</sup>

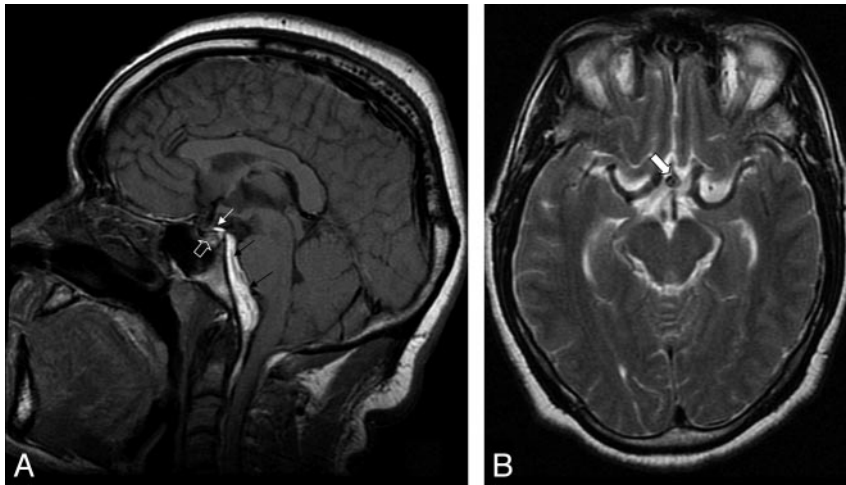
With respect to the location of the SDH in patients with ruptured intracranial aneurysm without SAH, all such cases demonstrated blood over the convexity, 7 cases showed blood tracking along the tentorium, and blood was seen within the interhemispheric fissure in 5 cases.<sup>3</sup> In the literature, there is only 1 case of an aneurysm of the AcomA with subdural hemorrhage along the dorsal aspect of the clivus, as well as the tentorium and convexity.<sup>4</sup> Our case is a unique example of a

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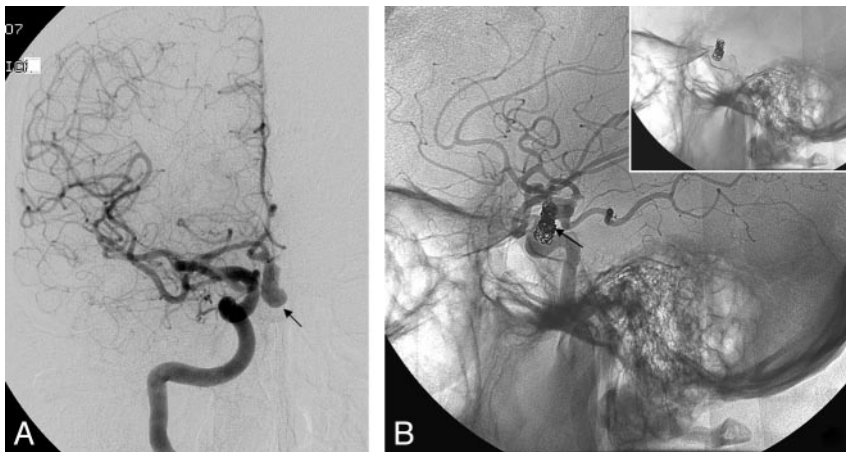
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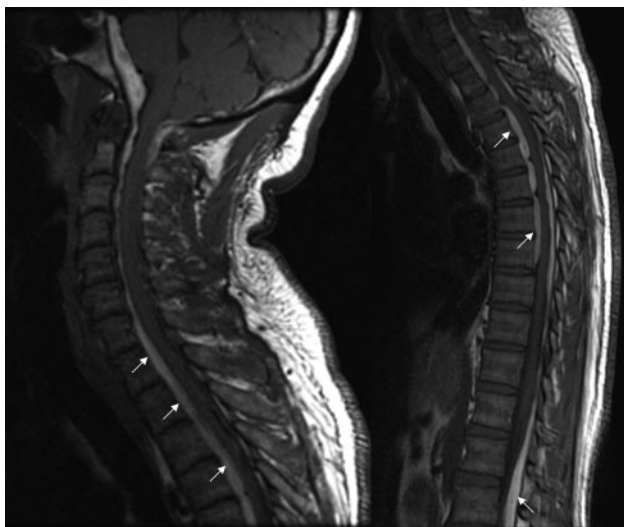
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**Fig 1.** A, Sagittal, noncontrast T1WI demonstrates an extensive SDH dorsal to the clivus (*black arrows*). Closer inspection reveals an inferiorly projecting flow void extending from the suprasellar region into the sella turcica (*open arrow*), which raises the possibility of an aneurysm of the AcomA. Increased signal intensity seen along the roof of the sella (*white arrow*) is consistent with hemorrhage dissecting along the diaphragma sella. B, Axial T2WI demonstrates a saccular outpouching (*white arrow*) consistent with an aneurysm arising from the AcomA.



**Fig 2.** Conventional angiogram obtained from a right internal carotid artery injection (A) precoiling AP view and (B) unsubtracted postcoiling lateral view demonstrates an aneurysm of the AcomA (*black arrows*) projecting inferiorly into the expected location of the sella turcica. Note the inset skull x-ray in the upper right-hand corner showing the coil mass in the sella turcica.



**Fig 3.** Sagittal noncontrast T1WI MR imaging of the cervical, thoracic, and upper lumbar spine demonstrates a circumferential high signal intensity (*arrows*) in the intradural extramedullary space consistent with a SDH of the spine.

ruptured aneurysm of the intrasellar AcomA junction with trace intrasellar hemorrhage and associated SDH along the diaphragma sella, extending along the dorsal clivus and migrating down into the spinal canal. There was minimal SDH over the convexity and within the interhemispheric fissure. We speculate that the inferiorly projecting AcomA aneurysm

ruptured within the sella, which resulted in a trace intrasellar hemorrhage. The hemorrhage then dissected along the diaphragma sella and migrated into the adjacent subdural space, posterior to the clivus in the prepontine cistern, before migrating along the convexity, interhemispheric fissure, and spinal canal. Hence, the subdural hemorrhage in our case was most prominently seen posterior to the clivus, rather than in the other intracranial subdural locations.

Migrating SDH of the spine has been described, with 5 case reports of intracranial SDH migrating to the spine.<sup>8,9</sup> Yamaguchi et al<sup>8</sup> reported a case of a spinal SDH after a SAH from an inferior and posterior projecting supraclinoidal internal carotid aneurysmal rupture. The authors speculated that the bleeding from the aneurysm projected mostly into the infratentorial space, and that the extensive SAH dissected the subdural space under the cerebellar tentorium. With the influence of gravity, the SDH migrated from the intracranial subdural space to the spinal canal.<sup>8</sup> Our case is the second reported case of a migrating SDH from a ruptured aneurysm. It should be an important consideration when a patient with a ruptured aneurysm complains of back pain.

The typical configuration of an aneurysm involving the AcomA is distal and superior to the plane of the circle of Willis. In the literature, there are only 4 reported cases of an aneurysm of the AcomA and 1 of an aneurysm of the anterior cerebral artery pointing posteriorly and inferiorly, and therefore situated in the sella turcica.<sup>10,11</sup> This configuration has significant

management implications because of reported technical difficulty in dissecting the neck of such aneurysms.<sup>11</sup>

In the absence of trauma, coagulopathy, and SAH, a spontaneous SDH presents a diagnostic challenge. The remote possibility of a ruptured aneurysm as a source of the bleed should be kept in mind and used to guide further management decisions. Moreover, in a patient with back pain and a SDH from a ruptured aneurysm, the possibility of a spinal SDH that has migrated downward should be considered.

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