

Subdural Hematomas Following Intracranial Aneurysm Rupture: A Rare Phenomenon

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ABSTRACT

Acute Subdural Hematoma (aSDH) due to aneurysm rupture and no subarachnoid bleeding are very rare with only 29 cases reported in literature. A 56-year female presented with headache and drowsiness and a previous history of loss of consciousness. Clinical examination revealed a GCS of 14 and a right sided hemiparesis. Workup revealed a pure subdural hematoma due to a middle cerebral artery aneurysm rupture with no subarachnoid hemorrhage. Laboratory workup was otherwise normal and she had no history of falls or head trauma consistent with the usual etiology of an aSDH. She underwent evacuation of the hematoma with clipping of the aneurysm. She had an uneventful recovery with good outcome and no residual neurological deficits at one-year follow-up.

Key Words: *Acute subdural hematoma. Cerebral aneurysm. Middle cerebral artery. Clipping. Coiling.*

INTRODUCTION

A subdural hematoma is defined as a collection of blood between the dura mater and the pia-arachnoid mater of the brain usually in the region of the cerebral hemispheres but can also be along the falx, the tentorium and even in the posterior fossa. Its appearance on a head CT is that of a collection that is crescent shaped with intensity signal varying according to the age of the hematoma. Acute hematomas are always hyperintense on a CT scan. Subdural hematomas represent one of the most common indications for surgical intervention even though outcome may not always be satisfactory.^{1,2}

The most common etiology for an acute subdural hematoma is a high speed impact to the skull resulting in tearing of a bridging vein with intracranial aneurysm rupture being an uncommon cause which is usually associated with some degree of subarachnoid hemorrhage as well. In the absence of a subarachnoid or intraparenchymal component, this is referred to as a 'pure' or 'spontaneous' acute subdural hematoma (sSDH or aSDH). Suspicion should arise if there is an acute subdural hematoma in the absence of external injury. A pure subdural hematoma resulting from this cause is decidedly rare.³ Understanding the pathophysiology, diagnosis and treatment options are essential for timely intervention and effective management of patients.

CASE REPORT

A 56-year female presented to the Neurosurgery service with a 3 days history of headache and drowsiness. She had an episode of loss of consciousness 10 days prior and regained consciousness on the second day. CT of the head had shown an acute subdural hematoma and a left MCA aneurysm. She had been treated at another hospital and was advised to undergo surgery after a 2-week period. However, 3 days after discharge, she developed headache and drowsiness and was brought to our setup 3 days afterwards. There was no history of falls or head injury and no external signs that would be associated with suspected trauma. There was no history of headaches prior to presentation. Symptoms were of sudden onset.

Physical examination revealed a Glasgow Coma Scale score of 14/15 (E=3, V=6, M=5). Vital signs were within normal range. There was right sided hemiparesis with power being 3/5 in right upper and lower limbs with exaggerated deep tendon reflexes. Left pupil was mid-dilated with sluggish reaction.

Laboratory investigations revealed a random blood sugar of 164 mg/dL, Hb% of 10.6 gm/dL, DLC and platelets were within normal limits. Prothrombin time was 16 seconds (control=14 seconds) and activated partial thromboplastin time was 36 seconds (control=34 seconds). INR was 1.4 and D-dimer < 250 ng/mL. Renal and liver function was within normal limits.

Emergent CT scan showed an acute subdural hematoma (Figure 1A). An MRI and an MR angiogram (Figure 1B, 1C and 1D) were done which showed biconcave abnormal signal intensities along both convexities of the cerebral hemispheres appearing hyperintense on T1 Weighted imaging, T2 Weighted imaging and FLAIR sequence. Intensity signals appeared equal on both sides suggestive of bleeding at

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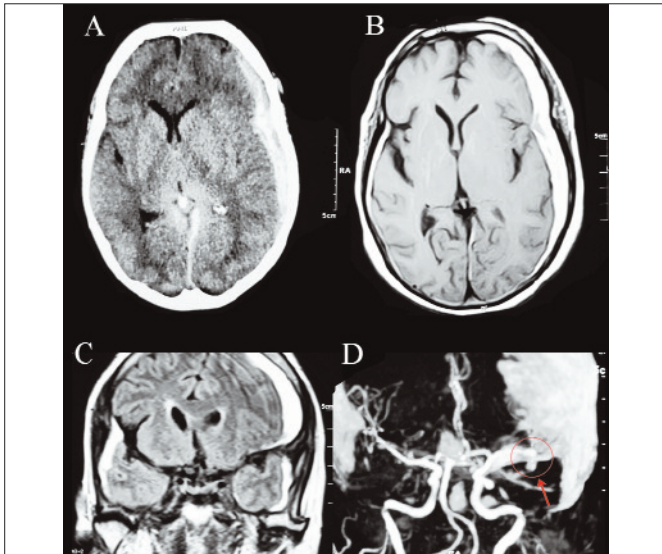


Figure 1 (A-D): (A) Preoperative CT scan showing left sided subdural hematoma. (B) Pre-operative MRI scan axial section at the level of the Sylvian fissure showing bilateral subdural hematomas. (C) Pre-operative MRI scan coronal section at the level of the Sylvian fissure showing bilateral subdural hematomas. (D) Magnetic resonance angiogram showing left Middle Cerebral Artery (MCA) aneurysm (circled in red).

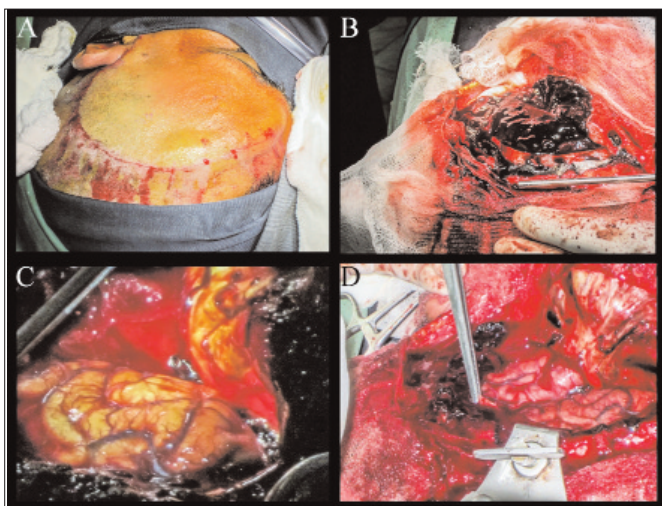


Figure 2 (A-D): (A) Intraoperative photograph showing incision line for a pterional craniotomy. (B) Subdural hematoma visible after opening the overlying dura. (C) YASARGIL aneurysm clip in position. (D) Intraoperative appearance following hematoma evacuation.

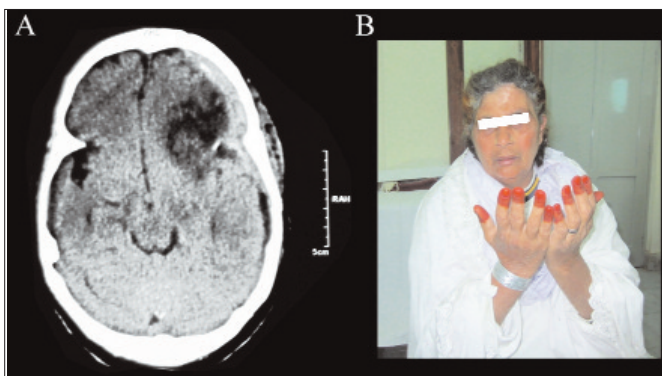


Figure 3 (A,B): (A) Pre-operative CT scan (B) Patient at one year follow-up.

the same time. The hematoma was 1.5 cm thick on the left side and 0.5 cm thick on the right side. Mild midline shift was noted towards the left (WFNS grade III). An aneurysm was evident in the left Middle Cerebral Artery (MCA) [Figure 1D]. No aneurysm was evident on the right side. There was no radiological evidence of blood in the subarachnoid space or ventricular system. She was diagnosed as a case of ruptured intracranial aneurysm with acute subdural hematoma and was admitted for observation. Her blood pressure was notably increased while under observation with a maximum of 170/100 mmHg recorded. She was planned for evacuation of the hematoma and clipping of the aneurysm in a single setting.

Approach was via a left pterional craniotomy (Figure 2A). Osteoplastic flap was raised and dura opened in a curvilinear fashion with the base of the incision lying over the middle meningeal artery. Opening the dura revealed the subdural collection which appeared clotted in appearance (Figure 2B). This was evacuated using suction. A lateral Sylvian fissure approach was used to access the middle cerebral artery. On opening the fissure, the sac of the aneurysm was encountered which was not bleeding actively. The aneurysm itself was found lying outside the arachnoid mater with no obvious adhesions and appeared to have pierced the arachnoid. The neck was dissected free and clipped successfully with an 8 mm YASARGIL® clip (Figure 2C and 2D). The aneurysm sac was ruptured with a needle to assess adequate occlusion of the neck. Hemostasis was secured and dura closed with water tight closure.

Postoperatively, the patient was nursed in an intensive care unit in a 45 degree propped up position. Her mean arterial pressure was maintained between 70 - 80 mmHg. She made an uneventful recovery. Her postoperative CT scan showed the clip in place with a surrounding dark area of infarction (Figure 3A). Her physical examination after 1 year at follow-up revealed a GCS of 15 with pupils equal in size and normally reactive to light and neurological examination revealed no deficits. Fundoscopy was normal with no indication of papilledema. Cognitive function was also normal. The outcome was deemed to be excellent (Figure 3B).

DISCUSSION

ASDHs arise spontaneously in the absence of head trauma and coagulopathy and the source is usually traced to small cortical arteries near the Sylvian fissure or an aneurysm with posterior communicating artery being the most common (46.6%).^{4,5} A typical intracranial aneurysm rupture will usually lead to a collection of blood in the subarachnoid space because of the relationship of the arteries to the meninges. Such bleeds will not extend into the subdural space. However, sometimes, an aneurysm can rupture with bleeding

directed into the subdural space. While the exact mechanism causing this is unclear, several propositions have been made. Repetitive minor bleeds may cause adhesions purportedly fixing the aneurysm to the dura and later a major bleed through the wall may result in a subdural hematoma. Operative evidence strongly points towards this mechanism.^{6,7} Another suspected mechanism is a high pressure bleed piercing through the pia-arachnoid and into the subdural space.⁶ Other proposed mechanisms are rupture of an internal carotid artery aneurysm in its course in the subdural space and in one particular case, erosion of the cavernous sinus wall due to enlargement of an intracavernous aneurysm following thrombosis.^{8,9} A superficially located aneurysm, theoretically, may be more likely to bleed into the subdural space.⁴

Timely diagnosis of such a case requires a high degree of suspicion for an aneurysm being the cause of an acute subdural hematoma in the absence of head trauma. A plain head CT will demonstrate a subdural hematoma. However, whenever there is lack of an obvious explanation for the condition, further workup is warranted especially in the absence of head trauma.¹⁰ Head trauma or a history of head trauma may be misleading in certain cases where it is the consequence and not the cause of the sSDH. A history of repetitive headaches may be a clue towards repetitive bleeds.⁷ The gold standard for demonstrating an aneurysm as the cause of an aSDH remains cerebral Digital Subtraction Angiography (DSA). However, because of its noninvasiveness and less time required, a CT angiogram or a MR angiogram may be performed (negative predictive value remains low). In the event of a negative CTA or MRA, a cerebral DSA becomes mandatory.¹⁰ Another modality available is a three dimensional CT angiogram.⁷ Investigations will not only show the location of the aneurysm but also orientation in order to devise a best approach to managing the aneurysm. Absence of a subarachnoid bleeds related to common aneurysm sites will confound the diagnosis. Most cases had a convexity hematoma with or without extension to the tentorium depending on the site of the aneurysm. In this case, the hematoma from an MCA aneurysm was extended to the tentorium and there was also a contralateral aSDH for which a causative aneurysm could not be identified. Its appearance placed it as being the same age as the left side hematoma.

A review of literature by Takada *et al.* and Koerbel *et al.* lists a total of 29 cases in published literature of pure aSDH due to aneurysm rupture. The most frequent were ICA-Pcom aneurysms (15 cases) and MCA aneurysms in 5 cases not including this case. Outcome was good in 17 cases; similar to this case, but poor in 10 patients with 5 dead and 5 disabled and 2 cases in which outcome was not described. As opposed to aSDH with SAH, pure aSDHs had better outcomes.^{4,7}

Any patient with an aSDH should undoubtedly be treated at a dedicated neurosurgical service. Current guidelines state that an aSDH should be surgically evacuated if it is greater than 10 mm in thickness or the midline shift is greater than 5 mm regardless of the neurological status. Patients with smaller hematomas may be treated based on the amount of neurological deficit.¹¹ In patients with sSDH, management depends on the underlying cause. A bleeding aneurysm will need to be dealt with to prevent morbidity and mortality. Several options have been used with aneurysm clipping the most common. Coiling is also an available alternative. Literature review of 29 previous cases shows that 26 patients underwent surgery with 17 having hematoma evacuation and clipping and 4 received clipping only.⁷ Again, a proper workup for the underlying cause is important to ensure the aneurysm is detected and treated as soon as possible to improve outcome. Koerbel *et al.* described a protocol for management of aSDH from aneurysm rupture regardless of SAH. The protocol describes that patients with rapid deterioration of neurological status should have a CT+CTA followed by sedation-osmotherapy with mannitol or hypertonic saline and if hemodynamically stable, should undergo hematoma evacuation and intraoperative DSA (if available) and clipping. Unstable patients should have delayed DSA and coiling/clipping. Patients who have a stable neurological status should have a CT+CTA+DSA and can undergo either hematoma evacuation and clipping or a coiling of the aneurysm followed by delayed evacuation.⁴

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