

CASE REPORT

Intracerebral aneurysmal bleed presenting as acute subdural haemorrhageG Menon,¹ MBBS, MCh, DNB; A Thomas,¹ MBBS, MMed; M Nzey,¹ MB ChB; B Luke,² MBBS, MD, FCP (SA)¹ Department of Neurosurgery, Tshewpong Hospital, Klerksdorp, South Africa² Department of Internal Medicine, Tshewpong Hospital, Klerksdorp, South Africa**Corresponding author:** G Menon (neuron1967@gmail.com)

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Acute subdural haemorrhage (aSDH) secondary to an aneurysmal bleed is a surgical emergency that requires urgent decision making. Major dilemmas are whether preoperative diagnostic studies should precede surgery and whether obliteration of the aneurysm should be performed during haematoma evacuation or as a separate delayed intervention after resuscitation. We successfully operated on a 44-year-old patient with aneurysmal aSDH, and discuss the challenges involved with such patients.

Case report

A 44-year-old man was brought unconscious to hospital by paramedics. Immediate family members or friends were not available to give a reliable history. On admission his Glasgow

Coma Score (GCS) was 6/15 (E1 M4 V1). His right pupil was dilated and unreactive to light and he had features of Cushing's reflex with bradycardia and hypertension. He was immediately intubated and underwent an urgent computed tomography (CT) scan (Fig. 1), which revealed a large, right fronto-temporo-parietal acute subdural haematoma with effacement of basal cisterns and evidence of mass effect and midline shift. The scan also showed thick blood in the right sylvian fissure and a thrombus in the superior temporal gyrus on the right side. Because of features of uncal herniation, an emergency craniotomy was performed immediately. On draining the aSDH, the brain became lax and pulsatile. The sylvian fissure was thereafter opened proximal to distal. The arachnoid membrane across the sylvian fissure could clearly be seen in

the proximal fissure, but distally, over the aneurysm, it was obliterated by the thick thrombus surrounding the fundus of a large bi-lobed aneurysm at the middle cerebral artery (MCA) bifurcation. The thrombi were carefully removed and the aneurysm neck was dissected out and clipped using a large curved Yasargil clip. Postoperatively he was ventilated for 24 hours and then gradually weaned off the ventilator. His postoperative recovery was slow but steady. On last follow-up six months after surgery he had made significant recovery with a modified Rankin score of 2. He was independent for activities of daily living but had not yet resumed his earlier vocation.

Discussion

aSDH from rupture of an aneurysm was first reported by Hasse in 1855.^[1] The reported incidence in autopsy studies (10 - 22%) is higher than in clinical studies (0.5 - 1.6%) owing to the high rate of mortality associated with this condition.^[1-3] Although a literature survey suggested more than 200 published case reports, a comprehensive meta-analysis was available only as late as 2012. Marbacher *et al.*^[3] specifically reviewed 20 published studies involving 82 patients, providing the most comprehensive summary of this difficult clinical condition.

In almost all reported series of aSDH caused by aneurysm, the most common aneurysm arises in the posterior communicating artery followed by the MCA. Other less common sites include the anterior communicating artery and internal carotid artery. A posterior communicating artery aneurysm rarely presents with a posterior fossa subdural haematoma.^[4] Patients with aSDH secondary to an aneurysm usually present in poor clinical grade, with World Federation of Neurosurgical Societies (WFNS) Grade 4 and 5 patients accounting for nearly 74.4% of cases.^[3] These patients are known to have a high incidence of associated

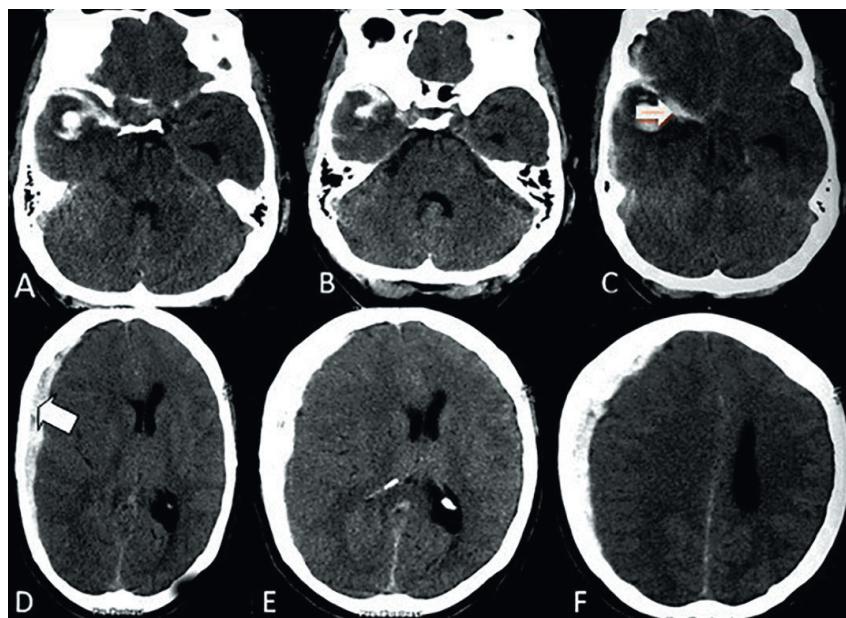


Fig. 1. Non-contrast CT scan images of the patient with aneurysmal acute subdural haematoma. A, B, C: axial CT scan images showing blood in the left sylvian fissure with a small temporal haematoma (arrowhead). D, E, F: axial CT scan images showing left fronto-temporo-parietal acute subdural haematoma (arrowhead) with mass effect and midline shift.

potentially fatal comorbidities, including cardiac arrhythmias and pulmonary oedema.

Different mechanisms have been proposed to explain the occurrence of an aSDH after a ruptured aneurysm.^[1,5,6] Successive small bleeding episodes may cause adhesion of the aneurysm to the adjacent arachnoid membrane, and the final rupture occurs into the subdural space. The other theory is that a haemorrhage under high pressure may lead to pia-arachnoid membrane rupture.

The initial management dilemma lies in differentiating an aneurysmal aSDH from a traumatic SDH. Patients are often brought to the emergency room unconscious and, in the absence of a clear history, diagnosis is difficult. A CT scan will reveal the presence of an SDH, and an associated subarachnoid haemorrhage (SAH) may/may not be seen.^[7] In the presence of an SAH, an aneurysm needs to be considered but it must be remembered that a traumatic SDH can be associated with a traumatic SAH as well. In the absence of an associated SAH, the diagnosis is extremely difficult, although cases of pure aSDH due to ruptured intracranial aneurysm are extremely rare. Only 20 cases have been reported so far, 14 of those during the last 2 decades.^[3,7,8] These patients pose a real clinical challenge.

The subsequent management dilemmas involve the decision to perform a diagnostic angiogram prior to craniotomy in poor-grade patients where the aSDH is causing a significant mass effect. Crucial time is often lost in organising an angiogram – be it a CT angiogram (CTA), a magnetic resonance angiogram (MRA) or a conventional digital subtraction angiogram (DSA). The alternative is a two-step procedure – stage-one craniotomy to evacuate the aSDH, followed by an angiogram and securing the aneurysm either by coiling or clipping. The concern regarding the latter option is that the craniotomy required to drain the SDH could release the tamponade and again predispose the aneurysm to rupture.

Clarke and Walton^[5] classified their patients into three groups, based on the amount of subdural thrombus and the clinical course: (Group I) cases with a massive and rapidly fatal intracranial haemorrhage; (Group II) cases with an insignificant quantity of subdural blood; and (Group III) cases with a clinically significant subdural haematoma that is not rapidly fatal. In Group II and III patients, where the level of consciousness is not depressed (WFNS Grade 1 - 2), management may proceed in a standard manner with angiography and elective surgery with coiling/clipping. For Group I patients, one option is to perform an urgent CTA along with the initial CT scan, followed by emergency surgery. The second option is to perform intraoperative angiography and tackle both the aneurysm and the SDH in the same sitting, and the third option is to do a two-stage procedure, as discussed above.

Our scenario was distinct in that the patient, with no definite history, was referred from another centre in poor grade and with features of impending herniation. A plain CT scan of the brain had already been done at the referring hospital and an emergency craniotomy was warranted as a life-saving procedure to drain the SDH. As we do not have facilities for intraoperative angiography, we explored the right sylvian fissure as this was where the SAH was concentrated. A large bi-lobed aneurysm was found, which was clipped, and the thrombi in the fissure and superior temporal gyrus were evacuated. The patient was treated with a triple H regimen and gradually improved. We propose this approach as an alternative

in centres where intraoperative angiography is not available and emergency angiography services are often not available, a common scenario in many developing countries. The advantages are that the risk of re-bleeding following drainage of an aSDH can be avoided and the patient is spared the trauma of a two-stage procedure. This alternative is, however, associated with its attendant risks of operating on an aneurysm with an unclear morphology. The patient carries a risk of complications due to excessive cortical resection, suboptimal clip placement and inadvertent branch closure, and worsening vasospasm due to vessel manipulation.

Outcome is uniformly poor in most case reports as patients generally present with a poor preoperative clinical grade.^[1-3,7] Generally, two-thirds of all patients with either poor-grade SAH or traumatic aSDH do not survive, and functional survival is rare.^[9-11] In Marbacher's series of 82 patients, the critical status of 19 patients (23.2%) on admission did not allow any surgical or endovascular intervention. Four (4.9%) patients died during resuscitation, two (2.4%) patients died immediately after diagnosis, and one (1.2%) patient received no further therapy as a result of prolonged hypoxia before admission. Of the remaining 63 patients who underwent invasive treatment, 39 (69.9%) achieved good outcomes (Glasgow outcome score (GOS) 5 and 4). Good-grade patients do well irrespective of the timing of intervention. Marbacher's meta-analysis also suggests that patients with pure aSDH due to a ruptured aneurysm demonstrated better outcomes than those who suffered aneurysmal aSDH associated with SAH, probably because of the lower risk of vasospasm and hydrocephalus.

Conclusion

A high degree of suspicion is needed to look for an underlying aneurysm in patients with aSDH when no definite history of trauma is available. In the absence of comprehensive management guidelines, treatment needs to be individualised. An urgent angiogram is advisable in all patients. Our case was unusual, as clinical deterioration precluded further investigation and direct surgery yielded a good outcome. Direct surgery in such an instance may increase surgical risk, but with the benefit of preventing early re-bleeding.

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