Typical Case History

A 45 year old woman suffers a sudden onset occipital headache, vomits three times and has associated neck pain and photophobia. Her medical history includes migraines and thus she concludes this episode is a severe migraine. Over the next two hours her left arm becomes heavy and she becomes somnolent. Her husband is worried she has suffered a stroke and calls the emergency services. On arrival the paramedics find her Glasgow Coma Score (GCS) 12/15 (E3V3M6) with a 0/5 left hemiplegia. She is blue lighted to her local hospital where a CT scan demonstrates a right temporal intra-cerebral haematoma (ICH) and subarachnoid haemorrhage (SAH) in the basal cisterns. On returning to resus her GCS drops to 9/15 (E2V2M5) with both pupils reacting normally to light. The ED registrar refers the patient to the on-call neurosurgery registrar located in a neighbouring tertiary hospital.

Epidemiology

The SubArachnoid Haemorrhage International Trialists (SAHIT) group reported 21% of SAH had associated ICH,[1] with a range of 9% to 38% in other series.[2,3,4,5,6] Whilst the middle cerebral artery bifurcation is the classical location it actually represents only 36% of cases.[1] Anterior cerebral artery aneurysms and ICA aneurysms comprise a further 33% and 15% respectively.[1] Internal carotid artery (ICA) aneurysms causing ICH are usually distal, that is, posterior communicating, anterior choroidal or ICA bifurcation. The clinical focus on the middle cerebral artery (MCA) aneurysm is likely because they cause significantly larger haematomas than non-MCA locations (mean: 32ml vs 9ml)[2] and thus are more likely to require haematoma evacuation.

Aneurysm size is another risk factor for ICH associated with SAH. Medium or large size aneurysms (>12mm) pose a significant risk for ICH compared to small aneurysms.[1,8] Larger aneurysms are also associated with larger haematomas.[9]

History

The history of SAH with ICH can be similar that of SAH without ICH namely thunderclap headache with meningism or sudden loss of consciousness. The distinguishing features with ICH are the presence of focal deficits which are otherwise relatively rare with a pure SAH. Hemiplegia or aphasia from a unilateral frontoparietal clot would be typical. In unconscious patients a unilateral fixed and dilated pupil may be seen.

Examination

The clinical findings in SAH with ICH are different to those without ICH. SAH with ICH are more likely to be poor World Federation of Neurosurgical Studies (WFNS) grade (21% vs 61%).[1] In the SAHIT series only 13% of patients with SAH and ICH presented GCS 15/15. Another series of 494 patients identified the median GCS of 8/15 for SAH with ICH versus 14/15 for SAH alone.[2] As one would expect, the haematoma volume has a significant influence on GCS with haematomas >50cm^3 having an average GCS 5/15 compared to 10/15 in haematomas <50cm.[3,9]

Many clinical grading systems are used for SAH, including the classic Hunt and Hess and more recent WFNS, and were developed incorporating both SAH with and without ICH. It is notable that WFNS grade 3 are relatively rare, but proportionately are mostly made up of SAH with ICH.

Investigation

All patients will have a CT scan demonstrating SAH with ICH, however we strongly recommend performing a CT angiogram in all cases. It is tempting to prioritise an emergency operation over another trip to the scanner; however, modern CT angiography can be performed quickly and even in extreme circumstances (e.g. a fixed pupil), the risks of delay can be mitigated with mannitol.

The role of the pre-operative CT angiogram in the context of a clot is threefold. Firstly, it confirms the ICH is due to a ruptured aneurysm making the surgeon mindful of its presence. Secondly, aneurysms bleed into the parenchyma where brain is stuck to the dome so they are on the periphery of the haematoma and CT angiogram will define from where haematoma can be safely removed. Thirdly, understanding the vascular anatomy including the aneurysm size, branching vessels and vessels adherent to the dome helps with surgical clipping.

Intra-Sylvian vs Intra-Parenchymal Haematoma

CT angiogram will help discern between a Sylvian fissure versus brain parenchyma haematoma. The primary radiological sign of an intra-Sylvian clot instead of a parenchymal clot is the presence of intra-haematomal contrast enhancing vessels, that is, vessels running through, rather than around, the haematoma.

This distinction is important to help manage surgical expectations. Unlike parenchymal clots, which are one uninterrupted mass, Sylvian clots contain adherent MCA vessels. The clot sticks these vessels and islands of pia and arachnoid together making it much more difficult to suck out. The resultant increased effort to remove the clot frequently results in avulsion of small vessels.

Sylvian fissure clots tend to present with higher GCS and lower volume than MCA parenchymal clots.[6,10] As a result Sylvian clots tend to result in a lesser improvement in GCS at seven days post-surgery.[10] It has been proposed that an aneurysm sac angle inline with the M1 segment is more likely to produce Sylvian haematoma whereas a sharper angle results in parenchymal clot.[6] The latter aneurysms are heavily adherent to pia/parenchyma whereas the former are seen free floating within the Sylvian fissure.

Differential Diagnosis

Any ICH with associated SAH or extending to the pial surface of the Sylvian/interhemispheric fissure/basal cisterns should be considered for underlying aneurysm. Young age and lack of comorbidity also make an aneurysmal cause more likely and warrant CT angiogram before ICH evacuation.

The most common differential diagnosis for the Sylvian clot is a large hypertensive basal ganglia clot extending to the Sylvian fissure. Other differential diagnoses for aneurysmal ICH include vascular lesions such as arterio-venous malformations or cavernomas, or neoplastic lesions. Other causes for superficial lobar ICH such as venous sinus thrombosis, cerebral amyloid or dural fistula are less likely to be mistaken for aneurysmal ICH as they do not extend to the basal cisterns/interhemispheric fissure.

Management

Box 1 Initial instructions to the referrer.

1) Blood pressure should be controlled with a systolic BP <180mmHg to reduce the risk of ongoing bleeding.
2) Nimodipine 60mg PO STAT. Can be started up to 96 hours post-ictus as per BRANT study[7] so patient transfer should not be delayed for this.
3) Osmotherapy. If the GCS is <12 or dropping, or there is a fixed pupil, then give mannitol 20% 0.5–1mk/kg or hypertonic saline 3% 3–5ml/kg.
4) Intubate + ventilate. If the patient has a low GCS then intubate for safe transfer.
5) Transfer the patient directly to the neurosurgery theatre as soon as possible. CT angiogram should be performed en route.