Management of sub-arachnoid haemorrhage

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Abstract

Spontaneous subarachnoid haemorrhage (SAH) is a neurovascular emergency with sudden onset, which requires rapid recognition and early treatment to minimize the occurrence of serious complications. The most common cause is a cerebral aneurysm, which develops at areas of turbulent flow, especially within the circle of Willis. Initial aims are to provide appropriate resuscitation to the patient and to maintain cerebral oxygenation and perfusion. Anaesthesia involves prompt airway control and precise management of physiological parameters to reduce further neurological injury, such as from re-bleeding or delayed cerebral ischaemia. Once stabilized SAH patients should be admitted to a neurointensive care unit and managed by a skilled multidisciplinary team. Definitive treatment then involves either endovascular coiling or surgical clipping, preferably in hospitals managing high volumes of SAH cases per year. Care should be also taken throughout to avoid non-neurological complications such as infections or venous thromboembolism.

Keywords Aneurysm; brain injury; cerebral ischaemia; cerebral vasospasm; headache; hydrocephalus; intracranial pressure; SAH; subarachnoid haemorrhage

Royal College of Anaesthetists CPD Matrix: 2F01; 1A02; 2C01; 2A08

Learning objectives

After reading this article, you should be able to:

- describe the perioperative anaesthetic management of patients undergoing neurosurgery and neuroradiology for a subarachnoid haemorrhage (SAH)
- recognize and treat complications after an SAH
- demonstrate understanding of neuro-critical care treatment of an SAH

As anaesthetists, we may be involved in the management of patients with a subarachnoid haemorrhage (SAH) at several different points during their presentation and subsequent treatment. This is a neurovascular emergency that requires rapid recognition and early treatment.

Definition and aetiology (Table 1)

SAH is defined as bleeding around the brain confined within the subarachnoid space, between the arachnoid membrane and the pia mater. The incidence of aneurysmal SAH is between 6 and 8 per 100,000 population per year. The overall mortality is approximately 50% and about 25% of these patients die before reaching hospital. The remainder succumb as a result of neurological and non-neurological complications within the first 3 weeks. Of those who survive, one-third will remain dependent, with the other two-thirds reporting a decreased quality of life even if they regain their independence.[1]

The aetiology of SAH can be divided into two main categories:

- spontaneous
- traumatic.

The most common spontaneous cause is a cerebral aneurysm (70–85%), but there are a number of other causes associated with an SAH (Box 1).

Aneurysms are seen in approximately 1% of men and 2% of women in the UK, and the risk of rupture is 0.7% per annum. They develop where there is turbulent blood flow at vascular bifurcations, especially within the circle of Willis. The most common sites for rupture are the junction of the internal carotid and posterior communicating arteries, and the anterior

World Federation of Neurological Surgeons grading of subarachnoid haemorrhage

Grade	Glasgow Coma Scale score	Motor deficit
I	15	Absent
II	13–14	Absent
III	13–14	Present
IV	7–12	Either
V	3–6	Either

Table 1

Causes of a spontaneous subarachnoid haemorrhage (SAH)

- Ruptured intracranial aneurysm (75–89%)
- Cerebral arteriovenous malformation
- Dural and pial arteriovenous fistula
- Dural venous sinus thrombosis
- Pretruncal/perimesencephalic non-aneurysmal SAH
- Cerebral artery dissection (internal carotid and vertebral arteries)
- Rupture of an infundibulum
- Pituitary apoplexy
- Coagulation disorder (e.g. blood dyscrasias and thrombocytopenia)
- CNS vasculitis
- Brain tumour
- Spinal arteriovenous malformation (cervical or high thoracic)
- Unknown or idiopathic

Box 1

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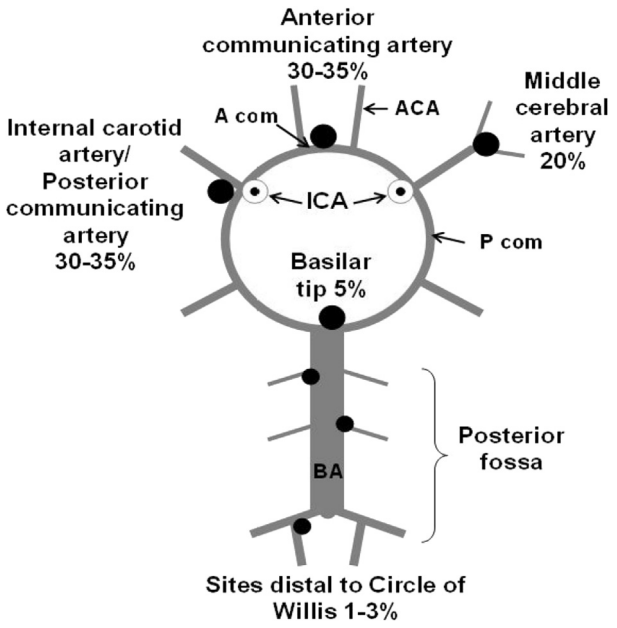

Figure 1 Common sites of aneurysms within the cerebral circulation. ACA, anterior cerebral artery; A com, anterior communicating artery; ICA, internal carotid artery; P com, posterior communicating artery; BA, basilar artery.

communicating arteries (Figure 1). Inflammation has been found to be a key component in the pathogenesis and growth of intracranial aneurysms, including tumour necrosis factor, macrophages, reactive oxygen species and nuclear factor K-light chain enhancer of activated B cells.[2]

The main modifiable risk factors are hypertension and smoking, although heavy alcohol use and illicit drug abuse (especially the sympathomimetics cocaine and methamphetamines) are also implicated.

Non-modifiable risk factors associated with an SAH include sex (female: male ratio is 3:2) age (peak incidence is between 40 and 60 years old), race (African—American), personal or family history of cerebral aneurysm, and previous history of SAH, stroke, or cerebrovascular disease. A small percentage (10%) have been shown to be attributable to a familial or genetic cause. Several inherited conditions have also been linked to SAH, including Ehlers—Danlos type 4, autosomal dominant polycystic kidney disease and familial intracerebral aneurysms.[3]

Clinical features

The classical presentation of an SAH is of a 'thunderclap' headache — this is of sudden onset, and immediately reaches peak intensity. Patients often describe it as the worst headache of their lives. It is due to the sudden rise in intracranial pressure which occurs at aneurysm rupture. A high level of suspicion for SAH should be maintained in all patients presenting with acute onset of severe headache.

The headache may occur in isolation, but also can be associated with other clinical features including nausea, vomiting, photophobia, neck stiffness, diplopia, focal neurologic deficit, back pain, seizures, loss of consciousness, and even cardiac arrest. Between 10% and 43% of patients may also describe a warning or 'sentinel' headache in the hours or days prior to the SAH. This is important as patients who report a sentinel headache have a 10-fold higher risk of early re-bleeding in the days following the initial insult.

Whilst patient's describing this type of headache should be presumed to have an SAH until proven otherwise, there are other differential diagnoses to be considered at the same time. These include migraine, meningitis, cerebral venous thrombosis, spontaneous intracranial hypotension, hypertensive encephalopathy, post-coital cephalgia and pituitary apoplexy.

The severity of clinical presentation is the strongest prognostic indicator for recovery after subarachnoid haemorrhage. Therefore early use of recognized scales to determine severity is recommended by the American Heart Association/American Stroke Association (AHA/ASA). One of the main clinical grades is the World Federation of Neurological Surgeons (WFNS) score, which allows for objective assessment of severity of SAH.

Imaging and diagnosis

Unenhanced (non-contrast) computed tomography (CT) is the first-line diagnostic modality, and has a high sensitivity for detecting blood in the subarachnoid space (close to 100% for the first 3 days). It should be performed as soon as possible after the patient presents with a headache, as once blood re-absorption has begun after 5—7 days the SAH may no longer be evident. In patients with a high index of suspicion for a haemorrhage but a negative CT results, further investigation should be carried out. A lumbar puncture is the next diagnostic test, but should only be performed 12 hours after the onset of symptoms to prevent transtentorial herniation. The characteristic finding is xanthachromia, a yellow discolouration of the cerebrospinal fluid.

The amount of blood seen on CT scans can be graded using the Fischer scale (Table 2), which is validated to predict likelihood of cerebral vasospasm with minimal inter-observer variability — the higher the grade the more likely the chance of spasm.

Once diagnosed, identifying the cause of the aneurysm requires further investigation with angiography, using either CT, magnetic resonance imaging or catheter angiography. Catheter angiography is the gold standard study for evaluation of aneurysms and occult arteriovenous malformations not detectable on other imaging modalities, giving additional information about flow dynamics within the corresponding vasculature.

A CT angiogram (CTA) is a non-invasive study, which can be performed with minimal risk to the patient. It shows detailed cerebral vascular anatomy, and has a specificity of 100% and a

Fischer scale

Grade	Computed tomography (CT) finding
1	No blood detected
2	Diffuse thin layer of subarachnoid blood (vertical layers <1 mm thick)
3	Localized clot or thick layer of subarachnoid blood (vertical layers >1 mm thick)
4	Intracerebral or intraventricular blood with diffuse or no subarachnoid blood

Table 2

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