Subarachnoid haemorrhage and anaesthesia for neurovascular surgery

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Abstract
Subarachnoid haemorrhage (SAH) is a life-threatening condition with multiple sequelae. The treatment of SAH requires urgent resuscitation and stabilization of the patient to prevent re-bleeding and to optimize cerebral oxygenation and perfusion. The perioperative care of these patients involves meticulous attention to maintain an appropriate fluid balance, cerebral blood flow and intracranial pressure. The majority of cases of SAH result from rupture of an intracerebral aneurysm and treatment involves obliteration of the aneurysmal sac either by surgical clipping or endoscopic coiling. Arteriovenous malformations may also cause SAH and often require a combination of radiological and neurosurgical treatments. Haematomas resulting from SAH may require surgical intervention, depending on the location of the haematoma and the clinical condition of the patient.

Keywords Arteriovenous malformation; intracranial aneurysm; subarachnoid haemorrhage

Subarachnoid haemorrhage
Subarachnoid haemorrhage (SAH) is defined as bleeding into the subarachnoid space, the area between the arachnoid and pia mater of the meninges enveloping the brain.

The incidence of SAH is around 9 per 100,000 population worldwide.1 It accounts for around 6% of all strokes, but is still one of the major causes of death from stroke. SAH carries a high mortality rate of around 50%, with one-third of survivors remaining dependent. Of those who remain independent, 70% report a decrease in their quality of life.

The most common cause for spontaneous (non-traumatic) SAH is rupture of a cerebral aneurysm (85%). Non-aneurysmal perimesencephalic haemorrhage accounts for another 10% and carries a better prognosis. In the remainder of cases, the cause is a variety of rare conditions including arteriovenous malformations (AVM) and septic (myotic) aneurysms.2

Risk factors for SAH are summarized in Table 1. Ten per cent of patients with cerebral aneurysms have a first-degree relative who has an aneurysm. These individuals are likely to have a SAH at a younger age and are at an increased risk of multiple and/or large aneurysms.

Cerebral artery aneurysms
Cerebral artery aneurysms occur in 1% of men and 2% of women in the UK population, although most will never rupture. The risk of rupture is 0.7% per annum in patients with no other risk factors.3 The most common site for these aneurysms to form is in the Circle of Willis (Figure 1). Up to one-third of patients have multiple aneurysms and the peak age for aneurysmal rupture is 55–60 years.

The definitive treatment for an aneurysm is obliteration of the aneurysmal sac. This may be achieved surgically by placing a clip over the neck of the aneurysm during craniotomy or by placing platinum coils into the sac during angiography. An endovascular treatment is favoured when possible as survival and disability rates at 1 year are superior; however, some cases may require surgical intervention because of technical problems with a radiological approach or because they require additional evacuation of a haematoma.4 It is important to note that endovascular treatment may be associated with a slightly increased risk of re-bleeding in the longer term.5

Arteriovenous malformations (AVM)
AVMs are congenital abnormal intraparenchymal clusters of arterial-venous communications. Up to 90% are supratentorial and the remainder are either infratentorial or dural. Patients usually present between 20 and 40 years of age. The risk of bleeding is up to 3% per year and the annual rate of death and disability is 4%. Most cases are treated by endovascular embolization, often in combination with neurosurgery and/or radiosurgery for obliteration of smaller superficial AVMs.

Presentation of subarachnoid haemorrhage
In 90% of cases the presenting symptom is a sudden onset of severe headache, often described as a ‘hammer blow’. In 40% of

| Risk factors for subarachnoid haemorrhage |
| Risk factor | Hypertension, pregnancy |
| General medical conditions | Smoking, alcohol, cocaine, ecstasy |
| Lifestyle | Oral contraceptive pill |
| Prescription drugs | Ehlers–Danlos Syndrome type III, Marfan’s syndrome, polycystic kidney disease |
| Genetic/collagen abnormalities |

Table 1

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cases there is a sentinel (warning) headache some weeks before the main SAH. The cause of sentinel headache is unknown, but it may result either from changes in the aneurysm wall without rupture or a small leak of blood.6

Other presenting symptoms and signs include nausea and vomiting (70%), diplopia (due to 3rd or 6th cranial nerve palsy), meningism (photophobia, neck stiffness), a decrease in conscious level, seizures (25%), papilloedema or retinal haemorrhages.2

Grading of subarachnoid haemorrhage

The most commonly used grading scale is that of the World Federation of Neurological Surgeons (WFNS) (Table 2). A higher WFNS grade is associated with worse outcome and it can be used to assess surgical risk and long-term prognosis.

Diagnosis

All patients with symptoms suggestive of an SAH must have an urgent brain computed tomography (CT) scan without contrast. This should be examined by an experienced radiologist. Blood located near common aneurysmal sites in the basal cisterns is diagnostic of SAH. Parenchymal blood in temporal or basal frontal lobes or interventricular haemorrhage is also highly suggestive of the diagnosis.

A CT scan may not always be diagnostic and in these cases a lumbar puncture should be performed. The characteristic finding is of xanthachromia (yellow discoloration) of centrifuged cerebrospinal fluid (CSF). If the CT scan has demonstrated subarachnoid blood, a lumbar puncture should not be performed as it may result in transtentorial herniation (coning). Once SAH has been diagnosed the patient must be transferred to a neurological unit where formal angiography can be performed.

Acute cerebral complications

Re-bleeding

The peak incidence of re-bleeding from an aneurysm occurs in the first 72 hours. After 6 months, up to 50% of patients who have suffered SAH will have had a further bleed.7,8

Hydrocephalus

Obstructive hydrocephalus, caused by blood entering the ventricles and blocking the flow of CSF, can occur in the first few days following the SAH and occurs in approximately 25% of patients. Treatment usually requires CSF drainage through a temporary external ventricular drain.

Cerebral vasospasm

The risk and severity of cerebral vasospasm is related to the volume of blood within the subarachnoid space. Vasospasm occurs within 3–12 days of the initial event and most commonly at 6–8 days. Although vasospasm can be identified
angiographically in 40–70% of patients with SAH, neurological deficit is only seen in 20–30% of patients. The diagnosis should be suspected when new neurological deficits occur and a repeated CT scan has excluded other causes. Transcranial Doppler ultrasound may suggest the diagnosis, but definitive diagnosis requires angiography.

The management of vasospasm aims to improve blood flow and includes several strategies. The administration of a cerebroselective calcium channel blocker, nimodipine for 3 weeks to patients who have had aneurysmal SAH, has been shown to reduce vasospasm and improve outcome. Once the aneurysm is protected by surgical clipping or endovascular coiling, ‘triple H’ therapy (hypertension, hypovolaemia and haemodilution) can be used to maintain blood flow through the narrowed vessel. This therapy may result in pulmonary oedema, hyponatraemia, exacerbation of cerebral oedema, increased intracranial pressure or haemorrhagic infarction. In severely resistant cases selective intra-arterial vasodilator infusions of papaverine or angioplasty may be required. Advanced age and poor clinical status are predictors of poor outcome in vasospasm.

Seizures
Generalized seizures may be due to vasospasm, re-bleeding or hydrocephalus and occur in approximately 25% of patients with SAH. Anticonvulsant treatment is usually started after the second witnessed seizure or if a seizure is prolonged.

Acute systemic complications
Cardiovascular dysfunction
Electrocardiography (ECG) changes are seen in up to 80% of cases of SAH and include sinus tachycardia, T wave inversion, Q and U wave abnormalities and a prolonged QT interval. They are often without corresponding myocardial compromise. In contrast, patients with higher grade SAH often suffer from life-threatening arrhythmias, ischaemic myocardial dysfunction and neurogenic pulmonary oedema. The precise mechanism is unclear, but may be related to direct trauma to the posterior hypothalamus resulting in abnormally high catecholamine release. Electrocardiogram changes return to normal but may persist for 6 weeks.

Pulmonary complications
Those patients with poor-grade SAH may present with neurogenic pulmonary oedema, requiring positive pressure ventilation and diuretic therapy. They may also be at risk of aspiration resulting from a decreased conscious level.

Fluid and electrolyte balance
Intravascular volume depletion and hyponatraemia is common after SAH. Hyponatraemia occurs in 40% of patients and results in decreased conscious level, cerebral oedema and seizures. Causes include inappropriate antidiuretic hormone release, cerebral salt wasting or administration of hypertonic fluids; the correct diagnosis is essential so that the required treatment can be given. Hyperglycaemia is associated with poorer outcomes after brain injury and tight glycaemic control should be maintained to a blood sugar of between 5.5 and 9 mmol/litre.

Initial management and resuscitation
Management is aimed at reducing complications and the first priority is to ensure adequate resuscitation. Early tracheal intubation is essential for airway protection if the Glasgow Coma Scale (GCS) score is below 8 or is decreasing or if there are persistent seizures. Mechanical ventilation of the lungs should maintain a PaCO2 of 4.5–5.0 kPa and a PaO2 above 13 kPa. Sedation and analgesia must be given to prevent surges in intracranial pressure (ICP) and to minimize the chances of re-bleeding.

The maintenance of circulating blood volume and close control of blood pressure are essential for optimal perfusion of the brain. Systolic blood pressure should be maintained below 160 mmHg with antihypertensive agents such as labetalol. Hypotension below 120 mmHg may result in cerebral ischaemia and must be treated aggressively with intravenous fluids and vasopressors, such as norepinephrine.

Anaesthetic management
The majority of patients requiring surgery will have suffered an SAH following aneurysm rupture, but a small proportion of patients will have had aneurysms diagnosed following CT/MRI as an incidental finding.

Premedication and preparation
Patients who have suffered a poor-grade SAH may already be intubated and sedated on the intensive care unit. In others, sedative premedication is usually avoided as it can impede neurological assessment, but anxious patients with a good grade may benefit from a short acting benzodiazepine. Haemorrhage can be extensive during neurovascular surgery and blood products must be available preoperatively.

Monitoring
The standard monitoring for neuroanaesthesia is required. In addition, a urinary catheter and a central venous catheter via the femoral vein guides fluid management and allows administration of vasopressors. More specialized intraoperative monitoring includes jugular bulb oximetry, transcranial doppler, oesophageal doppler, EEG, intraparenchymal probes and cerebral function monitors.

Induction and maintenance of anaesthesia
The induction agents of choice for neurovascular surgery are propofol or thiopental. Rupture of the aneurysm at induction of anaesthesia occurs in 1–2% of cases and carries a poor prognosis. Opioid analgesic agents (e.g. fentanyl 1–10 μg/kg) are used to obtund the hypertensive response to laryngoscopy. A non-depolarizing neuromuscular blocking drug with few haemodynamic side effects should be used to facilitate intubation of the trachea. Suxamethonium is avoided for elective surgery because it transiently increases ICP; however, it is the drug of choice if the airway needs to be secured rapidly in an emergency.

Most anaesthetists use a balanced technique with a volatile agent such as sevoflurane and an infusion of opioid such as remifentanil for maintenance of anaesthesia. Total intravenous anaesthesia with propofol has also been used.
ICP control
Brain relaxation is essential to optimize operating conditions and is achieved in several ways. A 30° head-up tilt encourages venous return and mild hyperventilation to a PaCO₂ of 4.5 kPa decreases cerebral blood volume and therefore ICP. In some cases ICP remains high and withdrawal of CSF by the neurosurgeon with an in-dwelling catheter improves operating conditions. Boluses of thiopental may be beneficial, as it causes cerebral vasoconstriction and reduces cerebral metabolism. In some centres the use of osmotic diuretic (mannitol) or loop diuretic (furosemide) are used to reduce brain bulk.

Temperature
Hypothermia decreases the active and basal components of cerebral metabolism and helps to protect the brain during times of ischaemia. There is no evidence that hypothermia during neurovascular surgery improves outcome and it is not without complications. It is usual to maintain normothermia during surgery and permissive hypothermia is reserved for the most challenging surgical cases.15,16

Fluid and electrolyte management
0.9% saline and Ringer’s lactate solutions are the usual choices for fluid maintenance as they help maintain plasma sodium levels (and osmotic pressure) in the normal range.

Emergence and recovery
Towards the end of the surgery, the neuroanaesthetist should ensure a normal blood pressure and PaCO₂, which will alert the surgeon to any bleeding points or brain swelling prior to closure of the dura. Excessive brain swelling following emergency surgery may require the bone flap to be left out, the insertion of an intracerebral pressure monitor and instituting a period of post-operative sedation and ventilatory support.

After uncomplicated surgery most patients are extubated under deep anaesthesia and are allowed to wake up. Adequate analgesia is vital and is usually achieved with a combination of simple analgesics (e.g. paracetamol) and opioids such as morphine given enterally or parenterally. Non-steroidal anti-inflammatory drugs are often avoided in the early post-operative period because of their effect on platelet function. Prophylactic anti-emetics such as ondansetron should be administered.

Blood pressure should be maintained at normal levels for the particular patient, unless the aneurysm has not been completely obliterated in which case, hypertension should be controlled appropriately to reduce the risk of re-bleeding. If the patient fails to recover to the expected GCS in the immediate postoperative period or recovers initially and develops worsening neurology, a further brain CT is required to exclude complications of surgery.

All patients are at high risk of venous thromboembolism and thromboembolic deterrent stockings should be worn with intermittent calf compression devices throughout the perioperative period. Pharmacological prophylaxis with low-molecular-weight heparin should be started on the second postoperative day providing there are no contraindications.17

REFERENCES