Subarachnoid haemorrhage

What is a subarachnoid haemorrhage? [1] [2]

A haemorrhagic stroke is defined as rapidly developing neurological dysfunction due to a focal collection of blood from within the brain parenchyma or ventricular system (intracerebral haemorrhage), or bleeding into the arachnoid space (subarachnoid haemorrhage) that is not caused by trauma.

The most common cause of a subarachnoid haemorrhage (SAH) is a ruptured cerebral aneurysm. Intracranial aneurysms are now considered to be acquired rather than congenital lesions and their prevalence is approximately 2%. Their cause is unknown.

90% of all cerebral aneurysms are less than 1 cm in size and have a relatively low risk of bleeding. 80-90% of all cerebral aneurysms are located in the anterior circulation of the brain (the internal carotid artery, the anterior and middle cerebral arteries, and their branches) and only 10-20% in the posterior circulation (the vertebral, basilar, and posterior cerebral arteries and their branches).

Unruptured aneurysms are usually asymptomatic, yet in 5% of cases they can give rise to epileptic seizures or, if large, to a thromboembolic event or a neurologic deficit due to mass effect (eg, an oculomotor nerve palsy).

Rarer causes include pathological vascular changes such as an arteriovenous malformation or fistula, vasculitis, arterial dissection, venous thrombosis, a tumour, or drug abuse.

How common is subarachnoid haemorrhage? (Epidemiology) [3]

Spontaneous SAH accounts for about 5% of all strokes. Ruptured aneurysms are the cause of 85% of spontaneous SAH. [4]

- SAH affects 7.9 people per 100,000 of the population per year and constitutes about 6% of first strokes.
- The relative risk for women is 1.3.
- SAH reported incidence declined by 40.6% in Europe between 1980 and 2010.
- This reduction may in part be attributed to a higher rate of CT scanning, excluding other haemorrhagic causes, although there is a clear correlation with the incidence of hypertension and smoking.
- Approximately 85% of patients bleed from intracranial arterial aneurysms, 10% from a non-aneurysmal peri-mesencephalic haemorrhage and 5% from other vascular abnormalities including arteriovenous malformation, vasculitis and abnormal blood vessels associated with tumour.
- SAH represents only 6% of cases of stroke but it is relatively far more important, as it tends to affect younger people, of whom about half die in that episode.
- The mean age is 50 years: most patients are under 60 years.
- Women have a higher risk than men: relative risk 1.6.
- Patients of Afro-Caribbean descent have a higher risk than white Europeans: relative risk 2.1:1
- Incidence is significantly higher in Finland and Japan.
- Spontaneous SAH is usually due to aneurysmal rupture but traumatic brain injury is a more common cause of blood in the subarachnoid space. Subarachnoid blood can be detected on CT scanning in as many as 60% of people with traumatic brain injury.

Risk factors

- Risk factors for spontaneous SAH are the same as for stroke in general, particularly smoking, excessive alcohol consumption, and hypertension. Genetic factors account for only a very small proportion of cases.

- The bigger the aneurysm, the more likely it is to bleed. However, as about 90% of aneurysms are small, the majority that bleed are less than 1 cm in diameter.[5]
- Hypertension - the global SAH incidence declined with every millimetre of mercury decrease in systolic blood pressure by 7.1%.[3]
- Smoking - the global incidence of SAH dropped by 2.4% for every 1% reduction in smoking prevalence.[3]
- Excessive alcohol intake roughly doubles the risk - its role has not been as well established as that of smoking, but drinking more than 37.5 units a week accounts for up to 1 in 5 cases of subarachnoid haemorrhage.[6]

Berry aneurysms[7]

- Berry aneurysms are common with a prevalence of approximately 4%.
- Most berry aneurysms under 7 mm do not rupture, but they grow unpredictably.
- 85% occur in the Circle of Willis. Multiple aneurysms are seen in 30% of patients.
- It is not clearly understood why some adults develop saccular aneurysms at arterial bifurcations in the Circle of Willis but most do not. There may be variation in the susceptibility of the elastic lamina of the arterial wall to the known stressors of hypertension and smoking.

Subarachnoid haemorrhage symptoms[8]

The most characteristic feature is a sudden explosive headache. The headache is sometimes referred to as a thunderclap headache.

Urgent investigation to confirm a diagnosis of subarachnoid haemorrhage facilitates early treatment to prevent re-bleeding from a ruptured aneurysm and minimises disability and death. The lack of clinical features distinguishing reliably between SAH and more innocuous headache means that a brief hospital consultation is needed for ALL patients with an episode of severe headache that comes on in minutes.

Where a person presents with unexplained acute severe headache, have a high index of suspicion for subarachnoid haemorrhage and take a careful history to establish the rate of onset and time to peak intensity of the headache.

A 'thunderclap' headache (a sudden severe headache, typically peaking in intensity within 1 to 5 minutes) is a red-flag symptom of subarachnoid haemorrhage.

Thunderclap headache is associated with other conditions or causes such as migraine, cough, coitus or exertion. Most people with a thunderclap headache do not have a subarachnoid haemorrhage, but this should not deter further investigation if subarachnoid haemorrhage is suspected.

People with subarachnoid haemorrhage can present with a range of non-specific symptoms and signs and are at greater risk of a diagnosis being missed. Other symptoms and signs include:

- Neck pain or stiffness.
- Photophobia.
- Nausea and vomiting.
- New symptoms or signs of altered brain function (such as reduced consciousness, seizure or focal neurological deficit).
- Limited or painful neck flexion on examination.

Refer people with suspected subarachnoid haemorrhage seen outside of acute hospital settings to an emergency department immediately for further assessment.

Warning symptoms and sentinel bleeds[9] [10]

A sudden, intense, and persistent headache, known as a warning or sentinel headache, sometimes presents during the days or weeks before aneurysmal SAH. These warning headaches have been interpreted as reflecting a minor or warning leak that arises from the first small bleed from an aneurysm before a major SAH.

The reported incidence of preceding warning signs in patients with SAH ranges from 15% to 60%. The true incidence of warning signs is difficult to establish because it is difficult or impossible to obtain complete information from patients in poor clinical condition or who die before reaching a hospital.

If a sentinel bleed is suspected, patients should be admitted urgently for investigations (treated as if an SAH has occurred).

Examination

Examination may reveal reduced conscious level. However, SAH patients can also walk into the surgery, complaining of sudden onset of headache. Neck stiffness may occur due to meningeal irritation by blood in the CSF, but it is not invariable.

Isolated pupillary dilation with loss of light reflex may indicate brain herniation as a result of rising intracranial pressure.

- Ophthalmoscopy may show intraocular haemorrhages.
- There may be focal neurological signs, suggestive of a stroke. Complete or partial palsy of the oculomotor nerve is well recognised, especially with rupture of aneurysms of the internal carotid artery at the origin of the posterior communicating artery.
- Oculomotor nerve impairment may indicate bleeding from the posterior communicating artery.
- Hypertension is a risk factor for the condition but a marked rise in blood pressure may also occur as a sympathetic reflex following intracerebral haemorrhage. This sympathetic reflex can raise blood pressure to life-threatening levels, and surges of adrenaline (epinephrine) may contribute to associated cardiac arrhythmias.

Differential diagnosis[11]

- Other causes of headache, particularly secondary headache and other causes of thunderclap headache - eg, migraine, cough, primary sexual headache or exertion headache.
- Other causes of stroke.
- Meningitis (rarely features thunderclap headache).
- Trauma.
- Cerebral venous sinus thrombosis.
- Cervical artery dissection.
- Carotid artery dissection.
- Hypertensive emergency (severely raised blood pressure).
- Pituitary apoplexy (infarction or haemorrhage of the pituitary gland).

Investigations[8]

All investigations are always performed in secondary care. The person should be referred for an urgent non-contrast CT head scan if review in secondary care by a senior clinical decision-maker confirms unexplained thunderclap headache, or other signs and symptoms that suggest subarachnoid haemorrhage. Diagnostic accuracy of CT head scans is highest within six hours of symptom onset. A subarachnoid haemorrhage is diagnosed if the non-contrast CT head scan shows blood in the subarachnoid space.

If a CT head scan done within six hours of symptom onset and reported and documented by a radiologist shows no evidence of a subarachnoid haemorrhage, a lumbar puncture should not be routinely offered and alternative diagnoses should be considered.

If a CT head scan done more than six hours after symptom onset shows no evidence of a subarachnoid haemorrhage, a lumbar puncture should be considered. There should be at least 12 hours after symptom onset before doing a lumbar puncture to diagnose a subarachnoid haemorrhage. Subarachnoid haemorrhage should be diagnosed if the lumbar puncture sample of cerebrospinal fluid (CSF) shows evidence of elevated bilirubin (xanthochromia) on spectrophotometry.

Alternative diagnoses should be considered if the lumbar puncture sample shows no evidence of elevated bilirubin (xanthochromia) on spectrophotometry.

A person with a diagnosis of subarachnoid haemorrhage should be urgently transferred to a specialist neurosurgical centre. The risk of re-bleeding is highest within 24 hours of the onset of symptoms.

CT angiography of the head should be offered without delay to people with a confirmed diagnosis of subarachnoid haemorrhage to identify the cause of the bleeding and to guide treatment. An aneurysmal subarachnoid haemorrhage if CT angiography of the head shows an intracranial arterial aneurysm and the pattern of subarachnoid blood is compatible with aneurysm rupture. Specialist opinion is required without delay from an interventional neuroradiologist and neurosurgeon if CT angiography of the head shows an intracranial arterial aneurysm and the pattern of subarachnoid blood is not compatible with aneurysm rupture.

If CT angiography of the head does not identify the cause of the subarachnoid haemorrhage and an aneurysm is still suspected, digital subtraction angiography (or magnetic resonance angiography if DSA is contra-indicated) should be considered. An aneurysmal subarachnoid haemorrhage should be diagnosed if digital subtraction angiography or magnetic resonance angiography shows an intracranial arterial aneurysm and the pattern of subarachnoid blood is compatible with aneurysm rupture.

Other diagnoses should be considered if digital subtraction angiography or magnetic resonance angiography does not show an intracranial arterial aneurysm.

Treatment[8]

Managing the culprit aneurysm

- Medical management: vasospasm is a serious and common complication of SAH which can lead to ischaemic brain injury, and which can be fatal. It affects around a third of admitted patients.Calcium antagonists help to reduce vasospasm. Nimodipine 60 mg four-hourly is generally used, as it has been shown to improve outcomes.[12] Enteral nimodipine should be considered for people with a confirmed subarachnoid haemorrhage. Intravenous nimodipine should only be used within a specialist setting and if enteral treatment is not suitable.
- Management also includes reducing the risk of venous thromboembolism and controlling raised blood pressure.
- An interventional neuroradiologist and a neurosurgeon should discuss the options, which are:
    - Endovascular coiling.
    - Neurosurgical clipping.
    - No interventional procedure, with monitoring to check for clinical improvement and reassess the options for treatment.

- If interventional treatment to secure the aneurysm is an option, offer endovascular coiling, or neurosurgical clipping if endovascular coiling is not suitable. Interventional treatment should be carried out at the earliest opportunity to prevent re-bleeding. The risk of re-bleeding is highest within 24 hours of the onset of symptoms.

Monitoring and managing complications

- For people with unexplained neurological deterioration after a subarachnoid haemorrhage, a non-contrast CT head scan should be offered as the first diagnostic investigation to determine the cause.
- Hydrocephalus: a diagnosis of acute or chronic hydrocephalus should be based on symptoms and signs, and on a comparison of current and previous CT or other brain imaging:
    - Acute hydrocephalus: drainage or diversion of cerebrospinal fluid should be considered for people with neurological deterioration caused by acute hydrocephalus.
    - Chronic hydrocephalus: for people with persistent or progressive symptoms and a clinical diagnosis of chronic hydrocephalus, drainage or permanent diversion of cerebrospinal fluid should be considered. If there is uncertainty about the likely benefit of permanent diversion, a trial of temporary drainage to assess the need for permanent diversion should be tried.
- Delayed cerebral ischaemia: ensure euvolaemia (normal blood volume) in people with delayed cerebral ischaemia after an aneurysmal subarachnoid haemorrhage and treatment with a vasopressor should be considered if symptoms persist. Clinical improvement from vasopressor treatment may be temporary.

Follow-up care

- Rehabilitation after aneurysmal subarachnoid haemorrhage should be offered in line with recommendations for stroke rehabilitation.
- Follow-up neuroimaging should be considered for people who have had an aneurysmal subarachnoid haemorrhage, taking into account the extent of their recovery and the suitability of further imaging.

Managing non-culprit (unruptured) aneurysms

The options for managing non-culprit (unruptured) aneurysms include endovascular coiling, neurosurgical clipping, and conservative management and follow-up monitoring.

Managing other conditions after discharge from hospital

- Management of hypertension.
- Do not withhold treatment with antiplatelets or anticoagulants solely on the basis of an aneurysmal subarachnoid haemorrhage if the culprit aneurysm has been secured by coiling or clipping. Balance the risks and benefits of treatment with an antiplatelet or anticoagulant, taking into account specialist assessment of the risk of a future subarachnoid haemorrhage.
- Smoking: encourage people who smoke to stop, and consider smoking cessation support.
- Management of headaches. Headaches in people with a history of aneurysmal subarachnoid haemorrhage are common and generally benign, but may be due to chronic hydrocephalus if the person has additional symptoms or signs, such as gait disturbance, incontinence, inco-ordination or cognitive impairment.
- Management of seizures.

Investigations to detect aneurysms in relatives

Routine testing to check for aneurysms in relatives has not been shown to save lives or prevent aneurysmal subarachnoid haemorrhages.

Testing for relatives is based on an assessment of the relative's own risk and is usually limited to people with at least two first-degree relatives (father, mother, sister or brother) who have had an aneurysmal subarachnoid haemorrhage.

Complications[2]

Immediately after an aneurysm ruptures and blood extravasates into the subarachnoid space, the intracranial pressure rises, sometimes to values above the diastolic blood pressure, up to 100 mm Hg, blocking further extravasation. The intracranial pressure usually falls again within a few minutes, although usually not all the way back to the pre-bleed level.

A second acute SAH due to re-bleeding of a ruptured aneurysm that has not yet been secured by clipping or coiling is a further clinically significant early complication (15% in the first 24 hours). Aneurysmal re-rupture and second bleeds are associated with a mortality of 70-90%.

Acute hydrocephalus, an intracerebral or (less commonly) subdural haematoma, and generalised cerebral oedema are treatable further sequelae that can cause acute and, often, potentially reversible neurological impairment in patients with acute SAH. About 30% of all patients with aneurysmal SAH develop hydrocephalus, reflecting disturbed circulation of the cerebrospinal fluid (CSF), at some time in the course of their disease, and go on to require a permanent CSF diversion procedure.

5% or more of patients develop epilepsy after discharge.

Cardiac complications are very commonly seen after acute SAH. Elevated catecholamine secretion after acute SAH can lead to myocardial necrosis and myocardial dysfunction. More than 90% of all patients with acute SAH have ECG abnormalities, and these can be very hard to distinguish from those of an acute myocardial infarction. Ischaemic signs (ST elevation) arrhythmias and prolongation of the QT segment may occur.

The cardiac stress resulting from SAH can lead to hypotension, which, in turn, exacerbates already existing cerebral hypoperfusion.

Electrolyte disturbances are also common, arising in approximately 30% of patients. The cause can be either:

- Cerebral salt-wasting syndrome: a renal loss of sodium during intracranial disease, leading to excessive loss of fluid and sodium in the urine, and therefore hyponatremia and a decrease in extracellular fluid volume (hypovolaemia); or
- Inappropriate ADH secretion (SIADH): unsuppressed release of antidiuretic hormone (ADH) from the pituitary gland or non-pituitary sources. The condition is characterised by impaired water excretion leading to hyponatraemia with hypervolaemia or euvolaemia.

Prognosis [1] [4]

Mortality rates in haemorrhagic stroke are significantly higher than in ischaemic stroke. Overall mortality at six months following SAH is more than 25%. Of people with SAH, 10-15% die before reaching hospital and 85% of people admitted to a neurosurgical unit with a confirmed aneurysm survive.

Most deaths occur as a result of re-bleeding from the same aneurysm. If untreated, re-bleeding occurs in 15% of people on day 1, and in 40% of people by one month after SAH.

Between 10% and 20% of all people with SAH (17%-46% of survivors) become dependent.
SAH results in cognitive impairment in a large number of people.

In a population-based study, 46% of survivors interviewed at one year reported incomplete recovery, with ongoing problems with memory, mood, and speech.

Factors associated with worse outcomes in haemorrhagic stroke include:

- Haemorrhage volume.
- Advanced age.
- Impaired consciousness at presentation.
- Rupture of the haematoma into the ventricular system.

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