

VASCULAR NEUROSURGERY

Subarachnoid haemorrhage

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'Spontaneous' SAH is usually the result of bleeding from a - ruptured aneurysm (approximately 80% of SAH) or an arterio - - venous malformation (A VM). Most ruptured aneurysms are located in the circle of Willis, at branch points in the arterial tree associated with turbulent blood flow (Figure 48.13 distinct subgroup of patients with SAH su ff er bleeds confined to the basal cisterns anterior to the midbrain and pons, without an underlying lesion evident on angiogram. This is termed perimesencephalic SAH , is believed to represent venous bleeding and has an excellent prognosis. Aneurysms may also develop as a result of infective infiltration of arterial walls in the context of bacteraemia (mycotic aneurysm), often in the setting of intravenous drug use or infective endocarditis. Pseudo- aneurysms may also develop after trauma or after surgery . Aneurysmal bleeding has an incidence of 10–15 per 100 /uni00A0 000 population per year. Risk factors include age, female sex, hypertension, smoking, cocaine abuse and a family his tory with two first-degree rela tives a ff ected. A range of genetic disorders, in particular adult polycystic kidney disease, fibro muscular dysplasia, neurofibromatosis type 1, Ehler s-Danlos and Marfan syndrome, are known to predispose patients to this condition. History and examination The typical presentation of an SAH includes a 'thunderclap' headache, which is both sudden and severe and is outside the patient's normal experience. Some patients describe prod romal headaches preceding the event, potentially representing aneurysm growth or subclinical bleeds. The sudden onset occurs commonly but not exclusively during e xertion, and may be associated with seizure (10%), unresponsiveness (50%) and vomiting (70%). Sometimes it is di ffi cult to establish whether SAH has caused a fall or a fall with head injury is responsible for the SAH. Approximately one-third of SAHs are incorrectly diagnosed at initial presentation. Patients are then at high risk of succumbing to early complications, especially a rebleed. Thomas Willis , 1621–1675, Sedleian Professor of Natural Philosophy at Oxford. Also the first anatomist to number the cranial nerves in the order used today . Edward Ehlers , 1863–1937, Professor of Clinical Dermatology , Copenhagen, Denmark. Henri Alexandre Danlos , 1844–1912, dermatologist, Hôpital St Louis, Paris, F Bernard Jean Antonin Marfan , 1858–1942, physician L'Hôpital des Enfants-Malades, Paris, France, described this syndrome in 1896. Albert Terson , 1867–1935, French ophthalmologist. Franciscus Sylvius , 1614–1672, a Dutch physician, chemist, physiologist and anatomist. grade') or the patient may have focal deficits and an impaired conscious level ('poor grade'). The World Federation of Neuro - surgical Societies (WFNS) grading of SAH is measured against the condition of the patient after resuscitation rather than at the time of ictus (Table 48.4). A painful third nerve palsy is typically the result of compression from a posterior communi - cating artery aneurysm. Meningitic features of neck sti ff ness and photophobia often develop over hours. Intraocular haem - orrhages, classically subhyaloid, may be visible on fundoscopy . The combination of SAH and vitreous

haemorrhage is known as Terson's syndrome and occurs in 15-20% of patients. Papilloedema should be sought, but may not be evident early in the course of a developing hydrocephalus. Investigation CT scan is the imaging of first choice; when performed within 12 hours of ictus, it will confirm bleeding in more than 98% of cases. This makes a diagnostic lumbar puncture unnecessary (Figure 48.14). Anderson, gave his account of this condition in 1908.

Anterior cerebral communicating
artery 36% 38% 21% Middle
cerebral Posterior artery
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Common sites of aneurysm in the
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Grade Glasgow Coma Scale Focal
de /f_ i cits I 15 - II 13-14 - III

13-14 + IV 7-12 ± V 3-9 ± a Focal

de /f_i cit = dysphasia or limb weakness. Figure 48.14 Diffuse subarachnoid bleeding from a ruptured anterior communicating artery aneurysm extends to the prepontine and ambi

ent cisterns around the brainstem and into both Sylvian /f_i ssures.

The sensitivity of CT scan, however, deteriorates to less than 50% at 1 week after a bleed. In light of this, patients with a suggestive history and negative CT scan will require lumbar puncture, especially where presentation is delayed. The CSF supernatant should be analysed by spectrophotometry (visual inspection is not reliable) for the spectra of haemoglobin breakdown products oxyhaemoglobin and bilirubin. These are present in samples taken at least 6 and preferably 12 hours after SAH, but not in CSF mixed with fresh blood due to trauma to exclude traumatic puncture and analysed immediately. Failure to do so with an adequate lumbar puncture sample can result in diagnostic confusion and overtreatment. Aneurysms can be visualised by CT and magnetic resonance imaging, but the gold standard remains digital subtraction angiography (DSA), which involves access to both vertebral and carotid arteries through the femoral artery under local anaesthetic. This allows visualisation of the vasculature by injection of contrast medium with simultaneous anatomical radiographic screening (Figure 48.15). The serious potential risks include ischaemic stroke or arterial dissection (1–2%), and renal failure or allergic reactions attributable to contrast.

Figure 48.15 There is a small saccular aneurysm of the pericallosal branch of the anterior cerebral artery.

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