Spontaneous intracranial haemorrhage

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Abstract

Spontaneous intracranial haemorrhage may present as a surgical emergency requiring rapid assessment of the patient and rapid access to diagnostic imaging. The site of the haemorrhage will determine whether there is global cerebral dysfunction or the development of a focal deficit. Hydrocephalus may complicate intracranial haemorrhage. Immediate decision-making as to the timing and means of treatment of the patient and the appropriateness of any intervention may be necessary. In this article we discuss the common types of intracranial haemorrhage, the significant features of the assessment and how these are used to guide treatment. Recent multicentre clinical studies have influenced decision-making for this group of patients.

Keywords Cerebellar haematoma; cerebral aneurysm; intracerebral haemorrhage; intraventricular haemorrhage; subarachnoid haemorrhage; subdural haemorrhage

Definition

Spontaneous intracranial haemorrhage occurs when there is spontaneous bleeding in the cranial cavity within the skull that is not caused by trauma. Intracranial haemorrhage may occur in any compartment within the cranial cavity and commonly consists of intraventricular haemorrhage (IVH), intracerebral haemorrhage (ICH), subarachnoid haemorrhage (SAH) and subdural haemorrhage (SDH). These forms of haemorrhage may co-exist and one may lead to one or more of the others (Figure 1).

Spontaneous **subdural haemorrhage** may be due to an iatrogenic hypocoagulable state and may be associated with warfarin or aspirin use. Haemorrhage from an underlying vascular abnormality such as an aneurysm (such as a middle cerebral artery aneurysm) may also be a cause.

Spontaneous **intraventricular haemorrhage** can either be due to extension of a subarachnoid or intracerebral haemorrhage into the ventricles or to bleeding from a lesion within the ventricle itself such as a tumour or arteriovenous malformation (AVM). In premature neonates IVH occurs as a consequence of fragile periventricular vessels. Haemorrhage into the ventricular system may cause obstructive hydrocephalus due to prevention of flow of cerebrospinal fluid (CSF) through the third ventricle, aqueduct or fourth ventricle. Communicating hydrocephalus may be caused by blockage of the arachnoid villi by blood thus preventing absorption of CSF into the venous sinuses.

Subarachnoid haemorrhage occurs when there is bleeding into the subarachnoid space from the medium-sized cerebral vessels which run between the pia mater and arachnoid mater. **Intracerebral haemorrhage** occurs when there is haemorrhage into the brain substance itself with destruction of brain tissue.

We will concentrate on the most commonly encountered forms of spontaneous intracranial haemorrhage in this article, that is, SAH and ICH.

Epidemiology

Spontaneous intracranial haemorrhage may be considered as a form of stroke and overall stroke mortality ranks third after cancer and heart disease in the UK. ICH may account for 20–30% of stroke and SAH about 10%.

The most common cause of **SAH** is an underlying cerebral aneurysm (85%). The global incidence of aneurysmal subarachnoid haemorrhage is 5–10 per 100,000 a year.[1] The peak age is between 40 and 60 years with a median age of 59 years old. It is more common in females than males by a ratio of 2:1. SAH is more common in Scandinavian countries and Japan. Smoking is a risk factor. Hypertension may be present in 60% of patients. The second most common cause of spontaneous subarachnoid haemorrhage is an arteriovenous malformation (AVM) (5%). Patients with an AVM are often younger with an average age of around 30 years. Other much rarer causes include vasculitides, tumours, arterial dissection and pituitary apoplexy. In about 15% no cause is found.

SAH may be associated with adult polycystic kidney disease, coarctation of the aorta, connective tissue disorders such as Ehlers–Danlos syndrome, Marfan's syndrome, and pseudoxanthoma elasticum. There may be a significant increase in family members where two or more first-degree relatives have proven aneurysmal SAH.

ICH may account for up to 30% of all strokes. The incidence is over twice that of SAH. Hypertension may be present in 70% of patients. Other risk factors include age (median age 73 years old), gender (more common in men), race (Afro-Caribbean twice as common), previous strokes, coagulopathies, alcohol consumption and use of illicit drugs such as cocaine. About 15% of ICH may be associated with warfarin use.[2]

Pathology and pathogenesis

Subarachnoid haemorrhage: the natural history of SAH and aneurysm development and rupture is not well known. In post mortem studies 0.5–2% of the population may harbour aneurysms yet the incidence of SAH is 10 per 100,000. SAH is associated with cardiovascular risk factors and theories of aneurysm development assume the development of atherosclerotic changes developing due to turbulence in a pre-existing weak area at the bifurcation of a medium-sized cerebral blood vessel (Figure 2). When an SAH first occurs there is a transient rise in intracranial pressure and reduction in cerebral blood flow. Many patients may be unconscious as a result at the time of the bleed, known as the 'ictus' and subsequently improve. Following an SAH delayed

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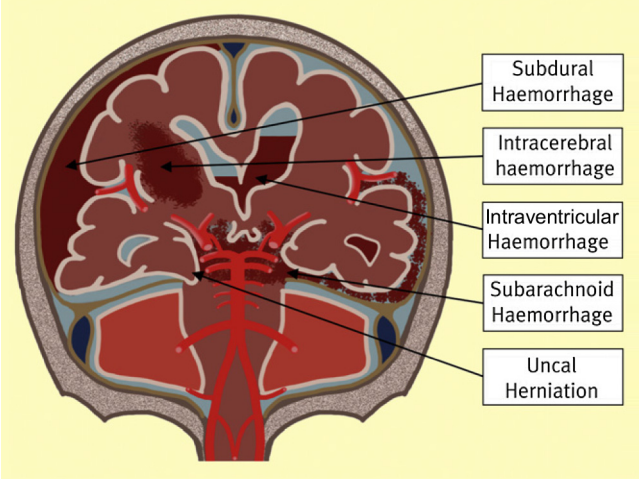

Figure 1

cerebral ischaemia 'cerebral vasospasm' may occur. This happens typically between 4 days and 10 days following SAH, but may last up to 3 weeks. There is vasoconstriction of medium-sized blood vessels which causes reduction of blood flow to the brain. This may cause neurological deficits which are potentially reversible. Cerebral vasospasm is caused by an imbalance between endothelin (a potent vasoconstrictor) and nitric oxide (NO) (a potent vasodilator). The chance of vasopasm resulting in an established stroke is reduced by administering 60 mg of nimodipine 4-hourly.

Intracerebral haemorrhage: 70% of ICH is associated with hypertension. These haemorrhages commonly occur in the basal ganglia and thalamus. Arterial rupture occurs in the lenticulostriate branches of the middle cerebral artery which have been weakened by fat and fibrin deposition in the media of the arterial wall. This may cause micro-pseudoaneurysms. ICH in normotensive patients may occur in the subcortical white matter. These haemorrhages may be due to a tumour, AVM, coagulopathy, cerebral amyloid or recreational sympathomimetic drug use (cocaine, amphetamine). ICH may also occur due to reperfusion of an ischaemic stroke causing a haemorrhagic infarct. The ICH itself causes destruction of cerebral tissue. There may then be subsequent haematoma growth with further neurological deterioration (a feature in approximately 30%). Surrounding the haematoma may be a penumbra of tissue that is compressed and ischaemic and as such is at risk of further cell death. Treatment strategies for ICH aim to prevent deterioration of this penumbra as well as preventing the consequences of raised intracranial pressure such as uncal herniation and brainstem compression ('coning').

Diagnosis (history, examination and investigations)

Subarachnoid haemorrhage is characterized by a sudden onset headache. This is typically described as the worst headache ever and builds in intensity over seconds. Patients often liken the headache to suddenly being hit on the back of the head. The headache may be associated with a transient loss of consciousness and with nausea and vomiting, photophobia and neck stiffness. The latter two are a direct result of meningeal irritation. A proportion of patients report a preceding headache in the previous few weeks which is usually of a lesser severity and is often referred to as a sentinel headache but still represents an SAH.

Neurological examination of the patient must include an assessment of the conscious level using the Glasgow Coma Scale (GCS) and an assessment of any cranial nerve deficit or limb weakness. On fundoscopy subhyaloid, retinal and vitreal haemorrhages are also associated with a spontaneous SAH.

Assessment of the patient should also include an assessment of respiratory function which may be compromised by neurogenic pulmonary oedema and blood pressure which may be elevated.

Patients may be categorized according to a grading scale such as the World Federation of Neurological Surgeons (WFNS) (Table 1). This scale is based on conscious level and presence or absence of a neurological deficit (excluding cranial nerve abnormalities). A patient in category 1–3 is often referred to as being in a good grade and a poor grade those in category 4–5. Poor-grade patients may require ventilation and treatment of hydrocephalus. The grade of patient may influence the timing and success rates of treatment as it reflects the physiological impact of the haemorrhage on the brain.

An unenhanced CT scan of the head is indicated in anyone presenting with a classical sudden onset headache irrespective of other symptoms and signs. This should be carried out as soon as possible as the sensitivity for SAH decreases with time (over 95% in the first 2 days). The CT scan (Figure 3) typically may show blood around the circle of Willis, in the basal cisterns, the interhemispheric fissure and the Sylvian fissure. There may be a coexisting ICH or SDH. Obstructive or communicating hydrocephalus may be present. If the CT scan is negative patients should undergo a lumbar puncture (LP) to determine whether there are breakdown products of blood. The gold standard is CSF spectrophotometry which would show a bilirubin peak if the patient had a SAH. The LP must ideally be carried out 12 hours after the onset of the headache to allow blood time to break down. It should be performed no later than 2 weeks as the sensitivity decreases significantly after that.

Once the diagnosis of SAH has been confirmed then imaging of the cerebral vasculature is required to determine the cause of the haemorrhage and any underlying vascular abnormality. CT angiography has replaced formal catheter angiography as the first vascular investigation in many centres (Figure 4a). The angiogram is examined for the presence of cerebral aneurysms, presence of vasospasm and variations in the anatomy of the Circle of Willis (which may be relevant when planning treatment).

Intracerebral haemorrhage: a spontaneous ICH presents most commonly with a sudden onset headache and is associated with a focal deficit that depends on the location of the haematoma. It is important in the history to establish the presence of risk factors such as hypertension and anticoagulant use. In the examination it is important to determine the degree of disability and neurological deficit as well as the blood pressure. There is usually no immediate improvement in the patient's condition following an ICH compared to an SAH.

