Medical Aspects of Subarachnoid Haemorrhage

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Although postmortem records in a general hospital showed that hypertensive cerebral vascular disease was the most common cause of spontaneous intracranial haemorrhage (Russell, 1954), a co-operative study in the United States (Sahs et al., 1969) indicated that intracranial aneurysms were the most frequent underlying lesions in cases of subarachnoid haemorrhage.

In their study of 5,836 patients who had had spontaneous non-traumatic subarachnoid haemorrhages, Sahs and his colleagues found that 51 per cent had aneurysms, in 15 per cent hypertensive arteriosclerotic vascular disease was the cause, and 6 per cent had arteriovenous malformations. There are many other causes of subarachnoid haemorrhage, including intracranial and spinal tumours, blood dyscrasias, inflammatory conditions of the brain and meninges, and the monoamine oxidase inhibitor drugs in association with amphetamines and tyramine-containing foods, but they are all rarities. It was a striking feature of their study that, despite careful clinical examination and either angiography or autopsy, no cause for the bleeding was found in 22 per cent.

The first subarachnoid haemorrhage from a single aneurysm occurs with a peak age frequency of 50 to 54 years; it is very rare in childhood and only some 3 per cent occur under the age of 15 years. Aneurysms are found more frequently in females than males, with a ratio of 3 : 2, but under the age of 40 years there is a predominance of males.

Subarachnoid haemorrhage is a common condition, having an estimated incidence of 6 per 100,000 in an area served by Atkinson Morley’s Hospital (Crawford and Sarner, 1965) and, as treatment, particularly that of an underlying aneurysm, improves the prognosis considerably, it is obviously vital to be able to recognise the condition clinically so that appropriate investigations may be undertaken. Walton (1956) pointed out that in his series of 312 cases, the correct diagnosis was made on clinical grounds by the referring doctor in only 46 out of the 162 cases in which the original letter of introduction was available. It is clear from this study that the clinical picture can and does present diagnostic difficulty.
CLINICAL FEATURES OF SUBARACHNOID HAEMORRHAGE

Symptoms
In various large series, it has been shown that premonitory symptoms and signs sufficient to warrant full investigation are produced in less than 10 per cent of aneurysm cases before rupture. Aneurysms at the junction of the internal carotid and posterior communicating arteries with the production of 3rd cranial nerve palsies, are most likely to do so, followed by lesions at the junction of the internal carotid and ophthalmic arteries, when orbital pain and disturbance of vision occur. Although unruptured middle cerebral aneurysms may produce focal fits and a hemiparesis, these are rare findings. Arteriovenous malformations, although much less common than aneurysms, relatively more often produce premonitory symptoms and signs in the shape of unilateral migraine, epilepsy which may be focal, hemiparesis, and intracranial bruits.

In about 90 per cent of patients the onset of the illness is abrupt, and in roughly one-third of them the first symptoms arise during sleep or repose, in one-third during random activity, and in one-third while the patient is carrying out actions liable to raise the arterial and venous pressures, such as lifting, bending, sexual intercourse, and straining at stool. Emotional turmoil may also play a part on rare occasions (Storey, 1969).

Headache is the first symptom of subarachnoid haemorrhage in 50 to 60 per cent of patients; indeed, only six of Walton’s (1956) cases, who were able to describe their symptoms, had no significant headache and in two of them the lesion was spinal. The most frequently used adjectives by patients in their description of the pain are sudden, bursting, excruciating, violent and like a blow on the head. The headache is most frequently felt in the occipital region or in the back of the neck, but may be situated anywhere and is of little localising value.

Loss of consciousness, which is usually sudden, occurs as the presenting symptom in 20 to 30 per cent of patients and is a feature at some stage of the illness in 50 per cent. The duration of the unconscious state is closely related to prognosis and patients do not often survive if it lasts longer than 24 hours.

Physical Signs
Some disturbance of consciousness is found in about 50 per cent of patients on admission to hospital and of these, about one-third are rousable, one-third withdraw to painful stimuli, and one-third show no reflex response. Even in those without clouding of consciousness, the mental state is normal in only about 25 per cent, mild confusion, irritability and amnesia occurring in the others.
Signs of meningeal irritation are a cardinal feature of subarachnoid haemorrhage; neck stiffness, which is almost invariably present, is a much more reliable index than Kernig's sign. It is usually associated with cerebral irritation, and the patient resents interference, often lying curled up with eyes turned away from the light.

Papilloedema is found in 10 to 15 per cent of patients. It may occur within minutes as the result of impairment of venous return from the retina caused by blood being forced into the optic nerve sheath, or it may appear later, secondary to raised intracranial pressure. A potentially more serious complication is the development of subhyaloid haemorrhages, which are usually large, have a brick-red appearance and may temporarily impair the eyesight. Complete recovery is the rule, although if there is rupture into the vitreous, organisation may occur, leading to permanent defects of vision.

Rather less than half the patients who have suffered a single subarachnoid haemorrhage show signs of a focal neurological deficit. In aneurysm cases, these signs are of more lateralising than localising value and this applies both to hemiplegia and cranial nerve palsy. Although rupture of a middle cerebral aneurysm is quite likely to produce a hemiplegia, and a 3rd nerve palsy to indicate a carotid aneurysm, these physical signs may result from rupture of aneurysms at other sites. Hemiplegia may be due to ischaemia secondary to arterial spasm rather than haemorrhage into the substance of the brain, and 3rd nerve palsy may be falsely localising, being brought about by temporal lobe herniation or constriction by blood clot. Subarachnoid haemorrhage secondary to an arteriovenous malformation is more likely to be associated with focal signs than if an aneurysm is the cause.

Crompton (1963) pointed out that micro-haemorrhages and regions of ischaemic necrosis in the hypothalamus are common after subarachnoid haemorrhage, particularly in cases of rupture of anterior and posterior communicating artery aneurysms. Stimulation of sympathetic centres within the hypothalamus and the release of catecholamines within the myocardium or systemically are thought to explain the hypertension, tachycardia and pulmonary oedema that may occur. The ECG changes may be indistinguishable from those of myocardial infarction. Other possible hypothalamic effects that may be seen are pyrexia, albuminuria, glycosuria, and haematemesis.
and subhyaloid haemorrhages. A 3rd nerve palsy on its own may be caused by pressure from a posterior communicating, carotid or even basal aneurysm and, in addition, may be produced by temporal lobe herniation against the tentorium. Third, 4th and 6th cranial nerve lesions on the same side almost always indicate an internal carotid aneurysm. A complete hemiplegia, which may be due to an intracerebral or subdural haematoma or infarction, does not indicate a particular site for an underlying aneurysm and, although a hemiparesis mainly affecting the arm and face suggests a temporal lobe herniation against the tentorium. Third, 4th and 6th cranial nerve lesions on the same side almost always indicate an internal carotid aneurysm.

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Arteriovenous malformations are suggested if there is a previous history of unilateral migraine, epilepsy, which is often focal, a history suggesting previous bleeds and the finding of physical signs indicating a lesion in the parietal or occipital lobes, a rarity in aneurysm cases. The presence of an intracranial bruit and dilated scalp vessels makes the diagnosis very likely.

In patients who are comatose, localisation of the underlying lesion on clinical grounds is usually impossible.

INVESTIGATIONS

Cerebrospinal Fluid

Examination of the cerebrospinal fluid by lumbar puncture is the means by which a clinical diagnosis of subarachnoid haemorrhage may be confirmed. The pressure, particularly in the early stages, is usually but not invariably raised, and the fluid is uniformly bloodstained. If specimens are taken in three bottles, there is progressively less blood when the tap is traumatic and there is no xanthochromia of the supernatant liquid, unless the amount of blood in the specimen is very considerable.

Spectrophotometer studies have shown that discoloration of the supernatant fluid by oxyhaemoglobin can occur within hours of subarachnoid haemorrhage, but that the darker colour of bilirubin does not appear for three to four days. On average, red cells disappear at about the ninth day after bleeding and the xanthochromia at about the twentieth. Initially, the white cells are proportional to the red, but later a lymphocytosis occurs.

A clear cerebrospinal fluid does not completely exclude a subarachnoid
haemorrhage, as tonsillar herniation or a circumscribed haematoma may seal off the area of haemorrhage from the lumbar sac (Heidrich, 1972).

**Radiology**
Radiological procedures, particularly angiography, are of the greatest importance in enabling the underlying lesion to be identified and localised. The details of the timing and range of these procedures are discussed by Mr Richardson (see page 245).

**Electroencephalography**
Binnie *et al.* (1969), using a detailed study of 35 criteria on each recording, were able to predict the site of bleeding of intracranial aneurysms with considerable accuracy. Of their 70 cases, full localisation was achieved in 57, partial localisation in 7, and in the remaining 6 they were unsuccessful. Even if the site of the bleeding aneurysm is determined by this method, the information obtained is obviously of much less value to the surgeon than angiography, but it does seem that if there are multiple aneurysms, the electroencephalogram is of help in deciding which one has bled.

**Other investigations**
If the underlying cause of the subarachnoid haemorrhage is not discovered by neuroradiological procedures, other investigations may have to be undertaken to exclude the presence of blood dyscrasias such as haemophilia, leukaemia, pernicious, aplastic and sickle cell anaemias, and polycythaemia vera.

**Prognosis**
Only the prognosis of those cases not treated surgically will be considered here.

**Mortality**
Of those patients in whom bleeding has been from a single aneurysm, 10 per cent die within 24 hours, 27 per cent within the first week, and 60 per cent within the first year. Re-bleeding is most frequent between the third and eleventh days, with a peak on the seventh, and carries with it a worse prognosis than the first episode, roughly 40 per cent dying.

If the haemorrhage is from an arteriovenous malformation, the outlook is better, the mortality figures for 24 hours, one week and one year being 7 per cent, 12 per cent, and 25 per cent respectively.

If all other causes are taken together, the corresponding figures are 12 per cent, 23 per cent, and 39 per cent. The hypertensive group does worst within
the first 72 hours and about 90 per cent of all those dying within this period of time have in addition an intracerebral, intracerebellar or subdural haematoma.

Bad prognostic signs are coma on admission to hospital, hypertension, focal neurological signs, particularly hemiplegia, and a history of a previous subarachnoid haemorrhage. Patients with a normal mental state who improve rapidly after the bleed have a good prognosis.

Morbidity
Roughly one-third of the survivors of subarachnoid haemorrhage recover completely, one-third are left with some physical disability, while the remainder are moderately or severely disabled. About two-thirds of the patients, however, are able to return to full employment.

Storey (1967, 1970) studied the psychiatric complications and found that 55 per cent of the survivors had some psychiatric disability. He found that intellectual impairment, apathy, euphoria and certain behaviour disorders depended on the amount of brain damage, whereas depression, anxiety and fatigue were more commonly related to the patient’s pre-morbid personality. The incidence of these abnormalities was less in those in whom no aneurysm was demonstrated. Storey (1967) also described an interesting group of 13 patients, 8 of whom had anterior communicating artery aneurysms, whose relatives described an improvement in their personality. He considered that this was due to a leucotomy effect.

Estimates of the incidence of epilepsy following subarachnoid haemorrhage vary widely in the various published series, from 6 per cent at one end of the scale to 52 per cent at the other. In a large series of 1,009 cases of ruptured aneurysms, Rose and Sarnar (1965) found that 53 of the 508 survivors (10.4 per cent) had fits. The seizures were usually generalised, but focal and temporal lobe attacks were also seen. Five of their patients had the first attack more than 18 months after the rupture. The likelihood of fits was greater in the younger age groups if the aneurysm was on the middle cerebral artery, and in the presence of an intracerebral haematoma. Those with residual signs of brain damage were particularly at risk, and they suggested that this group should be treated with anticonvulsants for a period of three years.

SUBARACHNOID HAEMORRHAGE IN PREGNANCY
Although many cases of subarachnoid haemorrhage have been reported in pregnancy, the evidence suggests that the association, in the absence of toxemia, is a random one. Labour does not appear to be the time when rupture of an aneurysm is particularly likely to occur and most authors recommend
that spontaneous labour should be allowed to occur, with forceps assistance in the second stage, in those with a previous history of subarachnoid haemorrhage during or before the pregnancy.

**SPINAL SUBARACHNOID HAE-MORRHAGE**

The clinical features of spinal subarachnoid haemorrhage have been well described by Henson and Croft (1956). They pointed out that the combination of sudden severe backache and root pain with signs of meningeal irritation were almost pathognomonic, but that diagnostic difficulty could occur when, in addition, cerebral symptoms and signs rapidly developed. Further good evidence is the appearance of a spinal cord lesion.

The most common cause of spinal subarachnoid haemorrhage is bleeding from an arteriovenous malformation, but intrathecal tumours, particularly ependymomas and neurofibromas, may also present in this way. More rarely, blood dyscrasias and polyarteritis nodosa have also been incriminated.

The nature of the underlying pathology has an important bearing on the prognosis and possible surgical treatment and investigation with myelography and in some cases spinal angiography should be undertaken.

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