The management of haemorrhagic stroke

Robin S Howard

Abstract

Intracerebral haemorrhage (ICH) accounts for 8–13% of all strokes and results from a variety of disorders. ICH is more likely to result in death or major disability than ischaemic stroke or subarachnoid haemorrhage. Rapid imaging allows early diagnosis and characterization of the localization and severity of the haemorrhage. Patients with acute ICH should be managed in an intensive care unit. Treatment entails general supportive care, control of blood pressure (BP) and intracranial pressure (ICP), prevention of haematoma expansion and, where indicated, neurosurgical intervention. The 30-day mortality from intracerebral haemorrhage (ICH) ranges from 35–52%. Among survivors, the prognosis for functional recovery depends upon the location of haemorrhage, size of the haematoma, level of consciousness, patient age, and overall medical health and condition. Rupture of arterial aneurysms is the major cause of subarachnoid haemorrhage (SAH). Aneurysm rupture releases blood directly into the cerebrospinal fluid (CSF) which spreads quickly within the CSF, rapidly increasing intracranial pressure. Death or deep coma ensues if the bleeding continues. The bleeding usually lasts only a few seconds but rebleeding is common and associated with a high mortality. Surgical clipping and endovascular coiling are effective in preventing rebleeding and generally should be performed early. Clinically significant vasospasm complicates 20–30% of aneurysmal SAH and is associated with delayed cerebral ischaemia and worse neurologic outcome.

Keywords Haemorrhagic stroke; intracerebral haemorrhage; subarachnoid haemorrhage

Royal College of Anaesthetists CPD Matrix: 2F01

Learning objectives

After reading this article, you should be able to:

- describe the initial assessment and managemant of acute haemorrhagic stroke
- identify the causes of haemorrhagic stroke
- identify the prognostic factors in acute haemorrhagic stroke

Intracerebral haemorrhage (ICH)

Approximately 10% of stroke is caused by brain haemorrhage with an annual incidence of 10–15/100,000 population. The incidence increases significantly after the age of 55 years and doubles with each decade to the age of 80. Intracerebral haemorrhage may be intracerebral, subarachnoid, subdural or extradural.

Causes and risk factors

ICH is strongly associated with a number of risk factors or structural abnormalities which include:

- hypertension – present in 40–60% of patients
- aneurysms (20%)
- vascular malformations (5–7%)
- anticoagulants
- coagulopathies
- tumours
- sporadic cerebral amyloid angiopathy
- secondary haemorrhage into a recent infarct (haemorrhagic infarction), including venous sinus thrombosis
- drugs of abuse (particularly, cocaine and amphetamine)
- vasculitis
- cerebral amyloid angiopathy (CAA).

Clinical presentation and syndromes of intracerebral haemorrhage

The rupture of a vessel or microaneurysm results in the sudden development of haematoma, of variable size which slowly enlarges over hours or days, especially in patients on anticoagulants, leading to progressive focal neurological deficit and then deterioration of conscious level secondary to mass effect. Headache, vomiting, and a decreased level of consciousness develop if the haematoma becomes sufficiently large. Seizures in the first days after ICH occur in <30%; the development of stupor or coma is an ominous sign. Neurologic signs vary depending upon the location of the haemorrhage. Bleeding into the putamen occurs in approximately 35%, subcortex in 30%, and, less commonly, into the cerebellum, thalamus and pons.

Deep haemorrhage

In putaminal haemorrhage (Figure 1) there is contralateral hemiparesis and conjugate deviation of the eyes towards the side of the haematoma. If the mass effect becomes critical then signs of herniation ensue and rupture into the ventricle may occur (Figure 2).

Lobar haemorrhage

In the frontal lobe, ipsilateral eye deviation, contralateral hemiparesis and/or hemisensory loss occurs, associated with dysphasia when the lesion is within the dominant hemisphere. Parietal lobe haemorrhage causes hemisensory loss and neglect/inattention syndromes. Bleeding into the dominant temporal lobe results in a fluent dysphasia with poor comprehension.

Infratentorial haemorrhage

Pontine haemorrhage (Figure 3) causes coma associated with pinpoint pupils, loss of horizontal eye movements and quadriparesis. Hyperpyrexia and irregular respiratory patterns ensue. Although a large haematoma here is often fatal, the outcome may be good in some patients. **Cerebellar haemorrhage** may result in

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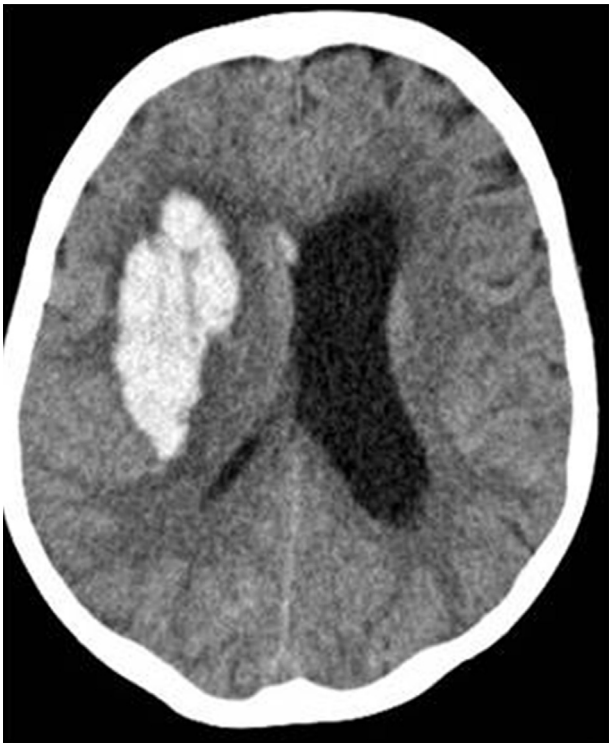

Figure 1 CT scan showing extensive ganglionic ICH.

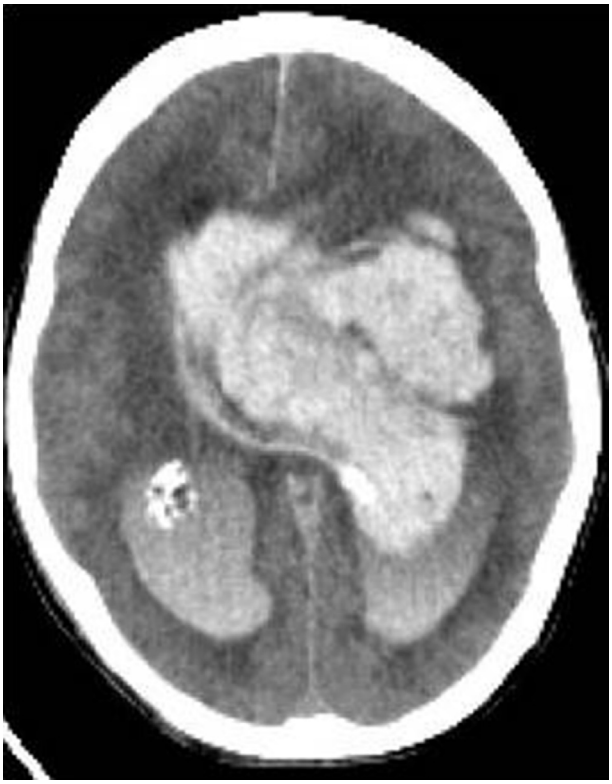

Figure 2 CT scan showing caudate haemorrhage which has ruptured into the ventricle.

secondary fatal brainstem compression and hydrocephalus. The usual picture is of acute headache and vomiting with unilateral ataxia.

Intraventricular haemorrhage

Intraventricular haemorrhage (Figure 2) mimics SAH with headache, vomiting, neck stiffness and depression of consciousness. There may be associated pyramidal signs, particularly if associated with a parenchymal haematoma.

Investigations

Acutely, a CT is essential to distinguish infarction from haemorrhage or reveal mimics of the stroke syndrome such as tumour or subdural haematoma. Haemorrhages seen on MRI shows complex changes depending on the image sequence; however, signal loss on susceptibility-weighted MRI is more specific and sensitive to new and old haemorrhages than the older MRI sequences. In appropriate patients it is necessary to exclude an aneurysm or underlying vascular malformations using CT or MR angiography. These may miss small lesions and catheter angiography is required in some cases.

Management (Box 1)

General supportive management issues are just as important as in ischaemic stroke. Medical treatment is aimed at correcting any underlying systemic disorder such as severe hypertension or coagulopathy, as well as preventing or limiting secondary complications such as pulmonary emboli, myocardial infarction, and pneumonia. Aspirin or any other medication with antiplatelet (clopidogrel, non-steroidal anti-inflammatory agents) or antithrombotic actions (dipyridamole) should be stopped.

Blood pressure

Early intensive lowering of blood pressure was shown to be safe in the second INTERACT trial. Secondary analyses suggested that this might improve outcome but the effects of rapid blood pressure reduction on cerebral autoregulation and perihaematoma ischaemia remain uncertain. At present, guidelines recommend a systolic BP < 160 mmHg (ATACH 2 trial).

Intracranial pressure should be kept <20 mmHg and cerebral perfusion >70 mmHg (see Box 1).

Haematoma expansion is a critical determinant of mortality and outcome. Anticoagulant-related haematomas often present as slowly evolving lesions. It is essential, even if they are small at initial imaging, to reverse warfarin immediately using vitamin K. The use of fresh frozen plasma (FFP) increases the risk of transfusion related lung injury so prothrombin complex concentrates (PCC) are preferable but costly. With increasing use of target-specific oral anticoagulants, improved point of care testing (e.g. TEG haemostasis analysis and platelet function tests) help direct most appropriate reversal. For patients with prosthetic valves the risk–benefit ratio is much in favour of anticoagulation reversal for a period of 2 weeks, after which the rebleeding rate is considerably lower. Newer anticoagulants such as rivaroxaban, apixaban or dabigatran may have a better safety profile but do not have specific antidotes.

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