Spontaneous intracerebral haemorrhage: a clinical review

This article provides a clinical overview of spontaneous intracerebral haemorrhage, focusing on clinical aspects of the aetiology, diagnosis and management (both in the emergency department and in a critical care environment) of this important and devastating condition.

Spontaneous intracerebral haemorrhage is the spontaneous, non-traumatic extravasation of blood into the brain parenchyma. This may extend into the ventricles and, rarely, the subarachnoid space. Spontaneous intracerebral haemorrhage is responsible for 10–20% of all strokes in the UK and has an estimated incidence of 10–20 per 100 000 among Caucasians, although this is higher in other ethnic groups (Broderick et al, 1992, 1999).

This article summarizes the classification, risk factors, clinical presentation and current management of patients with spontaneous intracerebral haemorrhage.

Classification

Primary spontaneous intracerebral haemorrhage occurs as a result of the spontaneous rupture of small blood vessels damaged by hypertension or amyloid angiopathy. This has an incidence of 7–17 per 100 000 and accounts for 78–85% of all cases (Gebel and Broderick, 2000). Secondary spontaneous intracerebral haemorrhage occurs in the presence of a pre-existing lesion such as a vascular or parenchymal abnormality, the most common cause being arteriovenous malformations. Other causes are listed in *Table 1*.

Aetiology of primary spontaneous intracerebral haemorrhage

A better understanding of the risk factors for primary spontaneous intracerebral haemorrhage can help prevent recurrence. These are discussed below, divided into modifiable and non-modifiable risk factors.

Table 1. Causes of secondary spontaneous intracerebral haemorrhage (in order of decreasing frequency)

Arteriovenous malformations
Cavernous haemangiomas
Intracranial aneurysms
Venous sinus thrombosis
Haemorrhagic transformation of ischaemic stroke
Coagulopathy
Intracranial tumours
Vasculitis

Non-modifiable risk factors

Age, sex and race

The risk of spontaneous intracerebral haemorrhage doubles every decade after the age of 35 years (Brott et al, 1986) and it is thought to occur more commonly in men, particularly after the age of 55 years. The incidence of spontaneous intracerebral haemorrhage also appears to be higher in the Japanese and Afro-Caribbean populations (Rincon and Mayer, 2008).

Cerebral amyloid angiopathy

Cerebral amyloid angiopathy is thought to be present in up to 50% of those over 90 years of age (Vinters and Gilbert, 1983), characterized by the deposition of β-amyloid protein in the media and adventitia of vessels in the cortex and the meninges. In the elderly, such deposition can lead to fibrinoid necrosis of the vessel wall, resulting in haemorrhage in the cortical and subcortical regions of the brain. The presence of cerebral amyloid angiopathy is significant as the risk of recurrence is increased in patients with cerebral amyloid angiopathy-related spontaneous intracerebral haemorrhage.

Modifiable risk factors

Hypertension

Hypertension is the most common and important risk factor for spontaneous intracerebral haemorrhage. It is thought that 75% of haemorrhages occur with a background of hypertension (Butcher and Laidlaw, 2003). Effective blood pressure control using antihypertensive medication significantly reduces the incidence of spontaneous intracerebral haemorrhage (Furlan et al, 1979).

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Serum cholesterol
Hypocholesterolaemia has been associated with an increased incidence of spontaneous intracerebral haemorrhage. The exact mechanism is not fully understood but it has been proposed that, in the context of hypertension, low cholesterol levels weaken the endothelial cells of intracerebral arteries and render them prone to rupture. However, this remains an area of controversy (Rincon and Mayer, 2008).

Alcohol
It is thought that heavy alcohol consumption can impair coagulation and distort the endothelial wall of the cerebral blood vessels, leaving them more fragile and prone to haemorrhage (Klatsky et al, 1989).

Smoking
Although smoking is a very well-established risk factor for ischaemic stroke and subarachnoid haemorrhage, its role in spontaneous intracerebral haemorrhage is more controversial. However, some studies have shown an increased risk of spontaneous intracerebral haemorrhage in smokers (Ruiz-Sandoval et al, 1999).

Anticoagulation
Randomized controlled trials have shown an up to fourfold increase in the risk of spontaneous intracerebral haemorrhage in patients taking long-term warfarin (Levine et al, 2001). This risk is thought to increase with intensity of anticoagulation, age and history of previous ischaemic stokes. Consideration of such factors may help when balancing an increased risk of spontaneous intracerebral haemorrhage with a reduced risk of thromboembolism during the initiation of anticoagulant therapy.

Pathophysiology

The pathophysiology of spontaneous intracerebral haemorrhage encompasses that of the primary haemorrhage itself along with the mechanisms of brain injury that ensue. In cases of primary spontaneous intracerebral haemorrhage, hypertension is the most commonly implicated cause of vessel disease. Hypertension leads to vasculopathy of small perforating arteries (diameter 50–400 μm) rendering these vessels prone to spontaneous rupture, particularly near sites of bifurcation (Takebayashi and Kaneko, 1983). Rupture of small penetrating arteries of the anterior, middle and posterior cerebral arteries and basilar artery leads to spontaneous intracerebral haemorrhage, occurring most commonly in the basal ganglia, thalamus, cerebellum, pons and cerebral lobes.

Primary brain injury in spontaneous intracerebral haemorrhage results from the haematoma itself spreading between white matter planes. This can lead to considerable mass effect with the catastrophic consequence of brain herniation possible.

Secondary brain injury results from various mechanisms, as discussed below.

Haematoma expansion

Haematoma expansion commonly occurs during the first 24 hours after onset of symptoms as a result of ongoing haemorrhage from the primary source, surrounding vessel disruption, acutely raised blood pressure and local coagulopathy (Brott et al, 1997).

Oedema

This occurs mainly within the first 5 days but may last for up to 2 weeks. Early oedema results from deposition of osmotically active serum proteins. Later oedema involves cytotoxic and vasogenic mechanisms.

Hydrocephalus

This may result from ventricular extension of the haematoma, or obstruction of the ventricular system caused by external pressure from displaced brain tissue, and is associated with worse outcome.

Ischaemia

Cushing (1902) proposed that brain injury in spontaneous intracerebral haemorrhage occurs as a result of the presence of the haematoma compressing surrounding tissue, leading to reduced perfusion and subsequent perilesional ischaemia. However, more recent studies have failed to support this theory (Qureshi et al, 1999).

Clinical features

These may be divided into generalized and location-dependent features (*Table 2*). Generalized features include reduced level of consciousness, which occurs as a result of a number of factors including increased intra-cranial pressure, compression of the reticular activating system and reduced benzodiazepine receptor binding. Nausea, vomiting, headache and acutely

Table 2. Clinical features of spontaneous intracerebral haemorrhage

Generalized	Reduced level of consciousness	
	Headache	
	Nausea and vomiting	
	Seizures	
	Acute hypertension	
	Meningism	
Location dependent	Supratentorial	Contralateral sensory-motor deficit
		Higher cortical dysfunction (dysphasia, hemianopia, neglect)
	Infratentorial	Contralateral motor deficit
		Cranial nerve dysfunction
		Gaze abnormalities
		Cerebellar signs (ataxia, nystagmus, dysmetria)

raised blood pressure may also result from increased intracranial pressure. Seizures may also be a feature of spontaneous intracerebral haemorrhage while symptoms of meningism may occur in the presence of ventricular blood.

Location-dependent features often involve a progressive neurological deficit with greatest deterioration during the first 24 hours because of the previously discussed mechanisms of secondary injury. Supratentorial lesions may result in contralateral sensory-motor deficit, because of the involvement of the internal capsule, and features of higher cortical dysfunction including dysphasia, hemianopia and neglect. In contrast, infratentorial lesions lead to contralateral motor deficit, cranial nerve dysfunction and abnormalities of gaze in cases of brainstem involvement, and ipsilateral ataxia, nystagmus and dysmetria in cases of cerebellar haemorrhage.

Diagnosis

The presence of headache, vomiting, hypertension or impaired consciousness is helpful in differentiating spontaneous intracerebral haemorrhage from cerebral ischaemia. However, clinical presentation is insufficient for a definitive diagnosis of spontaneous intracerebral haemorrhage, making imaging essential. Various imaging techniques used are discussed below. Routine investigations required to assess for the presence of any underlying pathology in spontaneous intracerebral haemorrhage are listed in *Table 3*.

Computed tomography

Computed tomography is the initial investigation of choice in cases of suspected spontaneous intracerebral haemorrhage to promptly determine the presence, location and size of the haematoma and to assess for the presence of intraventricular haemorrhage or hydrocephalus (*Figures 1* and *2*). Haemorrhage appears within an hour on brain computed tomography as an area of high attenuation associated with mass effect. This appearance lasts for several days. The haemorrhagic area then gradually loses its high signal until it eventually becomes hypoattenuating relative to the brain tissue.

Table 3. Routine investigations in spontaneous intracerebral haemorrhage

Full blood count
Urea and electrolytes
Liver function tests
Inflammatory markers
Coagulation
Blood glucose
Toxicology screen
Pregnancy test (women of childbearing age)
Electrocardiogram
Chest X-ray

Magnetic resonance imaging

The appearance of spontaneous intracerebral haemorrhage on magnetic resonance imaging is widely variable and depends on several factors, including the form of haemoglobin present, integrity of the red cell membrane and magnetic resonance sequence (T1 or T2 weighted images). Magnetic resonance imaging is generally more accurate than computed tomography in detecting struc-

Figure 1. Axial non-contrast enhanced computed tomography scan shows a hyperdense acute left lentiform nucleus haematoma surrounded by vasogenic oedema.

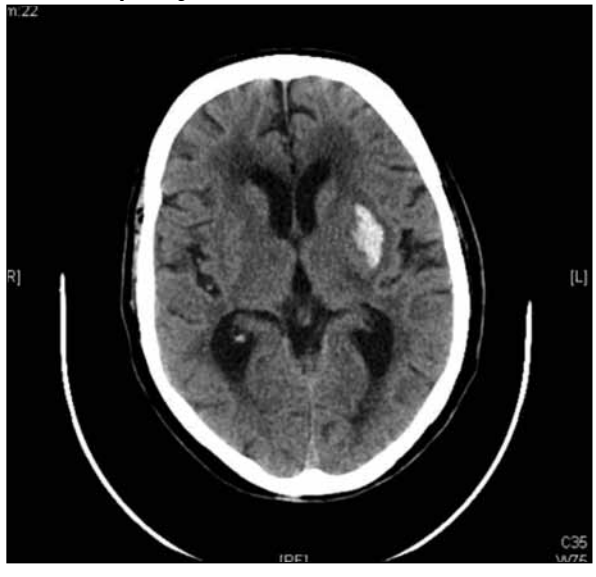
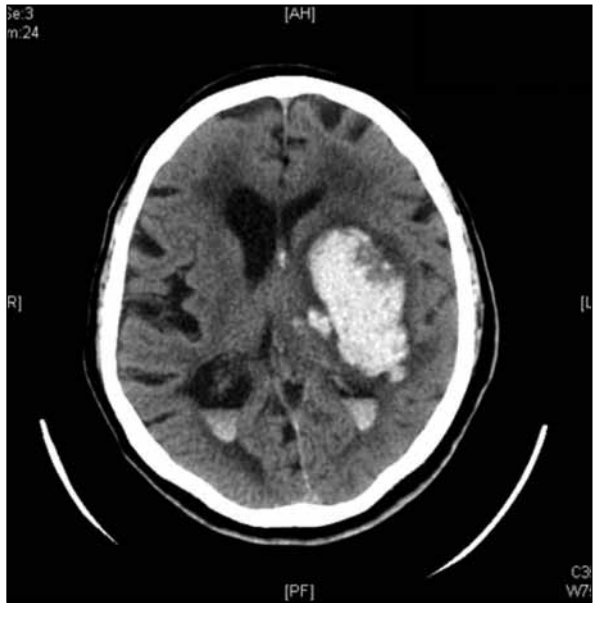


Figure 2. Computed tomography scan showing extensive left frontotemporal haemorrhage with a surrounding rim of vasogenic oedema, associated with significant mass effect, midline shift and intraventricular extension.



tural malformations, brain oedema, herniation and chronic bleeding (Broderick et al, 2007). Gradient-echo magnetic resonance is a sensitive sequence for diagnosing spontaneous intracerebral haemorrhage, and can be as sensitive as computed tomography for the detection of early spontaneous intracerebral haemorrhage (Kidwell et al, 2004).

Angiography

This invasive procedure is generally indicated in cases of lobar or primary intraventricular haemorrhage, regardless of age and blood pressure status, because of the high incidence of secondary causes of haemorrhage in these patients. In cases of putaminal, thalamic or cerebellar bleeds, angiography is usually reserved for patients aged 45 years or younger with normal blood pressure readings (Zhu et al, 1997). The American Heart Association recommends conventional angiography for all patients with no clear cause of haemorrhage (Broderick et al, 2007).

Computed tomography or magnetic resonance angiography

These non-invasive modalities are recommended as screening tests for the detection of underlying causes of spontaneous intracerebral haemorrhage such as major vascular anomalies and aneurysms; however, their sensitivity in detecting more subtle abnormalities is not well established (Huston et al, 1994). In the emergency setting, computed tomography angiography is less invasive and faster to perform than conventional angiography and is therefore more suitable for unstable and critically ill patients.

Management

Initial emergency department management

The emergency management of patients with spontaneous intracerebral haemorrhage should focus on maintaining the airway, breathing and circulation. Once this is completed, the patient should be fully exposed and examined looking for any signs of focal neurological deficit or external trauma, including any consequences of prolonged depressed consciousness. After the patient is stabilized, he/she should be transferred to an intensive care unit where the appropriate medical and surgical treatment can be commenced.

Critical care management

Owing to the risk of early deterioration in patients with spontaneous intracerebral haemorrhage, they should be treated in an intensive care unit for a minimum of 24 hours. The various aspects of management that must be considered are discussed below.

Medical

Blood pressure

The management of blood pressure for patients with spontaneous intracerebral haemorrhage is based upon balancing two rationales. The first stipulates that lowering blood pressure helps to reduce haematoma expansion while the argument against this states that lowering the blood pressure can reduce the cerebral perfusion pressure and thus exacerbate the ischaemic insult to the brain. There is currently no conclusive evidence for a specific blood pressure threshold in patients with spontaneous intracerebral haemorrhage. However, current guidelines state that patients with systolic blood pressure greater than 200 mmHg should be considered for aggressive treatment to reduce their blood pressure, although continual blood pressure monitoring is required (Broderick et al, 2007).

Intracranial hypertension

Raised intracranial pressure in patients with spontaneous intracerebral haemorrhage can impede cerebral blood flow and worsen brain ischaemia. Treatment goals are to keep the cerebral perfusion pressure above 70 mmHg (Chambers et al, 2001). Methods of reducing intracranial pressure include:

1. Head-of-bed elevation: elevating the patient's head to 30° is an important conservative method to help reduce intracranial pressure
2. Sedation: this should be adjusted to ensure patients remain in minimal pain but without affecting the clinical assessment of the patient
3. Osmotherapy: the use of mannitol and hypertonic saline solutions can help reduce brain oedema and lower intracranial pressure. Further research is required to determine specific thresholds and concentrations (Broderick et al, 2007)
4. Hyperventilation: Reducing carbon dioxide levels through hyperventilation is an effective method of reducing intracranial pressure, although its usefulness is limited by its transient effect. Target carbon dioxide levels are set at 30–35 mmHg (Stocchetti et al, 2005)
5. Barbiturate coma: the use of barbiturates can be considered in patients whose intracranial pressure remains elevated despite other treatment efforts (Broderick et al, 2007). However, patients should be carefully monitored because of the high risk of complications.

Haemostatic agents

There has been interest in the use of recombinant activated factor VII as a haemostatic agent to reduce haematoma growth in patients with spontaneous intracerebral haemorrhage. Early phase II studies looked promising (Mayer et al, 2005), but a larger phase III trial failed to show an improved clinical outcome (Al-Shahi Salman, 2009).

Body temperature

Since hyperthermia can worsen brain ischaemic damage, temperature should be kept within normal ranges in patients following spontaneous intracerebral haemor-

rhage, using cold baths, cooling blankets or medications such as paracetamol and acetaminophen (Broderick et al, 2007). Therapeutic cooling (temperatures of 32–34°C) is effective but carries high risks of complications (Schwab et al, 2001).

Seizures

Seizures are common following spontaneous intracerebral haemorrhage with rates as high as 18% in some studies (Cervoni et al, 1994) and should be treated with intravenous anticonvulsant medication. There is some evidence that early use of prophylactic anticonvulsants following spontaneous intracerebral haemorrhage can reduce early seizure rates (Passero et al, 2002).

Blood glucose

Hyperglycaemia on admission is associated with a worse short-term survival rate in patients with spontaneous intracerebral haemorrhage (Fogelholm et al, 2005). Current guidelines state that markedly raised glucose levels should be lowered to below 300 mg/dl (Broderick et al, 2007).

Surgical

Neurosurgeons play a vital role in treating spontaneous intracerebral haemorrhage and should be involved early in the patient's management. Their involvement can be broadly divided into intracranial pressure-specific procedures (CSF drainage and decompressive craniectomies) and craniotomies for haematoma removal.

CSF drainage

This is a very effective method of reducing intracranial pressure, and can be done intermittently following intraventricular catheter insertion to monitor intracranial pressure. The main complications are haemorrhage and infection.

Decompressive craniectomy

This surgical procedure involves removing part of the cranial vault to relieve raised intracranial pressure. There is a small amount of evidence of its effectiveness (Murthy et al, 2005), but more research is required.

Craniotomy

Craniotomies are the most studied surgical procedure for spontaneous intracerebral haemorrhage. The strongest current evidence for surgical removal of a haematoma is in patients with a cerebellar haemorrhage larger than 3 cm who are deteriorating neurologically or have brainstem compression or hydrocephalus (Broderick et al, 2007). A large randomized trial looking at the role of early surgical intervention for supratentorial spontaneous intracerebral haemorrhage showed that it had no overall benefit compared to initial conservative treatment (Mendelow et al, 2005).

Prognosis and outcome

Spontaneous intracerebral haemorrhage is associated with a higher mortality rate than other causes of stroke, with only 38% surviving 12 months (Dennis, 2003). Thirty-day mortality may be predicted by calculating the intracerebral haemorrhage score. This uses decreased initial Glasgow come scale, increasing haematoma volume, infratentorial location, ventricular extension and age above 80 years as predictors of increased mortality (Hemphill et al, 2001). This score has been validated in a small patient series in predicting 30-day mortality and it may have a use in predicting functional outcome at 1 year (Hemphill et al, 2009). Of the variables incorporated into the intracerebral haemorrhage score, initial Glasgow come scale and haematoma volume have been suggested to be the strongest predictors of 30-day outcome (Broderick et al, 1993).

Conclusions

Specialist care in critical care environments improves outcomes following spontaneous intracerebral haemorrhage (Barnett and Buchan, 2000). In survivors of spontaneous intracerebral haemorrhage, smoking cessation, limitation of alcohol consumption and blood pressure control are all recommended to reduce risk of recurrence (Broderick et al, 2007). However, evidence for other acute medical and surgical interventions is still lacking. There is still much work to be done in defining optimal treatment strategies to reduce morbidity and mortality in spontaneous intracerebral haemorrhage. **BJHM**

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KEY POINTS

- Spontaneous intracerebral haemorrhage is responsible for 10–20% of all strokes in the UK.
- Hypertension is the most important risk factor for primary spontaneous intracerebral haemorrhage.
- Spontaneous intracerebral haemorrhage commonly presents as a progressive neurological deficit accompanied by reduced level of consciousness.
- Angiography techniques play an important role in identifying treatable causes of secondary spontaneous intracerebral haemorrhage.
- Management of spontaneous intracerebral haemorrhage involves maintaining control of blood pressure, intracranial pressure, temperature, seizures and blood glucose and, in some cases, surgery.
- Management in specialist critical care environments improves outcome.
- Secondary prevention involves smoking cessation, reducing alcohol consumption and maintaining adequate blood pressure control.

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