An expanding intracerebral haematoma

Birkeland, Peter; Høgedal, Lisbeth; Rom Poulsen, Frantz

Published in: BMJ (Clinical research ed.)

DOI: 10.1136/bmj.h5014

Publication date: 2015

Document version
Publisher's PDF, also known as Version of record

Citation for published version (APA):
CASE REVIEW

An expanding intracerebral haematoma

Peter Birkeland senior clinical fellow1,2, Lisbeth Høgedal staff radiologist3, Frantz Poulsen senior consultant2,4

1 King’s College Hospital, Department of Neurosurgery, London SE5 9RS, UK; 2 Department of Neurosurgery, Odense University Hospital, Odense, Denmark; 3 Department of Radiology, Odense University Hospital; 4 Institute of Clinical Research, University of Southern Denmark, Odense

A 44 year old man presented to the emergency department with a sudden onset headache. He had no medical history of note and took no drugs. Urgent computed tomography of the head was performed (fig 1). A repeat scan (eight minutes later) was performed after administration of intravenous contrast (OptiRay) (fig 2). Upon return from the radiology department, he developed left sided hemiplegia. His blood pressure was 179/90 mm Hg. After another 46 minutes, computed tomography (fig 3) with angiography (not shown) was performed; no abnormal vessels were seen. Blood tests did not show an underlying coagulopathy. He subsequently deteriorated to localising to pain and making incomprehensible sounds to pain without opening his eyes (Glasgow coma score 8). His pupils remained equal and reactive to light.
The sequence of scans (figs 1-3) depicts an expanding intracerebral haematoma.

**Questions**

1. What causes of this bleed should be considered?
2. How should blood pressure be managed in patients with acute intracerebral haemorrhage?
3. When should surgical evacuation be considered?
4. What other management options could be considered?
5. How common is haematoma expansion?

**Answers**

1. **What causes of this bleed should be considered?**
   
   **Short answer**
   
   Aneurysmal subarachnoid haemorrhage should first be suspected in patients with a history of sudden onset headache. However, in this case the initial scan shows a small haematoma in the right basal ganglia rather than a subarachnoid haemorrhage. In patients with haemorrhage in the basal ganglia hypertension is the most common cause. Other causes include ruptured arteriovenous malformation, cavernoma, and tumour related haemorrhage.

   **Discussion**

   Aneurysmal subarachnoid haemorrhage should be at the top of the list of differential diagnoses in patients with a history of sudden onset headache, but if a haematoma originating from the right basal ganglia is detected there is no need for a lumbar puncture to rule it out. The sequence of scans shows a large deep seated haemorrhage originating from the right basal ganglia, which is a common site for hypertensive haemorrhages. Because the first computed tomogram (fig 1) did not suggest an underlying lesion, the haemorrhage is probably caused by previously unrecognised hypertension and the diagnosis is probably primary (hypertensive) intracerebral haemorrhage. Other common sites for hypertensive haemorrhages are the thalamus and cerebellum. Differential diagnoses include bleeding related to a micro- arteriovenous malformation.
cavernoma, or tumour. A contrast enhanced computed tomogram may not visualise an underlying tumour, and a computed tomogram or catheter angiogram may not detect an arteriovenous malformation owing to the mass effect of the haematoma. If there is no history of hypertension or the haematoma has an atypical location, the patient will need further investigation with interval magnetic resonance imaging or catheter angiography (or both) once the haematoma has resolved. Amyloid angiopathy is unlikely cause in this case because it usually manifests as lobar haemorrhages in older people.

2. How should blood pressure be managed in patients with acute intracerebral haemorrhage?

**Short answer**
Consider lowering systolic blood pressure to 140 mm Hg with a view to limiting haematoma expansion and improving outcome; however, there is limited evidence on which to base such a decision.

**Discussion**
Blood pressure often increases after haemorrhagic stroke. The rationale for lowering blood pressure is to limit the expansion of the haematoma and avoid an unfavourable outcome, but such an effect remains to be proved. American Heart Association/American Stroke Association recommendations state that in patients with systolic blood pressure of 150-220 mm Hg and without contraindications to acute blood pressure treatment, acute lowering of blood pressure to 140 mm Hg is safe and can improve functional outcomes.

If systolic blood pressure is above 220 mm Hg, it may be reasonable to reduce blood pressure with a continuous intravenous infusion.

Contraindications to acute lowering of blood pressure include increased intracranial pressure, Symptoms and signs of increased intracranial pressure are headaches, vomiting, and papilloedema (although papilloedema does not develop acutely) as well as Cushing’s response (hypertension, bradycardia, and slow irregular breathing).

Two randomised controlled trials have investigated whether rapid lowering of blood pressure with a target systolic pressure of less than 140 mm Hg would improve outcomes. INTERACT2 (Intensive Blood Pressure Reduction in Acute Cerebral Haemorrhage Trial 2) randomised 2839 patients with a spontaneous supratentorial intracerebral haemorrhage and raised systolic blood pressure within six hours of stroke onset to aggressive blood pressure reduction or blood pressure management with a target systolic blood pressure less than 180 mm Hg. Blood pressure was lowered by using agents of the physician’s choice, depending on local availability. INTERACT2 did not show that aggressive blood pressure reduction resulted in a better primary outcome (death or major disability). The second trial, ATACH II (Antihypertensive Treatment of Acute Cerebral Haemorrhage II), differed from INTERACT2 in several ways. The most notable differences were that treatment had to be started within three hours of the onset of stroke and the recommended drug was intravenous nicardipine. This trial is still recruiting.

3. When should surgical evacuation be considered?

**Short answer**
Surgery should not be considered at an early stage. In case of further deterioration, such as ipsilateral third nerve palsy suggesting impending transtentorial herniation, surgery may be undertaken at the discretion of a neurosurgeon to save life.

**Discussion**
The STICH (international Surgical Trial in Intracerebral Haemorrhage) trials were designed to answer the question of whether early surgery is beneficial. The first STICH trial, which investigated whether early surgery would improve outcome in spontaneous supratentorial haematomas, found no overall benefit from early surgery compared with initial conservative treatment. The STICH 2 trial included patients with intraventricular haemorrhages and included only those with a lobar haemorrhage less than 1 cm from the cortical surface (readily accessible surgical target). The STICH 2 trial largely confirmed the conclusions from the first STICH trial, that surgery should probably not be considered at an early stage. However, if the patient is likely to die from the mass effect of the haematoma, surgery may be undertaken even though it has no proved benefit. The STICH trials did not look at this clinical situation. Patients should have hourly neurological observations and if deterioration occurs further discussion with a neurosurgeon is warranted. Whenever possible the decision to proceed to surgery should be discussed with the patient, or, if this is not possible, the patient’s next of kin, to promote shared decision making. Factors that may favour a decision to proceed to surgery include in-hospital clinical deterioration, the need for intubation, superficially located haematoma, and absence of serious comorbidities.

4. What other management options could be considered?

**Short answer**
Treatment is largely supportive and the patient should be admitted to a dedicated stroke unit.

**Discussion**
The patient should be admitted to a dedicated stroke unit. Treatment is largely supportive, because no specific medical or surgical intervention has been shown to improve survival and outcome. If there is evidence of intracranial hypertension, an algorithm borrowed from traumatic brain injury guidelines can be considered.

The use of tranexamic acid is currently being investigated as a means to limit haematoma expansion and improve outcome. A spot sign (a focus of contrast enhancement within a haematoma on a computed tomography angiogram) is a predictor of haematoma expansion, and it be a useful as a tool to select patients who are likely to benefit from this intervention. Recombinant factor VIIa reduced haematoma growth in a phase III randomised controlled trial, but it did not improve overall outcome because patients receiving this factor had a higher risk of arterial thromboembolic events. Recombinant factor VIIa is therefore not currently used for this indication.

5. How common is haematoma expansion?

**Short answer**
Substantial haematoma growth occurs in about 40% of patients with spontaneous intracranial haemorrhage and most often in the first few hours after stroke.
Discussion

In a cohort of 103 patients presenting to hospital within three hours of haemorrhagic stroke, 38% of patients had more than 33% growth in haematoma volume.\(^{11}\) Another study found that in a cohort of 25 patients presenting within 4.5 hours of a haemorrhagic stroke, 10 patients had haematoma expansion of more than 12.5 mL during the first six hours in hospital.\(^{12}\) Although it is appealing to visualise haematoma expansion as being caused by a continuous bleed from a single vessel, it is more likely that expansion of the haemorrhage causes shearing and rupture of neighbouring vessels as a sort of avalanche effect.\(^{13,14}\)

Patient outcome

The patient underwent surgery to remove the haematoma. He remained intubated, sedated, and on mechanical ventilation for three weeks. Serial postoperative computed tomograms showed resolution of the haematoma and subsequent magnetic resonance imaging of the brain confirmed no underlying vascular disease. He was transferred to a local rehabilitation unit with a dense left sided hemiplegia. Nine months after the ictus he was discharged home and is now able to walk 200 m unaided.

Competing interests: We have read and understood BMJ policy on declaration of interests and declare the following interests: none.

Provenance and peer review: Not commissioned; externally peer reviewed.

Cite this as: BMJ 2015;351:h5014

© BMJ Publishing Group Ltd 2015