Intracerebral haemorrhage

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What is the epidemiology of spontaneous intracerebral haemorrhage (ICH)?

The incidence of ICH in the general population is 12–15/10000 per year, but reaches its peak of approx. 100/100000 persons per year in the population >75 years of age. It is responsible for 8–15% of all strokes in Europe and in North America, whereas in Japan and in China it accounts for 20–30% of all strokes. The 30-day mortality ranges from 35–50% and is thus 2–6 times that for ischaemic stroke [1, 3].

The management and prognosis of young patients presenting with ICH is entirely different as treatable pathologies are usually the underlying cause, e.g., vascular malformations (AVM, cavernomas etc.) or aneurysms, to name but a few.

Do prognostic criteria exist, and what are the consequences of spontaneous ICH?

The prognostic criteria associated with outcome are as follows:

– Initial level of consciousness, e.g., according to the Glasgow Coma Scale (GCS)
– Localisation of the haematoma
– Haematoma volume
– Additional ventricular haemorrhage
– Presence of midline shift
– Additional hydrocephalus
– Advanced age
– Neurological status on admission

Of these factors, low GCS grade and large haematoma volume are the most predictive of poor outcome. Mortality also relates to anatomical localisation of the haemorrhage, being highest in brainstem ICH.

Only approx. 20% of all affected patients may function independently six months after the ictus, and the lifetime cost per case is, according to the recent American literature, USD 124000. Chronic diseases of advanced age contribute negatively through increased rates of secondary complications in the course of the disease.

What are the causes and risk factors of intracerebral haemorrhage (ICH), and how does it occur?

Causes of ICH are as follows (frequent or rare according to the setting in a tertiary referral centre in Europe):

– Hypertensive haemorrhage (frequent)
– Amyloid angiopathy (frequent)
– Coagulopathy (frequent)
– Vascular malformations (AVM, cavernoma, aneurysm) (frequent)
– Haemorrhagic transformation of ischaemic infarction (frequent)
– Drug abuse (cocaine) (rare)
– Tumours (rare)
– Vasculitis / encephalitis (rare)
– Venous occlusion with consecutive haemorrhagic transformation (rare)

Risk factors of ICH:

– Hypertension
– Atherosclerosis
– Hypocholesterolaemia
– Previous brain infarction (increased 5–22 times compared with the normal population)
– Anticoagulants (increased 6–11 times compared with the normal population)
– Alcohol (increased 5 times compared with the normal population)
– Amyloid-angiopathy
– Advanced age (>65 years)

There seems to be a genetic disposition for lobar ICH in the presence of the apoEA2 or apoEA4 alleles. ICH, particularly in elderly patients, is frequently deeply located or ganglionic, and relates to long-standing arterial hypertension with concomitant atherosclerosis of small and fragile end-arteries. Even lobar ICH in the elderly may have its origin in a degenerative process of small arteries, so-called cerebral amyloidosis.

Once a haematoma has formed, the clot volume may increase in approx. 30% of patients during the first 24 hours. Morbidity and mortality are caused by the secondary effects exerted by the haematoma, such as neurotoxicity, neuronal ischaemia, and brain oedema: activation of the coagulation cascade with rupture of the blood brain barrier due to haemoglobin toxicity, for example, triggers immunologic-
What diagnostic measures should be taken in ICH?

After clinical assessment, the most important measure is cranial computed tomography (CCT). Recent CCT technology allows simultaneous angiography (CTa), and underlying vascular malformations can thereby be excluded. If in doubt, however, additional 3D digital subtraction angiography (DSA) should be performed if feasible. MRI plays a minor role in primary diagnostics due to limited access in ventilated patients. It may however be important during the further clinical course, e.g., for determination of brain damage, for perfusion imaging, and for exclusion or visualisation of vascular malformations.

It is of the utmost importance to rule out treatable pathologies such as cerebral AVMs, cavernomas, aneurysms, or dural AV fistulas as the underlying cause. Particularly in young(er) patients, ICH should be regarded as due to rupture of some form of vascular malformation, until proven otherwise.

What are the therapeutic options, and how should ICH be managed?

As in the case of supratentorial ICH, unfortunately no ideal and promising treatment regimen exists. Thus it is immaterial whether these patients are treated surgically (via open craniotomy, stereotactic aspiration and haematoma lysis, or endoscopically), or receive intensive care based on a conservative internistic/neurological regimen. Thus it is fully justified for neurosurgeons to refrain from expensive surgical therapy of whatever type (open craniotomy, stereotactic aspiration and haematoma lysis/drainage, or endoscopic aspiration). This almost fatalistic attitude has been substantiated by an international multi-centre study (STICH trial) in more than 1000 patients in whom no difference in outcome was noted between the groups, who received either initial surgical treatment or conservative treatment respectively [5]. However, if patients with additional VEH are excluded from the surgical group there may be some beneficial effect from surgery. It is intended to elucidate this further through the consecutive STICH II trial, which may help to identify a group which would possibly benefit from surgery. For example, the management of lobar haemorrhage in hypertensive patients aged between 45 and 70 is still controversial, and thus it is justifiable to decide according to department policy and/or on a case-by-case basis. The subgroup analysis of the STICH trial suggested that this group may benefit from surgical evacuation of the haematoma [6].

A large randomised trial of the use of recombinant factor VIIa (rFVIIa) showed that secondary haematoma growth could be reduced, but this did not translate into reduced mortality or a better outcome, due to increased frequency of thromboembolic events [4].

Management of supratentorial ICH remains, in conclusion, conservative and supportive. It should focus on reduction of increased ICP, restoration of cerebral blood flow, and avoidance of secondary complications [2, 3]:

- Take a precise clinical and patient history!
- Perform a precise neurological exam (including GCS)
- Assessment of respiratory and circulatory situation
- Keep upper part of body at 30°
- Ensure adequate oxygenation
- Sedation in unstable patients (to be maintained on neurological ICU)
- Reverse anticoagulation (if INR >1.4) (PCC, FFP, vitamin K)
- MAP <130 mm Hg (±180/110 in hypertensives)
- ECG surveillance
- Avoid excessive hypo-/hypertension (ensure physiological CPP!)
- Although the role of hyperosmolar substances is unclear, they may be administered
- Sensitive temperature management (fever worsens prognosis)
- Control laboratory results regularly (e.g., creatinine, INR, PTT, glucose, etc.)
- Perform frequent neurological/circulatory monitoring (apply cuff, or arterial line, and central venous line)
- (External drainage in case of concomitant hydrocephalus independently of estimated prognosis)

The position with regard to infratentorial, or cerebellar, ICH is different: surgical management of these haematomas improves prognosis and they should be operated upon if they do not extend into the brainstem [1].

Intense joint efforts are required from clinical and basic neuroscientists of all groups, together with internists and intensivists, in order to develop preventive measures or reasonable treatment options for ICH – particularly in our ageing societies.

Key words: ICH; conservative treatment; surgery; management

References