ICH for the Hospitalist

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Outline

- Who is going to get worse
- When do you (they) operate?
- What can you do medically
- Common issues
- A brief word of caution

What are we going to talk about?

- Intracranial/intracerebral hemorrhage
- Non-aneurysmal
- Non-traumatic

It’s a big deal

- >1,000,000 cases worldwide annually
- 63,000,000 DALY for ICH
- 39,000,000 DALY for ischemic stroke
Clinical Presentation

- Can’t reliably differentiate from ischemic stroke
- Clues - h/a, LOC, n/v
- Imaging - location

Prognostication

Volume of blood

| 30 day mortality | Volume ≤30cm³  
| GCS ≥9         | Volume ≥60cm³  
| GCS ≤8         |
|-----------------|----------------|
| 19%             | 91%            |

Broderick et al, Stroke 1993
Introduction

Infratentorial origin of ICH

Extravasation

<table>
<thead>
<tr>
<th>Component</th>
<th>ICH Score Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glasgow Coma Scale</td>
<td></td>
</tr>
<tr>
<td>3–4</td>
<td>2</td>
</tr>
<tr>
<td>5–12</td>
<td>1</td>
</tr>
<tr>
<td>13–15</td>
<td>0</td>
</tr>
<tr>
<td>ICH volume (cc)</td>
<td></td>
</tr>
<tr>
<td>( \geq 30 )</td>
<td>1</td>
</tr>
<tr>
<td>(&lt; 30 )</td>
<td>0</td>
</tr>
<tr>
<td>Presence of IVH</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Infratentorial origin of ICH</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
</tr>
<tr>
<td>( \geq 80 )</td>
<td>1</td>
</tr>
<tr>
<td>(&lt; 80 )</td>
<td>0</td>
</tr>
<tr>
<td>Total ICH Score</td>
<td>0–6</td>
</tr>
</tbody>
</table>

Table 2 The ICH Score [10]

See Spot Mortality

<table>
<thead>
<tr>
<th></th>
<th>All ICH</th>
<th>Supratentorial ICH</th>
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</thead>
<tbody>
<tr>
<td>Extravasation</td>
<td>63.5%</td>
<td>53.6%</td>
</tr>
<tr>
<td></td>
<td>33/52 ( p=0.01 )</td>
<td>15/28 ( p&lt;0.001 )</td>
</tr>
<tr>
<td>No Extravasation</td>
<td>16.4%</td>
<td>12.2%</td>
</tr>
<tr>
<td></td>
<td>10/61</td>
<td>5/41</td>
</tr>
</tbody>
</table>

Panel 2: Clues for underlying causes of intracerebral haemorrhage

Deep perforating vasculopathy
- Haematoma located in the basal ganglia or brainstem; microbleeds or old intracerebral haemorrhage in the basal ganglia or brainstem; white matter lesions; lacunes

Cerebral amyloid angiopathy
- Lobar intracerebral haemorrhage; cortico-subcortical microbleeds; cortical superficial siderosis; apolipoprotein E \( E^4 \); cognitive decline; transient focal neurological episodes

Brain arteriovenous malformation
- Extension to other brain compartments; flow voids; calcification

Infractorial arterial aneurysm
- Disproportionate subarachnoid extension

Cavernous malformation
- Small, homogeneous intracerebral haemorrhage with no extension to other brain compartments

Infractorial venous thrombosis
- Headaches preceding intracerebral haemorrhage onset; intracerebral haemorrhage close to sinuses or veins; high relative oedema volume; onset in pregnancy or postpartum

Dural arteriovenous fistula
- Subarachnoid or subdural extension; abnormal dilated cortical vessels

Haemorrhagic transformation of cerebral infarction
- Substantial areas of acute ischaemic lesions adjacent to the intracerebral haemorrhage or diffuse acute ischaemic lesions in other arterial territories

Severe clotting factor deficiency such as haemophilia
- Abnormal coagulation tests

Tumour (primary/metastasis)
- Large perivenous oedema

Vasculitis
- Headaches; small acute ischaemic lesions in different arterial territories; focal diffuse arterial stenosis

Infective endocarditis
- Acute ischaemic lesions in different arterial territories; small irregular arterial aneurysms; diffuse brain microbleeds

Posterior reversible encephalopathy syndrome
- Thunderclap headaches; perinatal and occipital asymmetrical oedematous lesions
Management

- Surgery
- Blood pressure
- Edema - Na, osmotic agents
- Sz
- Glucose
- VTE ppx
- Restart blood thinners

When do you operate?

- Major trials: STICH & MISTIE
- No difference in functional outcome, long term mortality
But

- Cerebellar hemorrhage >3cm
- Brainstem compression
- Hydrocephalus

Efficacy and safety of minimally invasive surgery with thrombolysis in intracerebral haemorrhage evacuation (MISTIE III): a randomised, controlled, open-label, blinded endpoint phase 3 trial

- 506 Patients with >30ml of ICH
- Spontaneous, non-traumatic, supratentorial
- Safe
- Better functional outcome if clot reduced to <15ml

Figure 3: Overall survival
Data was censored at day 365. Shaded areas show 95% CIs. HR-hazard ratio.
Early surgery versus initial conservative treatment in patients with spontaneous supratentorial lobar intracerebral haematomas (STICH II): a randomised trial

- 601 patients, parallel group
- Spontaneous ICH, no IVH
- ICH close to cortical surface 10-100 ml within 48 h
- 20% of initial conservative had surgery

Interpretation: The STICH II results confirm that early surgery does not increase the rate of death or disability at 6 months and might have a small but clinically relevant survival advantage for patients with spontaneous superficial intracerebral haemorrhage without intraventricular haemorrhage.

STICH 2

- Small benefit for superficial ICH without IVH
- No difference in death or disability
Future directions

- Seems like we’re getting close to
  - patients who might benefit
  - newer devices, MIS

BP parameters should be ordered with confidence and precision

ICH BP - the studies

- INTERACT & ATACH - SBP <140 is safe
- INTERACT 2
  - either SBP <180 or <140 for 7 days
- Improved mortality/disability with low bp

Current Guidelines

- For ICH patients presenting with SBP between 150 and 220 mm Hg and without contraindication to acute BP treatment, acute lowering of SBP to 140 mm Hg is safe (Class I; Level of Evidence A) and can be effective for improving functional outcome (Class IIa; Level of Evidence B).
- For ICH patients presenting with SBP >220 mm Hg, it may be reasonable to consider aggressive reduction of BP with a continuous intravenous infusion and frequent BP monitoring (Class IIb; Level of Evidence C).
ATACH 2

- 24 hrs of <180 or <140 with nicardipine
- no difference in death/disability
- With or without strict maintenance of intensive SBP goals
- Increased neurological deterioration
- Increase in cardiac-related adverse events

To AED or not

- What causes seizures in these patients?
- Incidence up to 16%

Don’t

- Empirically start AED’s

Do

- Have a low threshold for EEG
- Treat clinical seizures
- Treat electroencephalographic seizures
You’re a hospitalist

- Treat high or low sugars
- Treat fevers (probably)
- Use VTE ppx

VTE is Bad

- Intermittent pneumatic compression
- Document cessation of bleeding, then ok to use LMWH/Heparin at 1-4 days
- No great data on IVC filter vs. anticoagulation

Cerebral Edema

- Sodium
- Osmotic agents
- Monitoring - no exact metrics, consider for GCS ≤8, mass effect suggestive of high ICP, hydrocephalus

What are the options?

- Remove brain/lesion - surgeon
- Reduce CSF - ventric
- Reduce blood - hyperventilate, decr CMRO$_2$, raise HOB
- Reduce parenchymal volume - osmotic diuretics
hypertension, bradycardia, and irregular respirations or apnea (Cushing’s triad) although the concurrence of all three signs is an uncommon and often late finding.

Common sites for herniation are the cingulum (subfalcine herniation), medial temporal lobe (uncal herniation), and inferior cerebellum (tonsillar herniation).

The cardinal signs of transtentorial (uncal) herniation are an acute loss of consciousness associated with ipsilateral pupillary dilation and contralateral hemiparesis, resulting respectively from compression or displacement of ascending arousal pathways, oculomotor nerve (III), and corticospinal tract.

In a subset of patients, herniation-associated shift of the midbrain compresses the contralateral anterior cerebral peduncle (crus cerebri) against the tentorium, resulting in hemiparesis that is ipsilateral to the lesion (Kernohan’s false localizing sign).

Transtentorial herniation may cause ipsilateral cerebral infarction due to occlusion of the posterior cerebral artery.

**Neuroimaging**

In the emergent setting of a brain code, a cranial computed tomography (CT) scan should be obtained to identify a process that may require surgical intervention. Initial resuscitative measures and stabilization, including airway interventions, circulatory and ventilatory support, and initial hyperosmolar therapy, must occur prior to diagnostic imaging. Cranial CT is preferred over magnetic resonance imaging (MRI) due to availability and speed of imaging. In the majority of cases, CT will identify the underlying process (see Table 1), although MRI may subsequently be needed for further characterization. MRI should only be sought once the imminent risk of an additional brain code has been mitigated by medical and/or surgical intervention.

**ICP Monitoring**

ICP monitors are invasive and are of several different types, including intraventricular catheters as well as intraparenchymal, subdural, and epidural devices. The decision to proceed with ICP monitoring is determined by the

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**Restart Anticoagulation**

- Low risk, largely safe in the long term
- Decreases other cardio- and cerebrovascular events
- Timing is the question

The optimal timing to resume oral anticoagulation after anticoagulant-related ICH is uncertain. Avoidance of oral anticoagulation for at least 4 weeks, in patients without mechanical heart valves, might decrease the risk of ICH recurrence (Class IIIb; Level of Evidence B). (New recommendation)

If indicated, aspirin monotherapy can probably be restarted in the days after ICH, although the optimal timing is uncertain (Class IIa; Level of Evidence B). (New recommendation)

Anticoagulation after nonlobar ICH and antiplatelet monotherapy after any ICH might be considered, particularly when there are strong indications for these agents (Class IIb; Level of Evidence B).

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**Optimal timing in afib?**

Kuramatsu et al., European Heart Journal 2018
Afib

- Balance per day risk of stroke with per day risk of bleed
- Consider how you evaluate the bleed risk
- Exact risk unclear - no great data
- Many wait 4-6 weeks
- Some observational studies suggest 7-8 weeks may be optimal

Self-fulfilling prophecy

Aggressive full care and postponement of new DNR orders until the second full day of hospitalization “probably recommended”

Good Resources

- Neurocritical Care Society - ENLS
- Neurohospitalist Society
- Society of Hospital Medicine
- AHA/ASA guidelines