Neurointensive Care of Aneurysmal Subarachnoid Hemorrhage

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The traditional view: aSAH is a bad disease

• Pre-hospital mortality 16%
• 30-day case fatality 25-30%
  ➢ Higher with advancing age
• Frequent substantial morbidity in survivors
  ➢ Cognitive > physical
aSAH is not a functional disability sentence

<table>
<thead>
<tr>
<th>Study</th>
<th>Favorable outcome</th>
<th>Sample size</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISAT (2002) Coiling Clipping</td>
<td>76.4% 69.4%</td>
<td>2143</td>
</tr>
<tr>
<td>CONSCIOUS 2 (2011)</td>
<td>77.7%</td>
<td>1157</td>
</tr>
<tr>
<td>CONSCIOUS 3 (2012)</td>
<td>78%</td>
<td>571</td>
</tr>
<tr>
<td>BRAT (2013)</td>
<td>74%</td>
<td>403</td>
</tr>
<tr>
<td>STASH (2014)</td>
<td>72%</td>
<td>803</td>
</tr>
<tr>
<td>Mayo Clinic (2001-13)</td>
<td>71% (of all patients) mRS 0 = 32.7% mRS 1 = 30.6%</td>
<td>586</td>
</tr>
</tbody>
</table>
# A complex disease

<table>
<thead>
<tr>
<th>Neurological complications</th>
<th>Systemic complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aneurysm re-bleeding</td>
<td>Neurogenic pulmonary edema</td>
</tr>
<tr>
<td>Cerebral edema</td>
<td>Neurocardiogenic injury (apical ballooning syndrome)</td>
</tr>
<tr>
<td>Seizures</td>
<td>Cardiac arrhythmias</td>
</tr>
<tr>
<td>Raised intracranial pressure</td>
<td>Hyponatremia</td>
</tr>
<tr>
<td>Hydrocephalus (early and delayed)</td>
<td>Infection</td>
</tr>
<tr>
<td>Vasospasm (causing ischemic infarction)</td>
<td>Venous thromboembolism</td>
</tr>
</tbody>
</table>
Neurointensive care matters

• Care in an NICU led by a dedicated neurointensivist is independently associated with improved outcomes in aSAH:
  ➢ Shorter duration of stay in ICU and hospital
  ➢ Improved disposition
  ➢ Improvement in functional outcomes

Suarez et al Crit Care Med 2004;32:2311-7
Varelas et al Neurocrit Care 2008;9:293-9
Josephson et al J Neurosurg 2010;112:626-30
Time Course of SAH

Initial Stabilization

Delayed Complications
Initial Management

- Ensure adequacy of ABC
- Confirm diagnosis of SAH
- Fluids, analgesia, antiemesis
- BP control
- Treatment of hydrocephalus
- Identify early systemic complications
- Angiography
Initial Management

• Nimodipine
• Anticonvulsants
  - Only if seizures at onset
  - Try to avoid phenytoin
• Antifibrinolytics
  - Reduce risk of re-bleeding if started very promptly
  - No increased risk of ischemia if used for < 72 hr
Vasospasm: Prediction

- Amount of blood on the initial CT scan
  - Modified Fisher grade
- Young age
- Smoking
- Cocaine use
- Poor clinical grade
Cerebral Vasospasm: Diagnosis

- Transcranial Doppler
  - CO2 reactivity
- Angiography
  - Gold Standard for large vessel spasm
- Perfusion studies
- Multi-modality invasive brain monitoring
Cerebral Vasospasm: Medical Treatment

Hemodynamic Augmentation Therapy

Hypertension

Hypervolemia

Hemodilution
Cerebral Vasospasm: Endovascular Treatment

- **Balloon Angioplasty**
  - For large artery spasm

- **Super-selective intra-arterial infusion of vasodilators**
  - Papaverine
  - Verapamil
  - Nimodipine
New therapies: many unfulfilled promises

- Endothelin antagonists
- Magnesium sulfate
- Statins
- Prophylactic Angioplasty
- Human albumin
- Lumbar Drainage
- Nitrite infusion / Intrathecal nicardipine
Hypoperfusion and Vasospasm

• PET studies in 25 pts (652 vascular ROI); median day 7; median time from catheter angiogram 6 hr

• 24% of regions supplied by vessels with spasm (56% of pts had some spasm)

• CBF lower and OEF higher in regions supplied by vessels with spasm

• However, the majority of regions with hypoperfusion and with oligemia were not supplied by vessels with spasm

Dhar et al Stroke 2012;43:1788-94
Possible mechanisms of secondary injury in aSAH

- Vasospasm (large and small vessels)
- Intracranial hypertension
- Changes in autoregulation
- Subclinical seizures
- Cortical spreading depolarizations
- Microthrombosis
- Microembolism
- Delayed axonal degeneration
The poor-grade patient

• Coma, intubation, sedation = limited to no exam
• Multimodality monitoring
• Raised ICP
  ➢ Osmotic agents
  ➢ Hypothermia
  ➢ Decompressive craniectomy
Not all poor grade cases are the same

- Poor grade SAH at presentation
  - Early death
  - Still poor grade after resuscitation
  - Good grade after resuscitation
Early complications

- Rebleeding
- Hydrocephalus
- Intracranial hypertension
- Seizures (DDx: posturing)
- Pulmonary edema (often mixed)
- Labile BP
- Cardiac failure
In part, you decide
We love monitors
Sometimes too much
The importance of severity of systemic illness

- Various markers of systemic disease are associated with worse outcome in aSAH

<table>
<thead>
<tr>
<th>AA gradient of &gt;125 mm Hg</th>
<th>0.3544</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCO₃ of &lt;20 mmol/L</td>
<td>0.2594</td>
<td>2</td>
</tr>
<tr>
<td>Glucose of &gt;180 mg/dL</td>
<td>0.2529</td>
<td>2</td>
</tr>
<tr>
<td>MAP of &lt;70 or &gt;130 mm Hg</td>
<td>0.1334</td>
<td>1</td>
</tr>
<tr>
<td>Maximum score</td>
<td>1.0</td>
<td>8</td>
</tr>
</tbody>
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Claassen et al Crit Care Med;2004:32:832-8
Neurogenic Pulmonary Edema
TAKO-TSUBO Cardiomyopathy
(Apical Balloning Syndrome)
Hyponatremia

• Cerebral salt wasting syndrome (+/- SIADH)
• Avoid hypotonic solutions
• 1.5% saline
• Fludrocortisone
• Albumin
Fever / Infections

- SAH is a common cause of non-infectious (central fever)
- Nonetheless, always exclude infections (including ventriculitis)
- Fever is independently associated with worse outcome in SAH
  - Maintain strict normothermia
  - Very limited data on the value of hypothermia on most severe cases
Anemia and Transfusions

• Anemia is associated with worse outcomes, especially in patients with vasospasm

• Transfusions are associated with worse outcomes, especially in patients without vasospasm

• Transfusion strategies need further evaluation and individual application
Thromboprophylaxis

- SCDs from admission
- No solid studies evaluating the risk/benefit of LMWH or SQ heparin
- Value of surveillance ultrasounds?
Conclusions

• Patients with aSAH must be managed in an ICU
• Adequate early management is crucial
• Aneurysms should be assessed and treated following a team approach
• Perfusion scans are valuable in the evaluation of vasospasm
Conclusions

• Hypertension appears to be the most important H in triple H therapy
• Balloon angioplasty is recommended for refractory large vessel spasm
• We need to think of DCI rather than visible vasospasm
• Attention to systemic complications is crucial
SAH is a Neurological Disease!