Aneurysmal Subarachnoid Hemorrhage in the ICU

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FYI

• This is more of a handout that covers a majority of topics and provides references then the actual presentation that you will see.
• If you have any interest at all in this topic and only want one review to read then read the one cited below
  – Critical Care Management of Patients Following Aneurysmal Subarachnoid Hemorrhage: Recommendations from the Neurocritical Care Society’s Multidisciplinary Consensus Conference. Neurocrit Care (2011) 15:211–240
• Even with a consensus statement treatment of these patients remains variable
Making the diagnosis

• History and physical exam
  – Risk factors
  – LOC ?
• CT Head w/o contrast
  – Decrease of sensitivity with time
• LP
  – Xanthrochormia 12hrs -2 weeks
  – Lab technique (2)
• CT Angiogram of Head
  – A little better sensitivity for aneurysms <5mm
• Cerebral angiogram
  – Roughly 20% will be negative
  – Repeat cerebral angiogram to pick up the <5%
<table>
<thead>
<tr>
<th>Modifiable</th>
<th>Non-modifiable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cigarette smoking</td>
<td>Previous SAH</td>
</tr>
<tr>
<td>HTN</td>
<td>Polycystic Kidney Disease</td>
</tr>
<tr>
<td>Moderate to heavy EtOH consumption</td>
<td>Connective tissue disease</td>
</tr>
<tr>
<td>Cocaine use</td>
<td>Moya Moya</td>
</tr>
<tr>
<td>Endocarditis (formation of mycotic aneurysms)</td>
<td>AVM</td>
</tr>
<tr>
<td></td>
<td>Fibromuscular Dysplasia</td>
</tr>
<tr>
<td></td>
<td>Dissection</td>
</tr>
<tr>
<td></td>
<td>Neurofibromatosis 1</td>
</tr>
<tr>
<td></td>
<td>Pseudoxanthoma Elasticum</td>
</tr>
<tr>
<td></td>
<td>Family History (two 1st to 3rd degree</td>
</tr>
<tr>
<td></td>
<td>relatives)</td>
</tr>
</tbody>
</table>
## Hunt and Hess Grade

<table>
<thead>
<tr>
<th>Grade</th>
<th>Exam</th>
<th>Mortality (%)</th>
<th>Mortality (%) (27)</th>
<th>Mean Glasgow Outcome Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic, mild HA, some nuchal rigidity</td>
<td>1</td>
<td>3.5</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>CN Palsy, moderate to severe HA, severe nuchal rigidity</td>
<td>5</td>
<td>3.2</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>Mild focal deficit, lethargy, confusion</td>
<td>19</td>
<td>9.4</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis, early decerebrate rigidity</td>
<td>40</td>
<td>23.6</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, moribund appearance</td>
<td>77</td>
<td>70</td>
<td>2</td>
</tr>
<tr>
<td>Grade</td>
<td>GCS</td>
<td>Major Focal Deficit (Aphasia, Hemiparesis)</td>
<td>Associated Mortality %</td>
<td>Mean Glasgow Outcome Scale</td>
</tr>
<tr>
<td>-------</td>
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<tr>
<td>1</td>
<td>15</td>
<td>-</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>13-14</td>
<td>-</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>13-14</td>
<td>+</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>7-12</td>
<td>+/-</td>
<td>33</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>3-6</td>
<td>+/-</td>
<td>77</td>
<td>2</td>
</tr>
</tbody>
</table>
**Fisher Score (4, 5)**
1- No SAH
2- Diffuse Thin
3- Diffuse Thick or localized clot
4- No SAH or diffuse thin with IVH and/or ICH

**Modified Fisher Score (3)**
0- No SAH
1- Thin blood no IVH
2- Thin blood +IVH
3- Thick blood no IVH
4- Thick blood + IVH
Initial Complications

• Re-bleeding(5, 6)
  – Associated with high mortality (~50% immediately and another 30% die from subsequent complications) and morbidity
  – Risk
    • 4% in 24 hours
    • 20% in 2 weeks
    • 50% in 6 months
  – Treatment
    • Early securing of aneurysm
    • Antifibrinolytics
    • HTN control
    • Seizure prophylaxis
Initial Complications

• Hydrocephalus (1,7)
  – Can be communicating or non communicating
  – Acute development occurs in roughly 20%- 30%
  – Delayed development (after 3 days) is less common occurring <5%
  – Chronic development (>1 week) of hydrocephalus occurs in 10- 20%
  – Treatment
    • External Ventricular Drain
      – Triggers for placing and management can be variable
      – Directed by CT scan and clinical exam
      – Grade 3 and above
Initial Complications

• Cardiac complications (1, 8-14)
  – Abnormalities on EKG, Biomarkers, Wall Motion Abnormalities (WMA)
    • 50-70% will have one or a combination
    • EKG within 48 hours
      – P wave abnormalities, prolonged QT, ST, T wave abnormalities
    • Tn and CK MB typically rise in 24-48 hours
    • WMA as early as 3 hours
    • Possible link to higher mortality and morbidity
  – Arrhythmias
    • Roughly 5-10%
    • More commonly Tachy arrhythmias, brady arrhythmias, PAC, PVC
  – Treatment
    • Supportive care- pressors, inotropes, balloon pump
Delayed Neurological Deterioration (DND) (1)

• Any clinically detectable neurological deterioration in a aSAH patient following initial stabilization except new bleeding.

• Possible causes
  – Delayed Cerebral Ischemia
  – Vasospasm
  – Cerebral edema
  – Fever
  – Hyponatremia
Delayed Neurological Deterioration (DND)

• Delayed Cerebral Ischemia (DCI) (1)
  – Any neurological deterioration presumed related to ischemia that persist for an hour and cannot be explained by other physiologic abnormalities.

• Vasospasm(1)
  – Narrowing of blood vessels seen on a radiographic image or increased mean flow velocity on sonography
Delayed Neurological Deterioration (DND) (1)

• Vasospasm
  – Vasospasm seen in 70% of all aSAH patients after day 3
  – Only roughly 30-40% of these patients will continue on to DCI
  – Time Course
    • Between day 3-14
    • Peaks around day 6-8
  – Monitoring
    • Clinical exam
    • Trans Cranial Doppler
    • Other monitoring devices
      – Licox -pbO2
      – Micro dialysis
      – cEEG
Delayed Neurological Deterioration (DND) (1,15)

• A word about transcranial doppler ...
  – ~10% patient don’t have windows- meaning can’t get a waveform
  – Predictive of vasospasm not DCI
  – MCAs are the most reliable when very high >200cm/sec or low <120 cm/sec
  – Also helpful if significant increase in MFV
    • Doubling in 24- 48 hours
Delayed Neurological Deterioration (DND)

- Vasospasm treatment
  - Poiseuille Equation

\[ F \propto \frac{\Delta P \cdot r^4}{\eta \cdot L} \]

- \( F \) = CBF
- \( \Delta P \) = CPP
- \( r^4 \) = Radius
- \( \eta \) = Viscosity
- \( L \) = Length

HYPERTENSION/ HYPERVOLEMIA
HYPEROVOLEMIA/ HEMODILUTION
Delayed Neurological Deterioration (DND)

- Vasospasm treatment (Cont.)
  - From Poiseuille equation comes triple H therapy
  - The H that appears to be the most helpful is Hypertension in regards to improving cerebral blood flow
  - Increase sBP by at least 20mmHg, ceiling sBP 200-220mmHg or if the symptoms resolve
  - Judicious use of IV fluids.
    - Follow CVP, PADP, SVV, Echo/IVC
  - Intrathecal Cardene
    - shows improvement with mean flow velocity
  - Angiography
    - IA vasodilators
  - In difficult to examine patients further imaging is helpful to guide therapy.
    - CTA/P
**Delayed Neurological Deterioration (DND) (16-19)**

- The logical assumption is that DCI is dependent on vasospasm which is unfortunately not always true
  - Although nimodipine has been shown to improve outcome it has not been shown to reliably reduce vasospasm
  - Clazosentan (endothelin antagonist) and nicardipine have also been shown to reduce the relative risk vasospasm but it does not translate into decrease infarcts and better recovery
No Vasospasm  
n=497

Hypoperfusion  
n= 46

31 regions with hypoperfusion but no proximal vasospasm

Vasospasm  
n= 157

142 regions with vasospasm did not exhibit hypoperfusion

15 regions with hypoperfusion and vasospasm

Above is a Venn diagram of 25 aSAH patients who have undergone PET scan and cerebral angiogram in relatively quick succession which showed that true hypoperfusion was not routinely associated with vasospasm. (20)
Delayed Neurological Deterioration (DND) (20, 21, 24)

• DCI is more than just vasospasm
• Theories on other pathophysiologic states contributing to DCI
  – Early Brain Injury
    • Intracranial circulatory arrest / cerebral edema
  – Cortical Spreading Depression
    • Depolarization of grey matter that leads to hypoperfusion from cortical vasoconstriction
  – Microthrombus formation
  – Arteriolar constriction
Delayed Neurological Deterioration (DND)

• Fever (22,23)
  – Fever is common among the Neurologically critically ill
  – Up to 40% aSAH and typically not isolated (may persist for as long as 2 weeks)
  – Generally 50% can be attributed to infectious sources (pulmonary, uti)
  – Independently associated with death and severe disability
Primary Injury
- Axonal Damage
- Direct Cell Membrane Damage
- Metabolic Stress
- Break down of BBB
- Cortical spreading depression

Secondary Injury
- Glial swelling
- Endothelial dysfunction
- Delayed neuronal damage
- Cytokine release
- Increased inflammation
- Excitotoxicity

Outcome
Delayed Neurological Deterioration (DND)

• Sodium regulation (1, 24, 25)
  – Hyponatremia
    • 20-30% of aSAH
      – Cerebral salt wasting
        » Catecholamine release and high sympathetic state with increase release of naturetic peptides
      – SIADH
    • Differentiated by volume status
    • Associated with worse outcome
    • Polyuria has also been shown to correlate with vasospasm onset
  • Treatment
    – Goal is to maintain euvolemia therefore making fluid restriction difficult.
    – Increase Na intake (PO, IV, NG/OG), Fludrocortisone,
Delayed Neurological Deterioration (DND)

- Global Cerebral Edema
  - Initial or delayed
  - Has been shown to be a predictor of poor outcome and mortality
  - Global edema on admission
    - Loss of consciousness at ictus
    - Poor grade
  - Delayed global edema
    - Large aneurysm
    - Loss of consciousness at ictus
    - Use of vasopressors
- Treatment
  - Basic bedside manipulations → hypertonic NaCl/ Mannitol→ Sedation, TTM→ Burst suppression
Outcome (27-29)

• A recent retrospective review of a prospective collected database to determine reasons for aSAH patients death showed..
  – Withdrawal driven by primary effects of initial bleed, rebleeding and medical complications
  – Admission predictors of Mortality where age, LOC, GCS, APACHE II, mFS and large aneurysm
  – While several hospital complications increase risk of dying, DCI was not part of it
• The continued theme in outcome data of aSAH patients
  – Continued improvement 6 months out to a year. Even out to 36 months improvement can still be seen


