Thunderclap Headache and Subarachnoid Hemorrhage

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No conflicts of interest or disclosures
Subarachnoid Hemorrhage

Objectives

- **Recognize** the symptoms (thunderclap headache) and signs of SAH
- **Initiate workup** for suspected SAH
- **Initial management** of SAH patient
Case

- 39 year old woman presents with severe headache, uncertain time of onset, associated with nausea
- PMH: long history of migraine headaches
- Meds:
  - Reports taking ibuprofen prior to ED, with moderate improvement of headache
  - Taking warfarin for a pelvic DVT - year prior during pregnancy
- Exam: sleepy, yet follows commands, left eye droop and dilated pupil, moving all extremities
- BP 160/95 mmHg, RR 24/min, HR 98/min
- Breathing shallow and rapid, airway protection uncertain
<table>
<thead>
<tr>
<th>Task</th>
<th>Completed</th>
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<tbody>
<tr>
<td>Brain Imaging</td>
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<tr>
<td>Labs: PT/PTT, CBC, electrolytes, BUN, Cr, troponin, toxicology screen</td>
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<tr>
<td>12 lead ECG</td>
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<tr>
<td>Blood pressure goal established</td>
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<tr>
<td>Consult neurosurgery</td>
<td></td>
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<tr>
<td>Address hydrocephalus</td>
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</table>
SAH Algorithm

**SAH Symptoms and Signs**

<table>
<thead>
<tr>
<th>CLASSIC</th>
<th>NOT-SO-CLASSIC</th>
</tr>
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<tbody>
<tr>
<td>Abrupt onset of severe headache (HA), i.e. thunderclap</td>
<td>HA is not reported as abrupt (patient may not remember event well)</td>
</tr>
<tr>
<td>“Worst or first” headache of one’s life that is <em>instantaneously maximal</em> at onset (“thunderclap” after lightening strike)</td>
<td>HA responds well to non-narcotic analgesics</td>
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<tr>
<td>May have nausea, vomiting and neck pain</td>
<td>HA resolves on its own in few hours</td>
</tr>
<tr>
<td>May transiently lose consciousness, present in coma, or have focal deficits</td>
<td>40% patients with aneurysmal SAH will have normal neuro exam <em>without</em> meningismus</td>
</tr>
<tr>
<td>Nature of HA onset distinguishes from other forms of stroke</td>
<td>Do not necessarily appear acutely ill</td>
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</tbody>
</table>
SAH: Signs (Exam)

Key Exam Features:

- GCS
- Pupil exam
- Fundoscopic exam for vitreous/retinal hemorrhages (Terson’s syndrome)
- Neck exam for meningismus (versus neck pain)
- Hunt and Hess or WFNS score
## SAH: Clinical Severity Scales

<table>
<thead>
<tr>
<th>World Federation Neurological Scale</th>
<th>Hunt &amp; Hess Clinical Grading Scale</th>
</tr>
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<tbody>
<tr>
<td><strong>Grade</strong></td>
<td><strong>Criteria</strong></td>
</tr>
<tr>
<td>1</td>
<td>GCS 15</td>
</tr>
<tr>
<td>2</td>
<td>GCS 13-14, without neurological deficit</td>
</tr>
<tr>
<td>3</td>
<td>GCS 13-14, with neurological deficit</td>
</tr>
<tr>
<td>4</td>
<td>GCS 7-12</td>
</tr>
<tr>
<td>5</td>
<td>GCS 3-6</td>
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</table>
SAH-What’s Next?
SAH Brain Imaging

- Non-contrast CT imaging of the brain is the gold-standard for identifying SAH with sensitivity of 95-100% if:
  - Classic presentation with thunderclap HA
  - CT completed within six hours of HA onset
  - The patient is completely neurologically intact
  - The CT is read by an attending radiologist
- Sensitivity of CT decreases with time
- Falsely negative CT: time, anemia (HCT <30), low volume SAH, and a technically poor scan
SAH: CTA Brain Imaging

- Some physicians advocate a CTA at the time of the CT scan to look for an intracranial aneurysm.
- Caution regarding renal function and excessive contrast administration.
- Although CTA is helpful if an aneurysm is seen, the negative predictive value is less clear.
- One should not use a negative CTA alone to rule out aneurysmal SAH.
• MRI can be useful in patients who are imaged a few days or week or longer following the SAH

• Specific sequences (GRE, SWI, FLAIR) can be used to image subarachnoid blood even several days later
ENLS: SAH
Lumbar Puncture

- Must perform LP if CT is negative and history suggests SAH
- Rationale for LP is to confirm xanthochromia-staining of CSF by heme breakdown products
- Presence of xanthochromia is time dependent- takes several hours to develop
### Typical LP Findings

<table>
<thead>
<tr>
<th>Typical LP Findings</th>
<th>Atypical or Inconclusive</th>
<th>Not suggestive of SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ RBCs, No clearing from tube 1→ 4</td>
<td>Clearing of RBCs from tube 1→ 4</td>
<td>CSF clear of RBCs</td>
</tr>
<tr>
<td>&lt; 5 WBC, WBC:RBC ratio 1:700</td>
<td>↑ WBC:RBC ratio suggest another process, meningitis or encephalitis</td>
<td>Occasionally, rapidly expanding unruptured aneurysm may present with HA, recommend urgent consultation</td>
</tr>
</tbody>
</table>

### Xanthochromia

- Present (However if CSF Protein >100mg/dL may be false positive)
- Absent (Assuming LP is done more than 12 hours following headache onset).

### Opening Pressure

- Elevated (~2/3 patients)
- Normal

### Other

- CSF clear of RBCs
- WBC:RBC ratio suggest another process, meningitis or encephalitis
- Occasionally, rapidly expanding unruptured aneurysm may present with HA, recommend urgent consultation
- CSF clear of RBCs
Diagnosis of SAH confirmed. The goal is to reduce the chance of aneurysm re-rupture and expedite treatment of the aneurysm while preventing or minimizing medical or neurologic complications.
Once SAH is diagnosed, take these first steps:
• Bed rest
• Obtain pre-intervention labs: CBC, Platelets, PT/PTT, INR, electrolytes, BUN, Cr, cardiac enzymes
• 12-lead ECG
• Cardiac telemetry
• Nimodipine 60 mg po/ng (watch for hypotension)
• AED until aneurysm secured
• Consult Neurosurgery
**SAH**

**Seizure Prophylaxis & Management**

**Prophylactic Anticonvulsants - Controversial**
- **Pro**: seizures following SAH and prior to definitive treatment can be associated with re-rupture and can raise ICP
- **Con**: phenytoin use has been associated with worse cognitive outcomes in SAH
- One strategy is to administer a loading dose of phenytoin in the ED, and continue it until the aneurysm is secured, then stop the medication unless seizures have occurred
- Although frequently used by many centers there is limited data on use of alternative anticonvulsant agents such as levetiracetam

**Active Treatment of Seizures**
- Administer lorazepam for acute seizure management
- Administer loading dose of phenytoin
- More recently, many centers use levetiracetam
SAH
Coagulopathy

Correct underlying coagulopathies

- Goal INR < 1.4
- Goal platelets > 50,000
- Consider platelet transfusion for those on anti-platelets
- See ENLS pharmacology manuscript
SAH
Treat Pain and Anxiety

- It is important to avoid straining, Valsalva, and writhing, as they can potentially contribute to re-rupture of a tenuous aneurysm
- One must also be careful to not over-sedate the patient as one could mask the symptoms of hydrocephalus
- Use IV medication with short half-lives (fentanyl for example)
- Liberal use of anti-emetics is justified especially if vomiting occurs
- BP control is enhanced with adequate analgesia
- If anxiety seems to be the major issue, consider small doses of an anxiolytic such as lorazepam
SAH
BP Management

- Precise guidelines for BP management in SAH unfortunately do not exist
- Retrospective data suggest higher rates of re-bleeding with SBP > 160 mmHg
- Over treatment of BP can potentially lead to brain ischemia - especially if hydrocephalus or vasospasm is present.
- Pre-morbid BP should be taken into considerations
- **Experts recommend to aim for SBP < 160 mmHg, or MAP < 110 mmHg**, keeping principles above in mind
- Use short acting, titratable intravenous medications such as beta blockers or nicardipine.
- Avoid long-term nitroprusside due to concern of raising ICP
Back to our 39 y/o woman with a SAH

- Patients level of arousal abruptly declined requiring immediate intubation
- Blood pressure spiked to 220/115 mmHg
- Both pupils transiently dilated
- Repeat Head CT demonstrated a re-bleed associated with acute hydrocephalus
- Upon return from CT, patient had a generalized tonic-clonic seizure, requiring treatment with intravenous lorazepam
SAH
Decline in Neurological Status

Causes of Decline in Neurological Status
- Acute Re-rupture of the aneurysm
- Acute Hydrocephalus
- Seizure
- Cardiopulmonary complications
  - Neurogenic pulmonary edema
  - Neurogenic stress cardiomyopathy
  - Note cardiovascular collapse may be a sign of cerebral herniation

Re-rupture estimate 12-15% in the initial 24 hours
SAH Hydrocephalus

- Hydrocephalus is caused by blockage of CSF circulation and absorption within the ventricular system +/- increased CSF production and is readily diagnosed by the head CT scan
- If a patient is obtunded or comatose, a ventricular drain (EVD) can be placed. This treats the hydrocephalus and provides a monitor of ICP
- If a neurosurgeon is not available:
  - Transfer to facility with NS capabilities
  - Consider mannitol 1 gm/kg or a bolus of hypertonic saline in interim
SAH
Anti-fibrinolytic Agents

- Preventing re-rupture of the aneurysm is a goal of initial SAH management
- Antifibrinolytic agents such as ε-aminocaproic acid and tranexamic acid can reduce aneurysmal re-rupture
- These agents also raise the risk of thrombosis such as DVT, PE, and ischemic stroke, if they are continued beyond the acute SAH period
- If there is an unavoidable delay in obliteration of the aneurysm and the patient is free of recent MI, DVT/PE, or any known hypercoagulable state, many centers administer a time-limited course (<72 hours) of antifibrinolytic agents until the aneurysm can be secured
- Early and LIMITED use (<72 hours) of these agents appear to be safe
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<th>Checklist for SAH</th>
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<tbody>
<tr>
<td>☐ Airway status</td>
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<tr>
<td>☐ Hemodynamic Status and Blood Pressure Control</td>
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<tr>
<td>☐ Clinical presentation (level of consciousness, motor exam, pupil exam)</td>
</tr>
<tr>
<td>☐ WFNS and Hunt-Hess Grade</td>
</tr>
<tr>
<td>☐ Imaging/LP results</td>
</tr>
<tr>
<td>☐ Coagulopathy present?</td>
</tr>
<tr>
<td>☐ Hydrocephalus present?</td>
</tr>
<tr>
<td>☐ Medications given (dose and time administered), including sedatives, analgesics, seizure prophylaxis, anti-hypertensives, and nimodipine</td>
</tr>
<tr>
<td>☐ Coordination of other vascular imaging</td>
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Questions?