Aneurysmal Subarachnoid Hemorrhage – Presentation and Complications

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A Familiar Story...

- 46 woman, healthy except for high blood pressure and tobacco use.

- Suddenly collapses with a severe headache and shaking movements.

- She is intubated in the field by EMS and brought to the ED.

- Exam: obtunded, brain stem reflexes intact, grimaces only to deep sternal rub, minimal withdraw to noxious stimuli in limbs.
Another Familiar Story…

- > 30,000 cases/year in the US alone. 25% mortality.
- 80% SAH occur in people ages 40-65.

*Over 50% of these patient survive in long term severe disability.*
What do we do now?
Overview of Talk

- Basics of Aneurysmal SAH
- Hyper-acute Management
- Delayed Vasospasm & Ischemic Injuries
- Systemic Complications
Typical Presentations

- “Worse Headache of Life”: 80%
- ~20% describe sentinel headache
- Often associated with nausea/vomiting, stiff neck, loss of consciousness, or focal neurological deficits.
- Seizures: 20%
- Can induce cardiac arrhythmia and patients can present in cardiac arrest.
- Mis-diagnosis rate ~12% from recent data.
- Mis-diagnosis is associated with 4-fold increase risk for death or severe disability at 1 year.
SAH Diagnosis

- Non-contrast head CT: 98-100% sensitivity in the first 12 hrs after SAH, decreasing to 93% at 24 hrs and 57-85% by 6 days post SAH.

- Diagnostic lumbar puncture should be performed in patients with negative CT and high clinical suspicion of SAH.

- CSF profile also changes at different time points after SAH: from abundant RBC to xanthrochromia, to bilirubin in CSF.

- Emerging MRI technology (GRE, susceptibility, FLAIR) improves sensitivity for SAH, especially delayed presentation.
Aneurysm Basics

Saccular Aneurysm

Fusiform Aneurysm

Coil Procedure for Cerebral Aneurysm

Ruptured Aneurysm

Surgical Clip

Artery
Typical Locations
Typical Locations
## Basics of Aneurysmal SAH: Clinical Grades

<table>
<thead>
<tr>
<th>Grades</th>
<th>Hunt and Hess (H&amp;H)</th>
<th>World Federation of Neurological Surgeons Score (WFNS)</th>
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<tbody>
<tr>
<td>Grade 1</td>
<td>Asymptomatic or mild headache.</td>
<td>GCS of 15, motor deficit absent.</td>
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<tr>
<td>Grade 2</td>
<td>Moderate-to-severe headache, nuchal rigidity, and no neurological deficit other than cranial nerve palsy.</td>
<td>GCS of 13-14, motor deficit absent.</td>
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<tr>
<td>Grade 3</td>
<td>Mild alteration in mental status (confusion, lethargy), mild focal neurological deficit.</td>
<td>GCS 13-14, motor deficit present.</td>
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<tr>
<td>Grade 4</td>
<td>Stupor and/or hemiparesis</td>
<td>GCS of 7-12, motor deficit absent or present.</td>
</tr>
<tr>
<td>Grade 5</td>
<td>Comatose and/or decerebrate rigidity</td>
<td>GCS of 3-6, motor deficit absent or present.</td>
</tr>
</tbody>
</table>
Basics of Aneurysmal SAH: Radiologic Groups

Fisher 1
No blood.

Fisher 2
Diffuse SAH, thickness < 1mm.

Fisher 3
Localized clots, thickness >= 1mm.

Fisher 4
SAH with intraparenchymal or intraventricular
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Hyper-acute Management:
How Does SAH Kill?

- cardiac arrhythmia
- sudden ICP elevation
- Hydrocephalus
- Aneurysm re-rupture
- “Voodoo heart”
- Neurogenic pulmonary edema
- Delayed vasospasm and stroke
Hyper-acute Management 1:
Treat Acute Hydrocephalus

- Hydrocephalus is a clinical diagnosis!
- Acute hydrocephalus is high on the differential for **ALL** SAH patients with impaired mental status.
- Casting of 4th ventricle and/or cerebral aqueduct is high risk radiographic finding.
- Emergent external ventricular drain (EVD) placement SAVES LIVES.
- Many recommend keeping EVD clamped and only open it prn ICP > 20, before you the aneurysm is fixed.
EVD

Temporal-horn dilatation

Casting of cerebral aqueduct
Hyper-acute Management 2: Prevent Aneurysm Re-rupture

- Re-rupture is associated with 70% case-fatality rate.
- Re-rupture risk is maximal on day 1 post rupture (4%). Each additional day confers 1-2% bleeding risk in first 4 weeks post aneurysm rupture.
- Prevention methods (controversial) include: Strict BP control (SBP less than 140), bed rest, avoid over-draining CSF, and...
Hyper-acute Management 3:

FIX THE ANEURYSM ASAP

Pre-op aneurysm re-rupture rate:

- 0-3 days: 5.7%
- 4-6 days: 9.4%
- 7-10 days: 12.7%
- 11-14 days: 13.9%
- 15-32 days: 21.5%

Kassell NF et al, J Neurosurg 1990; 73:18-36
Hyper-acute Management 3: International Subarachnoid Aneurysm Trial

Clip vs. coil in anterior circulation aneurysms

Cumulative mortality

Cumulative re-bleeding risk

Molyneux A et al, Lancet 2005; 366:809-17
Overview of Talk

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Unique Time-line in SAH and Vasospasm

- **DAY 0** => aneurysm rupture
- **DAY 0-1** => fix aneurysm
- **DAY 0-4** => patient improves
- **DAY 5-6** => some patients develop neurological dysfunction
- **DAY 7-14** => visible vasospasm (VSP) fever, cerebral salt wasting.
- **DAY 14-21** => VSP resolution
Angiographic Vasospasm

Post SAH Day 0

Post SAH Day #7
Delayed Vasospasm: Impact

- 50% surviving SAH patients have angiographic VSP, and 32% have clinical, symptomatic VSP.
- Of those with symptomatic VSP, 15-20% suffer further stroke, severe disability, or die from VSP.
- 50% of SAH/VSP survivors suffer significant morbidity.

*Development of clinically symptomatic VSP requires urgent treatment.*

*In this disease, excellent neurocritical care support changes outcome*
Delayed Vasospasm:
ICU Management

- Hypovolemia and hypotension are associated with poor outcome in SAH and should be avoided.
- Hemodynamic augmentation and induce hypertension may reverse neurologic deterioration in patients with symptomatic vasospasm.
- There is no evidence that prophylactic use of hemodynamic augmentation improves outcome.
Intra-arterial Therapy

IA Vasodilator Injection:
- Treatment of choice in acute symptomatic VSP
- Ca channel antagonist: nicardipine, verapamil
- Phosphodiesterase III inhibitor: milrinone
- Often causes transient ICP elevation and hypotension
- May need repeat treatments for persistent vasospasm.

Balloon angioplasty
- Durable effects compared with IA injection
- Only available for proximal segment VSP
- Rare but serious complication of artery rupture
IA Vasodilator Injection

Pre

Post
Delayed Vasospasm:
Ischemic Infarcts

Post SAH day 5
Post SAH day 8
Post SAH day 5
Post SAH day 8
Overview of Talk

- Basics of Aneurysmal SAH
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- **Systemic Complications**
Neurogenic Stunned Myocardium

- 50-100% SAH have ECG changes (QTc prolongation, repolarization abnormalities) – not associated with poor outcome.
- 20-40% SAH have cardiac troponin elevation. Troponin release is associated with poor clinical grade, cerebral edema, intraventricular blood.
- Approximately 20% of SAH patients develop LV systolic dysfunction, cardiogenic shock, & cardiogenic pulmonary edema.
- Cardiac dysfunction is usually reversible.

Naidech A et al, Circulation 2005;112:2851-2856
“Broken Heart” Syndrome?

- NSM, Takotsubo cardiomyopathy, & “voodoo heart”: There is confusion of terms in literature and practice.
- Pathology: subendocardial contraction band necrosis.
- Thought to result from excessive release of nor-epinephrine.
- Typically not associated with coronary artery disease.
- Apical ballooning is often not seen in SAH patients with LV dysfunction and cardiogenic shock.

Bybee KA et al, Ann Intern Med 2004; 141:858-865
Cerebral Salt Wasting vs. SIADH

- Hyponatremia occurs in up to 30% patients.
- Difficult to distinguish between CSW and SIADH because we must maintain strict euvolemia at all times.
- Hypouricemia and increased FeUA more consistent with CSW.
- CSW in SAH is associated with elevated BNP.
- Fludrocortisone shown to be effective for hypoNa in SAH.

In Summary

- SAH is a serious but treatable condition if diagnosed correctly and treated timely.
- SAH presents with a constellation of severe neurologic and systemic symptoms.
- SAH treatment requires correct initial diagnosis, prompt alleviation of hydrocephalus, aneurysm obliteration, and critical care support through delayed vasospasm.
- Successful SAH treatment requires a collaborative team including emergency medicine, neurosurgery, interventional neuroradiology, and neurocritical care specialists.
Thank you
SAH Mimics

- Thunder-clap headaches in migrainers
- Sudden cardiac arrest
- Cocaine use
- Call-Fleming Syndrome
- Vasculitis of cerebral vessels
- Eclampsia/Post-partum angiopathy
Delayed Vasospasm:
Diagnostic Tools

1. Transcranial Doppler Ultrasound Monitoring
   - Useful as a surveillance tool (daily monitoring with pre-VSP baseline velocities known).
   - Sensitivity and specificity best for MCA spasm, and is operator dependent.
   - May only detect large vessel spasm
   - Lindergaard ratio: distinguish between VSP and hyperremia.
Transcranial Doppler Ultrasound

Pro: non-invasive – you can do this everyday; no contrast load.
Con: neither sufficiently sensitive nor specific – requires confirmatory studies.
Delayed Vasospasm:
Diagnostic Tools

2. **CT Angiography +/- CT Perfusion:**
   - **Pro:** non-invasive, anatomic study that can visualize mid size cerebral vessels.
   - **Con:** not yet validated (though new data are promising); contrast load.

3. **Conventional 4-vessel Cerebral Angiography:**
   - **Pro:** Gold-standard diagnostic tool; allows direct intervention if VSP is seen.
   - **Con:** Invasive; complications include stroke, vessel dissection, hematoma.