Subarachnoid Hemorrhage (SAH): Definition and Images

- Definition: extravasation of blood into the subarachnoid space between the pial and arachnoid membranes

SAH: Etiologies

1. Trauma – most common cause of SAH
2. Spontaneous
   - ruptured intracranial aneurysms cause 75-80% spontaneous SAHs
   - cerebral AVMs 4-5%
   - cerebral artery dissections (may be post-traumatic also)
   - coagulation disorders
   - spinal AVM
   - pituitary apoplexy
   - Sickle cell disease and other vascular pathologies
   - mycotic intracranial aneurysms

SAH: Miscellaneous Facts and Outcome Statistics

1. Peak age for aneurysmal SAH 55-60y/o with 20% between 15-45y/o
2. 30% occur during sleep
3. 50% have warning symptoms 6-20days before rupture
4. HA lateralizes to side of aneurysm 30% of cases
5. SAH complicated by IPH 20-40% cases and IVH 13-28% cases
6. 10-15% die before reaching medical care
7. 10% mortality in first few days
8. Overall mortality 32-67%
9. Among those surviving initial bleed without surgery, rebleeding is cause of major morbidity and mortality 15-20% risk in first 2 weeks
10. Vasospasm kills 7% and severe deficit in another 7%
11. Approx. 66% with successful clippings never return to their baseline quality of life

SAH: “The nitty-gritty”

1) Common causes: trauma vs atraumatic (>70% due to aneurysm or AVM)
2) Hunt Hess and Fischer Grading Scale for presentation
3) Labs: LP if HCT (-), watch for SIADH or cerebral salt wasting, monitor lysites and serum osmol q 6hrs, monitor ABGs if on vent, prevent hyperglycemia
4) Imaging: Cerebral Angiogram is gold standard after diagnosis with HCT or clinical presentation with xanthrochromia on LP, followed by daily TCDs
5) Treatment: Manage EVD, control ICPs, BP management not allowing hypotension, vasospasm prophylaxis with HHH therapy/ nimotop/ Mg gtt, art line and central line, maintain CPP >65, maximum CPP >90 or 70 (ask surgeon)
6) Surgeries: Angio/ coiling, possible clipping if unable to be coiled, EVD
7) Prognosis: variable depending on size of aneurysm and successful clipping, and postoperative care preventing vasospasm and stroke

You need to stay awake to remain in the circle of trust…
### Risk Factors for SAH

- Hypertension
- Oral contraceptives
- Substance abuse – EtOH, smoking, cocaine
- Pregnancy
- Increased risk of aneurysm rupture with arteriogram or LP
- Age over 40 and Advancing age
- Congenital abnormalities
- CNS infections
- Head Trauma
- Family History
- Gender Women 3:2

### Clinical presentation of SAH

1. HA – present in 97% of cases
2. Warning HA – 30-60% of cases; may be due to aneurysmal enlargement or hemorrhage within the aneurysm wall and clears within 1 day
3. Altered LOC or Coma – possible 2nd to elevated ICP, hydrocephalus, seizure, and ischemia due to decreased CBF
4. Meningismus – nuchal rigidity, Kernig’s or Brudzinski’s signs
5. HTN
6. Acute FNDs (focal neuro deficits)
7. Seizure
8. Ocular hemorrhage

### Aneurysmal SAH Images

#### Saccular aneurysm locations:

1. 85-95% in carotid system
   - AComm (34%) – CN II palsy affecting visual fields, leg weakness
   - ACA (5%) – leg weakness, incontinence, confusion
   - MCA (20%) – aphasia, hemiparesis, hemisensory loss, visual field deficit (CN II palsy), CN VII palsy, visual neglect
   - PComm (23%) – hemiparesis, CN III palsy, peri-orbital HA
   - ICA (4%) – CN II, III, IV, VI palsies, retro-orbital HA
2. 5-15% posterior circulation
   - Basilar (10%) – CN II, III palsies (vertical gaze), paresis, coma
   - Vertebral-PICA (~5%) – ataxia, vertigo

### Traumatic SAH Images

#### SAH with Rebleed and progression of Hydrocephalus

CT scan (top row) showed diffuse SAH with mild hydro HH grade 2. Pt rebled 12 hours after admission and deteriorate-> comatose decerebrate posturing with:

- Correlating CT scan (bottom row) showing rebleeding in anterior interhemispheric fissure and the lateral ventricles, global edema and effacement of the hemispheric sulci, and disruption of the hemispheric gray-white junction with diffuse peripheral fingerlike lucencies extending from the deep white matter.
Hey you…stop chopping logs

Evaluation and Diagnosis of SAH

1) HCT without contrast
2) If HCT (-) then proceed with LP – xanthochromia is positive (however false positives may occur)
3) MRI – not as sensitive as CT in first 24-48hrs
4) Cerebral angiogram – “gold standard”
5) Additional testing – MRA and CTA

Differentiating SAH from traumatic tap (TT)

1) RBC count – declines comparing 1st to last tube in TT
2) Ratio WBC:RBC – similar to ratio in peripheral blood on TT whereas SAH promotes leukocytosis
3) Clotting – SAH usually does not clot
4) Protein concentration - higher in SAH 1mg/1000 RBC
5) Repeat LP at higher level – remains bloody in SAH
6) Opening pressure – usually elevated in SAH

Hey you…on your cell phones

CT scan evaluation of SAH

1) Assess ventricular size: Hydrocephalus occurs acutely in 21% of aneurysmal SAH
2) Identify any additional hematomas – determine if needs evacuation and directs clinical management
3) Identify any Infarcts, or signs of ischemia
4) Assess amount of blood in cisterns and fissures: prognosticator for vasospasm (correlate with Fischer scale)
5) May identify aneurysm location in 70% cases and specify which bled if multiple aneurysms are present
   (a) Blood predominantly in the ventricles, especially the 3rd and 4th suggests PICA aneurysm or VA dissection
   (b) Blood in the anterior interhemispheric fissure suggests Acmm aneurysm
   (c) Blood in sylvian fissure c/w Pcomm or MCA aneurysm

Hey you…on your cell phones

“1 hear the day that technology will surpass our human interaction. The world will have a generation of idiots.” Albert Einstein
Grading SAH

Hunt and Hess grading
Grade 1 - Asymptomatic or mild headache
Grade 2 - Moderate-to-severe headache, nuchal rigidity, and no deficits other than possible cranial nerve palsy
Grade 3 - Mild altered MS (confusion, lethargy), mild FNDs
Grade 4 - Stupor or hemiparesis
Grade 5 - Comatose and/or decerebrate rigidity

Fischer scale (CT scan appearance)
Group 1 - No blood detected
Group 2 - Diffuse deposition of SAH, no clots, no blood ≥ 1mm thick
Group 3 - Localized clots and/or vertical layers of blood ≥1mm thick
Group 4 - Diffuse or no SAH, but ICH or IVH present

Initial Management of SAH

1) Prevent rebleeding – BP control and correction of coagulopathies
2) Hydrocephalus – EVD placement and mgmt
3) Vasospasm and ischemia prevention/treatment
4) Correction of electrolyte abnormalities such as hyponatremia which may be secondary to cerebral salt wasting or SIADH seen with SAH – this is also key in HHH therapy
5) Obtaining an arterial line – BP and ABG monitoring purposes
6) Obtaining a central venous line – monitoring CVP
7) Seizure prophylaxis
8) Coiling or clipping of aneurysm as soon as possible

Timing of Surgical Intervention

This topic has been controversial over the past 3 decades

Early surgery (0-3 d) has the following advantages:
• Prevention of rebleed, which is associated with a high mortality rate
• Possible vasospasm prophylaxis by removal of subarachnoid clot
• Prevention and treatment of ischemic complications
• Prevention of medical complications
• Decreased duration of hospitalization

Disadvantages of early surgery for SAH include the following:
• Technical problems associated with edematous brain tissue
• High risk of intraoperative rupture of fragile aneurysm
• Higher surgical morbidity and mortality rates

Delayed surgery for SAH (>10d post-bleed) has the following advantages:
• Brain tissue is less edematous.
• Lower risk of intraoperative aneurysm rupture
• Lower surgical morbidity and mortality rates
• Flexibility of scheduling

The disadvantages of delayed surgery are as follows:
• Increased rate of morbidity and mortality due to rebleeding
• Technical difficulties due to adhesions around the aneurysm

Why Serial Lytes and ABGs?

1) Hyponatremia – occurs in 10-40% of SAH and may compromise HHH therapy in aneurysmal SAH and lead to cerebral edema=–elevated ICP, decreased CBF and increase risk of vasospasm
2) Hypercapnia/ Hypoxia – may lead to vasodilation, increased cerebral edema, and elevated ICPs thus decreased CBF

Note: This goes for both traumatic (if diffuse) and aneurysmal SAH

Blood pressure and Volume Management

Mild elevation of BP with volume expansion using IVF reduces the risk of vasospasm. However, too hypertensive may lead to re-bleeding as well. The plan of care depends upon stability of the coiling/ clipping securing the aneurysm, and the stability of the hemorrhage(s).

HHH therapy is preventative of vasospasm and entails using pressors, albumin, and hypertonic saline at times indicated in addition to diluting with high rates of IVF to manage BP elevation and volume expansion.
Characteristics of Cerebral Vasospasm

1. Altered LOC
2. Delayed onset new focal neuro deficits (FNDs) including CN palsies and focal motor deficits (FMDs)
3. Onset 4-20 days post-SAH
4. Deficit correlates with involved arteries

Cluster syndromes:
ACA syndrome: drowsiness, delayed response, confusion, whispering, urinary incontinence, lower limb weakness and/or sensory loss
MCA syndrome: hemiparesis, monoparesis, aphasia, sensory apraxia, inability to use objects, neglect

Note: Higher Fisher grading correlates with increased incidence of vasospasm

Ancillary tests for Vasospasm

1) Transcranial Doppler (TCDs)
2) CTA
3) MRA
4) SPECT
5) Cerebral angiography – gold standard and method of treatment

Things to rule out when suspecting vasospasm

1) Rule out rebleeding
2) Rule out hydrocephalus if not already treated
3) Rule out cerebral edema
4) Rule out Metabolic disturbances: hyponatremia
5) Rule out hypoxia
6) Rule out infectious process

Vasospasm Preventative Management

1) HHH therapy – hypervolemia, HTN, hemodilution
   • IVF usually 150ml/hr or higher
   • normotension or 30% above baseline even without spasm
   • Target Hct > 33%
   • albumin if CVP <10
   • Hypertonic saline – shown to increase CBF
   • Strict I/Os
   • Pressors for subclinical and/or clinical vasospasm on TCDs
2) Strict ICU monitoring
   • neuro checks, daily CBC, serial lytes and ABGs
   • daily TCDs
   • A-line and central line
   • Monitor/ Manage EVD output as well as ICP/CPP
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3) Smooth Muscle relaxants – nimotop, cardene, Mg gtt, zocor
4) Imaging - HCTs or MRI as deemed necessary by NS and clinical presentation

Cerebral Vasospasm Treatment Regimens

NOTE: THE KEY IS CBF (factors at play here include MAP/CPP/ICP)

For Subclinical Vasospasm (Elevated TCDs):
1) Increase IVF
2) Give albumin boluses for CVP <10
3) Start IV pressors and titrate

For Clinical Vasospasm:
1) Treat as noted above
2) If refractory to ICU mg => Cerebral angiogram for injection of verapamil (lasts 48-72hrs)

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M&M Facts

• 80% of SAH patients die in the first 30 days
• About 10% die immediately without any warning; an additional 25% die or become disabled as a result of the initial hemorrhage.
• Hospitalized SAH patients have an average mortality rate of 40% in the first month.
• Rebleeding, a major complication => mortality rate 51-80%.
• Delayed cerebral ischemia due to vasospasm, the most deadly of all complications, affects 20% of angiographically visualized cases of vasospasm.
Thank you