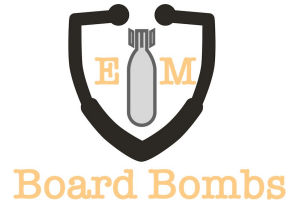


Authors: Blake Briggs, MD, Mary Claire O'Brien, MD



Introduction

Subarachnoid hemorrhage (SAH) is a terrifying, relatively frequent cause of intracranial bleeding and has a significant degree of morbidity and mortality. SAH makes up ~10% of all strokes. SAH can be aneurysmal (more common) or non-aneurysmal. Average age of aneurysmal rupture causing SAH is ~50 years old.

Aneurysm: protrusion from an artery that is prone to rupture

-**Saccular** (berry): thin-walled tunica media that protrudes from an artery. Defected elastic lamina.

-**Fusiform:** circumferential dilatation of the entire section of a particular artery.

Non-aneurysmal causes: trauma, AVM, vasculitis, amyloid, bleeding pathology, sympathomimetic abuse.

Biggest risks for aneurysm formation and SAH: smoking > HTN > family history (specific traits, Ehlers-Danlos, PCKD)

As disturbing as they are to patients, most cerebral aneurysms do not rupture. Their lifetime prevalence is ~5% in the US alone. About 85% of them are located in the anterior circulation, mainly on the Circle of Willis. 20-30% have multiple aneurysms.

Sometimes unruptured aneurysms can cause headaches. These headaches often mimic SAH.

There is no major consensus on which aneurysms need to be managed surgically. It is agreed that aneurysms >7 mm grow faster and have a higher rate of rupture (e.g. 5-year rupture rates for 7-12 mm risk was 2.5%; 13-24 mm, 14.5%).

We prefer to stay out of this debate (for obvious reasons). However, here's a summary where there is decent support for intervention: symptomatic unruptured aneurysms of all sizes, asymptomatic aneurysms >7-10 mm, remaining aneurysms of all sizes in those with SAH.

Triggers of rupture: not always identifiable. Some can occur during sleep. Often, some form of exertional event tied with many other confounding variables including consuming caffeine, sexual intercourse, competitive athletics. Emotional events alone have not been shown to be solo triggers.

Rupture of vessel → blood enters CSF rapidly → rapid increase in ICP with symptoms of intense headache

Presentation

Sudden, severe headache = 97% of cases.

Unilateral headache = 30% of patients.

Nausea and vomiting = 77% of patients.

Loss of consciousness = about 50% of patients.

Seizures = ~10% of patients. Arguably the most concerning symptom if present early on.

Sudden death = ~10-15% of patients. These rarely reach the hospital.

Clinical Pearl

Clinicians often struggle in asking patients to appropriately describe this headache. Instead of asking "is this the worst headache of your life?" to which many a patient might say yes, inquire the following, "do you typically get headaches? If so, is this headache like others you've had before? What is different about this one?"

EKG can show ischemic changes such as ST depression, QT prolongation, and deep T wave inversions. Of course, these are not very specific or sensitive for SAH and can be seen in countless systemic ischemic conditions.

Diagnosis

First test is always *CT head without contrast*.

Blood is found in the subarachnoid space 92% of the time if scan is <24 hours. If the CT head scan is done in <6 hours, the sensitivity is virtually 100%. Therefore, if a **patient's symptoms truly began <6 hours prior and the CT scan is negative, SAH workup is complete and no further diagnostic workup is warranted.**

The critical caveats we must mention: 1) the CT scan is reviewed by expert radiologists, 2) the CT scanner is a 7th generation (latest) model.

Lumbar puncture (LP): should be performed if there is negative head CT and patient presentation is >6 hours with a concerning story. Sensitivity is 100% and NPV 100% with a specificity of 65%. Classic findings from the LP:

-elevated opening pressure. Not always reliably present.

-RBC count >2000 in CSF.

-elevated RBC count that does not decrease from tubes 1→4. *If the RBC does diminish, this still does not mean it was a "traumatic tap"!* Always have a high index of suspicion!

-Xanthochromia (yellow tint from Hgb breakdown). This is the most specific finding and in the setting of a severe headache is virtually diagnostic of SAH. If unsure, compare the vial of CSF to a vial of tap water against a white background. Xanthochromia is rarely found <2 hours after symptom onset.

The absence of RBCs in the final LP tube and absence of xanthochromia >2 hours after symptom onset rules out SAH with a sensitivity of 100%.

Xanthochromia: aside from SAH, it can be seen in hyperbilirubinemia >10 mg/dL, and severely traumatic taps where there is typically >100k RBCs (oh my word!).

CTA and MRA can both equally detect aneurysms ~3-5mm or larger. New, multidetector CTA (64-slice) has improved to >97% sensitive/specific for SAH. In many institutions CTA has been seen as a reasonable alternative to LP (the main drawbacks of CTA are cost and radiation). LP is thousands of dollars cheaper however can be painful, time-consuming, and not without risk. Post-LP headaches are a rare but substantial source of patient suffering.

While there might be some debate between CTA and LP, this can be avoided in some populations with the application of the Ottawa Subarachnoid Hemorrhage Rule. The rule is validated and has been found to be 100% sensitive in ruling out SAH in patients aged 14-39 years old within 14 days of symptom onset for new onset severe atraumatic headache. As you can imagine, the criteria are very strict. So, what are the cutoffs in this rule? Well, we don't want you to memorize it because you do not need to, and you can always look it up online while on shift, but we need to mention it at least once.

Age \geq 40, neck pain or stiffness, witnessed loss of consciousness, onset during exertion, thunderclap headache (onset with max intensity in 1 second), limited neck flexion on exam.

If any one of these variables are present, then you cannot rule out SAH and you need to consider further testing like CT. If none of these signs or symptoms is identified, sensitivity for exclusion of subarachnoid hemorrhage is 100%. However, specificity is only 15%, so the tool should not be used to definitively diagnose subarachnoid hemorrhage.

Management

Admission to neurosurgical service with critical care monitoring. ~35% of these patients have worsening neuro functioning after admission.

Discuss intubation for those GCS $<$ 8, and/or need for better critical care monitoring in the setting of elevated ICP, hemodynamic instability, and/or need for heavy sedation/paralysis.

Digital subtraction angiography (DSA): gold standard for high resolution detection of intracranial aneurysm as the cause of the SAH. If negative, it is often repeated due to a substantial number of false negatives.

In most settings, CTA has virtually replaced conventional angiography in many institutions as initial "aneurysm-mapping" test due to its speed, lack of invasiveness, and cost.

Definitive management: surgical clipping and endovascular coiling. These procedures are outside the scope of this review.

High yield Neuro Critical Care:

Please see our handout on ICP management on our website for more details on general neurological critical care!

Consider ventriculostomy for direct ICP monitoring if enlarged ventricles, consider craniectomy in select patients.

BP control: guidelines are not clear. Goal of SBP $<$ 160 is reasonable. Labetalol, nicardipine, clevidipine are preferred. Benefit of lowering BP might be offset by risk of infarction (CPP = MAP - ICP). i.e. if ICP is high then the only variable maintaining perfusion is MAP. Patient's consciousness might be a helpful marker: alert and oriented = SBP \sim 140. Patient impaired = SBP \sim 160.

Seizure management: widely debated with no consensus. Higher risk patients should have a lower threshold to starting. There is no good evidence to support prophylaxis in patients post-clipping/coiling if they previously had no seizures and had a good initial grade.

Complications

Overall, a high mortality rate with average being 51%! The majority die within 30 days.

-Rebleeding: highest risk within the first 24 hours.

-Hydrocephalus: caused by obstruction of CSF by blood products. Early and late complication. Consider drain placement.

-Vasospasm: Delayed cerebral ischemia. Basically, a form of inflammatory complication where lysis of clots and endothelial damage cause smooth muscle contraction. Associated with poor neurologic decline and high mortality.

-Presentation: neurologic decline/LOC/AMS/focal deficits vs clinical silence. Peaks at 7 days post-hemorrhage. No sooner than 3 days.

-Prevention: all patients should receive nimodipine and statins (improves outcomes but no direct evidence actually shows it affects the incidence or symptoms of vasospasm).

All patients should undergo TCD (transcranial doppler) to monitor for vasospasm. DSA is more accurate but one must go to the OR.

-Hyponatremia: likely due to hypothalamic injury. Target euvolesmia and normal electrolyte balance. Isotonic saline is the crystalloid of choice (this is the *only* time we support the use of normal saline for IVF!).

Bottom line: any change in clinical status warrants a stat CT head scan!

Prognosis

The most important prognostic factors for good outcome are as follows: consciousness and neurologic exam on initial evaluation, younger age, the amount of blood on CT.

Long term complications for survivors include neurocognitive disability, epilepsy, and lasting focal deficits.

Unfortunately, those with SAH have a small risk of recurrence of SAH, despite successful repair.

Family members have a 5-fold risk of SAH compared to the rest of the population.

References: check out our website for a complete list of references under this topic heading. We didn't want to kill any more trees!