The Non-Neurosurgeon's Guide to Subarachnoid Hemorrhage

PART ONE: THE BASICS

Introduction

Subarachnoid hemorrhage is one of the most devastating forms of stroke. It is the result of arterial bleeding into the subarachnoid space and has a significant associated morbidity and mortality. Most (85%) subarachnoid hemorrhages (SAH) are due to aneurysmal rupture. Other causes include trauma, perimesencephalic bleeding, cocaine abuse, and dural arteriovenous malformations. This guide is primarily concerned with aneurysmal SAH, as this is the most serious type and requires aggressive treatment. Nonaneurysmal SAH has a much better prognosis and often requires nothing more than supportive care.

Initial Evaluation

As with everything else, the initial evaluation begins with the history. The classic description of an aneurysmal rupture is the "thunderclap headache," also known as the "worst headache of my life." This is usually associated with photophobia, vomiting, and neck stiffness. Headaches that occur during physical exertion, sex, or that wake the patient up from sleep are suspicious for SAH. So are headaches associated with syncope or chest pain. More serious initial presentations can include altered mental status and frank coma.

Three major modifiable risk factors have been identified for SAH: smoking, heavy alcohol abuse, and poorly controlled hypertension. A first-degree relative with a history of an intracerebral aneurysm is a risk factor as well. Patients with polycystic kidney disease may have congenital berry aneurysms, but the rate of rupture is similar to the general population.

Physical examination may reveal nuchal rigidity, cranial nerve palsy (an abducens nerve palsy is the most common), or peripheral deficits. Particular attention should be paid to the Glasgow Coma Scale and the patient's level of consciousness.

The **Hunt and Hess Scale** grades SAH based on clinical exam. This is important to determine for both therapeutic and prognostic purposes.

H&H Class	Clinical Findings
0	Unruptured aneurysm
1	Headache, may have nuchal rigidity, **alert**
2	Headache, cranial nerve palsy or motor deficit, **alert**
3	**Drowsy,** headache, cranial nerve palsy, motor deficits
4	**Stuporous** or **light coma**, hemiparesis or hemiplegia
5	**Comatose,** decorticate/decerebrate posturing, dense hemiplegia

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In the past, patients with a poor-grade SAH (H&H 4 or 5) were treated conservatively. Recent data suggests that with aggressive care, even those with significant disability on presentation have a good chance of a meaningful recovery (modified Rankin score 0-3). Don't be overly pessimistic, especially in the first few days.

Diagnosis

The cornerstone of diagnosis is a noncontrasted CT of the brain. If the CT is negative and the suspicion of SAH is moderate to high, a lumbar puncture should be performed. Xanthrochromia or a high red blood cell count that does not clear between the first and last tubes of CSF collected is diagnostic of SAH.

The **Fisher Scale** describes the appearance of the SAH on the CT scan. This classification, along with the Hunt and Hess Scale, is predictive of the likelihood of DCI (more on that later).

Fisher Class	CT Findings
I	No SAH seen
II	Diffuse SAH without clot or hydrocephalus
III	SAH with thick (> 1mm) clot in the suprasellar or basilar cisterns
IV	SAH with intraventricular extension or intraparenchymal clot

CT angiography is being used more frequently—it can diagnose aneurysms and arteriovenous malformations and is less invasive than a conventional angiogram. A four-vessel angiogram will still be necessary, but the CTA may limit it to one (for coiling) rather than two (one diagnostic, one for treatment). Of course, if the CTA doesn't show anything, a diagnostic angiogram will still be necessary. This will hopefully demonstrate the aneurysm (or aneurysms—20% of the time, there are more than one). If it doesn't, a repeat angiogram should be performed in 4-5 days to see if the first study missed a small ruptured aneurysm.

Repair

Most aneurysms are now treated with endovascular coiling. A minority will need clipping, which requires a craniotomy. Early repair is preferred before the risk of delayed cerebral ischemia sets in.

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PART TWO: THE INTENSIVIST'S ROLE

Initial Stabilization

Always keep the ABC's in mind!

A: Patients who have a GCS less than 8, who are seizing, or who have a rapidly declining condition should be intubated. Have a low threshold for protecting the airway. **The risk of rupture of the aneurysm outweighs the risk of just about anything else, so make sure that you deeply sedate the patient. Rapid-sequence intubation works well, and propofol is a great drug for the sedative. Transient hypotension is preferred over a spike in blood pressure.**

B: Full ventilatory support should be initiated, with a goal of normocapnia ($PaCO_2$ 35-40) and normoxia (PaO_2 90-100). Hyperventilation works, but it's transient and works by making the brain ischemic. Use it if the patient is herniating and you need to buy 5 minutes to get the mannitol running or 10 minutes to get the ventriculostomy placed.

C: Make sure you have adequate IV access. Most patients will need a central line for infusing caustic antihypertensives, fluids, insulin drips, etc. Blood pressure control is important—more about this in a minute! If the patient is going to need continuous infusions of antihypertensive drugs, place an arterial line.

D: Stands for "disability," or "da' Brain." Frequently reassess the patient's neurologic status, especially if he's on a ventilator. Use a short-acting sedative like propofol for sedation and a short-acting narcotic like fentanyl for pain control.

E: Pay attention to the patient's temperature—heat is not good for injured brain! Aggressively control hyperthermia with antipyretics and cooling blankets. The Arctic Sun is good for maintaining normothermia as well.

Blood Pressure Control

The most dangerous early complication of aneurysmal SAH (for those who survive to make it to the hospital) is **rebleeding**. The mortality for an aneurysm that rebleeds has been estimated to be as high as 75%. The risk of rebleeding is 4% on the first day post-bleed, and 1% every day thereafter for 30 days if the aneurysm is not repaired. For this reason, strict blood pressure control is important. The goal systolic blood pressure is 100-140, or a mean arterial pressure of 70-90.[1]

All patients with SAH should be given nimodipine 60 mg orally q 4º for 21 days—this is for prevention of delayed cerebral ischemia. Some patients will only need PRN

[1] This is *until the aneurysm is secured* by coiling or clipping. Once that's taken care of, the blood pressure should be allowed to autoregulate.

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antihypertensives (e.g., labetalol 10-20 mg IV q 1º or hydralazine 10 mg IV q 4º). Others may need a continuous infusion. Nicardipine, 5-15 mg/hr, is very useful and does not affect the AV node. Labetalol, 0.5-2 mg/min, is a good alternative, especially if the patient has cardiac dysfunction or is tachycardic. Do not use sodium nitroprusside! It lowers the blood pressure, but it is both an arterial and venous dilator, so it can impair cerebral autoregulation and worsen cerebral edema.

Hydrocephalus

Hydrocephalus is common with Fisher III and IV SAH, and persistent hydrocephalus is the major late complication of subarachnoid hemorrhage. Most of the time, the neurosurgeon will place a ventriculostomy (or EVD, "external ventricular drain") when the patient is first diagnosed. This is useful in preventing persistent hydrocephalus, and can be used during the intensive care management of the patient—the ICP can be monitored, and CSF can be drained for therapeutic purposes. Patients with persistent hydrocephalus may need to have the shunt internalized.

Delayed Cerebral Ischemia

What we used to call cerebral vasospasm is now called delayed cerebral ischemia (DCI). DCI is characterized by smooth muscle proliferation and hyperreactivity. It is thought to be due to breakdown of the blood products in the subarachnoid space, specifically the alteration of inducible nitric oxide synthase. DCI is the leading cause of morbidity following SAH and contributes significantly to the overall mortality. Oral nimodipine has been shown to reduce the risk of DCI (probably due to ameliorating delayed calcium toxicity)—that's why everyone gets it for 21 days.

DCI usually doesn't occur before Day 4 post-bleed, and almost never persists beyond Day 21. The peak incidence occurs between Days 7 and 10. DCI should be suspected whenever the patient develops one of the following:

- New neurologic deficits
- Difficult-to-control hypertension
- Respiratory distress or failure
- Pulmonary Edema
- Changes in level of consciousness, including significant agitation or combativeness[2]

DCI can be detected with transcranial Doppler monitoring or cerebral angiography (either CT or DSA). Remember that this isn't a focal process, though—only about 50% of people who have large vessel narrowing on angiography will have symptoms, and even with focal narrowing the deficits are more global. TCD monitoring is useful because it's done at the

[2] Frequently, patients will become disinhibited and will piss off the nurses. This may be the earliest warning sign of DCI. Be very wary of what looks like frontal lobe dysfunction!

bedside and gives you a trend to follow. If the peak velocities of the vessels are over 120 cm/sec, that should get your attention. A Lindegaard ratio (MCA:ICA) > 3 can also indicate arterial narrowing—this is helpful because as the systemic blood pressure rises, so does cerebral blood velocity.

The medical management of DCI is focused on ensuring adequate cerebral perfusion. By the time DCI sets in, the aneurysm should (hopefully) be secured, thus eliminating the risk of rebleeding. Allow the blood pressure to autoregulate—often, the brain can determine how much blood it needs. Induced hypertension is often started as well—dopamine, norepinephrine, and phenylephrine have all been used to get the SBP over 160-180, and sometimes as high as 220 (if the heart can handle it). Give enough fluids to maintain euvolemia, but don't push to the point of pulmonary edema. Cerebral angiography can be performed for therapeutic intra-arterial verapamil or nicardipine, or for balloon angioplasty.

Therapy with statins and intravenous magnesium has been studied, but the evidence is underwhelming. Prophylactic hypervolemia and angioplasty are also not supported by the evidence. RBC transfusion to a hemoglobin of 10 may be beneficial in those who are at high risk of, or actively having, DCI.

PART THREE: COMPLICATIONS

Cardiac Complications

Nearly all patients with SAH will have some kind of EKG change. Inverted T-waves, ST depression, and even ST elevation patterns consistent with acute MI can be seen. Troponin elevation is common. Conduction abnormalities can also be seen—QRS prolongation, Torsade de Pointes, bundle-branch block, atrial fibrillation, and ventricular tachycardia all may occur. Some patients may show signs of cardiac pump failure, even progressing to cardiogenic shock.

The important thing to remember is the mechanism—cardiac dysfunction is due to contraction band necrosis and demand ischemia due to the intense catecholamine surge associated with the SAH.[3] This is very different from the usual cause of acute MI—plaque rupture with thrombosis of a coronary vessel. Consequently, the patient does not require antithrombotic therapy (although you couldn't do that anyway, on account of the bleeding) or urgent cardiac catheterization. The best treatment is supportive, with pacing or inotropic therapy if needed and beta-blockade for tachydysrhythmias.

[3] Also seen with Tako-tsubo cardiomyopathy, pheochromocytoma, and "voodoo death."

Neurogenic Pulmonary Edema

Neurogenic pulmonary edema can be seen with SAH, intracerebral hemorrhage, brain tumors, and after craniotomy. The mechanism is related to the loss of neural regulation of pulmonary venous sphincter tone, leading to an abrupt increase in pulmonary venous pressures and flash pulmonary edema. This is noncardiogenic pulmonary edema and is best treated with positive pressure, not diuretics.

Seizures

Seizure prophylaxis is controversial—only 6-18% of SAH patients have seizures, and anticonvulsants are associated with cognitive dysfunction, fever, and DCI (especially phenytoin). Factors that increase the risk of seizures include an MCA aneurysm, an intracerebral hematoma, intractable hypertension, and previous seizures. In those cases, a brief period (3-7 days) of antiepileptic therapy is prudent. Levetiracetam or valproic acid are preferred over phenytoin.

For those patients who seize, therapy is needed. Lorazepam (0.1 mg/kg, or 8-10 mg IV) is the preferred drug to stop the seizure. Levetiracetam is preferred for suppressive therapy, and should be continued for at least 3 months. Nonconvulsive status epilepticus should always be considered in comatose SAH patients, and EEG monitoring may be necessary.

Cerebral Salt Wasting

Many patients with SAH will become hyponatremic, and this can worsen neurologic deficits. This is usually due to cerebral salt wasting (CSW), not SIADH. The distinction is important because, while SIADH is treated with water restriction and diuretics, CSW is treated with saline and fluid replacement. CSW causes intravascular volume depletion, and fluid restriction and diuretics could make this worse. Brain-injured patients do not tolerate hypovolemia very well. Check the urine osmolality and replace the fluid and sodium losses with a more hyperosmolar fluid—1.8% or 3% saline in many cases. Trying to replace sodium losses when the urine osmolality is 500 mOsm/L with normal saline (308 mOsm/L) is futile—better to use 1.8% saline (616 mOsm/L) or 3% saline (1026 mOsm/L). Fludrocortisone 0.2 mg bid may also help.

SIADH is normally a state of euvolemia and the urine output is low. CSW is characterized by hypovolemia and polyuria, with a urine osmolality higher than serum. The uric acid is low with SIADH, whereas it is normal in CSW. A Fractional Excretion of Uric Acid (FEUA) can be calculated to differentiate the two conditions—in both cases, the FEUA will be elevated (> 10%). When the hyponatremia is corrected, however, the FEUA will normalize in SIADH but remain elevated in CSW.

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Coagulopathy

Injured brain tissue releases tissue thromboplastin, which can lead to coagulopathy; in addition, many patients with SAH are on anticoagulants (Coumadin, Plavix, aspirin). These should be treated aggressively.

Hyperglycemia

Hyperglycemia and hypoglycemia are both very, very bad for brain-injured patients. Tight glucose control is important, even in non-diabetics—keep it 100-180.

Deep Venous Thrombosis

SAH patients are at high risk for DVT and should all have mechanical prophylaxis. As soon as the aneurysm is secured, start pharmacologic prophylaxis as well. Enoxaparin is the first choice.

Protein Malnutrition

Brain-injured patients are usually in a hypercatabolic state. With severe brain injuries, the metabolic demands can be as high as that seen in a 30-40% burn. Early enteral nutrition is a must, and be diligent about replacing anabolic electrolytes (potassium, magnesium, phosphorus) and ensuring a positive nitrogen balance.

Nosocomial Infections

Some of the biggest complications that SAH patients face are infectious—ventilator-associated pneumonia, sinusitis, UTI, line sepsis, etc. Follow infection-prevention recommendations and be vigilant for brewing infections.

Issues Unique To Palmetto Health Richland

Dr. Webb and Dr. Graham are the two physicians who perform interventional therapy for cerebral aneurysms. Dr. Webb is a neurosurgeon; Dr. Graham is an interventional radiologist.

Dr. Webb will admit her own patients and will provide the medical and surgical care for them. She may consult PCCM for ventilator management, but for the most part the MICU team will not be involved with her cases unless she specifically asks for assistance.[4]

Dr. Graham has asked the MICU team to provide medical care for his patients. He will perform the necessary diagnostic and therapeutic procedures, but the rest of the

[4] This applies to cerebrovascular patients only. Intracerebral hemorrhages, subdural hematomas, brain tumors, etc.—those will all be seen by the MICU team in collaboration with Neurosurgery.

management (i.e., most of this handout) will be handled by PCCM. Neurosurgery will see the patients every day and leave recommendations; if an EVD is needed, then any of the neurosurgeons will do it. Dr. Webb does not cross-cover Dr. Graham's patients, and vice versa. Both Dr. Graham and the Neurosurgery attending will communicate with the PCCM attending every day to discuss the plan of care.

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