SAH…
Current State of the Art…

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Introduction

• Signs and symptoms of a problem
• What are aneurysms
  – Why are they bad?
  – Because they rupture...and cause...
• Subarachnoid hemorrhage
• SAH is more a result or sign...don’t really treat it...
  – But do treat the cause
  – And its sequelae...
Cerebral vasculature
Big problem in small packages
Pathophysiology

- Weakness in the wall of an artery, typically at the branch point of a large intracerebral artery
  - Classically thought of as a congenital weakness in the wall of an artery
  - Current leading theory is repeated vascular injury

- Conditions with increased incidence of aneurysms
  - Atherosclerosis
  - Hypertension
  - High flow vascular lesions
  - Moya Moya
  - Polycystic Kidney Disease
  - Fibromuscular Dysplasia
  - Etc
Signs and Symptoms of a Problem…

- Worst headache of a patient’s life
- Neck stiffness
- Nausea
- Vomiting
- Encephalopathy (confusion)
- Focal neurological symptoms (weakness, numbness, cranial neuropathies)
Work-up & Diagnosis

- Head CT
  - If positive CTA or cerebral angiogram
- Lumbar Puncture
- Angio negative SAH
  - Benign perimesencephalic hemorrhage
  - Localized vasospasm
Subarachnoid Hemorrhage
So is this bad??

- 10% die before reaching medical attention
- 50% die within the first month
- Of the survivors, half will have a permanent neurological deficit
What do they look like??

• 80 to 90% with signs and symptoms of SAH
  – Worst headache of one’s life
  – Meningeal signs
  – Lethargy
  – Nausea/vomitting
  – LOC, altered MS, coma

• Expanding aneurysm
  – Third nerve palsy (posterior communicating artery aneurysm)
  – Decreased vision (peri-ophthalmic aneurysm)
Now What??

- SAH considered neurosurgical emergency
- Without treatment 25 to 50% of patients will have a second rupture with a mortality rate of 85%
- Immediate treatment
- Definitive Treatment options
  - Open surgery, and occlusion of the aneurysm
  - Endovascular embolization via cerebral angiography
First base...

- Stabilize the patient (not all patients are created equal…)
- Treatment really depends on the condition of the patient…
- Hunt & Hess SAH grade is a helpful descriptor and prognosticator
Be a doctor…

- **History - Get the story…**
  - Did the patient pass out and fall or fall and pass out…
  - Did the patient pass out and run their car into the telephone pole or did they hit the pole and pass out?
  - Premorbid neurological condition
    - **Co-morbidities**
      - CAD, COPD, CVD, DM etc…
    - **Medications/Allergies** (anticoagulation? Anti-platelett? Metformin?)
    - **Family Hx** – anyone else have an aneurysm?? Any sudden deaths due to brain hemorrhage?
    - **Social Hx** – hx of recreational pharmacology??

- **Hunt & Hess Grade**
• **DESCRIPTION**
  • Asymptomatic, mild headache, slight nuchal rigidity  
  • Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy  
  • Drowsiness / confusion, mild focal neurologic deficit  
  • Stupor, moderate-severe hemiparesis  
  • Coma, decerebrate posturing

• Some authorities will have a “1A” or “2A” to indicate grade plus a cranial nerve deficit
Rounding 1<sup>st</sup> going to 2<sup>nd</sup>

- Depends on grade, CT scan and comorbidities
  - ICP management
    - HOB > 30°
    - Mild hyperventilation if intubated
    - EVD if hydrocephalus
  - Seizure prophylaxis
- Start Nimodipine
- NS + 20KCl at 100-125/hr
- BP management (generally 120-140s)
- EKG – cardiac stun
- CXR
- TCDs, DVT prophylaxis, PUD prophylaxis, routine labs
Getting to 2\textsuperscript{nd} base…

• Keep calm! (recommended for both physician and patient!)
  – Try to avoid stimulation
  – Anxiolytics
  – Analgesics
  – May also use steroids that help with headache and nuchal rigidity

• Trying to prevent…
Re-rupture!

- Very high morbidity and mortality rate...
- Incidence is 4% in first 24hrs and about 15-20% in first 7 days
- Antifibrinolytic therapy
  - Tranexamic acid
  - Reduces rebleed rates…but at a cost – increased incidence of vasospasm and ischemic events and increased incidence of hydrocephalus…therefore use is discouraged.
- Best prevention is early definitive treatment (the best defense is a strong offense…)
Clip vs Coil...
Coiling

- **Pros** – less invasive, typically shorter surgeries, can treat aneurysms difficult to surgically clip (basilar tip), less recovery following the procedure.

- **Cons** – aneurysm is more likely to recur and recanalize...can occur many years after treatment. Intra-procedural rupture. Not able to remove the blood or substantial hemorrhage.
Clipping

• **Pros** – More durable, can treat wide necked aneurysms, less likely to have a residual and recurrence

• **Cons** – more trauma due to the operation, higher infection rate, more stressful to the body, higher initial complication rate.
International Subarachnoid Aneurysm Trial (ISAT), a prospective, randomized trial comparing surgery (craniotomy for clipping) to endovascular therapy (coiling) in the treatment of ruptured intracranial aneurysms

Conclusions

- Craniotomy and clipping had a 30.3 chance of poor outcome at 1 year
- Coiling had a 23.7 chance of a poor outcome at 1 year
- Therefore a relative risk reduction of 6.9%
Here come the critics …

- Patients case reviewed by a neurovascular team
- 9,559 with ruptured aneurysms were reviewed for the study
- Only 2,143 were randomized, namely the neurovascular team felt that either clipping or coiling would be an appropriate treatment
- 7,416 of patients did not get randomized, the majority of which underwent surgery
Now what??

• Once the aneurysm is definitively clipped (or coiled)…
• Post op monitoring
  – Vasospasm
  – ICP or hydrocephalus
  – Seizures
• Cardiac
• Pulmonary
• Renal function
Main culprit...vasospasm

- Pathophysiology (theory…) – breakdown product of the blood cause irritation in the arterial smooth muscle causing irritability and proliferation/hypertrophy
- Peak incidence is post bleed day 6-8
- Rarely seen before day 3 or after day 17
- Most significant cause of morbidity and mortality in patients that survive initial hemorrhage and are stabilized (mortality rate of 7-10%)
Diagnosis

- Clinically
  - In patient’s who have a clinical exam
    - Careful monitoring of neurological status
    - Monitoring for development of a new focal deficits
    - Overall decrease in sensorium
  - Intubated patients or those in coma
    - No clinical exam, therefore must depend on diagnostic tools
Diagnostic Tools

- **Transcranial Doppler studies**
  - Evaluates velocity of blood flow in main arteries of the cerebral vasculature
  - Compared to extracranial ICA as a ratio to correct for hyperdynamic flow

- **CT perfusion studies**
- **Cerebral Angiography**
Treatment...

• Nimodipine

• Avoidance of hyperventilation – Keep normocarbic
  – May need for ICP control...however must carefully balance needs/risks

• Gold standard....

  Triple H Therapy

• Hypertension

• Hypervolumia

• Hemodilution
Need to get physical??

• Generally, in patients with subtle initial deficits
  – HHH, generally increasing SBP
  – Typically the deficit will reverse…

• Pharmacology
  – To this point disappointing
  – Some evidence to the benefit of IV Mg…

• If medical therapy fails…off to the angio suite…
Getting tough…

- Intra arterial pharmacological treatment
  - Papavarin
  - Verapamil
  - Often temporal relief of the spasm
  - Needs multiple infusions

- Angioplasty
  - Gentle dilation of the arterial wall
  - But must push it so that the smooth muscle is torn…
  - Therefore permanent treatment of vasospasm…
  - But there is risk of arterial rupture…
On the Horizon

• General trend to the severity of vasospasm...the more blood and the worse the clinical exam, the higher risk of vasospasm...

• For this reason, there have been several “ideas” regarding the reduction of the SAH
  – Some good ideas
  – And of course some not so good...
Promising treatments...

- **Implants**
  - Nicardipine prolonged release implants
  - Placed intraoperatively into the cisterns
  - Medication leaches into the CSF at the site of the vasospasm had has shown promise

- **Clazosentan** – selective endothelin IA receptor antagonist...reduces frequency and severity of vasospasm

- **Statins**...3 small studies have shown benefit
The take home points

• SAH is bad...very bad...
• To clip or coil...not a foregone conclusion
  – Decision best made by a team with expertise in both treatment options

The Aftermath...

• Vasospasm
  – major source of morbidity and mortality
  – Monitored through exam, TCDs, perfusion studies
  – Symptomatic vasospasm, treated with Nimodipine, HHH, angiography
Quiz Questions

T or F  All ruptured aneurysm are best treated with coiling?

What is the most common cause of neurologic morbidity and mortality in a patient who has suffered a SAH and has had definitive treatment of the ruptured aneurysm?

T or F  In a randomized, double blinded study (funded by Obamacare), snail therapy has been shown to reduce the incidence and severity of vasospasm following aneurysmal SAH, and is quite cost effective…