Mobilization of patients with a Subarachnoid Hemorrhage in the Acute Care Setting

Joseph Borst, PT, DPT
William Pino, PT, DPT

March 14th, 2017
Good Shepherd Penn Partners at the Hospital of the University of Pennsylvania
Objectives

- Define subarachnoid hemorrhage (SAH)
  - Understand classifications of SAH
- Review of brain anatomy
- Understand complications associated with these patients
- Medical management of a SAH
- Treatment strategies with a patient with a SAH
- Considerations of mobility with an external ventricular drain
Case Study

- 50 year old African American female presents to ED with worst headache of life, photophobia.

- PMH significant for smoking, hypertension, EtOH abuse
“Bleeding within the subarachnoid space, which lies between the arachnoid and pia mater, typically filled with cerebrospinal fluid.”

Two types:
- Aneurysmal SAH
- Non-aneurysmal SAH
Aneurysmal SAH

- Occurs in 10-15/100,000 people in the U.S.
  - Slightly higher incidence in women than men
  - Mean age of rupture is 55 (however affects all ages)

- Saccular aneurysm accounts for 80-90% of all cerebral aneurysms

- Other vascular abnormalities include:
  - AVM rupture
  - Arterial dissections
  - Tumors
Symptoms of Aneurysmal SAH

- Primary symptom is sudden onset of severe headache, with or without photophobia
  - Worst Headache Of Life (WHOL) - 80%
- Photophobia
- Seizure
- Nausea
- Vomiting
- Loss of consciousness

Case courtesy of Dr Domenico Nicoletti, Radiopaedia.org, rID: 43168
Non-aneurysmal SAH

• A hemorrhage in which no structural cause can be identified on radiographic imaging.
  - Perimesencephalic hemorrhage
  - Non-perimesencephalic hemorrhage

• Gradual onset of headache
• Less incidence of hydrocephalus, vasospasm, or rebleeding
• Significantly improved prognosis
• Shorter length of stay
  - Typically discharged by post-bleed day #7
Perimesencephalic Hemorrhage

- Diagnosis of exclusion
  - Requires confirmation of negative vascular imaging

- Fits a commonly defined pattern
  - 38% of angiogram negative SAH fit this pattern
  - Centered around the basilar cisterns around the midbrain

- Etiology remains unknown
  - Some theories propose bleeding of venous origin or rupture of small perforating vessels
  - Research varies and is inconsistent

- Should be managed as if they have an aneurysmal SAH until definitive vascular imaging is obtained.
Perimesencephalic Hemorrhage
Epidemiology

- Hemorrhagic stroke accounts for 20% of all strokes
  - SAH totals 3% of all strokes
- 15-20% of patients with a SAH do not have an aneurysm preceding the bleed
- Disorders associated with weakened blood vessels:
  - Fibromuscular dysplasia
  - Polycystic kidney disease
  - Ehler-Danlos syndrome
  - Marfan’s syndrome
  - Alpha-1 antitrypsin deficiency
Risk Factors

- Cigarette Smoking*
  - Most preventable risk factor
- Hypertension
- Genetic Risk
  - 3x-5x increase if first-degree relative positive for SAH
- Alcohol*
- Drug Abuse*
  - Methamphetamine and Cocaine Use
    - Acute cocaine use may be associated with higher rates of rebleeding and in hospital mortality.
- Gender
Diagnosis

- Digital Subtraction Angiography (DSA)
  - Gold standard
- Cerebral Angiogram
- CT Angiogram
- Lumbar Puncture
- Non-contrast HCT

Case courtesy of A.Prof Frank Gaillard, Radiopaedia.org, rID: 36179
Classification - Hunt and Hess Scale

Classification for severity of subarachnoid hemorrhage (SAH)

- **Grade 1**: Asymptomatic or mild headache
- **Grade 2**: Cranial nerve palsy or moderate to severe headache/nuchal rigidity
- **Grade 3**: Mild focal deficits, lethargy, or confusion
- **Grade 4**: Stupor and/or hemiparesis
- **Grade 5**: Deep coma, decerebrate posturing, moribund appearance
Classification - Fisher Scale

- Index for vasospasm risk (but not clinical outcome) based on hemorrhage pattern seen on initial HCT
  - **F1**: No blood detected
  - **F2**: Diffuse disposition or thin layer with all vertical layers of blood (in interhemispheric fissure, insular cistern, or ambient cistern) less than 1mm thick
  - **F3**: Localized clots and/or vertical layers of blood 1mm or more in thickness
  - **F4**: Intracerebral or intraventricular clots with diffuse or no subarachnoid blood
FISHER SCALE FOR GRADING SUBARACHNOID HEMORRHAGE AND VASOSPASM RISK

Fisher grade 1
No SAH visualized
Low risk for vasospasm

Fisher grade 2
Diffuse SAH without clots
or vertical layers of blood 1 mm or more thick
Low risk for vasospasm

Fisher grade 3
Localized blood clots in the subarachnoid space or blood clot more than 1 mm thick in the vertical plane
(interhemispheric fissure, insular cistern, or ambient cistern)
High risk for vasospasm

Fisher grade 4
Intracerebral or intraventricular blood with only diffuse blood or without blood in the basal cisterns
Low risk for vasospasm
**Glasgow Coma Scale (GCS)**

- Used to gauge the severity of an acute brain injury

**Motor Response (M)**
- 6 = Normal
- 5 = Localized to pain
- 4 = Withdraws to pain
- 3 = Decorticate posture
- 2 = Decerebrate posture
- 1 = None

**Eye Opening (E)**
- 4 = Spontaneous
- 3 = To Voice
- 2 = To Pain
- 1 = None

**Verbal Response (V)**
- 5 = Normal Conversation
- 4 = Disoriented Conversation
- 3 = Words, but not coherent
- 2 = No words, only sounds
- 1 = None

\[ E + V + M = \text{GCS} \]

- **Severe**: 3-8
- **Moderate**: 9-12
- **Mild**: 13-15
Prognosis

• Aneurysmal
  • Neurologic Grade (Hunt and Hess)
  • Re-bleeding
  • Complications (ie. Vasospasm)
  • Patient Age
  • Amount of blood on initial HCT

• Non-aneurysmal
  • Typically have improved prognosis and shorter length of stay
Complications (Vasospasm)

- Narrowing of a cerebral blood vessel that can cause reduced blood flow distally
  - May lead to delayed ischemia and cerebral infarct if left untreated
- Associated with Fisher Grade upon admission
- Develops between 3 to 14 days
  - Peaks at 7-10 days post bleed
- Occurs in approximately 30% of patients with SAH
- Treated with maintaining euvolemia, induce hypertension, optimize cardiac output
- Trans-cranial dopplers (TCD)
  - Low frequency (2MHz) ultrasound to visualize intracranial vessels
  - Assesses for intracranial stenosis, detect emboli, and monitor for reperfusion.
Complications (Hydrocephalus)

- Non-communicating vs Communicating
- Occurs in 20-30% of SAH survivors
- Can cause acute and chronic neurological effects
- May require EVD placement (acute) or VPS (long-term)
  - 50%-80% of patients show neurological improvement with placement of EVD
- Increased risk for aneurysmal bleeding if CSF is rapidly drained
  - Secondary to significant change in intracranial pressure
Complications (Cardiac/EKG Abnormalities)

• Can occur in up to 50% of patients who experience a SAH
  • Often seen abnormalities include:
    • T-wave inversion
    • QT prolongation
    • ST segment changes

• Hypertension is also common after SAH
  • Maintain SBP<160

• May not be aggressively treated if mild to moderate HTN
• Fluctuating changes in pressure may be worse than an absolute pressure
Complications (Hyponatremia)

- Develops in 10%-34% of patients with SAH
- Almost always secondary to cerebral salt wasting
  - Different diagnosis than SIADH
- Characterized by hypovolemia and hyponatremia
- Should not be fluid restricted
- Treatment includes fluid and salt administration
Typical pathway upon admission with suspected SAH

- HCT in emergency room identified SAH
- Admit patient to Neuro ICU
- Assess **Hunt and Hess** Scale and **Fisher** Grade
- Obtain routine labs
- Perform 12-lead EKG, CXR, CT angiogram
- EVD will be placed at this time if indicated
  - Symptomatic hydrocephalus OR have GCS < 8
- Research coordinator is alerted
Typical Medical Management for SAH at Admission

- **Neurologic:**
  - EVD placement if applicable
  - Seizure prophylaxis
  - Anti-fibrinolytics

- **Renal:**
  - Maintain euvoolemia
  - Maintain Na+ within normal range
  - Maintain Mg+2 within normal range

- **Cardiovascular:**
  - Arterial line placement
  - Maintain SBP<160
    - Initiate anti-hypertensives if necessary, avoid fluctuations in BP
  - Start nimodipine and statins
  - Triple lumen subclavian or IJ placement
Typical Medical Management for SAH at Admission

- **Endocrine:**
  - Blood glucose monitoring
    - Target glycemic threshold: <200mg/dL
    - Follow HUP insulin infusion protocol if >200mg/dL

- **Prophylaxis:**
  - Stress ulcer prophylaxis
  - Venous thromboembolism prophylaxis
    - Anticoagulants held until 24 hours post craniotomy or aneurysm securing
  - Alcohol abuse withdrawal

- **Other:**
  - Analgesia
    - Lowest effective dose that does not limit clinical neurological examination
  - Anti-emetics
Surgical Interventions

**Surgical Clipping**

**Endovascular Coiling**

- Neurosurgery and interventional radiology decide which intervention will be given based on morphology and patient characteristics.

- Early surgery is associated with:
  - Lower rates of rebleeding
  - More aggressive treatment of vasospasms
Aneurysm Surgical Clipping
Aneurysm Endovascular Coiling
Post-Surgical Management

• Continue intracranial hypertension management

• BP management
  • SBP<160mmHg for 24 hours post-craniotomy
    • SBP=100-220mmHg thereafter
  • Team may liberalize blood pressure to SBP=100-220mmHg immediately if coiling takes place endovascularly
Transfer / discharge from ICU

- **Low grade (1-2)**
  - May transfer on post-bleed day 10 if no evidence of vasospasm for 48 hours

- **High grade (3-4)**
  - May transfer on post-bleed day 14 if no evidence of vasospasm for 48 hours

- May not be discharged on nimodipine
- May transfer directly from ICU to acute rehab specializing in brain injury
Treatment PT/OT

- Assessment of arousal, delirium
  - Use of GCS, JFK-CRS, CAM-ICU

- Assessment of mobility
  - PASS, mRS should be completed

- EVD protocol for mobility (if applicable)
  - Implications on the floor if mobility limited in ICU

- Progress functional mobility as appropriate

- Performance of ADLs (sitting versus standing)

- Cognitive assessment and treatment
  - MoCA and PHQ-2 should be completed
External Ventriculostomy Drain

- An external system for the temporary drainage of cerebrospinal fluid (CSF) from the lateral ventricles and monitoring of intracranial pressures.

- Monroe-Kellie hypothesis

- Risks associated with EVD include:
  - Infection
  - Hemorrhage
  - Drain occlusion from blood or tissue
External Ventriculostomy Drain

• Drainage is level dependent to tragus of ear

• Measured in cm H2o

• Most patients have the drain open at varying levels
  • Will perform clamp trials prior to VPS placement or discharging EVD
  • Clamping may also be performed during bed mobility and RN care
Progression of Mobility for Patients with EVDs

- Clamped at ALL times with RN present
  - RN may unclamp the proximal stopcock to obtain ICP
- Be mindful of hemodynamics with change of position
  - Hypotension, tachycardia, ICP
- Monitor for neuro changes throughout session
  - Take vitals throughout session not just pre-post
  - Talk to your patients!
- Have a plan
  - Chair set up along the walk not just at bedside
  - Use assistance when needed
References


