Code Stroke Intervention: Endovascular therapy for aSAH and management

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INTERVENTIONAL NEURORADIOLOGY
Disclosures

None
Part B. Objectives

- Epidemiology of aSAH
- Concept:
  - What is a brain aneurysm
  - Hunt and Hess grading scale
  - Know about severity of bleed: modified Fisher scale (from CT head)
- ED management
- Case
  - Management from RN perspective, after endovascular therapy
Introduction – Brain aneurysm

- Definition: Persistent pathologic dilatation of an arterial wall.
- An estimated 6 million people in the United States have an unruptured brain aneurysm, or 1 in 50 people.
- Aneurysms are estimated to occur in approximately 2% to 6% of the population (based on autopsy and angiographic studies).
- Non traumatic SAH represents about 3% of all strokes in the US.
- Worldwide incidence: 2 to 16 per 100,000 people.
- Women are more likely to have SAH → 1.24 : 1.
- Minority groups, particularly AA and Hispanic populations more frequently affected compared to white Americans.
Introduction

- Incidence of SAH increases with age
  - Mean onset of ≥ 50 years
- ~80% of SAH, a ruptured cerebral aneurysm is found
  - No source of bleeding in 15% of cases
  - Other etiologies such as AVM in 5%
Introduction – a SAH

- Mortality rates 8% to 67%
  - Median is 30% in the US
- Prehospital deaths estimated between 10% to 15%
- There has been a significant decrease in case-fatality rates of SAH across the globe
  - Likely owing to changes in managements of patients with SAH
  - Neurocritical care
  - Endovascular therapy
  - Refined microsurgical techniques
- However, despite the decrease in case-fatality rates, about half of survivors experience significant chronic reduction in health-related quality of life
Introduction

- A large proportion of survivors do not return to their previous level of employment, social independence and interactions, or personal or family relationships even 5 years after the event.

- This may be due to a combination of factors, including
  - Impaired physical functioning
  - Cognitive deficits particularly executive function and memory
  - Mood and emotional symptoms e.g. anxiety, depression and PTSD
  - Personality changes
Subarachnoid Space
Common locations of brain aneurysms

~75% of brain aneurysms
In these locations:
- Acomm
- Pcomm
- MCA bifurcation
Aneurysms can be classified by size and by configuration.
<table>
<thead>
<tr>
<th>Risk Factors for Subarachnoid Hemorrhage</th>
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</thead>
<tbody>
<tr>
<td><strong>Nonmodifiable Risk Factors</strong></td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Female sex</td>
</tr>
<tr>
<td>Prior history of aneurysmal subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Family history of subarachnoid hemorrhage</td>
</tr>
<tr>
<td>History of aneurysm in first-degree relatives (especially in two or more relatives)</td>
</tr>
<tr>
<td><strong>Modifiable Risk Factors</strong></td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Heavy alcohol use</td>
</tr>
<tr>
<td>Sympathomimetic drug use (eg, cocaine)</td>
</tr>
<tr>
<td><strong>Other</strong></td>
</tr>
<tr>
<td>Certain genetic disorders (eg, autosomal dominant polycystic kidney disease, type IV Ehlers-Danlos syndrome)</td>
</tr>
<tr>
<td>Anterior circulation aneurysms are more likely to rupture in patients who are younger than 55 years of age</td>
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<tr>
<td>Posterior circulation aneurysms are more likely to rupture in men</td>
</tr>
<tr>
<td>Significant financial or legal problems within the past 30 days</td>
</tr>
<tr>
<td>Cerebral aneurysms of more than 7 mm in diameter</td>
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</tbody>
</table>
Main emphasis when caring for patients with SAH

- Prompt evaluation and diagnosis
- Immediate transfer to appropriate centers
- Expeditious diagnosis and treatment of the bleeding source
- Overall good neurocritical care adhering to available treatment guidelines.
Clinical Presentation

- SAH typically presents with sudden and severe headache
  - Described as the worst headache ever
- Nausea, vomiting → d/t transient elevation in intracranial pressure (ICP)
- Photophobia
- Neck pain
- Loss of consciousness → d/t more sustained and severe ICP; can lead to coma and brain death.

Physical examination should include
- Determination of level of consciousness, fundoscopic evaluation
- Determination of meningeal signs
- Presence of focal neurologic deficits → in about 10% of cases and are associated with worse prognosis when thick SAH clot or parenchymal hemorrhage
Clinical presentation

- Occasionally, patients may present with seizures, acute encephalopathy and concomitant subdural hematoma and head trauma
  - → diagnosis of SAH may become more elusive

- A minority of patients may have a warning “sentinel” headache days to weeks before an aneurysmal SAH
  - → though to represent a small aneurysmal leak
  - Unfortunately this information is only obtained retrospectively as most of the time the headache is transient and head CT scanning is unrevealing in about 50% of cases
Step 1: Diagnosis

- **Head CT scan**
  - Sensitivity of 98%-100% for detection of SAH within 12 hours of symptom onset when compared to LP
  - Sensitivity decreases to 93% at 24 hours
  - Sensitivity only 50% at 7 days

- **LP**
  - Recommended in any patient with suspected SAH and negative or equivocal results on head CT.
  - CSF should be collected in 4 consecutive tubes and RBC count should be determined in tubes #1 and #4.
    - Diagnosis: Elevated opening pressure, elevated RBC count that does not significantly decrease from tube #1 to tube #4, and xanthochromia
Main focus initially is stabilization of airway, breathing and circulation

Patients who are unable to protect their airway should be intubated immediately
  - Most common indications: Coma, HCP, seizure, need for sedation for significant agitation

Once deemed stable → CT brain

Avoid hypertension
  - MAP <110 mmHg or systolic less than 140 mmHg until the ruptured aneurysm is secured
    - Use premorbid baseline blood pressure to refine targets and avoid hypotension
  - Pain control → best achieved with short acting opiates
    - I.v. labetalol 5mg to 20 mg pushes
    - Nicardipine 5 to 15 mg/h continuous
Step 2: Disease severity scoring

- What are the strongest predictors of neurologic complications and outcomes after SAH?
  - Severity of neurologic impairment on presentation
  - Amount of SAH on admission
<table>
<thead>
<tr>
<th>Grade</th>
<th>Symptoms</th>
<th>Other Neurological Signs</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Asymptomatic or mild headache</td>
<td>Minimal/slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Moderate to severe headache</td>
<td>Nuchal rigidity, no neurologic deficit</td>
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<td>(except for cranial nerve palsy)</td>
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<td>Drowsiness or confusion</td>
<td>Mild focal neurologic deficit</td>
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<td>4</td>
<td>Stupor</td>
<td>Moderate to severe hemiparesis</td>
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<td>5</td>
<td>Coma</td>
<td>Decerebrate posturing</td>
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Step 3: Admission to High-Volume Centers

- Transfer to high volume center, if not already in one
- Admit the patient to a dedicated neurocritical care unit
- Have the patient undergo a multidisciplinary evaluation for the management of an unsecured cerebral aneurysm
Case #1

- 45 y/o right handed woman presented to PSC with sudden onset of severe H/A, N/V and syncope 1 hour prior to presentation while she was moving furniture at her house
- PMHx of heavy smoking and cocaine use

- VS; BP 180/100 mmHg, HR 105’, SaO2 97% on room air, T 36.5 C

- GCS15, Hunt and Hess grade 2, modified Fisher scale score 3
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<th>Description</th>
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Case #1

Modified Fisher scale

- **Grade I:** no or min subarachnoid Blood, no IWH
  - 24%
- **Grade II:** Min subarachnoid Blood with IWH
  - 33%
- **Grade III:** Diffuse or focal, thick Subarachnoid blood, no IWH
  - 33%
- **Grade IV:** Diffuse or focal, thick Subarachnoid blood with IWH
  - 40%

*risk of symptomatic vasospasm*
Case #1

- She was started on a nicardipine drip to keep her SBP < 140 mmHg
- Transferred to a CSC
- DSA demonstrated an irregular, multilobed and wide-neck Acomm aneurysm
- After discussion with INR, NeuroSx and neuroICU, patient underwent surgical clipping
Clipping vs. coiling

Clipping

Coiling
Rerupture risk

- Rebleeding in unsecured aneurysms:
  - 1st 24 hours → risk is 4% to 15%
  - 1st 10 days → 2% to 4% per day for the first 10 days
  - 30% during the first 30 days
  - 2-4% per year thereafter

- Main risk factors associated with rebleeding:
  - SBP > 160 mmHg
  - Poor neurologic grade
  - Intracerebral or intraventricular hematomas
  - Ruptured posterior circulation aneurysms
  - Aneurysms > 10 mm in size

- Rebleeding rate in secured (treated aneurysms)
  - Coiling: 1-2%
  - Clipping ~1%
Best measure to reduce the risk of rebleeding

- Early treatment of unsecured aneurysms (as seen on previous slides)
- Coiling vs. clipping

### TABLE 1-6 Preferences for Treatment of Unsecured Aneurysms

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Preferred Treatment Modality</th>
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<tbody>
<tr>
<td>Advanced age</td>
<td>Endovascular coiling</td>
</tr>
<tr>
<td>Poor clinical grade</td>
<td>Endovascular coiling</td>
</tr>
<tr>
<td>Multiple underlying systemic conditions</td>
<td>Endovascular coiling</td>
</tr>
<tr>
<td>Aneurysms with wide neck-to-body ratio</td>
<td>Surgical clipping</td>
</tr>
<tr>
<td>Normal arterial branches arising from dome or body of aneurysm</td>
<td>Surgical clipping</td>
</tr>
<tr>
<td>Middle cerebral artery aneurysm</td>
<td>Surgical clipping</td>
</tr>
<tr>
<td>Top-of-the-basilar aneurysm</td>
<td>Endovascular coiling</td>
</tr>
<tr>
<td>Aneurysm associated with large parenchymal hematoma</td>
<td>Surgical clipping</td>
</tr>
<tr>
<td>High surgical risk</td>
<td>Endovascular coiling</td>
</tr>
<tr>
<td>Patient preference</td>
<td>Endovascular coiling</td>
</tr>
<tr>
<td>Clinical equipoise*</td>
<td>Endovascular coiling</td>
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* Unsecured aneurysm is considered equally suitable for either endovascular coiling or surgical clipping.
Step 4: Treatment of unsecured aneurysms

- Surgical clipping vs. Endovascular coiling
- Choice depends on several factors including:
  - Age
  - Aneurysm location
  - Morphology of the aneurysm
  - Relationship to adjacent vessels
- Because of the complexity of determining the most appropriate treatment for individual patients, it is recommended that a multidisciplinary team made up of cerebrovascular neurosurgeons, endovascular practitioners and neurointensivists confer to reach a consensus
- Overall, endovascular coiling should be preferred over surgical clipping whenever possible.
Step 5: ICU management

- More than 75% of SAH experience SIRS → likely as a result of elevated levels of inflammatory cytokines.
- SAH patients at higher risk for
  - HCP
  - Cerebral edema
  - Delayed cerebral ischemia (DCI)
  - Rebleeding
  - Seizures
  - Neuroendocrine abnormalities that lead to impaired body regulation of Na+, water and glucose
- SAH also unleashes hypothalamic-mediated changes, including increased sympathetic and parasympathetic drive, that result in cardiac and pulmonary complications. → EKG changes, arrhythmias, impaired cardiac contractility (eg, Takotsubo cardiomyopathy), troponinemia, and myocardial necrosis.
- Pulmonary complications: Neurogenic pulmonary edema
Seizures

- 20%-26% may present with seizure like episodes
- Patients with MCA bifurcation aneurysms, concomitant intraparenchymal hematomas, and poor clinical grade are at higher risk for seizures
- Patients treated with coiling have lower rates of seizures
- Long term risk for epilepsy is low
- Prophylactic anticonvulsants, 3 to 7 days.
- Frequency of subclinical seizure may be high in patients with poor-grade SAH → continuous EEG monitoring
Hydrocephalus

- Acute symptomatic HCP occurs in about 20% of patients with SAH, usually within the first few days after symptom onset.

What to look for?
- Decreased level of consciousness
- Signs of increased ICP such as impaired upward gaze and HTN

- Immediate f/u CT head is warranted
- EVD may follow
  - Some centers perform a lumbar drain
- Weaning the patient of an EVD should begin shortly after aneurysm obliteration or within 48 hours of insertion if patient is neurologically stable
- ~60% of patients who undergo EVD will have successful weaning; others may require VPS.
Hydrocephalus

Arachnoid granulations get clogged by subarachnoid blood causing hydrocephalus

CSF Pathways

- CSF is produced by modified ependymal cells in choroid plexus
- It circulates from lateral ventricles into the third ventricle through the foramen of monro.
- It then passes into the fourth ventricle through the narrow cerebral aqueduct.
- From the fourth ventricle, it passes slowly through median aperture (foramen of magendie) and lateral foramina (foramen of luschka) and enters the subarachnoid space over brain and spinal cord.
- It is reabsorbed into venous sinus blood via arachnoid granulations.
Hydrocephalus

NCCT
Massive hyper dense subarachnoid bleed in basal cisterns, Sylvain fissures and hemispheric cortical sulci with hydrocephalus
VASOSPASM – PROPHYLAXIS(?)

- Nimodipine 60 mg PO q 4 hours for 21 days
- Affords neuroprotection without decreasing the frequency of angiographic vasospasm
- Adverse effects: constipation and hypotension
- Maintain euvolemia
  - Hypervolemia has not been shown to improve CBF or decrease frequency of vasospasm/DCI
  - No established methodology as to how to achieve euvolemia
  - Many intensivists use a combination of methods: strict monitoring of fluid balance, central venous pressure, echocardiogram, and stroke volume variation
  - In practice, euvolemia can be achieved by replacing urine output and even administering fludrocortisone or hydrocortisone in patients with significant diuresis
Vasospasm and delayed cerebral ischemia (DCI)

<table>
<thead>
<tr>
<th>Modified Fisher Grade</th>
<th>Risk of Vasospasm</th>
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<tbody>
<tr>
<td>1 - Focal of diffuse thin (&lt;1mm) SAH, no IVH</td>
<td>24%</td>
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<td>3 - Thick (&gt;1mm) SAH, no IVH</td>
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Most likely, the main driver is the release of oxyhemoglobin and erythrocyte contents through hemolysis, which unleashes a host of inflammatory and proapoptotic factors.

The risk of vasospasm increases with:
- Thickness of SAH, density, location, persistence of the SAH
- Poor clinical grade, loss of consciousness at ictus, cigarette smoking, cocaine use, SIRS, hyperglycemia and HCP

However, predicting who will have vasospasm is difficult.
Vasospasm and delayed cerebral ischemia (DCI)

**FIG. 45.3.** The daily percentage probability for the development of symptomatic vasospasm (*solid line*) or rebleeding (*dashed line*) after SAH. Day 0 denotes day of onset of SAH.
Diagnosis of vasospasm/DCI

- Not easy
- Frequent neuro checks, at least q 2 hours
- Suspect DCI if new neurologic impairment of at least 2 points on GCS that lasts for more than 1 hour and cannot be explained by any other cause.

Monitoring: Multimodal and includes
- ICP, CPP, CBF, EEG
- TCD, DSA, CTA, CT perfusion
- MR brain, MRA, MR perfusion
Delayed Cerebral Ischemia (DCI)

- Once the diagnosis of cerebral vasospasm is confirmed (after ruling out other neurologic and systemic disorders) treat with:
  - Induced Hypertension
  - Endovascular therapy

- Goal: Complete resolution of symptoms.

- Management of DCI is carried out in stepwise fashion

- Final confirmation and treatment must be done within 2 hours of symptom onset.
Treatment

- I.v. bolus of NS 1 to 2 liters is administered
- Hypertension is induced with Phenylephrine or Norepinephrine
- Blood pressure augmentation progresses in stepwise fashion with frequent assessment of neurologic function at each 10 mmHg change in systolic (up to 200 mmHg) or MAP
- If neurologic deficits persist then patients undergoes CT/CTA/CTP followed by DSA for endovascular therapy
- Induced hypertension is maintained for at least 72 hours or until stability is achieved and is slowly weaned off after that
Case #1 continued

- Patient continued to evolve satisfactorily with normal Transcranial Doppler (TCD)
- On Postbleed day 6, TCD revealed an increase in Mean Flow Velocity (MFV) in the right MCA from 80 cm/s to 180 cm/s
Case #1 continued

- Patient continued to evolve satisfactorily with normal Transcranial Doppler (TCD).

- On Postbleed day 6, TCD revealed an increase in Mean Flow Velocity (MFV) in the right MCA from 80 cm/s to 180 cm/s.

- The next morning the patient developed sudden onset of left hemiparesis and confusion.

- CT brain showed no rebleeding, cerebral edema or HCP.

- She received a bolus of 500 mL of NS via i.v. and was started on norepinephrine drip with some improvement on her left hemiparesis but without resolution.

- BMP was normal, WBC was 14K.

- F/U TCD showed right MCA MFV of 220 cm/s and a Lindegaard ratio of 6.
CASE #1 CONTINUED
Case #1 continued

- Her SBP was maintained at greater than 180 mmHg for 3 more days.

- TCD showed improvement in MFV by day 9 to less than 100 cm/s; weaned off norepinephrine by day 10.

- On day 11 she developed a decreased level of consciousness without focal neurological findings except for limited upward gaze.

- F/U CT showed HCP requiring an EVD.
Case #1 continued

- Several attempts to wean off the patient from the EVD failed; she underwent a programmable VP shunt placement on day 15.

- She was transferred to a regular floor.

- Discharged to home on day 17 after clearance by physical and occupational therapies with instructions to continue on oral nimodipine to complete 21 days (for four more days).

- Schedule follow-up in vascular neurology and...
Medical complications

- Cardiopulmonary
  - Minor EKG changes to severe dilated cardiomyopathy and ARDS
  - Elevated troponin also frequent – up to 30% of patients with SAH

- Fever
  - Most common non neurologic complication seen in up to 70% of patients
  - Antipyretic medications $\rightarrow$ surface cooling or intravascular devices while avoiding shivering

- Thromboembolism
  - DVT incidence 2% to 20%
  - SCDS for everyone
  - Heparin SQ 24 hours after aneurysm obliteration
Medical complications

- Glucose abnormalities
  - Hyperglycemia is a common phenomenon. Maintain level between 80 to 200 mg/dL
- Hyponatremia:
  - The most common electrolyte disorder seen in up to 30% of SAH patients
  - Hyponatremia has been associated with the development of DCI and poor clinical outcomes
  - Can be secondary to cerebral salt wasting or inappropriate secretion of ADH
  - Traditionally the former is treated with volume infusion and the latter with fluid restriction
  - However, hypovolemia is associated with poor outcomes, so fluid restriction should be avoided in patients with SAH
    - Oral free water restriction and maintain euvolemia
    - Continuous infusion of hypertonic saline (1.5% to 3%)
    - Fludrocortisone if diuresis is active
Conclusion

- SHA is a neurologic emergency with high morbidity and mortality
- SAH is more frequent in women than men
  - More frequent in minority populations
- Main areas when caring for patients with SAH should be the following:
  - Prompt evaluation and diagnosis
  - Immediate transfer to appropriate centers
  - Expeditious diagnosis and treatment of bleeding source
  - Overall good neurocritical care adhering to available treatment guidelines
- Main neurologic complications: HCP, seizures, cerebral edema, delayed cerebral ischemia, and neuroendocrine disorders
- Patients with SAH frequently experience cardiopulmonary complications, which can be life threatening.