Neurosurgical Management of Aneurysmal Subarachnoid Hemorrhage

PVHMC STROKE SYMPOSIUM
2018
10/27/18

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Epidemiology of Subarachnoid Hemorrhage (SAH)

- Less than 10% of all stroke annually
- Acute mortality 25-50%; 30-50% morbidity
- Incidence of aneurysms ~5% general population
- Most SAH traumatic, but of non traumatic, majority due to aneurysm rupture (85%)
Risk Factors and Epidemiology of Aneurysmal SAH

- Females greater than males
- Peak age 55-60
- May be higher amongst blacks
- **Smoking**, hypertension, heavy alcohol use

(Note, these are not the same risk factors for aneurysms development which may include collagen-vascular disease and co-associated conditions such as polycystic kidney disease)
Types of Cerebral aneurysms

- SACCULAR
- FUSIFORM
- DISSECTING
Focal peripontine SAH and possible Vertebral artery dissection
Clinical features

- Sudden, severe headache “thunderclap” headache; Nuchal rigidity; Nausea/vomiting
- Subhyaloid vitreous hemorrhage
- Cranial nerve palsies, particularly IIIrd nerve palsy with PCOM aneurysms or other ocular cranial nerve palsies with giant cavernous carotid artery aneurysms
- Coma, convulsions at onset
Diagnosis

• Computed tomography: hyperdensity in SAS
  – Fisher rating scale:
    • 1: minimal or no blood
    • 2: diffuse thin
    • 3: thick layering in basal cisterns
    • 4: IVH or ICH out of proportion to basal cisternal blood

• Lumbar puncture: xanthochromia, constant red cell count b/w first and last tube

• FLAIR MRI increased signal on T2 FLAIR in SAS
Fisher grade on CT

Fisher 2
Diffuse thin

Fisher 3
Thick cisternal clot

Fisher 4
ICH, IVH > SAH
Clinical Grading Scales

• Hunt Hess grade
  – 1: mild headache*
  – 2: severe headache OR mild headache with cranial nerve palsy (e.g. dilated pupil)
  – 3: lethargy or focal neurological sign
  – 4: stupor
  – 5: coma

• World Federation of Neurological Surgeons (combination of GCS and presence or absence of focal symptoms)

• Occasional reference to H-H grade 0 (unruptured aneurysm), though technically this grading scale applies only to ruptured aneurysms.
Natural history of ruptured aneurysms

• Very high incidence of early rebleeding, upto 50% within first 6 months with 30% of that risk being within first 2 weeks
• Second bleed is often fatal
• “sentinel bleed”: initial asymptomatic or mildly symptomatic bleed followed by subsequent bleed of increased clinical severity
Clinical Management of SAH

Pre-aneurysm repair: Prevent rerupture
- Control BP
- Maintenance fluids
- Avoidance of anticoagulants
- Supportive therapy
- Treatment of hydro if present
- Nimodipine 60 mg q4
- Secure aneurysm

Post aneurysm repair: Prevent/treat VSP
- ‘permissive HTN’
- Fluid augmentation
- +/- pharm. VTE prophylaxis
- Supportive therapy
- Treatment of hydro/VSP if present/occurs
- Nimodipine x 21 days
Deciding on Repair procedure

- Decision based on aneurysm location, morphology, patient comorbidity factors
- Ex: basilar tip $\rightarrow$ endovascular treatment, vs MCA bifurcation $\rightarrow$ surgical clipping
- Wide necked $\rightarrow$ clipping
- Older patients, cardiopulmonary issues, etc $\rightarrow$ endovascular treatment
Clinical trials of aneurysm repair procedures

- **International Surgical Aneurysm Trial (ISAT)**
  - Good grade SAH patients
  - ‘clinical equipoise’
- **Barrow Ruptured Aneurysm Trial (BRAT)**
  - Single center study
  - not blinded
  - Significant crossover from endovasc to surg arm
ISAT

- 2143 patients, 22 UK centers
- 1 year Death/dependency ~24 vs 31% (EVE vs MSC)
- 10 years: 83% coiled pts alive vs 79% clipped; 82% coiled vs 78% clipped pts independent
- Combined OR 1.34, 95% CI, 1.07-1.67
- Slightly higher rate rehemorrhage endo group
Barrow Ruptured Aneurysm Trial

- 700 screened → 500 eligible → 403 eval
- 358 patients underwent treatment (remainder died pre-Tx or angio negative)
- 1 year data: Poor outcome in 33.7% clipped vs 23.2% coiled. OR 1.68, 95% CI 1.08-2.61, p=0.02
- 3 years: fav outcome, 5.8% absolute difference for coiled groups, P=0.25
Aneurysm Repair

ENDOVASCULAR ANEURYSM TREATMENT
Endovascular Aneurysm Treatment

- **Intrasaccular treatments**
  - Coil embolization
  - Liquid embolic embolization

- **Extrasaccular (neck treatments)**
  - Flow diversion
  - Neck coverage
Development of Neurovascular Coils

• Guglielmi & Vinuela, UCLA, 1989-1990
• FDA approved 1995, Target Therapeutics
• Numerous manufacturers since then
• ‘bioactive’ vs bare platinum coils
Other general considerations

- Coils are MRI compatible
- Stents/Flow diverters are MRI compatible
OPEN NEUROSURGICAL ANEURYSM TREATMENT
Efficacy and risk factors of surgery

• More than 90% complete occlusion rate
• Mortality 0-3% in various series
• Morbidity 2-11% quoted in large studies
• Risk of surgery exceeds the 7.5 year risk of bleed in those aneurysm which are <10mm
Factors affecting surgical outcome

• Aneurysm related factors
  • Aneurysm size (>2.5).
  • Location (A comm, ICA bifurcation).
  • Orientation

• Patient related factors
  • Age
  • Ischemic cerebrovascular diseases
  • Diabetes mellitus
RUPTURED ANEURYSMS

- Sixty percent of patients either die or disabled.
- 20-30% rebleed in 30 days.
- 4% rebleed rate on day 1.
- More than 70% who rebleed, die.
- Aneurysm occlusion either surgical or endovascular is the only answer.
Options for definitive treatment

- **Surgery.**
  - Simple Clipping
  - Wrapping
  - Parent vessel occlusion
  - Revascularization procedures

- **Endovascular methods.**
  - Destructive procedures
  - Reconstructive procedures

- **Endoscopy**

- **Conservative**
Timing of surgery:

• Anterior circulation: early surgery has good results compared to late

• Posterior circulations:
  • Easy aneurysms: early surgery
  • Difficult aneurysms: after two weeks

(Haley EC jr et al. the international cooperative study on the timing of aneurysm surgery; the north American experience. Stroke 23:205-214;1992)
Early surgery

- Virtually eliminates re-bleed
- Facilitates treatment of vasospasm
- Allows removal of vasospasmogenic material
- Though operative mortality higher, but overall outcome is better
- Factors favoring early surgery:
  - Good medical condition of patient
  - Good neurologic condition
  - Large clot, blood
  - Early rebleed, multiple episodes
  - Imminent rebleed signs
Disadvantages

- Inflammation and brain edema causes more difficult and traumatic retraction
- Acute clot makes dissection difficult
- Risk of intraoperative rupture is high
- Vessel injury may aggravate vasospasm
- Factors favoring late surgery:
  - Poor medical neurological condition
  - Difficult aneurysms
  - Significant edema on CT
  - Active vasospasm
Technical considerations of aneurysm surgery

Intraoperative objectives

- Prevent rupture
- Further enlargement
- Preserve normal vessels
- Minimize injury to the brain

Intraoperative surgical images of a large intracranial aneurysm (A) successfully treated by placing an aneurysm clip around the neck of the aneurysm (B).
Technical considerations of aneurysm surgery

- Clip too low- may occlude parent vessel
- Distal placement- aneurysmal rest
- Aneurysmal rest expand in future and may rebleed
- Surgical exposure:
  - avoid retraction
  - Brain relaxation- hyperventilation, CSF drainage, lumbar spinal drainage, cisternal drainage
  - drugs
POST ANEURYSM REPAIR CONSIDERATIONS

NEUROLOGICAL SEQUELAE OF SAH AND CLINICAL MANAGEMENT
Brain Injury and Neuro sequelae post SAH

- Ultra-early injury (initial hours)
  - ICP crisis, cerebral circulatory arrest
  - Microthrombosis
  - Cortical spreading ischemia
- Early injury (first few days)
  - Vasospasm
  - Hydrocephalus
- Late/subacute (first 6-12 months)
  - Depression
  - Fatigue
  - Cognitive problems
- Delayed complications (remote from incident event)
  - Normal pressure hydrocephalus
34 M with sudden LOC, L vertebral artery aneurysm
Vasospasm and HHT

- Constriction of blood vessels due to inflammatory effect of blood breakdown products
- Peak incidence days 3-10 post SAH
- Diagnosed by TCD with MFV in major intracranial vessels > 120 cm/sec
- Treated by ‘triple H therapy’ (hypertension, hypervoleemia, hemodilution) or first two arms, intra-arterial vasodilator administration, intracranial angioplasty
HHT

- Many patients ‘auto-hypertense’
- Induced hypertension with vasoactive medications, inotropes
- In extreme cases IABP
- Hypervolemic therapy to maintain CVP > 8 mm Hg with albumin, NS, 3% HTS
- Early move to definitive therapy for refractory cases (no improvement w/in 1 hour of HHT, or despite max HHT)
Magnesium therapy

- Experience with eclampsia patients
- Neuroprotectant (glutamate antagonist)
- 5 g bolus
- MgSO4 2 gm/250 cc NS at 23 cc/hr
Endovascular Tx of VSP

- IA vasodilators: papaverine (very short duration of action), verapamil, nicardipine, NTG
- Intracranial angioplasty more durable, with low or no incidence of recurrence of VSP at angioplastied segment
Global cerebral edema

- More common in poor grade patients
- Extension of white matter hypodensity to cortical surface on HCT
- *May be exacerbated by HHT therapy*
Ischemia in Subarachnoid Hemorrhage

- VASOSPASM
- SILENT CEREBRAL ISCHEMIA
- DELAYED CEREBRAL ISCHEMIA

Acute and delayed ischemic neurological deficit

70% angiographic VSP

30%

?3-20%
Prevention/Treatment of Cerebral Ischemia in SAH

• Detection/treatment of vasospasm
• Nimodipine
• Negative/equivocal trials: clozasentan, cilostazol, dantrolene, albumin, statins, magnesium, intra-thecal nimodipine
POST ANEURYSM REPAIR CONSIDERATIONS

MEDICAL COMPLICATIONS OF SAH
EKG changes in SAH

- T wave inversions
- Diffuse ST elevations or depressions
- (changes that do not respect coronary artery territory)
- Prolonged QT interval
- *Torsade de pointes*
32 M sudden seizure, SAH
Neurogenic stunned myocardium

- Sympathetic surge leads to catecholamine release and global depressed cardiac function
- More common among middle aged women
- Echocardiography may show hypokinesis which does not follow pattern of coronary artery distribution
- May have elevation of cardiac enzymes
- Can be reversible
Differentiating Etiology of Myocardial Dysfunction

**Neurogenic**
- Age < 50
- F > M
- Diffuse/global dysfunction

**Cardiogenic**
- Older
- M > F
- Regional wall motion abnormalities
Neurogenic pulmonary edema

- Can develop within minutes of CNS injury
- Causes: changes in pulmonary capillary hydrostatic pressure and permeability due to catecholamine surge
- Lower PCWP than with non-neurological causes of pulmonary edema

Fever in SAH

- May be more common in poorer grade SAH patients and those with IVH
- Mees et al, *Stroke*, 2008: 42% patients experienced fever, 34% infections
- Fernandez et al, *Neurology*, 2007: for every 1°C over 37, 9x inc risk of death, 3x inc risk disability
- Circuit controlled temperature regulatory mechanisms
- Acetaminophen, NSAIDs, cyproheptadine
Cerebral Salt Wasting

- Sodium derangements (hyponatremia) common initially thought to be due to SIADH, now understood to be far more commonly due to cerebral natriuresis
- Hypervolemic therapy rather than fluid restriction
- Hypertonic saline in refractory cases
- PO salt tabs (not very effective)
- Avoidance of hypotonic fluids
Standard ICU orders for SAH patients (pre-Tx)

- Hourly neurological checks
- A-line, maintenance of BP $\leq 140/90$ mm Hg
- Ventilatory support for lethargic patients
- EVD drainage for lethargic patients, pts with hydrocephalus, EVD at 15 cm H20 to tragus
- Dilantin/leviteracetam or other AED through first few days
Pre-Tx orders ctd

- Nimodipine 60 mg q4 (can give 30 mg q4 for lower BP)
- IVF (NS, no hypotonic fluids)
- Stool softener
- PPI
- Non medical DVT prophylaxis
ICU orders (post Tx)

- Liberalization of BP parameters
- Consider starting SQ heparin for DVT prophylaxis
- Consider lower EVD reservoir level
- Consider D/Cing AEDs at day 7-14 for grades 1 and 2 without intraparenchymal clot
- Steroids not routine, but may alleviate chemical meningitis pain
Differential of Worsening Neurological Status in SAH

- Rebleeding
- Worsening hydrocephalus
- Vasospasm
- Developing edema or other mass effect
- Seizures (up to 30% poor grade patients with SAH may have subclinical status epilepticus)
- Worsening of medical comorbidities/dysfunction
Management of Neurological Decline in SAH

- Treatment of medical comorbidities
- Clinical assessment, pupillary exam, look for clinical signs of herniation, if present treat as for ICP crisis
- Early CT scan
- If rebleeding or concern for VSP, angiography and treatment
- Additional monitoring (EEG)
Management of ICP crisis

- Osmotic therapy (mannitol, hypertonic saline)
- Hyperventilation to PCO$_2$ 35 mm Hg
- Sedation (propofol, extreme cases, barbiturates)
- CSF diversion or other surgical therapy
Predictors of Poor Outcome

• McDonald et al, Toronto, Canada:
  – Worse Hunt and Hess/WFNS scores
  – Leukocytosis at presentation
  – Hemodynamic instability

• Mayer et al, Columbia, NY, NY
  – Hypoxemia, Aa gradient > 125 mm Hg
  – $\text{HCO}_3^- < 20 \text{ mmol/L}$
  – Glucose > 180 mg/dL
  – MAP < 70, > 130 mm Hg
Which Unruptured Aneurysms Should be Treated?

- All newly discovered aneurysms should be referred for further evaluation
- ISUIA: Low annual risk of hemorrhage for anterior circulation aneurysms < 7 mm, and posterior/PCOM < 5 mm
- In patients with prior SAH and multiple aneurysms, higher risk of hemorrhage for small aneurysms compared with other pts
- 2+ Family history → 25% lifetime risk SAH
Common Intracranial Aneurysm Locations

![Diagram of intracranial arteries showing common aneurysm locations.](image)
PVHMC pt Intracranial Aneurysm Locations

- 2, 7.4%
- 8, 29%
- 1, 3.7%
- 4, 14.8%
- 3, 11.1%

Dural AVF: 1, 3.7%
Angio neg: 4, 14.8%
Post circ. 4, 14.8% (1 with assoc. AVM)
Mortality by Intracranial Aneurysm Locations

- 1/2, 3.7/12.5/50%
- 1/8, 3.7/12.5/12.5%
- 1/1, 3.7/12.5/100%
- 1/3, 3.7/12.5/33%
- Post circ. 4/4, 14.8/50/100%

% of total # of SAH patients/% of expired patients/% w/in this ANEU group
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<tr>
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<th>Age</th>
<th>Female</th>
<th>HH 1-2</th>
<th>HH 3-5</th>
<th>misdx</th>
<th>Delay hosp. arr.</th>
<th>&gt;1 aneu</th>
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<tbody>
<tr>
<td>Total</td>
<td>58</td>
<td>16, 59%</td>
<td>8, 29.6%</td>
<td>19, 70.4%</td>
<td>3, 11.1%</td>
<td>9, 33.3%</td>
<td>3, 11%</td>
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<td>Mort.</td>
<td>46</td>
<td>5, 62.5%</td>
<td>1, 12.5%</td>
<td>7, 87.5%</td>
<td>1, 12.5%</td>
<td>4, 50%</td>
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<td>8, 29.6%</td>
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65 F H/A, prior R cerebellar infarction, ?? R VAD, expired SAH day 7, sepsis, cardiopulmonary arrest

- Severe atherosclerosis with dense calcification of R VA
- Inflammatory thrombus of the R VA associated with focal vasculitis
Conclusion

- SAH highly morbid subset of stroke, mortality rates 40%
- Early aneurysm repair
- Treatment of concomitant hydrocephalus
- Close ICU observation during vasospasm period and treatment if it occurs essential
- Treatment of other medical comorbidities