Neurosurgical Management of Stroke
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Overview
• Hemorrhagic Stroke
• Ischemic Stroke
• Aneurysmal Subarachnoid hemorrhage
• Initial management
• In hospital management

Hemorrhagic Stroke
• Loosely defined as sudden loss of brain function due to bleeding
• Presentation, prognosis, cause and hence treatment vary depending on the location of bleeding
• Major Types: epidural, subdural, subarachnoid, intraparenchymal, intraventricular

Intracerebral hemorrhage
• Sudden onset focal neurologic abnormality
• More often associated with HA, nausea, loss of consciousness, acute severe HTN
• Non contrast head CT best and fastest method to distinguish from hemorrhagic stroke from ischemic stroke
Intracerebral hemorrhage

- 10-15% of all strokes
- Potential causes include:
  - Hypertension (majority)
  - Amyloid Angiopathy
  - Arteriovenous malformation (AVM) or dAVF
  - Aneurysm
  - use of sympathomimetic drugs (cocaine, methamphetamine)
  - Vasculitis
  - Moyamoya disease

Hemorrhagic stroke - Hypertension

- Most important and prevalent modifiable risk factor
- 60-70% of spontaneous intracranial hemorrhage
- Brainstem and deep hemispheric locations most common
Hemorrhagic Stroke - Amyloid

- Major cause of lobar hemorrhage
- Amyloid protein in media and adventia of arteries, arterioles, and capillaries
- Increases with age
- 5-8% 60-69 yrs
- 57-58% 90yrs or older
- Associated with Apolipoprotein E

Hemorrhagic stroke - Anticoagulant therapy

- Oral anticoagulant therapy for atrial fibrillation common and growing
- Increases risk of ICH (0.3-3.7%)
- Increases severity and mortality of ICH
- Mortality may be as high as 67%

Hemorrhagic stroke other risk factors

- Heavy alcohol use
- Current tobacco use
- Diabetes
- Prior cerebral infarct 5-22 fold increase
- Hypercholesterolemia
  - Reduced platelet function
  - Fragile vessels
  - Comorbidities
- Family history

Hemorrhagic stroke - AVM

- 0.5/100,000
- Abnormal arteries draining directly into veins without capillary bed
- Mean age at diagnosis 40
- Risk factors:
  - Prior hemorrhage
  - Deep location
  - Deep venous drainage
  - Venous restrictive disease
  - Associated aneurysm
Hemorrhagic stroke - Moyamoya

- Progressive stenosis or occlusion of the circle of Willis arteries
- Numerous neovessels arise “puff of smoke”
- Dilated neovessels form microaneurysms

Moyamoya

- 0.06/100,000 whites
- 0.54/100,000 in Japan
- Women 2x higher than men
- Multiple hemorrhages
- Associated with Down’s syndrome
Treatment of Moyamoya

- Surgical bypass
  - STA-MCA bypass
  - EDAMS (Encephalo-Dural-Arterio-Myo-Synangiosis)
  - EDAS (Encephalo-Dural-Arterio-Synangiosis)

Intracerebral Hemorrhage - Prognosis

- Location of hemorrhage
- Presence of IVH
- Volume of hemorrhage
  - Best predictor of outcome

ICH Grading scale – Hemphill 2001

- Glasgow Coma Scale:
  - 3-4 2
  - 5-12 1
  - 13-15 0
- ICH Volume
  - >30 cc 0
  - <30 cc 2
- Infratentorial
  - Yes 1
  - No 0
- Age
  - >80 1
  - <80 0

More current data?

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<th>GCS (0-15)</th>
<th>Mortality rate, %</th>
<th>Mortality rate 95% confidence intervals</th>
<th>Withdrawal of care, %</th>
<th>ICH score 95% confidence intervals</th>
<th>Surgery rate, %</th>
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*Note: no patient in our series had an ICH score of 6.*
ANEURYSMAL HEMORRHAGE

Cerebral Aneurysms and SAH

- SAH accounts for 3-4% of all strokes (10/100,000)
- Fatality approx 50%
  - 10-15% with sudden death at initial bleed
  - 40% die within 1 month of admission
- 30% of survivors with morbidity
- 6% of the population may harbor an aneurysm(s)
- ½ of SAH in patients younger than 55

Epidemiology

- Modifiable risk factors for SAH:
  - Hypertension, smoking, excessive alcohol (undefined)
    - Possibly methamphetamine, cocaine use
  - Modifiable risk factors double risk of SAH, contribute to 2/3 cases
  - Genetic risk factors important in 10% of cases

Cerebral Aneurysms

Subarachnoid hemorrhage (SAH)

- Typical presentation
- Sudden onset WORST HEADACHE of Life
- Rapidly followed by nausea, vomiting, confusion or coma
- High risk of re-rupture
- Hydrocephalus

Case Illustration

- 36 F with no medical history
- Sudden onset headache, nausea, vomiting after eating dinner
- No neurologic deficits.
Grading Schemes

Hunt Hess Clinical Grade
1. Asymptomatic, mild headache, slight nuchal rigidity
2. Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
3. Drowsiness / confusion, mild focal neurologic deficit
4. Stupor, moderate-severe hemiparesis
5. Coma, decerebrate posturing

Fisher Grade: Appearance of hemorrhage on CT
1. None evident
2. Less than 1 mm thick
3. More than 1 mm thick
4. Any thickness with intraventricular hemorrhage or parenchymal extension

Case Illustration

• 63M with EtOH and tobacco abuse who presented with sudden onset worst headache.
SAH and Vasospasm

- High early mortality followed by delayed morbidity/mortality
- Large vessel spasm with subarachnoid hemorrhage first described in 1951 (Ecker and Rimenschneider) and further elaborated in 1978 by Weir.
- Now termed Delayed Ischemic Neurologic Deficits

SAH and Brain Injury

Hemorrhagic stroke

- Goals of early intervention
  - Establish an airway
  - Blood pressure control
  - Reverse coagulopathy
  - Establish the etiology of the hemorrhage
  - Surgical intervention if indicated

Acute Hypertensive Response

- Dramatic rise with ICH
- Remains elevated for 24 hours
- Current recommendation **SBP<140**
  - Nicardipine, Hydralazine, Labetalol
- Too low BP may exacerbate ischemic injury of adjacent brain

Hemorrhagic Stroke

- ICU care is supportive
- Aimed at preventing secondary injury
- Aggressive ICU care in first 48 hours improves survival and outcomes
- Dedicated stroke unit improves survival and recovery
Evacuation of clot

- Early evacuation associated with slight increase in re-hemorrhage rate
- May shorten hospital stay but has no proven impact on long term outcomes except for mass effect causing further injury
- Surgery aimed at preventing secondary injury

Subarachnoid Hemorrhage

Delayed Ischemic Neurological Deficits

- Develops 5-15 days after SAH
- Risk factors:
  - Fisher Grade
  - Age <68 years (elderly may become symptomatic with little spasm)
  - Smoking, Cocaine use, Hypertension
- Genetics: variations in eNOS system

Delayed Ischemic Neurological Deficits

- Symptoms may be vague: sleepiness, lethargy or stupor, unexplained fever, diuresis
- May be localizing: Hemiparesis, language disturbance, abulia, gaze impairment etc.
- Confounders: poor grade, sedation, etc.

Neuro ICU

- SAH associated with derrangements of multiple brain systems
- Careful monitoring and early intervention critical to preventing infarcts
- Improved patient outcomes with dedicated Neuro-ICU

DIND Therapies

- Nimodipine: Antagonist to L-type Ca Channels
- Only FDA approved tx
- RCT (Picard et al. 1989) demonstrated decreased cerebral infarction and poor outcomes
Hemorrhagic Stroke

- Recovery can take months
- Long-term outcomes are difficult to predict early on in disease course
- Multi-disciplinary rehabilitation important

ISCHEMIC STROKE

Ischemic Stroke

- Time is Brain!
- Time of onset
- Neurologic exam
  - Aphasia, hemiplegia, gaze deviation
  - FAST – Face, Arms, Speech, TIME

Ischemic Stroke – case illustration

- 49F with no medical problems
  - Possibly methamphetamine user
  - Sudden onset left sided hemiplegia, facial droop, right gaze deviation
Ischemic Stroke

• Get the patient to a Stroke center.
  – Primary vs Comprehensive
  – Non contrast head CT to rule out hemorrhage
  – If ischemic, get perfusion imaging
  – Thrombectomy for large vessel occlusion

Conclusion

• Primary goal of treatment is to prevent secondary injury
• Prolonged ICU stay expected
• Many patients can make meaningful recoveries
• Individual recovery can be hard to predict
• SAH sets up complex pathological processes that are poorly understood
• Future studies needed