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Introduction to cerebrovascular diseases
SAH
Subarachnoid haemorrhage

Blood within the CSF

- **Trauma**: most common.
- **Spontaneous**
- Causes:
  - Ruptured berry aneurysms; 75-80%
  - AVM; 4-5%
  - Vasculitis, tumours, carotid dissection, HTN, 3-5%
  - Unknown; 10-15%
- Annual rate: 10-28/100,000
- 10-15% die before reaching the hospital
Some facts

• Among survival rebleeding is the major cause of M&M risk is at 38%
• 8% die from progressive deterioration from the initial bleed
• 7% die from vasospasm
• Another 7% will have severe deficit
• Overall one third will have good results
• 85-95% of aneurysms occur in ICA
• 5-15% in posterior circulation
• 20-30% of aneurysm patients have multiple aneurysms
Incidence of aneurysms are 5% of population
Aneurysm rupture 6-12/ 100 000/ y
Ruptured to unruptured ratio 5:3
Peak age is 55-65
  females more than males 3:2
30% of aneurysmal SAH occurs during sleep
50% of patients have warning symptoms 1-3 weeks before SAH
SAH complicated with ICH 20-40%, IVH in 15-35%
The 30 day Mortality rate is 50-60%
Those who survive initial bleed 6 month mortality rate is 60%
Aetiology

- Congenital predisposition due to defect in the arterial wall
- Atherosclerosis
- HTN presumed to be the most implicated factor for initiation, growth and subsequent rupture due to variable hemodynamic changes
- Infectious or mycotic aneurysm
- Traumatic as in dissecting aneurysms
- Genetics as seen in familial aneurysms is well established such as in PCK. Marfan’s, EDS.
Risk factors

HTN
Diurnal variation in blood pressure
Smoking
Alcohol consumption
Coffee consumption
Oral contraceptives
Drug abuse
Clinical features

- 1-rupture 95-97%
- Sudden onset of severe headache
- Associated with vomiting and kneeling
- LOC
- Focal cranial nerve deficits
- Back pain
- Nuchal rigidity, Kernig's, Brudzinski's sign
- Sentinel haemorrhage causes warning headache
Continued

• *2- mass effect 2-3%*

• Giant aneurysm as any mass lesion

• Compression of neural structure 3ed CN as in P-com
CONTINUED

3- Incidental finding
Natural history of ruptured aneurysm if treated conservatively: studies in 60’s

- Of 100 patients, 15% die before reaching hospital.
- 15% die in first 24 hours in hospital.
- 15% die between 1-14 days.
- 15% die between 2-8 weeks.
- 15% die between 2-24 months.
- 25% may survive > 2 years.
Evaluation

• Non contrast high resolution CT scan is positive in 95-98%
• Ct can also assess the following
  • Hydrocephalus which occurs in 21%
  • ICH
  • Infarction
  • Amount of blood in cisterns which is a prognosticato in vasospasm
• Ct may predict the aneurysm location
continued

CSF
- in questionable cases
- Pressure is elevated
- 3 tube test
- Xanthochromia after 6 hours

MRI
- is not sensitive acutely
- May be helpful after 4-10 days
Angiogram DSA

- Demonstrate the cause of the SAH usually aneurysm in 85-95%
- Study the 4 vessels to rule out additional aneurysms and collateral circulation
- 3 views for each vessel
- Obtain 3-D views for aneurysm to assess the actual size, shape and the N:F ratio
Grading SAH

• **Hunt & Hess**
  • 0: unruptured
  • 1: asymptomatic, mild headache, slight nuchal rigidity
  • 2: Cr N palsy, severe headache & nuchal rigidity
  • 3: focal deficit, lethargy or confusion
  • 4: stupor, hemiparesis, decerebrate
  • 5: deep coma moribund appearance
## WFNS Grading

<table>
<thead>
<tr>
<th>WFNS grade</th>
<th>GCS Score</th>
<th>Major deficit</th>
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<tr>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1</td>
<td>15</td>
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</tr>
<tr>
<td>2</td>
<td>13-14</td>
<td>absent</td>
</tr>
<tr>
<td>3</td>
<td>13-14</td>
<td>present</td>
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<tr>
<td>4</td>
<td>7-12</td>
<td>Present or absent</td>
</tr>
<tr>
<td>5</td>
<td>3-6</td>
<td>Present or absent</td>
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Fisher grading system

correlates between blood on CT and the risk of vasospasm

<table>
<thead>
<tr>
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<th>Description</th>
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<tr>
<td>1</td>
<td>No blood detected</td>
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<tr>
<td>2</td>
<td>Diffuse &lt; 1 mm thick</td>
</tr>
<tr>
<td>3</td>
<td>Localized clot or and &gt; 1mm</td>
</tr>
<tr>
<td>4</td>
<td>ICH, IVH</td>
</tr>
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</table>
Initial management

• Once SAH is documented admit to ICU
• Arterial & Venous catheters, bloods
• Intubation if necessary
• EVD ?
• VS with crani checks q 1 hr
• BR, Head up 30, foley
• I’S & O’s
• 100-125 cc/h N/S vs. D/W+ 20 meq kcl
continued

- **Medications**
- Codeine phosphate 30-60 mg q3h
- Stool softeners, H2 blockers
- Ca channel blockers Nimodipine 60mg q 4h PO **OR** 0.2 mg/kg/h IV
- Dexamethasone may help with headache and neck pain
- Prophylactic anticonvulsant usually phenytoin is controversial
Blood pressure volume management

- **Unsecured aneurysm:**
  - gentle volume expansion & hemodilution may help prevent vasospasm
  - HTN must be avoided  keep at 120-150

- **Clipped aneurysm**
  - Aggressive 3 H treatment is recommended
  - Keep SBP at 140-160  may be as high as 180
  - Hyponatremia is common
  - Over hydration or SIADH vs CSWS
REBLEEDING

• 1\textsuperscript{st} day 4%, then 1.5 % /d
• Mortality rate is 70%
• 15-20% within 2 w
• 50% will bleed within 6 m
• 3% will bleed annually with 2% mortality
• 50% of deaths occur in the 1\textsuperscript{st} m
• Early surgery prevents rebleeding
Vasospasm

- It is also called cerebral angiopathy or DIND.
- First introduced in 1951 by Ecker.
- Commonly seen after aneurysmal SAH but may occur with trauma.
- Never before day 3 post SAH peak day 5-7.
- The most significant cause of M&M in patient surviving SAH long enough to reach medical care.
- Mortality rate is 7%, severe morbidity 7%.
- Blood clot is spasmogenic when indirect contact with the proximal 9 cm of ACA, MCA.
- The high grade the high risk.
- The more blood on CT scan the more risk.
continued

• Clinical vasospasm 20-30% of patient symptoms usually develop gradually
• Criteria: increased symptoms headache and lethargy, new focal deficits, or hyponatremia
• ACA > MCA
• Radiological vasospasm 30-70%. Arterial narrowing demonstrated on angiogram with slowing of contrast filling
Pathogenesis

• Poorly understood
• Proposed mechanisms
• Contraction of the smooth muscle as a result of the vasoconstrictors or vasoactive substances released into the CSF
• Neuronal mechanism via nervi vasorum as a result of sympathetic hyperactivity
• Impairment of endothelial derived relaxant facto
• Mechanical phenomenon
• Components implicated: oxyhemoglobin, iron, noradrenalin, thromboxane-A2 and free radicals
Management for vasospasm

- Non contrast CT scan to rule out hydrocephalus, oedema, infarction or rebleed
- Electrolytes and ABGs
- Angiogram?, TCD?
- Numerous treatment have been proposed medical and invasive
- Calcium channel blockers Nimodipine vs Nicardpine for 21 days
- May improve the outcome, more beneficial in neuroprotection than in preventing vasospasm
- 3 H Protocol
- Dexamethasone
- Balloon angioplasty
- Intra-arterial papaverine
- ICP monitor
Treatment options

- Best treatment depends the patient’s condition, anatomy of the aneurysm, ability of the surgeon

- **Clipping of the aneurysm** at the neck to exclude it from the circulation is considered the optimal treatment

- **Goal of surgery to prevent rupture** or further enlargement while preserving all normal vessels and minimizing injury to brain
Alternatives:

- Wrapping
- Trapping
- Hunterian ligation
- Endovascular techniques
- GDC
- Intra-aneurysmal placement of detachable balloon
Coiling
coiling
Decision regarding the management by GDC vs surgical repair

- **Unruptured** aneurysm are not to be coiled as the long term efficacy has yet to be established
- **Ruptured** aneurysm
  - Following aneurysms are considered for GDC
  - Posterior circulation (except PICA and BASA pointing up), A com.
  - If the N:F ratio <2:1 or neck is >4mm those are not for GDC
  - Multiple aneurysms which can be dealt with same approach
Timing of surgery

• Controversy exists between what so called Early surgery within first 3 days and late surgery after 10 days.
• Early is advocated for following reasons
• Reduce the risk of rebleeding
• Facilitate treatment of vasospasm
• May remove potentially vasospasomgenic agents
• Factors favoring early
• Good medical and neurologic condition
• Associated ICH
• Rebleeding or imminent rebleeding
CONTINUED

- Against early brain is red and swollen,
- may increase vasospasm, high incidence of rebleeding
- Factors favoring late
- Poor condition
- Difficult aneurysm because of site and size
FOLLOW UP

• Patients should be followed up on regular basis as follows 3, 6, 12, 24, 36 months
• Investigation of asymptomatic first degree siblings is recommended
• Genetic screen is under evaluation
Giant aneurysms

- Less than 1 cm is small
- 1-2.5 large
- More than 2.5 is giant
- Saccular and fusiform
- 3-5% of all aneurysms
- Peak 30-60  F: M 3:1
- 35% present with bleeding
- The rest present with TIAs or seizures or mass effect
- Angiogram often underestimates the actual size because of the thrombus
- CT, MRI with and without contrast are more informative
- Treatment options are variable:
  - direct clipping is the ideal if applicable, clipping with EC-IC ICA bypass, trapping, ligation
Vascular malformation

• 4 types described by McCormick
• Arterio-venous malformation
• Cavernous angioma
• Venous angioma
• Capillary telangiectasia
AVM
AVM

Definition:

A collection of blood vessels wherein arterial blood flows directly into draining veins without normal interposed capillary beds
- Appears as a tangle of vessels
- A congenital lesion progresses from low flow at birth to high flow in adult hood

Presentation
- **Bleeding** 50%, mainly from small lesions peak age 15-20 y, mortality is 10% from each bleeding, morbidity is 30-50%, risk of bleeding is 4% per year
- **Seizures** more frequent with large AVM
- **Mass effect**
- Headache, bruises, heart failure, hydrocephalus as seen in vein of Galen aneurysm
- 7% of AVM patients have aneurysms
Evaluation

- CT flickers of calcifications
- MRI flow void on T1,T2, feeding arteries, and draining veins, ischemic changes, size and site, better for cavernous angiomas
- Angiogram reveals tangle of vessels (nidus), feeders and draining veins
- Can not show up cavernous angiomas
### Spetzler and Martin grading system

<table>
<thead>
<tr>
<th>GRADED FEATURE</th>
<th>POINTS</th>
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<tbody>
<tr>
<td><strong>Size</strong></td>
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<tr>
<td>Small &lt;3cm</td>
<td>1</td>
</tr>
<tr>
<td>Medium 3-6cm</td>
<td>2</td>
</tr>
<tr>
<td>large &gt;6</td>
<td>3</td>
</tr>
<tr>
<td><strong>Eloquence of adjacent brain</strong></td>
<td></td>
</tr>
<tr>
<td>Non eloquent</td>
<td>0</td>
</tr>
<tr>
<td>eloquent</td>
<td>1</td>
</tr>
<tr>
<td><strong>Pattern of venous drainage</strong></td>
<td></td>
</tr>
<tr>
<td>Superficial</td>
<td>0</td>
</tr>
<tr>
<td>deep</td>
<td>1</td>
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</table>
Treatment

• Excision is the treatment of choice
• Adjunctive treatment may help includes conventional radiotherapy, radiosurgery and embolization
• Pre op consider B- blockers dexamethasone, and phenytoin
Cavernous angiomas

• Sinusoidal vascular channels located within the brain but lacking intervening neural parenchyma, feeders and draining veins
• usually present with seizures, bleeding, mass effect or incidental
• CT, MRI show the lesion
• Symptomatic lesion should be excised