ENDOVASCULAR TREATMENT OF ARTERIOVENOUS MALFORMATIONS

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NEUROVASCULAR NP SANTA BARBARA COTTAGE HOSPITAL
ETIOLOGY OF AVM

- Prevalence is 15-18 per 100,000.

- Autopsy data suggests as few as 12% are symptomatic during a life time.

- Congenital vascular lesion consisting of abnormal direct connections between arterial and venous systems.
DIFFERENT TYPES OF VASCULAR MALFORMATIONS
INCIDENCE

OF DETECTION

• Data from the New York islands AVM hemorrhage study suggests that the detection rate is 1.21/100,000 person-years.

OF HEMORRHAGE

• The incidence of AVM hemorrhage is 0.42/100,000 person-years.

• Data from the Manhattan stroke study suggests the incidence for first ever hemorrhage is 0.55/100,000 person-years.
BLEEDING AND REBLEEDING

- AVM hemorrhage approximately 2%-4% per year.
- 1\textsuperscript{st} episode associated with mortality rate of 10% increases to 15% with 2\textsuperscript{nd} hemorrhage and 20% 3rd.
- Rate of rehemorrhage increases to 6% 1\textsuperscript{st} year, returns to 2%-4% per year thereafter.
- Grade IV and V have a lower rate of hemorrhage about 1.5% per year.
Typically diagnosed before the age of 40. Most common presentation is hemorrhage >50%.

Second is Seizure 20-25%.

Less common is headaches 15%.
IMAGING

- Head CT, head and neck CTA, first and best studies in a hemorrhagic AVM emergent setting.

- MRI, not the first choice secondary to the time required to obtain images, however MRI will provide superior images of surrounding brain structures.
Angiography gold standard for diagnosis, treatment planning and follow up after treatment.
Cerebral angiography helps to evaluate nidus size and both arterial and venous flow patterns.
Cerebral angiography evaluates the feeder arteries and veins.

High blood flow and shunting of high pressure arterial blood causes the feeder arteries and veins to dilate.

Weakened veins are susceptible to hemorrhage; feeder arteries become susceptible to aneurysms.
<table>
<thead>
<tr>
<th>GRADED FEATURE</th>
<th>POINTS ASSIGNED</th>
</tr>
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<tbody>
<tr>
<td>Size</td>
<td></td>
</tr>
<tr>
<td>Small</td>
<td>1</td>
</tr>
<tr>
<td>Medium</td>
<td>2</td>
</tr>
<tr>
<td>Large</td>
<td>3</td>
</tr>
<tr>
<td>Eloquence of Adjacent brain</td>
<td></td>
</tr>
<tr>
<td>Non-eloquent</td>
<td>0</td>
</tr>
<tr>
<td>Eloquent</td>
<td>1</td>
</tr>
<tr>
<td>Pattern of Venous Drainage</td>
<td></td>
</tr>
<tr>
<td>Superficial</td>
<td>0</td>
</tr>
<tr>
<td>Deep</td>
<td>1</td>
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GOAL OF AVM TREATMENT

• Eliminate the risk of hemorrhage and all related symptoms. Such as seizures and neurologic deficits.

• Without any or with only minimal morbidity or mortality to the patient.
TREATMENT OF AVMS

- SURGICAL RESECTION
- EMBOLIZATION
- RADIATEGGERY
- COMBINATION THERAPY
Grade I, II, III AVMs have a low treatment-associated morbidity.

Grade IV AVMs have a 31.2% treatment-associated morbidity and the rate of permanent deficit is 29.9%.

Grade V AVMs have a treatment-associated morbidity of 50%.
**EMBOLIZATION**

- **Complete obliteration** inversely proportional to AVM volume and the number of feeding arteries.

- **Achieved** by using a nonbiodegradable agent to occlude the AVM nidus.

- **Confirmed** by both immediate angiogram and follow-up angiography.
Complete obliteration of AVMs by using embolization as the sole method of treatment is between 5% and 40%.

These AVMs are usually smaller than 3 cm in diameter and have only one or two feeding arteries.
TRUFILL® n-BCA
N-Butyl-Cyanoacrylate
High viscosity, long term efficacy
Mixed with ethiodized oil to visualize
Rheology can be controlled
Permanent occlusion

ONYX
Dimethyl sulfoxide solvent
Ethylene-vinyl alcohol, tantalum
Non-adhesive but cohesive
Longer injection time
More control over polymerization
AVMs with a nidal volumes less than 10ml are frequently curable by radiosurgery with complete obliteration at 2yrs about 80%-88%.

Hemorrhage risk remains during this time or can increase up to 11-16% during the first 6 months.
Efficacy of embolization followed by XRT not fully established

X-RT: 1 - 3 years to obliterate AVM, bleeding rate 3.7% / year
The goal of combination therapy is to maximize the benefits of each option and decrease the overall morbidity and mortality of AVM treatment.
Neuro-Angio Suite

- Biplanar Angio: Siemens, Philips, GE
- 3D visualization technology
- Flat panel technology with Dyna CT
- Neuroanesthesiology
- Specialized technologists & nurses
BENEFITS OF EMBOLIZATION BEFORE MICROSURGERY AND RADIOSURGERY

<table>
<thead>
<tr>
<th>MICROSURGERY</th>
<th>RADIOSURGERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Occludes the deep and surgically inaccessible feeding arteries.</td>
<td>• Decreases the size so radiosurgery can be performed.</td>
</tr>
<tr>
<td>• Decreases blood flow and nidal size, less blood loss during surgery.</td>
<td>• Treat AVM-associated aneurysms prior to radiosurgery which may not be effective for the aneurysms.</td>
</tr>
<tr>
<td>• Onyx or n-BCA in arteries provides a good road map.</td>
<td>• Smaller residual AVMs have a higher radiosurgical cure rate.</td>
</tr>
<tr>
<td>• AVM associated aneurysms treated prior to surgical resection.</td>
<td></td>
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AVM PRE EMBO
AVM POST EMBO and after resection.
M 15, left fronto-parieto-occipital AVM, no hemorrhage

Flow directed catheters, NBCA, Onyx

S/P Embolization (x 2)  Final Results: Surgery/Embo
29M, grand mal seizure, Spetzler grade IV AVM, left parieto-occipital
X 3 Onyx/NBCA embolizations
Surgical resection of AVM (15 hour surgery)
CONCLUSION

• Endovascular procedures have changed how AVMs are treated.
• Embolization has made surgical resection safer and radiosurgery possible for larger vascular lesions.
• Grade III and IV AVMs can be completely obliterated with combination therapy.
Neuroendovascular Treatment of Cerebral Aneurysms

Emily Rorden MSN, RN, ACNP-BC, CNRN, SCRN, CCRN
• Aneurysm rupture can occur at any age
• Most commonly in the 5th decade
• Incidence approximately 10 in 10,000 a year
• Arise more commonly in anterior cerebral circulation
Circle of Willis
Saccular Aneurysm

- 85-95% Carotid system
  - 30% Anterior Communicating Artery (single most common)
  - 25% Posterior Communicating Artery
  - 20% Middle Cerebral Artery
Saccular Aneurysm

- 5-15% Posterior Circulation
  - 10% Basilar artery
  - 5% Vertebral artery

- 20-30% have multiple aneurysms
• Smoking
• Hypertension
• Atherosclerosis
• Connective tissue disorders
• Alcohol
• Hemodynamic stresses
• Illicit drugs
• Genetics/Family History

Risk factors for Aneurysm Formation
Diagnosis

- **Unruptured**
  - Chronic headaches
  - Third nerve palsy
  - Visual loss
  - Ill-defined neurological disorder
- **Ruptured**
  - Acute onset of headache
  - Meningismus
  - Photophobia
  - Nausea and Vomiting
## 5 Year Rupture Risk

<table>
<thead>
<tr>
<th>Location</th>
<th>&lt;7mm no hx of SAH</th>
<th>&lt;7 mm w/hx of SAH</th>
<th>7-12 mm</th>
<th>13-24 mm</th>
<th>25 mm+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cavernous</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>6.5</td>
</tr>
<tr>
<td>AC/MC/IC</td>
<td>0</td>
<td>1.5</td>
<td>2.5</td>
<td>14.5</td>
<td>40</td>
</tr>
<tr>
<td>Post, Pcomm</td>
<td>2.5</td>
<td>3.5</td>
<td>14.5</td>
<td>18.5</td>
<td>50</td>
</tr>
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</table>
• Aneurysmal SAH accounts for 6-8% of all stroke

• Incidence of aSAH 8-10 cases per 100,000 annually

• Risk of aneurysm rupture and aSAH is positively correlated with:
  ▫ Aneurysm size
  ▫ Hypertension
  ▫ Smoking

**Subarachnoid Hemorrhage**
Mortality and Morbidity

- Approximately 10-15% aSAH die before obtaining medical attention
- For those who survive another 30-60% will die because of the initial hemorrhage or secondary sequelae
- Thirty day mortality is approximately 50%

Mortality and Morbidity
<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Asymptomatic or mild H/A and slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Cr. N palsy, moderate to severe H/A, nuchal rigidity</td>
</tr>
<tr>
<td>3</td>
<td>Mild focal deficit, lethargy, or confusion</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis, early decerebrate rigidity</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, moribund appearance</td>
</tr>
</tbody>
</table>

*Add one grade for serious systemic disease (e.g. HTN, DM, severe atherosclerosis, COPD) or severe vasospasm on arteriography*
• Admission Hunt and Hess Grade I or II 20%

• Patient taken to O.R. (for any procedure) at H&H Grade I or II 14%

• Major cause of death in Grade I or II is rebleed

• Signs of meningeal irritation increases surgical risk

Hunt & Hess Grade Mortality
<table>
<thead>
<tr>
<th>Coiling</th>
<th>Clipping</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Elderly Patients</td>
<td>• Younger age</td>
</tr>
<tr>
<td>• Poor clinical grade</td>
<td>• MCA bifurcation aneurysms</td>
</tr>
<tr>
<td>• Inaccessible ruptured aneurysms</td>
<td>• Giant aneurysm</td>
</tr>
<tr>
<td>• Aneurysm configuration</td>
<td>• Symptoms due to mass effect</td>
</tr>
<tr>
<td>• Patients on Plavix</td>
<td>• Small aneurysm</td>
</tr>
<tr>
<td>• Technically difficult to clip</td>
<td>• Wide aneurysm neck</td>
</tr>
</tbody>
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Coil Embolization Procedure

- Vascular access via the femoral artery
- Vessel is engaged with guide catheter
- Several Techniques
  - Stent-assisted coiling
  - Balloon-Assisted coiling
  - Flow diverting stents
Coil Embolization

• Goal is to occlude the fundus of the aneurysm completely

• Maintain the patency of the parent vessel

• The ability to embolize an aneurysm fully is largely determined by its anatomy
Coil Embolization
Stent Assisted Embolization

- Stent is deployed across the aneurysm neck
- Deployment of coils through the stent
Stent Assisted Embolization

- Advantage: stent serves as a permanent buttress for the coil mass

- Disadvantage: long term use of antiplatelet agents and risk of in stent stenosis
• Inflation of a balloon in the parent vessel while coils are deployed within the aneurysm

• Once the coil or coils have been deployed, the balloon is deflated

• This technique is especially useful for treating ruptured wide-necked aneurysms
Balloon Assisted Embolization

• Advantage:
  ▫ No antiplatelet agents are required

• Disadvantage:
  ▫ ischemic complications
  ▫ Dissection
Flow Diversion

- The newest technologies for aneurysm embolization
- Primarily used for wide necked giant aneurysm
- Need long term antiplatelet therapy
Flow Diversion
Potential Complications

- **Aneurysm Rupture**
  - Uncommon < 1%

- **Aneurysm Recurrence**
  - As high as 20% with bare platinum coils

- **Thromboembolic Complications**
  - Stroke is the biggest risk in non ruptured aneurysm

- **Cranial Neuropathies**
  - Neuropathies may worsen after embolization from further compression of the CN by the coil mass

Potential Complications
• Admission to a neuroscience observation unit or ICU
• Hourly neurological exams, vital signs, and arteriotomy site checks
• Keep access site immobilized
• Resume diet
• Adequate hydration to improve renal clearance of dye load
• Check hematocrit and creatinine level next day
Postprocedural Care: Ruptured

- Admission to ICU
- Hourly neurological, vital signs, and arterial line monitoring
- EVD when hydrocephalus is present or ICP/Licox monitoring when needed
- Strict monitoring of fluid balance
- Daily Transcranial doppler to assess vasospasm
- Echocardiogram and EKG
- Head CT
• There are no established guidelines for follow up imaging

• Cerebral angiogram or MRA are more useful imaging studies

• Suggested follow up Imaging from time of coiling
  ▫ 3, 9, 15, 24, 36, 48, 60 (months)
71F, SAH, H&H 2, Fisher # 3, IVH, R-Pcom and L-SHA Aneurysms, Coil embo R-Pcom, Cordis Orbit coils.
Conclusion

• Cerebral aneurysms are complex

• A craniotomy is no longer the only treatment option

• Endovascular surgery is definitely the better treatment option in those with high surgical risks
THANK YOU