ARTERIOVENOUS MALFORMATION

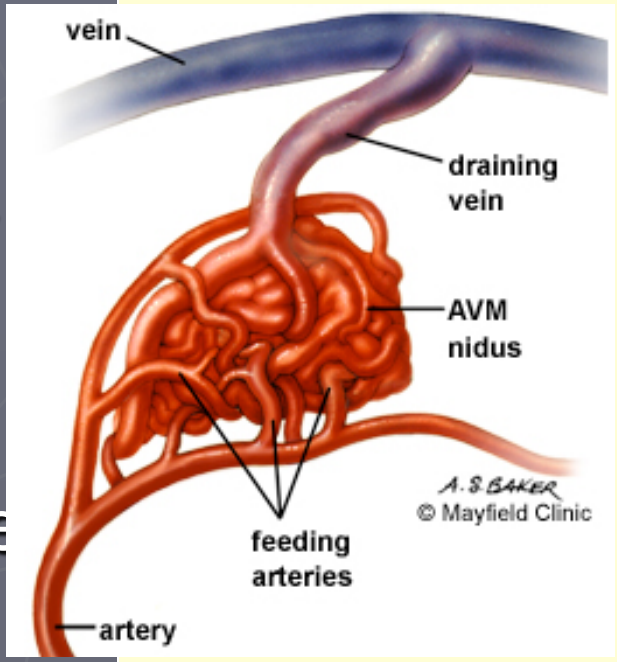
AVM-Introduction

Vascular malformation:

- AVM
- Venous malformation
- Cavernous malformation
- Capillary telangiectasia
- AVF

AVM-introduction

- Most dangerous vascular malformation
- Congenital lesion
- Abnormal collection of vessels wherein arterial blood flows directly into draining veins without the normal capillary beds
- Feeding arteries/ Nidus/ Draining ve
- Static/ Grow/ Regress



AVM-Presentation

- Hemorrhage(50%)
- Seizure
- Mass effect
- Ischemia; steal phenomenon
- Headache
- Bruit
- HCP
- Peds: hydrocephalus, heart failure

AVM-Hemorrhage

- Peak age: 15-20 y/o
- 10 % mortality; 30-50% morbidity
- ICH(80%)/IVH/SAH
- Risk of hemorrhage:

  High feeding a. pressure/V. outflow obstruction/Size/Location/Aneurysm/ Pregnancy

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Hemorrhage related to AVM size

- Small AVMs are more lethal than larger ones
- Small AVMs tends to present more often as hemorrhage than do larger ones [1]
- Small AVMs are thought to have much higher pressure in feeding artery [1, 2]

1. Crawford P M, West C R, et al: Arteriovenous Malformation: Natural History in Unoperated Patients. **J Neurol Neurosuurg Psy** 49:1-10,1986
2. Spetzler R F, Hargraves R W, et al: Relationship of Perfusion Pressure and Size to Risk of Hemorrhage from Arteriovenous Malformations. **Neurosurgery** 37: 851-5, 1995

Annual & Lifetime risk of Hemorrhage

- Lifelong risk of bleeding: 2-4% per yr
- A study of 166 symptomatic AVMs with 24 year follow-up found the risk of major bleeding was constant at 4% per year, independent of whether the AVM presented with or without hemorrhage [3]
- The AVM Study Group:

  Annual rate of rehemorrhage was 18% among pts who had hemorrhage at presentation; 2% among pts with no history of bleeding (306 cases) [4]
- Rebleeding rate significantly lower than aneurysms.

3. Ondra SL, Troupp H, et al: The natural history of symptomatic cerebral arteriovenous malformation: A 24-year follow-up assessment. **J Neurosurg** 25:387-91, 1990

AVMs & Associated Aneurysms

- 7% of pts with AVMs have aneurysms
- 75% are located on major feeding artery; probably from increased flow [1]
- The symptomatic one is treated first
- Although 66% of related aneurysms will regress following AVM removal, this does not always occur [4]

4. Cunha M J, Stein B M, et al: The Treatment of Associated Intracranial Aneurysm and Arteriovenous Malformations. **J Neurosurg** 77: 853-9, 1992.

Hemodynamic Effects of AVM

Pre-op effects:

- Steal phenomenon
- AVM & aneurysm
- High-flow angiopathy [7]

Post-op effects:

- Normal perfusion pressure breakthrough
- Occlusive hyperemia

7. Pile Spellman JM, Baker KF, et al: High flow angiopathy: cerebral blood vessel changes in chronic arteriovenous malformation. Am J Neuroradiol 1986; 7:811-5

Cerebral Steal Phenomenon

- Autoregulation curve shifts to left
- Despite cerebral arterial hypotension, focal neurological deficits are rare(<10%)
- More likely to be local mass effect

Normal perfusion pressure breakthrough (NPPB)

- Peri-/Post-op swelling or hemorrhage
- Loss of autoregulation[4] ?[5]
- Less than 5%
- Should be diagnosis of exclusion
- Mx: prevent post-op hypertension

4. Spetzler R F, Wilson C B, et al: Normal perfusion breakthrough theory. **Clin Neurosurg** 25: 651-72, 1978

5. Young W L, Kader A, et al: Pressure autoregulation is intact after arteriovenous malformation resection. **Neurosurgery** 32: 491-7, 1993

Evaluation-MRI

- Flow void on T1WI
  or T2WI
- Feeding arteries
- Nidus
- Draining veins

Evaluation-Angiography

- Tangle of vessels
- Large feeding artery
- Large draining veins
- Not all AVMs show up on angiography!

  Angiographically occult vascular malformation (AOVM)

Evaluation-Grading

- Spetzler-Martin grade
- Outcome based on Spetzler-Martin grade:

  100 consecutive cases operated by Spetzler

TABLE 1. THE SPETZLER–MARTIN SCALE FOR EVALUATING THE RISK OF SURGERY IN PATIENTS WITH ARTERIOVENOUS MALFORMATIONS.*

CHARACTERISTIC	NO. OF POINTS ASSIGNED
Size of lesion	
Small (maximal diameter, <3 cm)	1
Medium (maximal diameter, 3–6 cm)	2
Large (maximal diameter, >6 cm)	3
Location	
Noneloquent site	0
Sensorimotor, language, or visual cortex; hypothalamus or thalamus; internal capsule; brain stem; cerebellar peduncles; or cerebellar nuclei	1
Pattern of venous drainage	
Superficial only	0
Any deep	1

*A score of 4 or 5 is associated with the highest risk of persistent neurologic deficits after surgery. Data are from Spetzler and Martin.[60]

Treatment

- Multidisciplinary approach
- Primary goal: decrease the risk of bleeding

1) Surgery: mainstay
2) Stereotactic Radiosurgery (SRS):
   high-risk for surgery
3) TAE: adjunct to 1) & 2)

Surgery

American Stroke Association recommends:

- Low grade (I & II)- surgery alone
- Higher grade(>III)-TAE before surgery

- Eliminates risk of bleeding immediately, seizure controls improves
- Invasive, risk of surgery

Surgery

- Pre-op propranolol 20mg po QIDx3d to minimize post-op normal perfusion pressure breakthrough (NPPB)
- Peri-op labetalol to keep MAP 70-80mmHg

Surgery

- Craniotomy
- Dural opening
- Identify the borders
- Cautery of feeding arteries

Surgery

- Deep dissection of the nidus
- Securing the ventricle
- Obliterate the draining veins
- Final removal of AVM
- Post-resection BP challenge

  Hemostasis/ Residual nidus/ Areas prone to NPPB
- Immediate post-op/ Peri-op angiography

Intra-Op Complication

- Premature division of venous drainage
- Extensive bleeding along the deep margin
- Post-resection NPPB/ Residual AVM

- Pack the wall with Avitene & Gelfoam
- Immediate removal of the entire AVM

Post-Op Complications

- Subgaleal fluid collection
- Sterile meningitis
- Wound infection
- Intracerebral hematoma

Post-op Deterioration

- Normal Perfusion Pressure Breakthrough [4]
  post-op swelling or hemorrhage
  loss of autoregulation[4] ?[5]
  Mx: prevent post-op hypertension
- Occlusive Hyperemia [6]
  immediate: obstruction of venous outflow
  delayed: venous or sinus thrombosis
  Mx: adequate post-op hydration
- Rebleeding from a retained nidus
- Seizures

Radiation treatment

- Conventional radiation:
  effective in< 20% of cases
- SRS: for small (Nidus<3cm) & deep AVMs
- Radiation-induced endothelial cell proliferation→Obliteration, thrombosis
- Gamma knife/ Linac

- Non-invasive, gradual reduction of flow
- Takes 1-3 yrs to work, limited to small lesion

Endovascular Approach (TAE)

- Op inaccessible deep or dural feeding a.
- Usually inadequate if used alone for AVM; may recanalize

- Facilitates OP (less bleeding) & possibly SRS
- Can't be used alone, acute hemodynamic change, multiple procedures

Endovascular Approach (TAE)

- Glue: N-butyl cyanoacrylate (nBCA), Lipiodol,

  tantalum powder, D5W
- Embolization of the nidus through the feeders without any significant glue entering the draining veins
- In general, only 2-3 vessels are embolized per session.

Endovascular Approach (TAE)

- Anesthesia: MAC/ GA
- Induced hypotension with vasoactive agents, general anesthesia, or even brief adenosine-induced cardiac pause at the time of embolization to allows the glue to set
- Provocation test:

  Sodium amytal & cardiac lidocaine injection to determine that embolization will not result in neurologic deficit

Anesthesia-related Considerations for Cerebral AVMs

- Extensive blood loss
- Pharmacological brain protection
- Non-pharmacological brain protection

Anesthesia-related considerations for cerebral arteriovenous malformations
Hashimoto T, Young W L, et al
Departments of Anesthesia and Perioperative Care, Neurosurgery, and Neurology, Center for Cerebrovascular Research, UCSF
Neurosurg Focus 11 (5): Article 5, 2001

Monitor

- EKG/SpO_2/$ETCO_2$/BT/CVP
- Measurement of vascular pressure

  differentiate a. from v.

  decision of whether a vein can be sacrificed

Anesthetic Technique
Choice of Agents

- Avoid cerebral vasodilators!!!
- General condition
- Isoflurane/N_20
- Additional Barbiturate loading
- Metabolic suppression- propofol, etomidate

Brain Relaxation

- Good head position
- CSF drainage
- Diuretics/Osmotherapy
- Avoid excessive cerebral vasodilator!!!
- Modest hypocapnia with hyperventilation

Euvolemia & Pressure Control

- Euvolemia
- Optimal cerebral perfusion pressure

Induced Hypotension

- Aneurysm/ AVM
- Large AVMs with deep a. supply
- Barbiturate therapy

Fluid and Electrolyte Management

- Isotonicity

  Stable cardiovascular status

  Prevention of cerebral edema

  Aggressive isotonic crystalloids may worsen brain edema by decreasing colloid oncotic pressure. [6]
- Euglycemia

  less than 200mg/dl

6. Drummond JC, Patel PM, et al: The effect of the reduction of colloid oncotic pressure, with and without reduction of osmolarity, on post-traumatic cerebral edema. **Anesthesiology** 88:993-1002,1998

Toleration of Modest Hypothermia

- Mild hypothermia(34-35° C);
  cerebral protection
- SE: drug metabolism
  increased rate of myocardial ischemia
  infection
  arrhythmia
  coagulopathy

Emergence & Recovery

- Post-resection BP challenge;
  Hemostasis/ Residual nidus/ Areas prone to NPPB
- BP control: most important
- NE

Postoperative Management

- BP control

  SBP< 120mmHg x 2d
- BT control

Any Comment or Question?


Thanks for Your Attention
&
Have a Good Day!!!