Topics

1. Myocardial infarction and subarachnoid hemorrhage
2. Risks of therapeutic embolization in the presence of a patent foramen ovale
3. Cardiac catheterization related stroke and emergent intracranial thrombolysis
**Topic #1:** Myocardial Infarction and Subarachnoid Hemorrhage

- **Issues**
  - What is incidence of myocardial injury after SAH?
  - What is the relationship to patient outcomes?
Subarachnoid Hemorrhage and Myocardial Infarction

- **Research**
  - NIH R01 Myocardial Ischemia and Vasospasm in Aneurysmal SAH. 2004-2009. $2,273,652 (PI: M. Horowitz, MD)

- **Articles**
Subarachnoid Hemorrhage and Myocardial Infarction

**Study #1**

- 47 consecutive patients ages 27-85 (mean 52) with aneurysmal SAH
- Serum cTnI checked at admission and at 24 hours
- EKG at admission and at 24 hours
- Transthoracic echocardiogram performed in those with positive cTnI (>1.5ng/ml)
FINDINGS

8/47 (17%) had cTnI >0.4 ng/ml
- Ages 27-69 (mean 45)
- No patients had a history of prior myocardial ischemia
- EKG available for review in 7/8 patients
  - 6 EKGs abnormal
    - 4/6 abnormal EKGs consistent with cardiac ischemia
    - 2/6 abnormal EKGs had borderline evidence of ischemia
    - cTnI levels in those with abnormal EKGs ranged from 1.5-9.9 ng/ml
    - 1 patient died of cardiogenic shock prior to echocardiogram
    - Echocardiograms performed in 4/5 remaining patients with abnormal EKGs
    - 3 of 4 patients undergoing echocardiograms had wall motion abnormalities

39/47 patients had cTnI <0.4 ng/ml
- 24/37 had abnormal EKGs 14 of which were suggestive of ischemia
- 0/47 had cardiac complications during admission
- No association between elevated cTnI and abnormal EKG in this group
Subarachnoid Hemorrhage and Myocardial Infarction

- **Study #2**
- 81 patients with acute SAH
  - Cardiac complications defined as elevated cTnI >1.4 ng/ml), ECG changes, pulmonary edema
  - 33% had cardiac complications (30% had ECG rhythm changes and pulmonary edema)
  - No statistical difference in mortality between those with and without cardiac complications
  - Neurologic outcome worse at 6 months in those with cardiac complications
Subarachnoid Hemorrhage and Myocardial Infarction

- **Study #3**
  - 103 patients with SAH
  - 29% had mildly elevated cTnI (0.1-1.0 ng/ml)
  - 23% had highly elevated cTnI (>1.0 ng/ml)
  - 48% had negative cTnI
  - Highly positive cTnI associated with worse neurologic condition, longer ICU stay, slightly depressed EF (51% vs 59%)
  - cTnI >1.3 ng/ml associated with abnormal wall motion on echocardiogram
  - cTnI greater than 0.1 ng/ml associated with acute diastolic dysfunction
  - Pulmonary congestion seen in 79% with cTnI > 1.0 ng/ml), in 53% with cTnI > 0.1 ng/ml, and in 29% with negative cTnI
Topic #2: Risks of therapeutic embolization in the presence of a patent foramen ovale

- In the fetal heart, the foramen ovale allows blood to enter the left atrium from the right atrium. It is one of two fetal cardiac shunts, the other being the ductus arteriosus (which allows blood that still escapes to the right ventricle to bypass the pulmonary circulation).
- In most individuals, the foramen ovale closes at birth. It later forms the fossa ovalis.
- Patent foramen ovale (PFO) is a persistent valve like connection between the right and left atria.
- Autopsy studies find a 17-35% incidence.
Risks of therapeutic embolization in the presence of a patent foramen ovale

- PFO is associated with paradoxical cerebral emboli
- When particulate emboli are injected during an embolization procedure, some particles can travel from the arterial to the venous circulation leading to paradoxical cerebral embolization
Risks of therapeutic embolization in the presence of a patent foramen ovale


- Case Report
  - 14 year old right handed boy with juvenile nasopharyngeal angiofibroma (JNA)
  - Embolization requested prior to resection to reduce surgical blood loss
  - Procedure done under GETA
  - Neurophysiologic monitoring of somatosensory evoked potentials (SSEP) and EEG during procedure
  - Embolization of right internal maxillary artery and right ascending pharyngeal artery
  - Each vessel tested with 4 mg IA methohexital without changes in EEG or SSEP
  - Mass embolized with 300-500 micron Embospheres
  - SSEP reduced bilaterally by 50%
  - Patient awoke confused and became mute.
  - MR imaging demonstrated patchy infarcts of the caudate nuclei, deep white matter, and cerebellar hemisphere
  - Echocardiogram with bubble study showed a patent PFO during coughing (equivalent to the 2.5 mm Hg PEEP created by the anesthesia respirator)
  - Chest CT showed no pulmonary arteriovenous fistulas
  - Over the next 3 days the patient returned to his neurologic baseline
**Topic #3:** Cardiac catheterization associated embolic stroke treated with emergent IA thrombolysis


- Case Report
  - 65 year old woman with history CAD, MI, PVD, Rcor A stenting 15 months earlier. 45 pyhx smoking presented with chest pain and ECG changes.
  - During cardiac catheterization patient became acutely less responsive and mute
  - Patient was emergently intubated
  - Stroke team called
  - In cardiac cath lab diagnostic cerebral arteriogram demonstrated occlusion of the basilar apex with no posterior communicating arteries
  - Microcatheter placed into basilar artery (BA)
  - Patient administered 5000U heparin IV, and 75,000U urokinase into BA clot IA
  - 2mm x 10 mm coronary angioplasty balloon placed across the occlusion
  - Post angioplasty vessel opened then reoccluded
  - 15 mg Integrilin administered IV
  - Repeat angioplasty failed to open vessel
  - Additional 75,000U urokinase administered IA into BA
  - 2 mm x 10 mm BioDyvisio stent deployed across occlusion
  - Stent angioplasty using 2 mm x 10 mm Ranger balloon
  - 15 hours later patient extubated, following commands
  - Repeat angiography demonstrated patent posterior circulation
  - CT showed right PCA infarct
  - Patient maintained for 6 months on 325 mg ASA and 75 mg Plavix per day. After 6 months only ASA