Circulatory Derangements II
Thursday, April 24, 2008
1:00 PM

- **Fate of Venous Thrombi**
  - Resolution – Leukocytes chew up thrombus, dissolves and dislodges
  - Embolization – thrombus breaks off, becomes thromboemboli, migrates, and lodges in lungs
    - Can be so small that they are asymptomatic, or so large that they cause sudden death
  - Organization/Recanalization – inflammatory response to clot incites formation of granulation tissue
    - Vessel can “seal up” with a gran. tissue scar if resolution/organization happen concurrently
    - Organization advances around 1 week after clot forms
  - Phlebolith – “venous stone” – clot calcifies and forms a solid (atherosclerosis often?)
  - Propagation – additional Lines of Zahn form, clot grows

- **Fate of Arterial Thrombi**
  - Can come from heart valves, wall of heart (mural thrombus), aorta (usually atherosclerosis)
  - Occlusion – thrombus clogs artery
  - Embolism – movement of something (air, fat, amniotic fluid, cholesterol, foreign body, tumor, etc.)
    - Embolus – the “something”, most often a thrombus, being moved
  - All emboli result in infarction

- **Thrombus vs. Embolus vs. Post-mortem Clot**
  - Thrombus – has Lines of Zahn, is a cast of blood vessel, and is attached to vessel wall (can organize)
  - Embolus – has Lines of Zahn, does not fit vessel, but can still attach to vessel wall (and organize)
  - Post-mortem Clot – has No Lines of Zahn, forms in layers as blood settles, and looks like “chicken fat” or “currant jelly”

- **Venous Emboli** – usually come from veins of lower extremity; thrombi in pulmonary artery extremely rare
  - Thromboembolus reaches pulmonary artery
  - Sudden death
  - Lung looks histologically normal
  - If not dead, pt experiences extreme SOB and chest pain

- **Arterial Emboli**
  - Occur on left side of circulation, usually coming from aorta or heart
  - Symptoms – always appear “downstream” from origin
  - Atherosclerotic Plaque Embolus – always arterial, two sources:
    - Can be thrombotic embolus from a clot from a plaque fissure
    - Can be atherosclerotic debris breaking off from the plaque itself – see crystals
    - Diabetes: atherosclerotic emboli travels to small vessels of foot --> toes die; neuropathic pt can't feel anything
  - Iatrogenic factors – from surgery

- **Cardiogenic Emboli**
  - 20% of ischemic strokes caused by cardiogenic emboli (other 80% are due to atherosclerosis in the carotids
  - Chamber – number of sources:
    - Can be mural thrombus (from infarction, myocarditis, valve disease)
    - Can be tumors (atrial myxoma – can break off, or form thrombus on it to break off)
    - Can be from chronic atrial fibrillation – stagnant blood forming thrombus
  - Valves – emboli have a number of sources:
    - Can be septic (clouds of bacteria & PMNs, macrophages, from endocarditis)
    - Can be marantic (hypercoagulability, brought on by cancer in elderly)
    - Can be from calcified valves (rheumatic heart disease, on mitral valve)
  - Emboli may block coronary artery opening --> MI

- **Paradoxic Embolus** – thromboembolus from right side of circulation bypassing lungs; come through patent atrial septal defect to enter left heart --> the only way this can happen

**QUIZ:** Will be given infarct result, must work backwards to figure out where it comes from
  - Thrombi --> Emboli --> Occlusion --> Infarction
  - An arterial occlusion – causes coagulative necrosis

- Infarcts
  - Hemorrhagic “Red” Infarct – partial occlusion leads to infarction but still some blood flow
    - Liver and lung especially b/c of dual blood supply
  - Pale Infarct – full occlusion leads to infarction and no blood flow
• **Myocardial Infarct Stages**
  - **0-4 hours** – not much, some waviness in fibers at border of infarct, staining defect
  - **4-12 hours** – some dark mottling, begin to see **coagulative necrosis, edema, hemorrhage**
  - **12-24 hours** – dark mottling, **coagulative necrosis**
  - **1-3 days** – mottled, yellow center, **coagulative necrosis** --> **karyolysis**, PMN infiltration
  - **3-7 days** – hyperemic border w/ yellow center, **disintegration of dead myocytes**, PMNs die, macrophages
  - **7-14 days – demolition phase** – very yellow-tan, **phagocytosis** by macrophages, **granulation tissue**
    - **Demolition phase** most vulnerable to rupture (dehiscence), very acellular
  - **2-8 weeks** – gray-white scar, **increased collagen deposition; angiogenesis**
  - **> 2 months** – scar complete, **dense collagen**

• **Central Hemorrhagic Necrosis, Dead Bowel**
  - **QUIZ: CHN** - See liver slide, indicates **heart failure**
  - **Dead Bowel** – embolus to blood supply to bowel, causing ischemic colitis, caused by **left side embolus**