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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**)
 - **Occulsion** – 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
- **Paradoxical 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**