Cellular Injury and Death I - Lecture
Tuesday, April 08, 2008
1:00 PM

- Genetic Storage Disease: Gaucher Cells
  - Gaucher cells – macrophages accumulating too much shit
    - Look like crumpled tissue paper, lack a fast enough metabolism, occur where macrophages are:
    - Lymph nodes, liver, spleen, bone marrow
  - Bone marrow – holes in bone marrow from Gaucher cells cause rarefaction of bone marrow:
    - Hematopoiesis slowed, (small dark cells = hematopoietic cells, lacking)
    - Bone density decreases, and thus greater likelihood of fracture
  - Spleen – Gaucher cells can create a HUGE spleen, causing trapped blood, petechiae/ecchymosis
    - Gaucher cells are eating up platelets, create a huge enlargement necessitating splenectomy

- Genetic Storage Disease: Fabry Disease
  - Fabry Disease – lysosomal storage disease
  - Fabry Disease affects kidneys & myocardium – appear evacuated here
  - Myocardium – myofibrils pushed aside, Fabry’s accumulates --> “jelly rolls” form in myocardium

- Genetic Storage Disease: Glycogen Storage Disease
  - Myocardium – myocytes greatly expanded due to high [glycogen]

- Hydropic/Vacuolar Change
  - Kidney tubules & glomeruli can accumulate water = hydropic change
  - Vacuoles accumulate in epithelial cells = vacuolar change (same thing?)

- Steatosis
  - Steatosis is in your liver, caused by alcohol, steroids, trauma... looks like fatty blobs of various sizes
    - Storage disease of lipids in liver
  - Steatohepatitis – steatosis accompanied by fibrosis (excess connective tissue forming = scar tissue)
  - NASH – Non-alcoholic steatohepatitis – another kind on the rise, risks include obesity, diabetes, HTN

- Cholesterolosis
  - Occurs in gall bladder, core filled with foamy-looking substance in plicae = cholesterol
  - “Strawberry gall bladder” – has many yellow dots everywhere = cholesterol
  - Benign

- Hyaline Droplets
  - Red, glassy droplets in kidney = hyaline droplets, coming from glomeruli leaking filtrate; no actual damage
    - b/c protein should be reabsorbed in tubules
  - Kidney takes up leaky filtrate, exhibits proteinurea
  - Mallory hyaline – can accumulate in liver, hepatocytes seriously injured

- Constipated Plasma Cells
  - Russell Bodies – constipated plasma cells, have way too much protein to handle, swell up w/ hyaline

- Intracellular Pigments
  - Blood breakdown – erythropagocytosis --> iron storage overload = hemosiderin, RBCs broken down by macrophages, leave clusters of brown crap around.
  - Wear and Tear – lipofuscin formed; generally in tissues with slow/no cell turnover – liver, neuron, muscle
    - Accumulates during starvation & atrophy – hepatocytes will break down & accumulate
  - Melanin – melanoma--> pigmented cells in cancerous tumor, notice cancer cells have funky nuclei (pleomorphic, mitotic bodies --> malignancy)
  - Bile – cholestasis --> bile buildup in canaliculi, Kupffer cells, etc., brown pigment everywhere
  - Exogenous – anthracotic pigment of lung (smoking/miner) --> carbon buildup, very black
  - Formalin precipitate – reacts with blood, appears as artifact – brown gunk

- Nuclei Necrosis
  - Pyknosis – nuclei lose water, become dark & shriveled
  - Karyolysis – nuclei dissolve, become barely visible even with hematoxylin stain
  - Karyorrhexis – nuclei fragment
  - True necrosis during life, and not just post-mortem would be accompanied by inflammation!

- Necrosis vs. Apoptosis
  - Apoptosis – controlled cell death; see as “haloed” cells; isolated cells, no overt inflammation
  - Necrosis – no order to death, passive process, heavy inflammation
  - Note that these aren’t mutually exclusive – necrosis can often trigger apoptosis, for example

- Myocardial Infarct
○ An area of necrotic tissue in heart muscle will leave **myocardial infarct** if ischemic for too long
○ **Nitro blue tetrazolium** – black stain for **lactic dehydrogenase (LDH)** – normal in healthy tissue
  ▪ Stains tissue black if LDH present; **absent in infarct**
○ **Creatinine Kinase** – look for in muscle necrosis
○ **Amino Transferase** – look for in liver necrosis
○ **Lipase** – pancreas
○ Fate of necrotic tissue:
  ▪ Demolition & Repair – leukocytes break down dead tissue to allow for rep
  ▪ Separation – necrotic tissue might just slough off from live tissue (ulcers, toes)
  ▪ Perisistence/Calcification – remains in body and calcifies
  ◆ **Dystrophic calcification** – ensues when dead tissues aren’t rid of
  ◆ Atherosclerosis, lymph node & heart valve hardening
  ◆ **Metastatic calcification** – previously normal tissue becomes calcified due to metabolic abnormalities (from necrosis?)
  ◆ **Calculi** – “stones” precipitating in organs from weird chemical reactions, such as gallstones, kidney stones, kidney “stag-horns”