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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)
 - **NAFLD/NASH** – Non-alcoholic steatohepatitis – another kind on the rise, risks include obesity, diabetes, HTN
- **Cholesterosis**
 - 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 **proteinuria**
 - **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 – **erythrophagocytosis** --> 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** (pleiomorphic, 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)
 - Persistence/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”