ECVP/ESVP Summer School in Veterinary Pathology

Summer School 2014 – Toxicological Pathology (178)

Slide 3. H-1 (86-623) Mouse

Description (10)

Liver (also lung on section).
Coagulative necrosis with prominent karryorhexis (3 for description), centrilobular (2)
Hemorrhage and neutrophilic infiltration rimming areas of necrosis (2)
Hepatocytes at periphery of necrosis undergoing vacuolar change (1) and some cells show karyomegaly (1)
Prominent sinusoidal or Ito cells (1)

Morphologic Diagnosis(es) (4)

Hepatic necrosis (coagulative) with hemorrhage, central lobular, severe, acute

Give possible mechanism(s) with examples (4)

P450 mediated toxicity - bioactivation of toxicant by cytochrome P450 enzymes - acetaminophen (CYPIIEI), bromobenzene, carbon tetrachloride, etc (2)

Hypoxia/ischemia – primary or secondary to cardiac toxicant e.g., gossypol (2)

Bile acid carrier mediated toxicity - uptake of microcystin LR (Bonus point 1)

Discuss repair mechanism(s) (2)

Normally see hepatocyte or oval cell (in rodents) proliferation. Further impairment by endothelial cell damage in this case disrupts normal architecture and can lead to fibrosis.

Experimental information: Balb/C mouse given acetaminophen (paracetamol) 24 hr previously. Additional lesions include necrosis of olfactory and transitional nasal epithelium and Clara cells.

Jeffery, E. H., and **W. M. Haschek**. (1988) Protection by dimethyl sulfoxide against acetaminophen induced hepatic but not respiratory toxicity in the mouse. Toxicol. Appl. Pharmacol.* 93:452-461.