WSC 2011-2012
Conference 3, Slide 1

Tissue from a guinea pig.

(NOTE: Some sections contain only spleen.)

MICROSCOPIC DESCRIPTION: Liver: Scattered throughout the liver, there are multiple abscesses **(1 pt.)** (in various stages of maturity) ranging up to 5mm in diameter, which replace pre-existent hepatic architecture. Mature abscesses have a central core of viable and degenerate of neutrophils **(1 pt.)** admixed with fewer macrophages, abundant cellular debris, small amounts of fibrin, and large colonies **(1 pt.)** of 2-4um bacilli **(1 pt.)**. Macrophages contain phagocytized cellular debris and occasionally intact neutrophils. The periphery of the abscess is bounded by concentric rings of fibrous connective tissue containing plump fibroblasts, prominent vessels, and low to moderate numbers of macrophages, neutrophils, lymphocytes and plasma cells. **(2 pt.)** The fibrous connective tissue surrounds and entraps hyperplastic bile ducts **(1 pt.)**, and fingers into surrounding hepatic plates. Scattered throughout the rest of the section, there are areas of lytic necrosis **(1 pt.)** containing numerous degenerate neutrophils, cellular debris, and within these areas, hepatocytes are shrunken with brightly eosinophilic cytoplasm and pyknotic to karyorrhectic nuclei (necrosis) **(1 pt.)**. Occasionally, hepatocytes are outlined by a deeply basophilic granular material (mineral) **(1 pt.)**. Throughout the remainder of the section, hepatocytes are swollen with single or multiple clear clearly defined vacuoles within their cytoplasm (macrovesicular fatty change) **(1 pt.)**. Portal areas are often expanded by variable combinations and concentrations of loosely arranged fibrous connective tissue, plump fibroblasts, and lymphocytes and plasma cells **(1 pt.)**. Hepatic sinusoids contained increased numbers of circulating neutrophils (neutrophilia), and Kupffer cell nuclei are prominent.

MORPHOLOGIC DIANGNOSIS: 1. Liver: Hepatitis, necrosuppurative, chronic-active, multifocal to coalescing, moderate to severe, with multiple abscesses and large colonies of bacilli. **(3 pt.)**

2. Liver, hepatocytes: Lipidosis, macrovesicular, diffuse, moderate. **(2 pt.)**

CAUSE: *Yersinia pseudotuberculosis* or *enterocolitica* **(2 pt.)**

O/C: **(1 pt.)**

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Conference 3, Slide 2
Tissue from a dog.

MICROSCOPIC DESCRIPTION: Liver: There is diffuse massive **(2 pt.)** necrosis of hepatocytes throughout the section. In all sections of the hepatic lobule, there is dissolution of hepatic cords, with disassociation, individualization and rounding up of hepatocytes. **(2 pt.)** Hepatocytes are hypereosinophilic with vacuolated cytoplasm, mildly shrunken, and contain either karyolytic or pyknotic nuclei or lack nuclei **(2 pt.)**. Sinusoids are moderately to severely dilated **(1 pt.)**with marked congestion and hemorrhage within centrilobular and midzonal areas**(1 pt.)**. There are large numbers of macrophages **(2 pt.)**and fewer neutrophils scattered amongst the necrotic hepatocytes which have phagocytized cellular debris. Portal areas, when identifiable, contain hemorrhage, low numbers of macrophages and neutrophils, and cellular debris, **(1 pt.)** and there is mild diffuse biliary hyperplasia **(1 pt.)**. The hepatic capsule is undulant as a result of parenchymal collapse, and subcapsular lymphatics are dilated. **(1 pt.)**

MORPHOLOGIC DIANGNOSIS: Liver, hepatocytes: Necrosis, massive, diffuse, acute, with hemorrhage and stromal collapse. **(3 pt.)**

NAME THREE POSSIBLE CAUSES: *Amanitin*, microcystin, imidocarb, xylitol, acetaminophen toxicosis**,** mebendazole **(3 pt.)**

O/C: **(1 pt.)**

WSC 2011-2012
Conference 3, Slide 3

Tissue from a dog.

MICROSCOPIC DESCRIPTION: Liver: Diffusely, hepatic architecture is altered by concurrent processes of bridging portal fibrosis **(2 pt.)** and nodular hepatocellular regeneration **(1 pt.)**. Throughout the section, portal areas are expanded and incompletely bridged by varying amounts of mature fibrous connective tissue, which often breaches the hepatic limiting plate and entraps adjacent mildly atrophic hepatocytes. In addition to increased amounts of collagen, portal areas contain increased profiles of biliary ductules (biliary hyperplasia, ductular reaction) **(1 pt.)**, moderate numbers of lymphocytes and plasma cells**(1 pt.)**, and macrophages laden with a brown granular pigment. This fibrous connective tissue entraps large nodules of hepatocytes with abnormal sinusoidal architecture**(1 pt.)**; centrilobular veins are often contracted or inapparent. Hepatocytes, especially in centrilobular and midzonal areas are markedly swollen **(1 pt.)** by accumulation of numerous discrete clear distinct round cytoplasmic vacuoles that are smaller in diameter than the nucleus (microvesicular steatosis) **(1 pt.)**. Bile canaliculi are often plugged by a homogenous brown material (bile) **(1 pt.)**, Kupffer cells often contain a similar pigment (hemosiderin, bile) **(1 pt.)**. Scattered thoughout the nodules, there are low numbers of hepatocytes which are shrunken, darkly eosinophilic, and have karyolytic nuclei **(1 pt.)** and are often surrounded by few neutrophils (necrosis) **(1 pt.)**. Multinucleated hepatocytes are scattered throughout the nodules. The hepatic capsule is markedly undulant **(1 pt.)**, there is subcapsular hepatocellular loss, and marked dilation of subcapsular lymphatics. Lymphatics surrounding sublobular and hepatic veins are also markedly dilated. **(1 pt.)**

MORPHOLOGIC DIAGNOSIS: Liver: Macronodular hepatocellular regeneration, diffuse, moderate to severe, with microvesicular steatosis, necrosis, cholestasis, and bridging portal fibrosis **(3 pt.)**

CAUSE: Aflatoxin **(2 pt.)**

O/C: **(1 pt.)**

WSC 2011-2012
Conference 3, Slide 4

Tissue from a horse.

MICROSCOPIC DESCRIPTION: Liver: Diffusely, portal areas are markedly expanded by abundant loosely arranged fibrous connective tissue and plump fibroblasts **(1pt)** which breach the limiting plate, surround and replace portal hepatocytes**,** and occasionally bridge adjacent portal areas **(1pt)**. There is moderate biliary proliferation **(1pt)**, and epithelium lining hyperplastic ductules is pale and swollen. Portal areas contain small numbers of lymphocytes and plasma cells and rare hemosiderin-laden macrophages, and portal lymphatics are moderately ectatic **(1pt)**. There is distortion of the remaining hepatocellular architecture and hepatocytes are often enlarged up to 2-3 times normal **(2pt)**, Hepatocyte cytoplasm ranges from a lacy, pink, ground glass appearance (glycogenosis) **(1pt)** to microvacuolated (microvesicular steatosis) **(1pt)**, and both changes may be seen in the same hepatocytes. Many hepatocytes contain nuclei which are three time the size of surrounding hepatocytes (megalocytosis) with marginated chromatin and a prominent nucleolus **(2pt)**. There are occasional multinucleated hepatocytes. Bile canaliculi are distended with dark brown waxy pigment (bile) (canalicular cholestasis) **(1pt),** and hepatocytes often contain brown granular pigment. There are rare necrotic hepatocytes which are shrunken, hypereosoinophilic and are occasionally surrounded by low numbers of neutrophils**(1pt)**. The overlying capsule is mildly undulant and mesothelial cells are hypertrophied.

MICROSCOPIC DIAGNOSIS: Liver: Fibrosis, portal and bridging, diffuse, moderate, with hepatocellular anisocytosis and megalocytosis, necrosis, and cholestasis. **(4pt)**

CAUSE(S): Pyrollizzidine alkaloid toxicosis **(3pt)**

O/C: **(1pt)**