WSC 2016-2017 Conference 2.

Case 1. Tissue from a rabbit. (One section is a normal control).

MICROSCOPIC DESCRIPTION: Liver: There is diffuse massive **(1pt.)**. necrosis **(1pt.)**.of hepatocytes throughout the section of liver, but hepatic architecture is largely preserved (absence of stromal collapse) **(1pt.)**. In a few lobules, periportal hepatocytes remain **(1pt.)**.; these hepatocytes are arranged in cords or rarely, small nodules, and are swollen with accumulation of moderate amounts of glycogen **(1pt.)** and small amounts of lipid, as well as rare mitotses.. These hepatocytes are often in close association with proliferating oval cells **(**and bile ductules **(1pt.)** (Desmet reaction 2A). There is extensive expansion of portal areas by fibrosis which often bridges portal areas **(1pt.)**. and marked biliary hyperplasia **(1pt.)**. Midzonal and centrilobular hepatoctyes are diffusely absent **(1pt.)**, and these areas of the portal are expanded by massive edema **(1pt.)**. which separates pre-existent stromal fibers, throughout which are scattered areas of hemorrhage **(1pt.)** and fibrin and numerous hemosiderin-laden macrophages **(1pt.)** fewer heterophils, as well as low to moderate numbers of fibroblasts. There is marked distention of lymphatics **(1pt.)**.within sublobular veins due to edema ("rose-window effect.). Focally, a large caliber hepatic vessel contains a fibrinocellular thrombus (1pt.)The hepatic capsule is markedly expanded up to 1.5 mm **(1pt.)**. by fibrous connective tissue and fibroblasts separated by abundant edema and dilated lymphatics, and blends imperceptibly with the underlying damaged parenchyma. **(1pt.)**.

MORPHOLOGIC DIAGNOSIS: Liver: Hepatocellular necrosis, massive, diffuse, severe with marked bridging portal fibrosis, biliary hyperplasia, edema, and capsular fibrosis. **(3pt.)**

CAUSE: Hepatic lobar torsion: **(1pt.)**

Further reading: Desmet, V. Ductal plates in hepatic ductular reactions. Hypothesis and implications. I. Types of ductular reaction reconsidered. Virchows Arch (2011) 458: 251-259.

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Case 2. Tissue from a cat.

MICROSCOPIC DESCRIPTION: Lymph node: The subcapsular, paracortical and medullary sinuses **(1pt.)** are moderately expanded **(1pt.)** by an infiltrate of large numbers of macrophages **(2pt.)** which occasionally infiltrate and rarely efface the cortex and paracortex **(1pt.)**. Macrophages are polygonal to spindled and range up to 10 um **(1pt.)** in diameter and contain multiple intracytoplasmic **(1pt.)** 2-4um irregularly round yeasts **(2pt.)** with a prominent clear cell wall. Macrophages are separated by moderate amounts of edema fluid. The paracortex is moderately expanded by low numbers of intermediate - to large lymphocytes **(1pt.)** and moderate numbers of plasma cells **(1pt.)**. Regionally, medullary and paracortical sinuses are expanded with edema, small amounts of hemorrhage, and large numbers of hemosiderin-laden macrophages **(1pt.)**, some of which exhibit active erythrophaogocytosis **(1pt.)**. There is mild follicular hyperplasia. **(1pt.)**. Rare megakaryocytes are scattered throughout the medullary sinuses.

MORPHOLOGIC DIAGNOSIS: Lymph node: Lymphadenitis, histiocytic (granulomatous OK), diffuse, severe, with mild reactive lymphadenitis, hemosiderosis, and edema.

CAUSE: *Histoplasma capsulatum var. capsulatum* **(3pt.)**

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

Case 3. Tissue from a foal.

MICROSCOPIC DESCRIPTION: Kidney: Randomly scattered within the cortex and extending into the medulla **(1 pt)**, are low numbers of **(1 pt)**, multifocal to coalescing microabscesses **(1 pt)** that often center on and efface glomeruli **(1 pt),** linearly accompany vessels and extend into adjacent interstitium and surround and replace adjacent tubules **(1 pt)**. Abscesses are composed of abundant necrotic debris admixed with numerous degenerate and rare viable neutrophils **(1 pt)** and rare macrophages. Thoughout the section, glomeruli are expanded by fibrin thrombi within glomerular capillaries, and the tuft contains low numbers of neutrophils admixed with small amounts of cellular debris. Occasionally, Bowman's space contains polymerized fibrin. Occasionally, glomerular capillaries contain bacterial emboli composed of large colonies of basophilic 2x3um coccobacilli. **(1pt)** In areas adjacent to suppurative inflammation, tubular epithelium is markedly swollen with numerous vacuoles (degeneration) **(1 pt)** or brightly eosinophilic, shrunken and fragmented, with pyknotic nuclei (necrotic) **(1 pt)**. Necrotic tubules often contain sloughed epithelial cells admixed with degenerate neutrophils and cellular debris **(1 pt)**. Other tubules often contain abundant protein within their lumen, or rarely hemorrhage. **(1 pt.)** Rarely within areas of interstitial inflammation, there are areas of hemorrhage, hemosiderin-laden macrophages, large colonies of coccobacilli as previously described. **(1 pt)**.

MORPHOLOGIC DIAGNOSIS: Kidney: Nephritis, suppurative **(1 pt.)**, embolic **(1 pt.)**, with mild fibrinosuppurative glomerulitis **(1 pt.)** and rare large colonies of bacilli **(1 pt.)**

CAUSE: *Actinobacillus equuli* **(3 pt)**

O/C – **(1 pt.)**

Case 4. Tissue from a calf.

MICROSCOPIC DESCRIPTION: Lung: Diffusely, alveoli are expanded and filled by variable combinations and concentrations of polymerized fibrin **(2 pt.)**, viable and degenerate neutrophils **(1 pt.)** (which often have streaming and smudgy nuclei (oat cells) **(2 pt.)**, macrophages, edema, and cellular debris. **(1 pt.)** Rarely, alveoli contain colonies of 2-3um bacilli. **(1 pt.)** Intervening alveolar septa **(1 pt.)** are expanded up to three times normal by similar inflammatory components as well as marked congestion, small amounts of edema and fibrin, and prominent Type II pneumocyte hyperplasia. **(1 pt.)** Diffusely, bronchiolar lumina **(1 pt.)** are expanded by large numbers of degenerate neutrophils and macrophages admixed with abundant cellular debris. Bronchiolar epithelium is mildly hyperplastic, and there is multifocal epithelial necrosis with low numbers of infiltrating neutrophils admixed with cellular debris.; **(1 pt.)** some airways are lined with attenuated epithelium. there is a focal area of acute coagulative necrosis (infarct) **(1 pt.)** which is bounded by a dense band of basophilic cellular debris **(1 pt.)**. Within this area, pulmonary parenchyma, including both aleoli and airways are pale and exhibit diminished differential staining. Interlobular septal and to a lesser extent, pleural connective tissues are markedly expanded up to ten times normal **(1 pt.)** by distended lymphatics **(1 pt.)** (which often contain fibrinocellular thrombi **(1 pt.)**), edema, and moderate to large numbers of infiltrating neutrophils and macrophages. Rare viral syncytia ranging up to 20um with up to eight nuclei are present within alveoli (not seen in all slides).

MORPHOLOGIC DIAGNOSIS: Lung: Bronchopneumonia **(1 pt.)**, fibrinosuppurative **(1 pt.)** and necrotizing, diffuse, severe, with infarction **(1 pt.)** and rare bacilli. **(3 pt.)**

CAUSE: *Mannheimia hemolytica* **(3 pt.) (***Mycoplasma mycoides subsp. Mycoides, Biebersteinia trehalosi, Pasteurella multocida OK***)**

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