WSC 2020-2021
Conference 23 Case 1.

Tissue from a mouse.

MICROSCOPIC DESCRIPTION: Liver: Scattered randomly **(1pt.)** throughout the section are multifocal to coalescing areas of lytic necrosis **(1pt.)** in which hepatocyte architecture is lost, and are infiltrated by numerous degenerate neutrophils **(1pt.)** admixed with cellular debris and brightly eosinophilic fibrin, as well as granular mineral **(1pt.)**. Numerous 2x4 bacilli are visible throughout necrotic foci, most prominently at the border with adjacent normal tissue. **(1pt.)** Hepatocytes adjacent to these foci are mildly swollen and contain one to multiple discrete lipid vacuoles **(1pt.)** within their cytoplasm and and nuclei are shrunken, mildly hyperchromatic, with clumped chromatin adjacent to the nuclear membrane (degeneration) **(1pt.)**. There are few lymphocytes and plasma cells within portal areas, and scattered islands of extramedullary hematopoiesis within the parenchyma. Throughout the section, Kupffer cells are mildly hypertrophic and sinusoids contain increased numbers of circulating neutrophils and histiocytes. Occasional binucleated hepatocytes are present. There is mild mesothelial cell hyperplasia overlying areas of subcapsular hepatocellular necrosis.

Spleen: Approximately 80% of the section is effaced by coalescing areas of lytic necrosis **(1pt.)** which indiscriminately affect both red and white pulp **(1pt.)**. Areas of necrosis are infiltrated by large numbers of viable and necrotic neutrophils and macrophages admixed with large amounts of cellular debris **(1pt.)**, hemorrhage and polymerized fibrin. **(1pt.)** There is lymphocytolysis in remaining areas of white pulp **(1pt.)** and numerous tingible body macrophages. In areas of necrosis, vessel walls are also necrotic and there are occasional occlusive fibrin thrombi **(1pt.)**.

MORPHOLOGIC DIAGNOSIS: 1. Liver: Hepatitis, necrotizing **(1pt.)**, random, multifocal, marked, with numerous extra- and intracellular bacilli. **(1pt.)**

2. Spleen: Splenitis, necrotizing **(1pt.),** multifocal to coalescing, severe, with thrombosis.

CAUSE: *Listeria monocytogenes* **(3pt.)**

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

WSC 2020-2021
Conference 23 Case 2.

Tissue from a rat.

MICROSCOPIC DESCRIPTION: Kidney: Effacing the pole **(1pt.)** of the kidney, there is a nodular, unencapsulated, infiltrative, moderately cellular, well-demarcated neoplasm. **(2pt.)** Neoplastic cells are composed of epithelial cells in cords **(1pt.)**, and smaller nests and packets on a fine fibrovascular stroma **(1pt.)**. Neoplastic cells are polygonal **(1pt.)** with distinct cell borders and a large amount of a granular to vacuolated **(1pt.)** eosinophilic cytoplasm. There are numerous cells with a large central vacuole **(1pt.)** which ranges from clear to containing granular basophilic material **(1pt.)** (signet ring cells **(1pt.)**). Nuclei are central with finely clumped chromatin and a single central prominent eosinophilic nucleolus **(1pt.)**. There are occasional karyomegalic cells scattered throughout the neoplasm. **(1pt.)** Mitoses are rare. **(1pt.)** There is extensive single cell necrosis and areas of central necrosis **(1pt.)** of neoplastic cells nests scattered throughout the neoplasm. There is mild dilation of the renal pelvis. **(1pt.)** There is tubular and glomerular compression at the interface with the adjacent cortex, as well as mild fibrosis and small aggregates of lymphocytes and plasma cells. **(1pt.)**

MORPHOLOGIC DIAGNOSIS: Kidney: Renal cell carcinoma **(3pt.)**, amphophilic-vacuolar type.

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

WSC 2020-2021
Conference 23, Case 3.
Tissue from a guinea pig.

Salivary gland: Approximately 70% of the parotid and sublingual gland are affected by various combinations of infarction, atrophy, and regeneration. Approximately 50% of the parotid gland is replaced by a large area of coagulative necrosis **(1pt.)** characterized by retention of tissue architecture with a loss of differential staining **(1pt.)** (infarct) and there are several smaller areas elsewhere in the gland as well. The periphery of the infarcted areas contain variable combinations and concentrations of hemorrhage, fibrin, edema, granulations tissue **(1pt.)**, and moderate numbers of neutrophils **(1pt.)**, and eosinophilic and karyorrhectic debris (lytic necrosis) which multifocally extends into the interlobular connective tissue, capsule, and periglandular fat. **(1pt.)** Acinar cells outside the area of the infarct and in other lobules are often shrunken with loss of mucus, and loss of distinct cellular borders **(1pt.)** (atrophy) **(1pt.)**. Atrophic acini often have no distinct lumina, or the lumen is filled with brightly eosinophilic cellular debris. **(1pt.)** Ducts at the margin of the area of infarction are multifocally surrounded by fibrosis and few lymphocytes **(1pt.)** and plasma cells and are segmentally lined by a) epithelial cells with swollen, pale, vacuolated cytoplasm (degeneration) **(1pt.)**, necrotic **(1pt.)** cells with pyknotic or karyorrhectic nuclei or that have sloughed into the lumen, darkly basophilic cells **(1pt.)** with a high mitotic rate that occasionally pile up and fill the duct lumen (hyperplasia) **(1pt.)**, or rarely by multiple layers of flattened epithelium (squamous metaplasia). Affected ducts often contain brightly eosinophilic cellular debris or sloughed epithelium within their lumina. **(1pt.)**

MORPHOLOGIC DIAGNOSIS: Salivary gland, submandibular: Coagulative necrosis (infarct) **(1pt.)**, multifocal, with ductular degeneration, necrosis, regeneration**(1pt.)** and squamous metaplasia **(1pt.)** and acinar atrophy.

CONDITION: Necrotizing sialometaplasia **(2pt.)**

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

WSC 2020-2021
Conference 23, Case 4.

Tissue from a hamster.

MICROSCOPIC DESCRIPTION: Lung: There are multifocal to coalescing areas of consolidation scattered randomly throughout the section. **(1pt.)** Within these areas, alveolar septa are markedly expanded by hypertrophy of alveolar intraseptal macrophages **(1pt.)**, numerous circulating neutrophils, lymphocytes (occasionally in aggregates) **(1pt.)**, small amounts of congestion and edema, and profound Type II pneumocyte hyperplasia **(1pt.)** with marked anisocytosis and anisokaryosis and rare multinucleation.. **(1pt.)** There is rare septal necrosis **(1pt.)** characterized by loss of discontinuous septa, hemorrhage and fibrin within adjacent alveoli, and cellular debris. Diffusely **(1pt.)**, alveolar septa are expanded by congestion, hypertrophied macrophages, and circulating neutrophils. Alveolar lumina in inflamed areas contain numerous foamy macrophages **(1pt.)** and neutrophils **(1pt.)** admixed with edema, small amounts of fibrin, and cellular debris. In inflamed areas, bronchiolar epithelium is variably attenuated **(1pt.)** to hyperplastic **(1pt.)**, and small airways often contain reflux of alveolar contents. **(1pt.)** Airway epithelium is occasionally infiltrated by low numbers of lymphocytes and/or neutrophils. Peribronchial and perivascular tissues are infiltrated by low to moderate numbers of lymphocytes and perivascular tissue is often further expanded by edema. **(1pt.)** Pleura are multifocally expanded by a combination of fibrous connective tissue, small numbers of lymphocytes and plasma cells, and overlying hyperplastic mesothelial cells. **(1pt.)**

MORPHOLOGIC DIAGNOSIS: Lung: Pneumonia, bronchointerstitial **(1pt.)**, neutrophilic and histiocytic, **(1pt.)** multifocal to coalescing, subacute (1pt.), severe, with marked Type II pneumocyte hyperplasia and syncytia formation**(1pt.)** and multifocal septal necrosis.

CAUSE: SARS-CoV-2, **(2pt.)** (Influenza, any other respiratory coronavirus or paramyxovirus OK)

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