

WSC 2022-2023
Conference 17, Case 1
Tissue from a piglet.

MICROSCOPIC DESCRIPTION: Mesenteric vascular plexus (1pt): The adventitia of the arterioles **(1pt)** of the vascular plexus surrounding mesenteric lymph nodes is infiltrated by moderate numbers of lymphocytes **(1pt)**, macrophages **(1pt)**, and plasma cells. These inflammatory cells multifocally extend into the mural smooth muscle. Arterioles within affected areas of smooth muscle are fragmented hypereosinophilic, and pyknotic (necrosis) and there is moderate intramural edema. Occasionally, there is small amounts of eosinophilic protein within arteriole wall (fibrinoid necrosis). **(1pt)** There are rare non-occlusive fibrin thrombi within arteriolar lumina. Larger aggregates of lymphocytes, macrophages, and plasma cells expand the connective tissue between arterioles. There is follicular loss within the adjacent nodes, but expansion of the paracortex. **(1pt)** Large numbers of hemosiderin-laden macrophages **(1pt)** are present within subcapsular and medullary sinuses and scattered throughout the medullary cords and paracortex.

Lung: Alveolar septa are diffusely and markedly expanded by hypertrophy of septal macrophages **(1pt)**, congestion, edema, and fewer lymphocytes and plasma cells. There is multifocal hypertrophy of type 2 pneumocytes **(1pt)**. Alveolar spaces contain moderate numbers of macrophages admixed with few neutrophils and small amounts of basophilic protein. **(1pt)** Similar components are refluxed into airways. There is mild perarteriolar edema and infiltration of the adventitia with low to moderate numbers of lymphocytes, macrophages, and plasma cells **(1pt)**. Interlobular septa are expanded by similar cells and small amounts of edema. **(1pt)**

Heart: The adventitia of coronary arteries is infiltrated by low to moderate numbers of lymphocytes, macrophages, and plasma cells. **(1pt)** Similarly to the mesenteric vascular plexus, there is occasional mural infiltration, smooth muscle necrosis, and eosinophilic fibrin within the inner layers of the arteriolar smooth muscle. There is mild pericardial edema with lymphatic dilation.

MORPHOLOGIC DIAGNOSIS: 1. Lung: Pneumonia, interstitial **(1pt)**, histiocytic **(1pt)**, diffuse, severe, with interlobular edema and lymphohistiocytic arteritis.
2. Mesenteric vascular plexus, heart: Arteritis **(1pt)**, histiocytic **(1pt)**, diffuse, moderate
3. Lymph node: Reactive hyperplasia, diffuse, mild, with marked hemosiderosis.

CAUSE: Porcine circovirus-3 (PCV -2 is fine as well.) **(2pt)**

O/C: (1pt)

WSC 22-23
Conference 17, Case 2
Tissue from a puppy

MICROSCOPIC DESCRIPTION: Lung: There is mild autolysis. Diffusely alveolar septa **(1pt)** are markedly expanded by edema **(1pt)**, moderate number of macrophages **(1pt)**, fewer neutrophils, lymphocytes, hypertrophied intraseptal macrophages and cellular debris. Septa are lined by bizarrely hypertrophic **(1pt)** Type II pneumocytes **(1pt)** which have abundant pink vacuolated cytoplasm. Type II pneumocytes often contain multiple nuclei (viral syncytia) **(1pt)** and one or multiple 2-4 irregularly shaped intracytoplasmic **(1pt)** viral inclusions **(1pt)**, and rarely, a 2-3um intranuclear viral inclusion **(1pt)** surrounded by a clear halo which peripheralizes the chromatin. Alveoli contain numerous foamy alveolar macrophages **(1pt)** and regionally, brightly eosinophilic multinucleated alveolar macrophage viral syncytia ranging up to 22um in diameter with similar intracytoplasmic and rare intranuclear inclusions **(1pt)**, admixed with edema, fibrin, and cellular debris. Airway lumina **(1pt)** are filled with similar refluxed material, and airway epithelium undergoes a range of changes from necrosis, attenuation, and viral syncytia formation. Small number of lymphocytes and macrophages infiltrate necrotic airway epithelium. There is mild edema and low number of lymphocytes, macrophages, and plasma cells within the interlobular and peribronchiolar fibrous connective tissue. **(1pt)**

MORPHOLOGIC DIAGNOSIS: Lung: Pneumonia, interstitial **(1pt)**, proliferative **(1pt)** and histiocytic **(1pt)**, diffuse, severe, with marked type II pneumocyte hypertrophy **(1pt)**, and intraepithelial and intrahistiocytic viral syncytia, intracytoplasmic and intranuclear inclusions **(1pt)**.

CAUSE: Canine morbillivirus **(2pt)**

WSC 2022-2023
Conference 17, Case 3
Tissue from a dog.

MICROSCOPIC DESCRIPTION: Cerebrum: Within cortical white matter tracts, **(1pt)** there are extensive and coalescing areas of spongiosis **(1pt)**. Within these areas, myelin sheaths are dilated up to 50um and occasionally coalesce (digestion chambers) **(1pt)**, and contain dilated axons (spheroids) **(1pt)**, myelin debris, and Gitter cells. There are numerous hypertrophic microglia. **(1pt)**. In more severely affected areas, there is rarefaction of the intervening white matter with infiltration of numerous foamy Gitter cells **(1pt)**, increased numbers of hypertrophic microglia **(1pt)**, (demyelination) **(1pt)**. Multifocally, neurons occasionally are swollen and chromatolytic, and few are shrunken and pyknotic (necrosis) **(1pt)**. Rare neurons and astrocytes contain 2-3um intranuclear **(1pt)** and/or intracytoplasmic **(1pt)** eosinophilic viral inclusions. In these areas, there is marked gliosis **(1pt)**, with microglial hypertrophy, scattered glial nodules **(1pt)**, and few hypertrophic astrocytes **(1pt)**. Blood vessels are often lined by hypertrophic endothelium and surrounded by 2-3 layers of lymphocytes and plasma cells. **(1pt)**

mMORPHOLOGIC DIAGNOSIS: Cerebrum: Demyelinationyy **(1pt.)**, multifocal to coalescing, marked, with spongiosis **(1pt)**, gliosis, and neuronal and astrocytic intranuclear and intracytoplasmic viral inclusions **(1pt)**

CAUSE: Canine morbillivirus **(2pt)**

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

WSC 2022-2023
Conference 17, Case 4.
Tissue from a dog.

MICROSCOPIC DESCRIPTION: Kidney: There is mild autolysis. Multifocally and linearly **(1pt)**, extending from the cortex down into the medulla and centered on vasa recta **(1pt)**, there are numerous, occasionally coalescing foci of necrosis and/or granulomas. The lesions within the cortex are most commonly well-formed granulomas **(1pt)**, characterized by a central area of eosinophilic granular cellular debris **(1pt)**, surrounded by multiple layers of epithelioid macrophages **(1pt)**, which is in turn surrounded by lamellations of fibrous connective tissue **(1pt)** which contain low numbers of lymphocytes and plasma cells. **(1pt)** As foci of inflammation proceed more deeply into the medulla, granulomas are less well formed. Within the medulla, there are foci of variable combinations and concentrations of macrophages **(1pt)**, lymphocytes **(1pt)** and plasma cells **(1pt)** surrounding thrombosed vessels. The inflammatory cells extend into the surrounding renal parenchyma, effacing normal parenchyma and expanding the medullary interstitium. **(1pt)** Within these extensive areas, there is necrosis of medullary tubules **(1pt)** – remnant tubules contain sloughed epithelium admixed with macrophages and cellular debris in tubular lumina. Tubules are lined with a combination of swollen epithelium (degeneration), pyknotic (necrotic) or attenuated epithelium. **(1pt)** Within the unaffected cortex, there is segmental mineralization of Bowman's capsule and capillary basement membranes. **(1pt)**

MORPHOLOGIC DIAGNOSIS: Kidney: Nephritis, granulomatous **(1pt)** and necrotizing **(1pt)**, multifocal to coalescing, marked.

CAUSE: Mycobacterium sp. **(3pt)**

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