

Case 1 – Tissue from a horse.

MICROSCOPIC DESCRIPTION: Lung: Within a focally extensive area approximating 90% of the section, alveolar septa are diffusely and markedly thickened up to 500um **(1pt)** by abundant mature collagen **(1pt)**, plump fibroblasts **(1pt)**, low numbers of neutrophils, histiocytes, congested capillaries, and often lined by hyperplastic type II pneumocytes **(1pt)** and scattered aggregates of siderophages. All areas of remodeling are similar in terms of remodeling, and it is often interrupted by the presence of interlobular septa. **(1pt)** Alveolar spaces are distorted by the fibrosis (alveolar remodeling) **(1pt)**, often expanded, and filled by various combinations and concentrations of viable and degenerate neutrophils **(1pt)**, foamy macrophages **(1pt)**, fewer eosinophils, sloughed degenerate type II pneumocytes, cellular debris, fibrin, and edema fluid **(1pt)**. Rarely, alveolar macrophages contain a single, 4-6 um, smudgy basophilic **(1pt)** intranuclear viral inclusion **(1pt)** which is often surrounded by a clear halo. Bronchioles are often filled with sloughed respiratory epithelium (due to the moderate autolysis in this slide) and occasionally with cells refluxed from adjacent alveoli. **(1pt)** In affected areas, the pleura is markedly thickened **(1pt)** up to 2mm and there are numerous thick-walled arterioles with a loosely arranged, myxomatous tunica media scattered throughout this fibrous connective tissue.

MORPHOLOGIC DIAGNOSIS:

Lung: Pneumonia, necrotizing **(1pt)** and sclerosing **(1pt)**, interstitial, focally extensive, severe, with marked alveolar remodeling, type II pneumocyte hyperplasia **(1pt)** and rare intrahistiocytic intranuclear viral inclusions **(1pt)**.

Name the disease: Equine multinodular pulmonary fibrosis **(1pt)**

CAUSE: Equine herpesvirus-5 **(2pt)**

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

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Case 2 – Tissue from a dog.

MICROSCOPIC DESCRIPTION: Lung: Expanding the bronchiole **(1pt)**, expanding into and compressing the surrounding atelectatic lung, there is a 1cm area of necrotizing and pyogranulomatous inflammation. The luminal inflammation is centered on a combination of innumerable 3-4um, parallel-walled, septate **(1pt)** hyphae **(1pt)** with segmental clear swellings up to 6 um, and acute angle dichotomous branching **(1pt)**. In one area of the section, admixed with innumerable fungal hyphae, numerous darkly pigmented **(1pt)** stalked fruiting bodies **(1pt)** are present, which range up to 125 microns in diameter. The masts of hyphae and enclosed fruiting bodies are surrounded by immense numbers of viable and degenerate neutrophils **(1pt)** admixed with fewer histiocytes **(1pt)**, abundant cellular debris, hyphae, and moderate amounts of polymerized fibrin. Scattered throughout the exudate and in densest concentration at the periphery are numerous 10-25um birefringent fan-shaped oxalate crystals **(1pt)**. The exudate surrounds and separates bronchial cartilage and effaces normal bronchiolar architecture, extending into the adjacent alveolar parenchyma, where there is increased hemorrhage and fibrin, as well as the formation of a compression capsule maturing collagen which effaces normal pulmonary architecture. **(1pt)** In adjacent, less affected tissue, alveolar septa are expanded up to 5x normal by congestion, edema, and moderate amounts of polymerized fibrin **(1pt)** and alveoli are multifocally expanded by various combinations and concentrations of viable and degenerate neutrophils, cellular debris, macrophages, and edema. **(1pt)** Airways contain moderate amounts of a similar exudate **(1pt)**.

MORPHOLOGIC DIAGNOSIS: 1. Lung, bronchiole: Bronchiolitis **(1pt)**, pyogranulomatous **(1pt)** and necrotizing, focally extensive, severe, with numerous fungal **(1pt)** hyphae, fruiting bodies, and oxalate crystals **(1pt)**.

CAUSE: *Aspergillus* **(2pt)** niger

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

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Case 3 – Tissue from a cat.

MICROSCOPIC DESCRIPTION: Lung: Affecting approximately 75% of the section **(1pt)**, there is a diffuse process of fibrosis arising from the pleura which gives the section a vaguely multinodular histologic appearance **(1pt)**. Nodules of affected pulmonary parenchyma are often surrounded by dense bands of mature collagen **(1pt)** containing numerous fibroblasts/myofibroblasts **(1pt)**. Within these areas, the alveolar architecture is lost **(1pt)**, alveoli are collapsed and septa are expanded **(1pt)** by moderate amounts of immature collagen **(1pt)**. Remaining alveolar spaces are enlarged **(1pt)**, contain moderate numbers of foamy macrophages **(1pt)** and fewer neutrophils and are often lined by type II pneumocytes **(1pt)**. Airways are largely collapsed with effacement of their lumina by the fibrotic process and are surrounded by markedly hyperplastic smooth muscle **(1pt)**. There is moderate mural smooth muscle hyperplasia of entrapped arterioles **(1pt)**. Aggregates of moderate numbers of lymphocytes and fewer plasma cells are often present at the periphery of the nodules within the dense bands of fibrosis. Regionally, there are disorganized bronchioles in close proximity **(1pt)** (bronchiolarization) lined by flattened, often cuboidal basophilic epithelium with rare foci of squamous metaplasia; their lumina often contain small amounts of sloughed epithelium, viable neutrophils and cellular debris. A large dilated bronchus (traction bronchiectasis) **(1pt)** lined by low cuboidal to attenuated epithelium traverses the section. Alveolar septa within remaining less affected tissue are expanded by congestion, edema, and polymerized fibrin and at the interface with adjacent fibrotic tissue, immature collagen. Alveoli contain increased numbers of foamy macrophages. The pleura is multifocal expanded by fibrosis as well.

MORPHOLOGIC DIAGNOSIS: Lung: Fibrosis **(1pt)**, interstitial **(1pt)**, supleural, multinodular, severe, with alveolar loss, bronchiolarization with epithelial metaplasia **(1pt)**, and airway and arteriolar smooth muscle hyperplasia.

NAME THE CONDITION: Feline idiopathic pulmonary fibrosis **(1pt)**

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

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Case 4 – Tissue from a alpaca.

MICROSCOPIC DESCRIPTION: Lung: There are multiple sections of lung on this slide, each looking worse than the last! Diffusely, airways are filled with innumerable viable and degenerate neutrophils **(1pt)**, admixed with fewer macrophages and abundant cellular debris. Lining epithelium is multifocally infiltrated by inflammatory cells and occasionally necrotic, but largely intact. **(1pt)** Areas of airway epithelial necrosis are largely covered by a layer of attenuated epithelium. Surrounding alveoli are often expanded by variable numbers (often large) of viable and degenerate neutrophils and macrophages **(1pt)** which are admixed with abundant edema, polymerized fibrin, and cellular debris. Occasionally, alveoli contain colonies of 2-3um coccobacilli which are often present within macrophage cytoplasm. **(1pt)** Septa are variably expanded **(1pt)** by edema, fibrin, and hypertrophic epithelial cells, and septal necrosis **(1pt)** is common. In some sections, there are well-demarcated areas, often bounded by a dense band of cellular debris **(1pt)**, in which there is distinct coagulative necrosis **(1pt)** of all parenchymal elements, with extensive septal necrosis as well as flooding of the alveoli with fibrin **(1pt)** in addition to the exudate seen in other areas (pulmonary sequestra). The interlobular septa are markedly expanded by abundant edema, fibrin and hemorrhage, dilated lymphatics, congested vessels, and low numbers of neutrophils. **(1pt)** Veins and lymphatics in proximity to areas of necrosis are partially to totally occluded by fibrin thrombi **(1pt)**, and some vessels in areas of inflammation exhibit vasculitis **(1pt)** or are totally necrotic. The pleura is multifocally expanded **(1pt)** by edema, hemorrhage and neutrophils, and multifocally covered by a thick mat of fibrin. **(1pt)**

MORPHOLOGIC DIAGNOSIS: 1. Lung: Bronchopneumonia, **(1pt)** necrosuppurative **(1pt)**, diffuse, severe, with vasculitis **(1pt)** and rare intra-and extracellular bacilli.
2. Lung: Pleuropneumonia, interstitial, fibrinous, diffuse, moderate.

CAUSE: This one is *Burkholderia pseudomallei*, but any traditional cause of ruminant shipping fever is acceptable. **(2pt)**

O/C: (1pt.)