



ECVP/ESVP Summer School in Veterinary Pathology



Marie Curie Training Courses

Summer School 2006 – Emerging Infectious Diseases Case 1(3)

CASE 1 Provided by: Dr. Jerry Ritchey, Oklahoma State University, USA.

Signalement: Domestic short-haired cat (*Felis catus*), 1 year, intact male

History:

Owner has lost 5-6 cats over the past 6 months. The cats lose weight despite normal eating habits and activity. The owner finds the cats dead without other signs. Owner is concerned about possible toxicity.

Gross Findings:

The animal was in poor nutritional condition.

Mucous membranes and subcutaneous tissues were mildly pale and icteric.

The spleen was enlarged 3-5 times normal with widely scattered, 2-3mm white foci throughout the splenic capsule and cut surface.

Mesenteric and submandibular lymph nodes were 2-5 times expected size; their cut surfaces bulged slightly and had scattered, discrete white foci similar to those seen in the spleen.

The lungs failed to collapse entirely, were mildly firm and diffusely reddened.

Sections of spleen and lymph node were collected fresh and submitted for bacterial culture.

Histology: Tissue from a cat.

1. DESCRIPTION OF HISTOLOGIC FINDINGS

Spleen. There are randomly distributed, variably sized areas of cells with loss of cellular details, hypereosinophilic cytoplasm, karyorrhexis and karyolysis (necrosis), between which there are erythrocytes, neutrophils, macrophages (some of which exhibit erythrophagocytosis) and fibrillar eosinophilic material (fibrin). These focal areas are relatively abruptly demarcated from adjacent red pulp. Within the red pulp there are focal aggregates of erythrocytes (haemorrhage) and moderate numbers of multinucleated cells (megacaryocytes; extramedullary haematopoiesis). as well as moderate numbers of macrophages, some of which are degenerate, with hypereosinophilic cytoplasm and pyknotic nuclei.

Small intestine. There is diffuse loss of villous architecture with blunting, fusion and loss of surface epithelium. Focally, there is extensive loss of cellular detail, hypereosinophilia, karyorrhexis and karyolysis (necrosis) of the entire mucosa with fibrin deposition on the luminal surface and within fibrin and cellular debris, multifocal accumulations of short (3-4 μ m) rod-shaped bacteria, often in short chains. Adjacent to the necrotic material, extending into the lamina propria, submucosa, and focally transmurally, there is an extensive diffuse infiltration by lymphocytes, plasma cells and macrophages; numerous degenerate cells are seen. There are moderate numbers of extracellular erythrocytes (haemorrhage) and macrophages with intracellular erythrocytes (erythrophagocytosis). Mesothelial cells generally appear enlarged and bulge outward (diffuse activation) and focally, inflammatory infiltrates and fibrin are present within the adjacent adipose tissue. [In the second sections from the small intestine, there is also necrosis and depletion of the lymphatic tissue (Peyer's patches).]

2. MORPHOLOGIC DIAGNOSIS

Spleen; severe, acute, multifocal necrosuppurative splenitis.

Small intestine; severe, subacute (multi)focal fibrinonecrotic enteritis with myriad coccobacilli and (with necrosis/depletion of Peyer's patches) and focal inflammation of the adjacent mesentery.

Mesenteric lymph node; severe, acute, multifocal, necrotising lymphadenitis.

Tonsil; severe, acute, multifocal, necrotising tonsillitis.

3. NAME THE DISEASE

Tularaemia

4. ETIOLOGY

Francisella tularensis