

**The Armed Forces Institute of Pathology  
Department of Veterinary Pathology  
WEDNESDAY SLIDE CONFERENCE  
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**CONFERENCE 14  
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**CASE I - D01-7839 (AFIP 2789808)**

**Signalment:** Tissue section is from a 10-year-old spayed female, mixed breed canine.

**History:** The dog was presented with heartworm disease of unknown duration. One week before presentation the dog was treated with a filaricide. There were no adverse effects during or following treatment. The day before presentation, severe respiratory compromise developed. The clinician suspected possible pulmonary embolism. The animal did not respond to treatment with dexamethosone sodium phosphate and prednisone. The dog expired following severe respiratory compromise and cardiovascular collapse.

**Gross Pathology:** Necropsy revealed adult heartworms in the pulmonary vasculature.

**Laboratory Results:** None reported.

**Contributor's Morphologic Diagnosis:** Dog, lung: Interstitial pneumonia, marked, diffuse, suppurative with thrombosis, microfilaria and adult *Dirofilaria immitis*.

**Contributor's Comment:** A large area of the lung section is characterized by necrosis, hemorrhage and infarction. In adjacent viable lung, alveoli contain neutrophils, macrophages and red blood cells. Within large pulmonary arteries, thrombosis is evident and associated with intravascular nematodes. A sagittal section of an adult *Dirofilaria immitis* illustrates coelomyarian muscles, gravid uterus and gut. Microfilariae are evident within the small vessels and capillaries of the lung.

Heartworm disease is a common infection of Canidae and Felidae of the southern and eastern coastal area of the United States. There are many genera of filarial parasites that can infect both man and animal. *Dirofilaria immitis* is the filarial parasite of most importance.

The adult worms of *Dirofilaria* lodge in pulmonary arteries and right ventricle of the heart interfering with blood circulation and causing many, and sometimes fatal, clinical manifestations. These manifestations are shortness of breath, weakness, cardiac enlargement, hepatomegaly, ascites and hypertrophic pulmonary osteoarthropathy. The lodged adult female worms discharge microfilariae into the bloodstream where mosquitoes ingest them during feeding. These microfilariae then mature into the infective state (L3 larvae). Given proper environmental conditions, the microfilariae are deposited into the skin of an animal during the mosquito's next feeding. The L3 larvae then molt and migrate to the pulmonary arteries and mature into adult nematodes.

Diagnosing dirofilariasis is possible with the following procedures: Knott's test, ECG, thoracic radiographs, IFA for microfilariae, ELISA for adult antibody (cats only), ELISA for adult antigen and arterogram. Treatment of infected animals requires adulticide, microfilaricide and preventative for re-infection with microfilaria.

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**AFIP Diagnosis:** Lung: Pneumonia, necrosuppurative, diffuse, severe, with hemorrhage, fibrin, thrombi, microfilaria, and intravascular adult nematodes, etiology consistent with *Dirofilaria immitis*, mixed breed, canine.

**Conference Comment:** This case was reviewed in consultation with Dr. Chris Gardiner, parasitology consultant to the AFIP. Identification of this filarid is based on its size, thick cuticle, paired uteri, and a very small intestinal diameter. On some oblique sections, the lateral internal ridges in the lateral chord area can be identified. Some slides show pyogranulomatous inflammation associated with microfilariae. There are aggregates of free and phagocytized debris from dead microfilariae.

Conference attendees discussed the presence of smooth muscle hypertrophy in smaller vessels. In addition to pulmonary hypertension-induced smooth muscle hypertrophy, the role of platelet-derived growth factor (PDGF) was discussed. Vascular injury disrupts the balance between growth inhibition and growth promotion, favoring smooth muscle cell growth. When endothelial cells are injured PDGF is released, mediated by thrombin, promoting the migratory and proliferative activity of smooth muscle cells.<sup>4</sup>

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**CASE II - 25683-03 (AFIP 2893040)**

**Signalment:** Two-day-old, male, cross-breed calf, (*Bos taurus*).

**History:** Calf appeared to be normal at birth and nursed well. Unexpected death.

**Gross Pathology:** Heart and skeletal muscles were reported to be pale in appearance.

**Laboratory Results:** No ancillary tests were performed.

**Contributor's Morphologic Diagnoses:** Severe acute to subacute multifocally disseminated necrotic myocarditis.

**Contributor's Comment:** These sections of heart are characterized by randomly arranged broad areas of necrosis. There is modest diffuse interstitial hemorrhage throughout the necrotic areas. Nuclear pyknosis, karyorrhexis, loss of striations, and granular eosinophilic cytoplasm are noted in necrotic myocytes. Hypercontraction bands are present in low numbers of necrotic myocytes. Occasional aggregates of mineral can be seen in necrotic myocytes. There are foci of interstitial inflammation consisting of neutrophils and macrophages in some areas. Fibrinoid necrosis of tunica media of occasional arteries can be seen.

These lesions are consistent with what has been reported for nutritional cardiomyopathy due to selenium or vitamin E deficiency.<sup>1</sup> This case is somewhat unusual compared to what is described for the typical age of onset of this disease.<sup>1</sup> A report of a recent investigation indicated bovine fetal deaths with myocardial lesions and heart failure were associated with selenium deficiency.<sup>2</sup> Liver lesions of heart failure were also reported.<sup>2</sup> In the present case, there were histologic changes in the liver consistent with heart failure, mainly periportal fibrosis.

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**AFIP Diagnosis:** Myocardium: Necrosis, multifocal and coalescing, with fibrinoid vasculitis and edema, cross-breed, bovine.

**Conference Comment:** Vitamin E is a fat-soluble vitamin and an important antioxidant, acting as a free radical scavenger. Selenium is an essential component of glutathione peroxidase, which catalyzes the breakdown of free radicals. A deficiency of either vitamin E or selenium results in increased levels of free radicals, which cause membrane damage and cellular destruction.<sup>3,4</sup>

Common gross lesions of vitamin E/selenium deficiency in different species were discussed. Typical gross findings of skeletal muscle necrosis (white muscle disease) may be seen in most species, including cattle, sheep, goats, horses, pigs, dogs, mink, rats, mice, rabbits, guinea pigs, nonhuman primates, and humans. Mulberry heart disease and hepatosis dietetica are diseases of young swine. Nodular panniculitis and steatitis (yellow fat disease) are most common in cats, mink, and piscivorous birds. Intestinal lipofuscinosis (brown dog gut) is a characteristic finding in dogs. Chickens develop encephalomalacia (crazy chick disease) and turkeys develop encephalomalacia with hemorrhage (cherry red cerebellum).<sup>4</sup>

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**CASE III - 2-1000 (AFIP 2888622)**

**Signalment:** 4 month old, female, DSH kitten.

**History:** This kitten presented with a 5-day course of vomiting and mucoid diarrhea. It was also febrile and had experienced significant weight loss. On physical examination the animal was emaciated and slightly icteric. Abdominal palpation revealed a firm, small abdominal mass.

**Gross Pathology:** On laparotomy, there was a focal enlargement of the distal ileum extending into the ileocecal junction and the ileocecal lymph node complex was moderately enlarged. No intussusception was noted. A block resection of the ileocecolic region was submitted.

**Laboratory Results:** None reported.

**Contributor's Morphologic Diagnoses:**

Intestine/Cecum:

1. Enteritis/typhlitis, necrotizing and ulcerative, transmural, with extensive submucosal expansion by multifocal and coalescing nodules of pyogranulomatous inflammation & edema and extension into adjacent mesentery (some slides also have focally extensive regions of mucosal ulceration associated with a severe necro-suppurative process).
2. Angiitis, with areas of fibrinoid vascular wall necrosis.

Mesenteric lymph node:

1. Lymphadenitis, necrotizing and pyogranulomatous with extensive nodal effacement and areas of fibrinoid necrosis and angiitis.
2. Lymphoid depletion, diffuse, moderate-marked.

**Contributor's Comment:** The microscopic lesions varied somewhat in the different conference slides submitted, depending on what portion of the block resection submission (i.e. ileum, cecum and/or mesenteric lymph node) was represented.

The clinical presentation, gross lesions and microscopic findings are all consistent with an uncommon, but well described variant of the non-effusive form of FIP in which the disease manifests initially as a localized/segmental transmural swelling in the ileocecal region<sup>1</sup>. This process is readily detectable clinically as a palpable abdominal mass.

Immunohistochemical evaluation of these tissues revealed strongly positive staining for antigen in the cytoplasm of macrophages in pyogranulomas located in the submucosa of the intestine as well as mesenteric lymph nodes.

Feline Infectious Peritonitis (FIP) is a progressive, fatal disease caused by a coronavirus. The condition is described in numerous felids and develops predominantly in younger animals, although any age may be affected. Disease exists in two clinical forms, effusive (wet) and non-effusive (dry)<sup>2</sup>. The wet/effusive form manifests as a characteristic effusion in the thoracic or abdominal cavity associated with pyogranulomatous inflammation. This presentation occurs when there is a weak cell-mediated immune response and a strong humoral immune response by the host. The dry/non-effusive form is more variable with respect to lesions, often presenting more localized granulomas or pyogranulomas within solid abdominal organs, lungs, eye or CNS tissue. It develops when there is an inflammatory but non-protective cell-mediated immune response<sup>3</sup>. In general, cats with FIP virus (FIPV) infection that develop disease have significant depletion of both T and B cells in lymphoid tissue, whereas cats with FIPV infection and no disease show distinct lymphoid hyperplasia.<sup>4</sup>

The reason for the localized manifestation of this form of the disease (vs. the multi-organ pyogranulomatous disease process generally seen in cats affected with the non-effusive form of FIP) is not clear. The pathogenesis may involve a partial cell-mediated immune reaction that initially restricts the virus to macrophages in focal segments of the intestine, but does not eliminate the virus - causing the development of a localized, chronic-active inflammatory process. In all cats reported with this condition, the lesions

progressed to multisystemic FIP. This variant, therefore, does carry the same grave prognosis as other forms of the disease.

Submitting veterinarians often tentatively diagnosis this condition as lymphosarcoma, based on the presence of a relatively circumscribed firm mass in the ileocecal region. Although the prognosis for both conditions (FIP and intestinal lymphoma) is poor, the distinction is critical to prevent exposure and infection of other susceptible cats.

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**AFIP Diagnoses:**

1. Intestine: Enteritis, pyogranulomatous, transmural, diffuse, severe, with multifocal vasculitis, domestic shorthair, feline.
2. Lymph node and associated mesentery: Lymphadenitis and serositis, pyogranulomatous, diffuse, severe, with necrosis and vasculitis.

**Conference Comment:** In sections with intestine, conference attendees had either sections of ileum, with villus blunting and fusion, or colon. There is variation in vasculitis among slides, from fibrinoid necrosis to vasculitis with neutrophilic infiltrates and necrotic debris. Conference attendees discussed how both type III and type IV hypersensitivity reactions are involved in the pathogenesis of FIP.

Cats are likely infected by exposure to exogenous virus via the oro-nasal route or by mutation of an endogenous enteric coronavirus. The virus replicates in the tonsil, lymph nodes, and intestine. After primary replication in lymphoid tissues a viremia occurs resulting in infection of macrophages in many tissues. As the contributor mentioned, disease occurs when the host fails to mount adequate cell-mediated immunity. If there is no cell-mediated immune response, macrophages infected with the virus accumulate in the perivascular spaces and interstitium of serous surfaces resulting in the wet, or effusive, form of FIP. If there is a weak cell-mediated immune response, fewer macrophages accumulate and there is decreased production of virus, resulting in the dry form of FIP. In either the wet or dry form of the disease the pathologic changes are induced by excess formation of antigen-antibody complexes (type III hypersensitivity). These complexes are phagocytized by macrophages and deposited in vessel walls. Complement fixation followed by neutrophil chemotaxis and macrophage activation culminates in tissue destruction. This Arthus reaction permits the effusion of protein-rich fluid and is most pronounced in the serosal surfaces, liver, and kidney.<sup>5,6</sup>

Due to similarities with other immunological granulomatous diseases, it is suggested that type IV hypersensitivity also plays a role in the pathogenesis of FIP. Immunohistochemical findings in such lesions include the presence of CD4+ T lymphocytes uniformly distributed throughout the lesion and CD8+ T lymphocytes at the periphery.<sup>7,8</sup> A type IV hypersensitivity pattern has also been detected in focally induced lesions of FIP, demonstrated by the progressive activation of CD4+ T lymphocytes and the presence of macrophages.<sup>8</sup>

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**CASE IV - UFSM-1 (AFIP 2897023)**

**Signalment:** One-year-old, castrated male, Holstein, bovine.

**History:** A disease characterized by severe respiratory distress was diagnosed in a herd of 23 dairy cattle in a small farm in southern Brazil. The first clinical signs developed one day after a batch of sweet potatoes (*Ipomoea batatas*) was introduced into the animals' feed, and the disease ran a clinical course of 2-4 days. Clinical signs included extended head and labored breathing, rapid respiratory rate (120 breaths/min) and rhythmical flaring of the nostrils. Five cattle were affected and three of them died (including the case cited in this report).

**Gross Pathology:** Gross lesions were similar in the 3 necropsied cattle, being restricted to the lungs and consisting mainly of emphysema and edema. The lungs were firm, rubbery, distended and failed to collapse when the thorax was open and were firm and rubbery (Fig. 1). Interstitial emphysema characterized by numerous air bubbles in the interlobular and subpleural spaces was prominent (Fig. 2). At the cut surface, the lobules and the interlobular and peribronchial spaces were distended by

gelatinous, light yellow material (edema) and air bubbles (Fig. 3). Multifocal, small (1-2 mm) white foci were distributed throughout the cut surface of the lung. Abundant white froth was observed within the trachea and major bronchi. Mediastinal lymph nodes were enlarged and moist.

**Laboratory Results:** Mycological cultures of the damaged sweet potatoes that were fed to the cows yielded *Fusarium solani* and *F. oxysporum*.

**Contributor's Morphologic Diagnosis:** Interstitial pneumonia with pulmonary edema and emphysema, acute, severe, Holstein, bovine.

**Etiologic diagnosis:** Toxic pneumonia

**Etiology:** Moldy sweet potato toxins

**Contributor's Comment:** The slides submitted are representative of the lesions found in all three cases, although they were from a single animal (a 1-year-old-calf). The interlobular septa are markedly distended by edema and emphysema, and the alveolar septa are thickened by edema, mononuclear infiltrate, and few neutrophils. Hyperplasia and hypertrophy can be observed in the epithelium of terminal bronchioles and alveolar ducts, imparting an adenomatous appearance to these structures. Numerous desquamated pneumocytes (occasionally forming syncytia) can be seen in the airways and alveolar spaces. Some alveoli have hyaline membranes. Reactive hyperplasia is observed in the mediastinal lymph nodes (slides not included).

The diagnosis of interstitial pneumonia caused by the ingestion of moldy sweet potatoes in the cases described here is based on epidemiology, clinical signs, laboratory results, necropsy findings and histopathology, all of which are similar to the description of this condition by several authors<sup>2,3,6,8,9</sup>.

The great majority of cases of interstitial pneumonia associated with the consumption of moldy sweet potatoes (*Ipomoea batatas*) are caused by contamination with the fungus *Fusarium solani*, although *F. fimbriata* and *F. oxysporum* are occasionally implicated<sup>10</sup>. It has been demonstrated that these *Fusarium* species have a stimulant effect on toxin production by sweet potatoes. Sweet potatoes, under stress caused by mechanical injury, insect invasion, treatment with exogenous chemicals, or microbial infection can produce 3-substituted furans; toxins that have the ability to cause lesions in lung cells of cattle, rats, rabbits and guinea-pigs<sup>3,6,9,10</sup>. These toxins are collectively referred to as "lung edema factor" and include 1-ipomeanol, 4-ipomeanol, 1,4-ipomeanol, 1,4-ipomeadiol and ipomeanine, which are responsible for the acute pulmonary edema and emphysema that occurs upon consumption of *Fusarium*-infected sweet potatoes. The pathogenesis of the poisoning by moldy sweet potatoes in cattle is similar to that of other interstitial pneumonias involved in ARDS. It consists basically of the generation of free radicals within type I pneumocytes and bronchiolar epithelia, which result in the death of these cells. Upon its arrival in the lung, 4-ipomeanol is activated through mixed function enzymes (oxidases) into potent lung toxins<sup>3</sup>. In



addition to the destruction of pneumocytes, 4-ipomeanol causes edema by the destruction of endothelial cells leading to the formation of hyaline membranes<sup>1</sup>. As time goes by, type II pneumocytes undergo cellular division, proliferate in great numbers and line the alveoli imparting the adenomatous histopathological appearance, characteristic of affected cattle. In those cattle that survive the more acute phase, there is accumulation of inflammatory cells and fibroblasts in the pulmonary interstitium.

Ingestion of moldy sweet potatoes has caused interstitial pneumonia in cattle in the USA<sup>8</sup>, Japan<sup>10</sup>, Australia<sup>3</sup>, Uruguay<sup>9</sup> and Brazil<sup>6</sup>. In the USA there was an outbreak of interstitial pneumonia associated with the consumption of hay of the pink half-runner bean (*Phaseolus vulgaris*), contaminated by the fungus *F. semitectum*<sup>5</sup>.

Clinical signs of interstitial pneumonia in cattle caused by the consumption of mold-damaged sweet potatoes are acute in onset and include tachypnea, tachycardia, hyperpnea and dyspnea. Loud expiratory grunting, frothing at the mouth, extension of the head and neck, and flaring of the nostrils will also be seen<sup>2,3,5,6,10</sup>. Signs usually occur within one day of exposure and death usually occurs 2 to 5 days later<sup>10</sup>.

Gross lesions and histopathology are rather characteristic of the condition and, when associated to right epidemiological, clinical and laboratory data, allow for a definite diagnosis. There are, however, several other causes producing clinical signs and lesions in cattle remarkably similar to those described here and should thus be included in the differential diagnosis. These conditions were grouped in the past under the term "atypical interstitial pneumonia" (AIP), because many causes of these pneumonias were unknown. In recent years however, the term AIP tends to be replaced by the designation "acute respiratory distress syndrome" (ARDS) of cattle. The various causes of ARDS include<sup>4</sup> 1) ingestion of moldy sweet potatoes; 2) extrinsic allergic alveolitis (hypersensitivity pneumonitis) caused by exposure to the dust from moldy hay or other plant matter contaminated by *Micropolyspora faeni* or *Thermoactinomyces vulgaris*<sup>4</sup>; 3) acute bovine pulmonary edema and emphysema (ABPE), also known as "fog fever" which occurs in cattle which are changed from dry, sparse forages to green pastures, and is caused by the conversion of L-tryptophan present in the lush green forages to 3-methylindole; 4) reinfection syndrome, i.e., hypersensitivity to the lungworm *Dictyocaulus viviparus* infection; 5) poisoning by plant fungal toxins such as perilla (*Perilla frutescens*) ketone, stinkwood (*Zieria arborescens*), rape, kale and turnip tops (*Brassica* spp.), *Crotalaria* spp., *Acremonium lolii* contaminated ryegrass<sup>7</sup>; and 6) poisonous gases such as nitrogen dioxide produced by anaerobic fermentation of green plant material ("silo gas"), zinc oxide, chlorine, and manure gases (mixture of hydrogen sulfide, ammonia, carbon dioxide, methane and carbon monoxide).

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**AFIP Diagnosis:** Lung: Pneumonia, interstitial, acute, diffuse, severe, with interstitial edema and emphysema, hyaline membranes, and type II pneumocyte hyperplasia, Holstein, bovine.

**Conference Comment:** The contributor gives an excellent review of pneumonia caused by moldy sweet potatoes and the differential diagnosis for bovine interstitial pneumonia. Conference attendees discussed the prominent hyaline membranes in this case. Hyaline membrane formation, typical of the acute phase of interstitial pneumonia, is evidence of significant cellular injury caused by the mixture of protein-rich edema fluid and remnants of necrotic cells.<sup>11</sup>

Conference attendees discussed the importance of the P450 enzyme system in Clara cells in the pathogenesis of acute bovine pulmonary edema (fog fever, named for regrowth - "foggage" - after hay or silage has been cut). When cattle are moved from dry to lush pasture with high concentrations of tryptophan, the tryptophan is converted in the rumen to 3-methylindole (3MI) and disseminated throughout the body. This metabolite is transformed by the P450 enzyme system of the lung to 3-methyleneindolenine (3MEIN), which damages cell membranes of bronchiolar cells and type I pneumocytes, and increases alveolar permeability leading to edema and interstitial pneumonia.<sup>12,13</sup>

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