The Armed Forces Institute of Pathology
Department of Veterinary Pathology

Conference Coordinator:
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WEDNESDAY SLIDE CONFERENCE 2008-2009

Conference 14

14 January 2009

Conference Moderator:

Dr. Bruce Williams, DVM, Diplomate ACVP

CASE I – Case 05-2373 (AFIP 3026964)

Signalment: Twelve-week-old female Chihuahua dog (*Canis familiaris*)

History: This puppy presented for evaluation of non-responsive hypoglycemia of several weeks duration. She subsequently developed diarrhea. On presentation blood glucose was 38 mg/dL (73-116), total protein was 2.6 g/dL (5.5-7.2), albumin was <1.0 g/dL (2.8-4) and PCV was 18%. Coccidial oocysts were found on fecal flotation and therapy was initiated. The puppy deteriorated over the next two days and eventually died.

Gross Pathology: The examined puppy was in decreased nutritional condition (BCS 1.5/5) with moderately to markedly reduced subcutaneous and intra-abdominal adipose tissue. There was scant adipose tissue present in the peri-renal mesentery and within the coronary groove of the heart. The stomach contained a small amount of tan mucoid ingesta, the small intestines segmentally contained scant beige pasty ingesta and there was no fecal material present in the colon. No other remarkable gross lesions were present.

Laboratory Results: None

Histopathologic Description: Jejunum: Diffusely there is marked blunting and fusion of villi **(Fig. 1-1).** The lamina propria is expanded by a cellular infiltrate composed of numerous neutrophils, lymphocytes, plasma cells, and macrophages that multifocally extend into the submucosa. Multifocally crypts are distended and filled with similar inflammatory cells admixed with pyknotic and karyorrhectic debris (crypt abscesses). Segmentally along villi there are small aggregates of 1x2 um bacilli that are intimately attached to the apical tips of enterocytes **(Fig. 1-2)**. Multifocally crypts are filled with aggregates of spirochete bacteria. Free in the lumen of the jejunum and attached to the apical surface of remaining villous epithelium are scattered 3-6um diameter round, amphophilic protozoal organisms (cryptosporidium) **(Fig. 1-3).**

Contributor's Morphologic Diagnosis: Diffuse, severe neutrophilic and lymphoplasmacytic enteritis with intralesional bacilli, cryptosporidia and spirochetes

Contributor's Comment: This puppy suffered from a severe, malabsorptive protein-losing enteropathy secondary to multiple pathogens. The most pathogenic organism in this case is likely enteroadherent (attaching-

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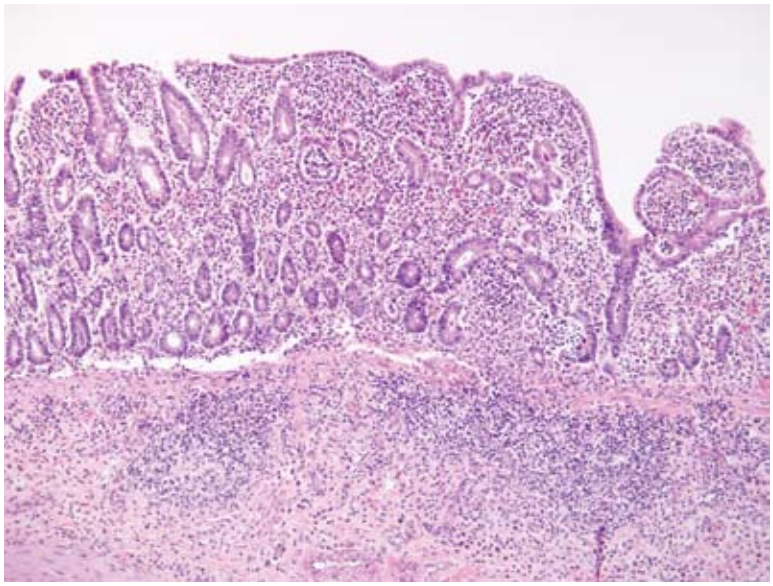
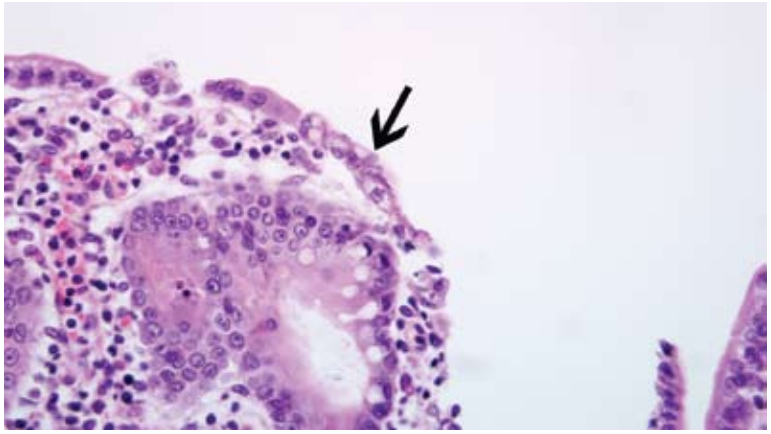
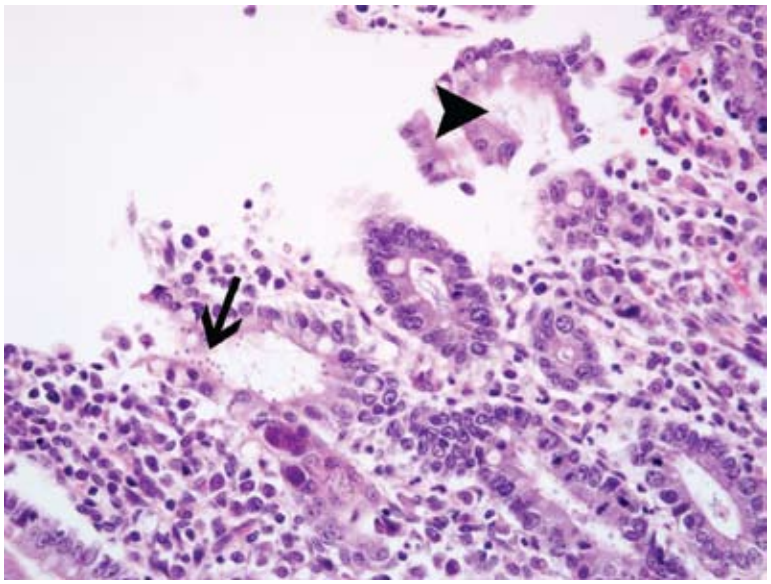


1-1. Small intestine, dog. Diffuse atrophy, blunting, and fusion of the villi with severe crypt loss. (HE 100X)



1-2. Small intestine, dog. Multifocally adherent to the enteric mucosa are small numbers of 2-6 micron round apicomplexan schizonts and gamonts (arrow). Crypts contain numerous helical bacteria which measure approximately 1x8 microns (arrowhead). (HE 400X)



1-3. Small intestine, dog. Randomly attached to the villar epithelium are small aggregates of robust bacilli which measure up to 2x3 microns (arrow). (HE 400X)

effacing) *Escherichia coli*. These bacteria are categorized as enteropathogenic *E. coli* (EPEC) and colonize the mucosa by a nonpilus adhesion termed EPEC adhesive factor(1). Concurrent infection with cryptosporidium and coccidia has been reported in an immunosuppressed puppy and they were considered opportunistic pathogens.[6] Cryptosporidium in dogs is seldom reported in the United States and typically occurs in immunosuppressed puppies. *Cryptosporidium parvum* and *C. canis* have been isolated from naturally infected dogs.[4] There was prominent lymphoid depletion present in the spleen and mesenteric lymph nodes, indicating this puppy was likely immunosuppressed secondary to viral infection or a primary underlying immunosuppression. There were no lesions of parvovirus or distemper.

No coccidial organisms were seen in multiple sections of intestine; however, there may have been low numbers of organisms due to the previous treatment. The numbers of spirochete bacteria are impressive in this case, but they are not a primary pathogen and this likely represents secondary opportunistic overgrowth. Weakly beta-haemolytic intestinal spirochaetes identified as *Brachyspira pilosicoli* have been isolated from puppies and dogs with diarrhea.[3,5] *Brachyspira canis* has been isolated from clinically healthy dogs, suggesting it is a commensal organism. [5]

AFIP Diagnosis: Small intestine (jejunum): Enteritis, subacute, diffuse, severe, with marked villus atrophy, fusion, and blunting, crypt necrosis and loss, and attaching bacilli, apicomplexans and intracrypt helical bacteria

Conference Comment: There are several different types of *E. coli* that affect domestic species and each type has virulence characteristics that manifest as varying disease entities. "Enteropathogenic" *E. coli* (EPEC) attaches to the mucosa and causes a malabsorptive diarrhea. Some strains of EPEC do not produce toxins, but they do cause blunting and fusion of villi with subsequent diarrhea. The nomenclature for *E. coli* can be extremely confusing, and this type of *E. coli* is also known as "enteroadherent" *E. coli* (EAEC), and "attaching and effacing" *E. coli.*[2]

EPEC attaches to a host enterocyte via long fimbria and subsequently releases proteins known as adhesins to form a secure attachment to the surface epithelium. Translocated intimin receptor, another protein produced by EPEC, is transported from the bacteria into the host cell. This causes a conformational change in the host cell's cytoskeleton. The affected enterocyte forms a "pedestal-like structure" beneath the bacteria and this unfortunate cell also loses its surface microvilli. The pathogenicity of EPEC is largely determined by the density of organisms on the surface of enterocytes. As the contributor mentioned, co-infections are common and lead to a much worse clinical picture. Bacterial attachment is most prolific in the distal small intestine and large intestine. Profuse diarrhea with EPEC results from a combination of maldigestion and malabsorption.[2]

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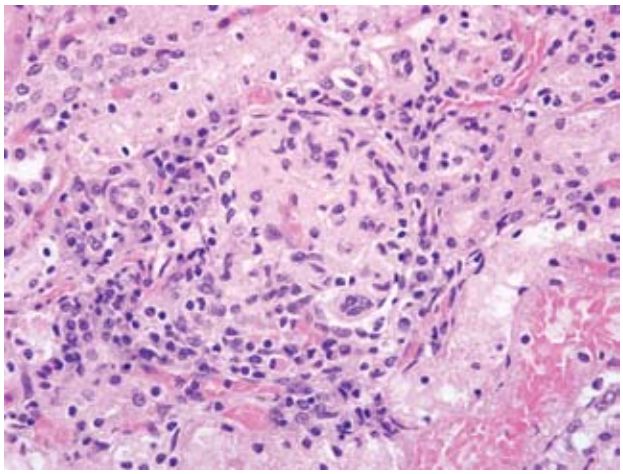
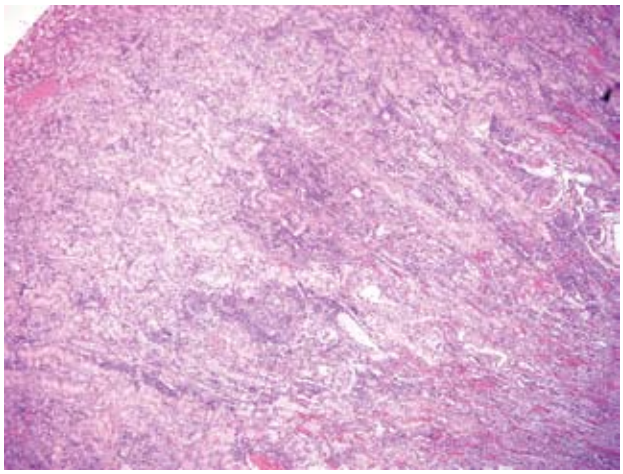
CASE II – G07-120059 (AFIP 3106379)

Signalment: Juvenile, female, Mahogany, (*Mustela vison*), mink

History: Farmed mink from a ranch with 9700 kits are experiencing reduced feed intake, increased mortality in this season's juveniles (averaging 10 deaths per week, recently up to 10 deaths per day). This ranch was cleaned and restocked 1.5 years ago. Kits are vaccinated with a four-way vaccine (against botulism, distemper, mink enteritis virus, *Pseudomonas aeruginosa*) at 10 weeks of age; no Aleutian disease testing has been done since restocking.



2-1. Kidney, mink. Multifocally, the cortical perivascular interstitium is expanded by a cellular infiltrate which occasionally surrounds, separates, or replaces tubules and glomeruli. (HE 40X)



2-2. Kidney, mink. There is increased cellularity of the glomerular tufts with glomerular and periglomerular infiltrate of low numbers of neutrophils, macrophages, and lymphocytes; the mesangial matrix is expanded by a homogenous eosinophilic material. (HE 400X)

Gross Pathology: This female mink exhibited pulmonary congestion and edema, splenic enlargement and an empty gastrointestinal tract on gross postmortem.

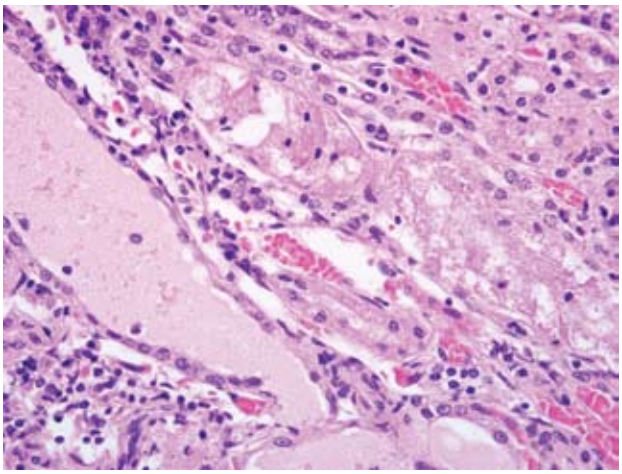
Laboratory Results: Seven of eight serum samples submitted tested POSITIVE for Aleutian disease parvovirus antibodies by counterimmunoelectrophoresis (CIE).
Spleen tissue from this mink tested positive for Aleutian disease parvovirus by PCR.

Histopathologic Description: Kidney: Glomeruli throughout the section exhibit a variety of lesions, including increased cellularity of the glomerular tuft with segmental to diffuse mesangial hyperplasia and hyalinization, and thickened glomerular basement membranes and Bowman's capsules **(Fig. 2-1)**. Some glomeruli are obsolescent. There are prominent interstitial infiltrates of plasma cells and lymphocytes, with associated tubular degeneration **(Fig. 2-2, 2-3)**. Many tubules contain protein or cellular casts. Occasional small to medium-sized arterioles display fibrinoid necrosis of vessel walls, with infiltrating inflammatory cells and cuffs of mixed inflammatory cells **(Fig. 2-4)**.

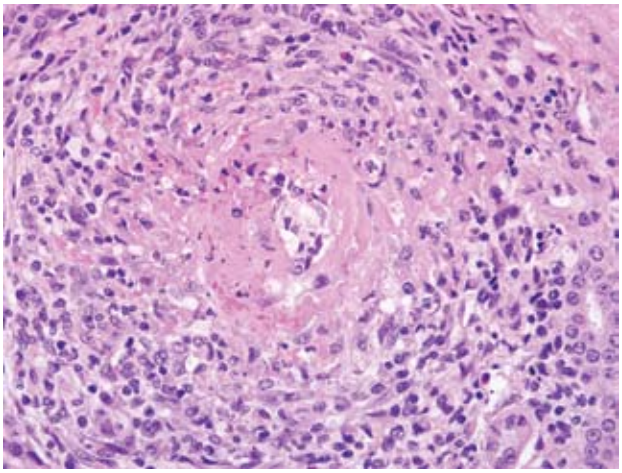
Urinary bladder: Arteries within the muscular wall and adjacent mesentery are surrounded by thick concentric cuffs of mixed inflammatory cells, including macrophages, plasma cells, neutrophils and eosinophils. There is fibrinoid necrosis of the vessel walls, some with infiltrating inflammatory cells.

Contributor's Morphologic Diagnosis:
Generalized, segmental to diffuse glomerulonephritis
Interstitial nephritis
Necrotizing arteritis, kidney, urinary bladder

Contributor's Comment: The histologic lesions in this case are compatible with Aleutian disease. This diagnosis is supported by the positive serology testing and confirmed by PCR. Aleutian disease, a chronic progressive disease of mink caused by the Aleutian disease parvovirus, is considered the most important disease affecting mink production worldwide, causing increased early kit mortality and chronic disease with high mortality in juveniles and adults.[3] Disease severity and development of lesions is dependent on the age and genetics of the mink, and the virulence of the strain of infecting virus.[2] While kits succumb to interstitial pneumonia and respiratory failure, the chronic form of the disease is result of immune complex glomerulonephritis and vasculitis, and death is usually a result of renal failure. The humoral immune response plays a direct role in the pathogenesis of Aleutian disease.[1] Antiviral antibody can be detected as early as five days post-infection, with development of a marked, progressive polyclonal hypergammaglobulinemia. While antibody can have a partially protective role via viral neutralization and incomplete restriction of virus replication in ACV-infected mink kits, it does not prevent infection. In older animals, antiviral antibody is thought to exacerbate disease through a variety of mechanisms, including antibody-dependent enhancement of infection (where antibody bound to virus facilitates viral entry into macrophages via Fc receptors;



2-3. Kidney, mink. The walls of small vessels contain abundant brightly eosinophilic proteinaceous material, cellular debris, and low numbers of neutrophils and lymphocytes which occasionally occludes vessel lumens (fibrinoid necrosis). Perivascular interstitium is expanded by moderate numbers of neutrophils, lymphocytes, plasma cells, and histiocytes. (HE 400X)



2-4. Kidney, mink. Multifocally, renal tubule epithelia are attenuated, degenerative, or necrotic, and tubules are often ectatic and contain a brightly eosinophilic proteinaceous material. (HE 400X)

Fc receptor binding stimulates IL-10 production, which inhibits interferon signaling, promotes antibody production and suppresses cytotoxic T-cell-mediated killing of virus-infected cells), and formation and tissue deposition of immune complexes, with subsequent development of immune-complex vasculitis and glomerulonephritis.

AFIP Diagnosis: Kidney: Glomerulonephritis, membranoproliferative and necrotizing, diffuse, moderate with multifocal necrotizing arteriolitis, subacute interstitial nephritis, and rare protein casts

Conference Comment: Aleutian disease was first reported in 1956 in Aleutian mink homozygous for the gene that is responsible for their steel- blue color. Mink and ferrets are susceptible to Aleutian mink disease (ADV). Genetic susceptibility plays a major role in ADV infections. Mink homozygous for the autosomal recessive Aleutian gene are more severely affected by ADV often resulting in death. ADV can cause disease in other types of mink ranging from death to subclinical infection.[2]

During the conference discussion Dr. Williams stated that the lesions in this case were quite striking and more severe than most cases of ADV he has seen. As the contributor mentioned, kits get an interstitial pneumonia, and type II pneumocytes are the primary site of replication leading to death of infected cells and a fulminant pneumonia with death. Rarely intranuclear inclusions are found in pneumocytes, and severe cases of pneumonia can lead to alveolar hyaline membrane formation.[1] Infection of adults leads to an insidious chronic form with splenomegaly, lymphadenopathy, hypergammaglobulinemia, and acute interstitial nephritis.[1] Chronic disease causes death via uremia and kidney failure.[3]

Gross lesions in the chronic form include ulceration of the mouth, tongue, footpad, and stomach secondary to uremia.[3] Histologic lesions in the chronic form are impressive and consist of lymphoplasmacytic interstitial nephritis, marked glomerulonephritis, and a necrotizing vasculitis of the small and medium sized arteries.[3]

Contributing Institution: Animal Health Laboratory, University of Guelph, Guelph, Ontario, Canada
http://ahl.uoguelph.ca

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CASE III – 06-34302 (AFIP 3027307)

Signalment: 3-week-old female miniature donkey, (*Equus asinus*)

History: Found in recumbency, treated with IV fluids and antibiotics. Brown urine was discharged shortly before death.

Gross Pathology: The lungs were edematous. Several ulcers up to 5mm length were present in the squamous mucosa of the stomach. The urinary bladder contained brown urine.

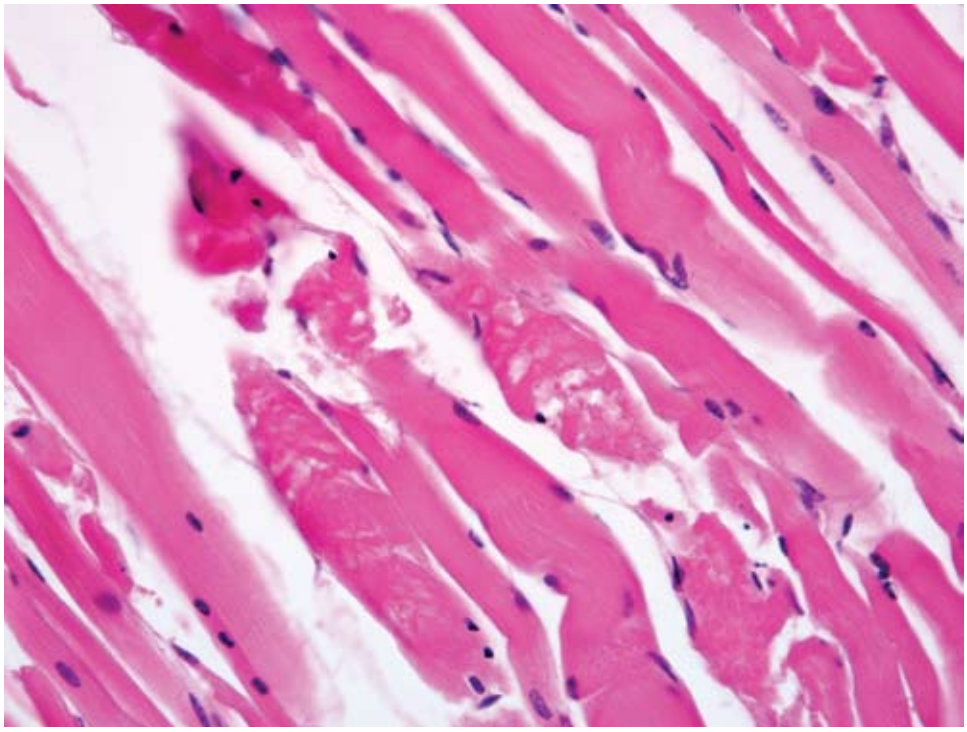
Laboratory Results: Liver selenium concentration was 0.7 ug/g (reference range 0.7-2.0 ug/g); liver Vitamin E concentration was < 2.5 ug/g (reference range 15.0-25.0 ug/g).

Histopathologic Description: There is diffuse to segmental degeneration and necrosis of skeletal muscle fibers, characterized by fragmentation of fibers and condensation of the sarcoplasm into hypereosinophilic coagulum with loss of cross striations **(Fig. 3-1)**. There is eosinophilic, granular to fibrillary debris within the fibers and extending into the interstitium around the affected fibers. Rarely, macrophages or satellite cells surround affected fibers.

Contributor's Morphologic Diagnosis: Skeletal muscle myofiber degeneration

Contributor's Comment: Nutritional myopathy was diagnosed in this donkey foal based on the muscle lesions and extremely low concentration of vitamin E in the liver. Selenium concentration in the liver was in the low normal range. Nutritional myopathy usually occurs in young animals on diets deficient in selenium and/or vitamin E, with or without the conditioning factors such as an excessive quantitiy of polyunsaturated fatty acids in the diet.[5] Vitamin E is an antioxidant that prevents oxidative damage to sensitive membrane lipids by decreasing hydroperoxide formation.[3] Selenium is an essential component of the enzyme glutathione peroxidase (GSH-PX). GSH-PX catalyzes the breakdown of hydrogen peroxide and other organic hydroperoxides produced by glutathione during the process of redox cycling.

Microscopic lesions similar to those in skeletal muscle were also present in the heart of this donkey, but were more mild. Granular eosinophilic material compatible with hemoglobin or myoglobin was present in tubular lumens of the kidney. This material is most likely myoglobin released from the affected skeletal muscle fibers. Although gross lesions were not detected in the heart or skeletal muscle of this foal, pale areas in the heart and skeletal muscle can be observed in cases



3-1. Skeletal muscle, donkey. Multifocally, there is random individual myocyte degeneration characterized by sarcoplasmic swelling, pallor, and vacuolization, or myocyte necrosis characterized by sarcoplasmic hypereosinophilia, loss of cross-striations, fragmentation, pyknosis, and karyorrhexis. (HE 400X)

of nutritional myopathy of foals, however is not always observed.[7] Degenerate muscle may be very difficult to detect grossly when it is uncalcified, and is likely to escape detection.[3] Mineralization of muscle fibers is not always present in cases of nutritional myodegeneration in foals.[7]

There were approximately 50 female donkeys in this herd, with this being the only donkey diagnosed with nutritional myopathy, although foals had died in previous years with similar signs. The female donkeys were fed brome grass hay year-round, never being pastured on green grass, or supplemented with vitamins or minerals. Green, growing forages should provide adequate vitamin E as α-tocopherol, but the vitamin E content is greatly reduced in grass that is dried for hay. Mature plants contain less α-tocopherol than younger plants, and mature grass cut for hay can have loss of up to 80% of the tocopherols when dried in the sun for 4 days.[1] Plasma vitamin E status of horses is highest from May to August when fresh grass is being grazed and lowest when horses are fed harvested or stored feed during the same period.[2]

AFIP Diagnosis: Skeletal muscle: Degeneration and necrosis, multifocal, moderate

Conference Comment: Differentials for this lesion in horses were discussed during the conference and included white muscle disease, capture myopathy, exertional rhabdomyolysis, and toxic myopathy due to toxic plants and ionophores. Nutritional myopathy causes a polyphasic, multifocal lesion in affected muscle while ionophore toxicity causes monophasic, multifocal lesion thus allowing for microscopic differentiation of these two conditions.[6]

Nutritional myopathy is common in calves, lambs, swine, and foals.[6] It can be caused by a lack of dietary intake of vitamin E or selenium or from competitive binding of selenium by copper, zinc, silver, or tellurium. Foods high in polyunsaturated fats such as fish require intake of more vitamin E to minimize oxidative damage from the metabolic processing of these foods.[6]

Vitamin E and selenium are important in preventing damage from free radicals from both within and from outside the cell. Free radicals are molecules with an odd number of electrons produced during normal cell functions or from tissue radiation, drug reactions, or inflammation. Free radicals are highly reactive molecules that can cause damage to mitochondria, endoplasmic reticulum, or the cytosol via damage to important cellular proteins or peroxidation and damage of cellular lipid membranes. When cellular membranes are damaged, ion gradients can not be properly maintained. Extracellular calcium moves into the cytosol and the cell responds by attempting to protect calcium-sensitive myofilaments by pushing calcium into mitochondria. Mitochondria quickly accumulate excess calcium and lose their ability to produce energy for the cell. Myofibrils exposed to leaking calcium hypercontract leading to degeneration and necrosis of myofibers.[6]

The attached table provides a non-comprehensive list of diseases considered to be associated with an imbalance or deficiency in either selenium or vitamin E.

Cattle	Nutritional myopathy Retention of fetal membranes
Horse	Nutritional myopathy
Swine	Mulberry heart disease Hepatosis dietetica Exudative diathesis Iron hypersensitivity Nutritional myopathy Anemia
Sheep	Nutritional myopathy Infertility Poor growth potential
Dogs	Intestinal liposfuscinosis
Cats; mink; birds; pigs; rabbits; reptiles	Steatitis (yellow fat disease)
Chickens and Turkeys	Encephalomalacia (superficial cerebellar hemorrhage – crazy chick disease)

[4,5]

Contributing Institution: Kansas State University, http://www.vet.k-state.edu/depts/dmp/

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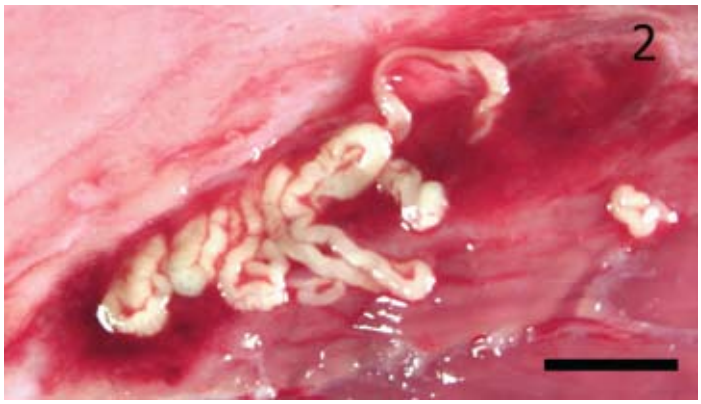
CASE IV – 07-52047 (AFIP 3096747)

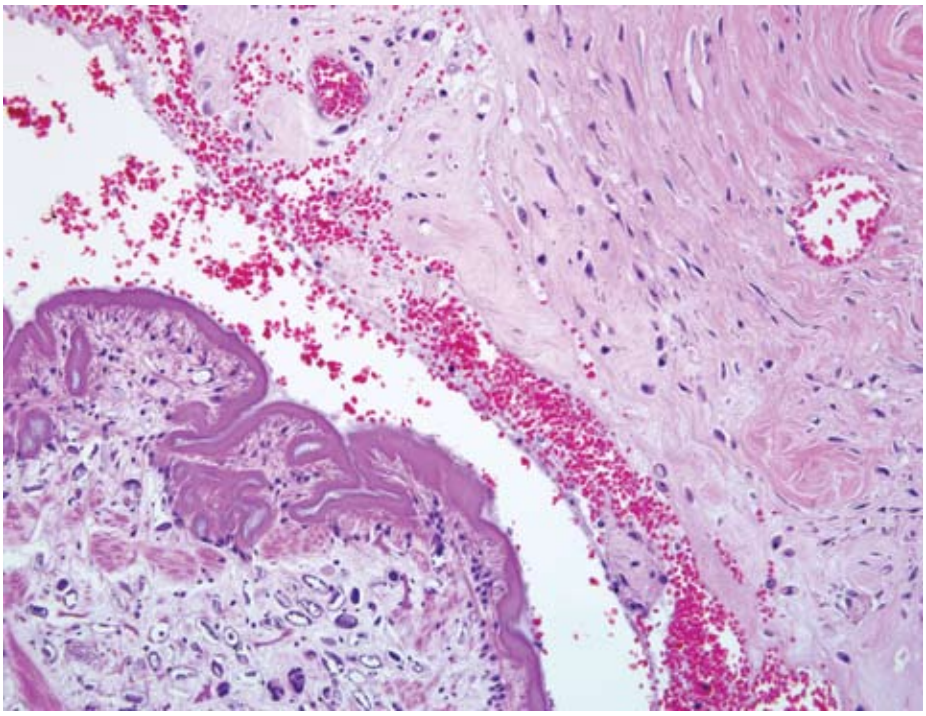
Signalment: 21-month-old spayed female border collie, (*Canis familiaris*) dog

History: The dog lived near Tampa, Florida and was maintained in a fenced yard containing a small area of marshy terrain. The presenting history included progressive lameness, pain, and subcutaneous edema of the right forelimb. Over an eight-week period, the dog developed worsening fever, dyspnea, mature neutrophilia, and hypoproteinemia that did not respond to symptomatic treatment or antibiotic therapy. The skin of the right axilla and forelimb contained several well defined areas of deep red discoloration overlying 1-3 cm diameter, fluctuant subcutaneous nodules that extended into subjacent soft tissues. Radiographs revealed linear areas of radiolucency in the soft tissue of the right shoulder region. Surgical pathology of a nodule revealed fibroplasia and chronic panniculitis. Later, a surgical incision into a nodule of the right axilla revealed a 2-cm diameter cystic cavity containing coiled aggregates of many (>10) intact and fragmented white worms ranging from 10-30 mm in length and 2-4 mm in width. The dog was treated with praziquantel (generic, Phoenix Scientific, Vedco, 20mg/kg PO SID x 5 days) and fenbendazole (Panacur®, DPT Laboratories, Intervet, 50 mg/kg PO SID x 3 days), in addition to antibiotics. Upon identification of the worms as plerocercoids (spargana) of a pseudophyllidian tapeworm, anthelmentic therapy was changed to cefpodoxime proxetil (Simplicef®, Pfizer, 2.5 mg/kg PO SID x 10 days), metronidazole (generic, PLIVA, Inc, 24 mg/kg PO BID x 7 days), and fenbendazole (100 mg/kg, PO, BID). Praziquantel (Praziject, IVX Animal Health, Inc., 50 mg/kg SQ divided among six sites) was given once a week for 3 weeks. After two weeks of clinical improvement, new nodules developed over the ventral chest, neck, right axillary region, and these encompassed cystic spaces containing many spargana. A right pleural cavity effusion developed, and approximately 500 ml of cloudy serosanguinous fluid were removed by thoracocentesis. Complete blood count and serum biochemical profile revealed mild anemia, normal WBC with mild monocytosis, and moderate hypoalbuminemia. Bacterial culture and sensitivity of the fluid identified *Pseudomonas aeruginosa*, and enrofloxacin therapy (Baytril®, Bayer, 3.5 mg/kg PO BID) was initiated. Praziquantel (Biltricide®, Bayer) was administered at 30 mg/kg PO SID for 8 days. Within two weeks, the dog developed a peritoneal exudate. The dog's overall condition continued to deteriorate and the owners authorized euthanasia and necropsy.

Gross Pathology: The subcutis and intermuscular fascia of the right forelimb, right axilla, ventral thoracic midline, and ventral cervical region contained many inflammatory tissue cysts filled with nodules of entangled intact and fragmented ribbon-shaped white larval cestodes (spargana) surrounded by red, cloudy, thick fluid **(Fig. 4-1)**. There was severe atelectasis of the right lung, and the right pleural cavity contained about 150 ml of thick, cloudy, tan fluid with two larval cestodes and scattered

4-1. Skeletal muscle, dog. Forming a cyst within subcuticular skeletal muscle are multiple cestode larvae. Photograph courtesy of the Department of Pathobiology, College of Veterinary Medicine, Auburn University, Auburn, Alabama.





4-2. Skeletal muscle and subcutis, dog. The cestode larva is characterized by a 7-10 micron thick tegument with small fibrilated projections (microtriches), a spongy parenchymous body cavity without a pseudocoelom, a row of somatic cell nuclei immediately subjacent to the tegument, numerous calcareous corpuscles, and numerous tortuous branching invaginations of the tegument (excretory ducts). The cyst wall is composed of fibrous connective tissue admixed with moderate amounts of hemorrhage and fibrin, and low numbers of neutrophils, eosinophils, macrophages, and lymphocytes bounded by large amounts of granulation tissue. (HE 200X)

white, friable fragments. There was partial atelectasis of the left lung, and the left pleural cavity contained about 100 ml of cloudy tan fluid with several free-floating larval cestodes. Microscopic examination of the pleural fluid after Wright-Giemsa staining revealed many bacteria both extracellularly and within neutrophils. The peritoneal cavity contained about 250 ml of red cloudy fluid in the caudoventral region containing many bacteria and segmented neutrophils. There were many fibrous adhesions between the omentum and serosal surfaces of the small intestine, spleen, and stomach. At least two larval cestodes were present in the peritoneal fluid.

Laboratory Results: Samples of the parasites were submitted to the Diagnostic Parasitology Service, Department of Pathobiology, College of Veterinary Medicine, Auburn University for identification. The worms were identified as larval cestodes (plerocercoids or spargana) of a pseudophyllidian tapeworm, most likely, *Spirometra* sp. Nucleic acids were extracted from intact, frozen spargana with a robotic extractor (Maxwell® 16, Promega Corporation) and used as template in PCR of an 18S rDNA fragment employing eucestode primers 84 and 90 as previously described.[7] Amplicons were sequenced (courtesy of Dr. Susan E. Little and M.D. West, Oklahoma State University) using an ABI3730 capillary sequencer and the sequence compared to those previously reported from *Spirometra erinacei* (D64072), *Diphyllobothrium latum* (AM778553), *Mesocestoides corti* (AF286984), and *Taenia solium* (DQ157224). Sequence of the spargana from this dog (EU392209) most closely resembled (99.4% identical) that previously reported from *Spirometra erinacei*, a pseudophyllidian cestode.

Histopathologic Description: Tissues from the skin and soft tissues of the right axilla are submitted. The deep dermis and subcutis of haired skin contain a parasitic cyst with a wall comprised of fibrous connective tissue and a lumen partially filled with larval cestodes, blood, fibrin, and proteinaceous fluid **(Fig. 4-2)**. Macrophages and neutrophils are rare in the region of the cyst, and there is congestion of surrounding vasculature. The larval cestodes lack discernible scolices or suckers and have shallow invaginations of the deeply eosinophilic tegument, resulting in segmentation and formation of tortuous parenchymal cavities (excretory ducts) filled with intensely eosinophilic granular substance. Microtriches occasionally project from the surface of the tegument. Columnar cells (subtegumentary cells) are often located in a parallel row beneath the tegument. The larval body is comprised of a fine fibrillar stroma with many calcareous corpuscles and a few striated muscle fibers, sometimes arranged as loose bundles beneath the tegument. The overlying skin is characterized by hyperkeratosis, epidermal atrophy, follicular keratosis, dermal edema, and venous congestion.

Contributor's Morphologic Diagnosis: Tissue from right axilla: Subcutaneous parasitic cyst, with intralesional larval cestodes and mild focal granulomatous

panniculitis

Contributor's Comment: The progressive disease in this young adult dog was attributed to proliferative sparganosis, caused by proliferating larval cestodes (spargana) of the organism *Sparganum proliferum*.[9,12] The spargana were widely distributed throughout the subcutis and intermuscular fascia of the cranial half of the body, the pleural cavities, and the peritoneal cavity. Morbidity resulted from widespread parasitism and septic pleuritis and peritonitis due to *Pseudomonas aeruginosa* infection. Bacteria were presumably introduced through tracts established by the encysted parasites.

Sparganosis is a disease characterized by the presence of larval pseudophyllidian cestodes in the host's tissues.[13] Tapeworms may be characterized in tissue sections by the absence of a digestive tract, the presence of a thick layered cuticle with a basement membrane, the presence of calcareous corpuscles, and evidence of a segmented body. [4] Plerocercoid larvae are usually solid, club-shaped forms in which scolices and suckers are absent.

Spargana were located in tissue cysts. In histologic sections, there is abundant space around individual spargana (arrows) and the discernible capsule (C) is comprised of eosinophilic amorphous material and relatively few inflammatory cells. Spargana are characterized by invaginations of the tegument resulting in segmentation. Columnar subtegumentary cells form a row beneath the densely eosinophilic tegumentary syncytium, which is covered by a row of dense microtriches. The body is comprised of evenly distributed loose parenchyma with calcareous corpuscles (arrowheads), muscle fibers (M), and excretory ducts (E). Muscle fibers are loosely arranged in a discontinuous row that is oriented parallel to the tegument. Scolices or suckers are not evident.

There are two forms of sparganosis: non-proliferative and proliferative.[7,10] Most infections are of the non-proliferative type associated with the presence of a single larva of either *Spirometra erinaceieuropaei* or *Spirometra mansonoides*. Proliferative sparganosis is caused by the asexual replication of larvae of *Sparganum proliferum* in host tissues and the migration of these larvae to new tissues where they grow and repeat the process, ultimately resulting in the death of the host.[3] In 2001, *Sparganum proliferum* was identified phylogenetically as a new species in the order Pseudophyllidea.[9] The first human infection by *S. proliferum* in the United States was reported in 1908.[14] Infection by *S. proliferum* has been reported in cats, dogs, and feral hogs, but this appears to be the first case of canine proliferative sparganosis in North America.[1-3,6]

The life cycle of *S. proliferum* has not yet been confirmed [12] but probably resembles that of other members of a related pseudophyllidian tapeworm, *Spirometra* spp. Adult tapeworms reside in the intestinal tract of a carnivorous definitive host, where they shed operculated eggs in the feces following discharge from the uterine pore of adult tapeworms. The operculated eggs then hatch in water, releasing a ciliated intermediate form (coracidium). The coracidium is ingested by the first intermediate host, a copepod crustacean (*Cyclops* sp.), where it develops into the procercoid stage. After the infected copepod is ingested by any one of a broad array of possible second intermediate hosts (any vertebrate other than a fish), the procercoids develop into plerocercoids (spargana) and migrate throughout the soft tissues of the body. If the second intermediate host is eaten by another non-fish vertebrate serving as a transport host, the plerocercoids migrate through the tissues but may remain as plerocercoids. The larval *Spirometra* can infect and survive in a series of transport hosts until finally consumed by a carnivore definitive host.[7] The ova are released from the uterine pore and are evident in the host's feces 10-30 days after infection.[1,6,7,11] Infection of the dog can occur through three different ways: ingestion of contaminated water, direct infection of open wounds with plerocercoids, or ingestion of plerocercoids in intermediate vertebrate hosts [11,12] Due to its requirement for an aquatic primary intermediate host, clinical disease is usually associated with exposure to aquatic environments. Sparganosis is zoonotic; thus, precautions should be made to block human infection by preventing consumption of infected water and insufficiently cooked fish or game, or the application of infected medicinal poultices to wounds. It is interesting to note that the first human case of proliferative sparganosis in North America was reported in 1908 in a Florida resident living in the same geographic region as the current canine case.[14]

Currently, there are no products labeled for treatment of *Spirometra* spp infections.[7] The lack of treatment options for proliferative sparganosis warrants a poor prognosis for survival. Infection of dogs is best controlled by preventing the consumption of infected water or the ingestion of vertebrates that could serve as secondary intermediate hosts.

AFIP Diagnosis: Skeletal muscle: Rhabdomyositis and panniculitis, pyogranulomatous and eosinophilic, focally extensive, mild with encysted larval cestodes

Conference Comment: The contributor did an outstanding job of describing this parasite in depth and in full, so this comment will focus on distinguishing

trematodes from cestodes and some common larval forms of cestodes found in domestic animals.
Adult cestodes are normally present in the intestine of the final host with larval forms present in tissue or body cavities of unfortunate intermediate hosts. Cestodes are split into segmented sections called proglottids that contain both female and male reproductive organs. Both larval and adult cestodes have suckers on their anterior end that may also have hooks depending on the species of cestode.[5]

Several types of cystic larval cestodes are often seen in tissue sections, and these include cysticercoids, cysticercus, coenurus, and the hydatid cyst. Cysticercoids are tiny larvae with a very small bladder and scolex that is encircled by parenchymous tissue. Cysticercus can be identified by a bladder with an inverted neck and scolex that always has four suckers. Coenurus is very similar in appearance to cysticercus but has more than one scolex. Hydatid cysts have a bladder with large numbers of very small scolices. [5]

In tissue section trematodes and cestodes look very similar, but to the trained eye they can be differentiated by a few key features. Both cestodes and trematodes are described as having a spongy parenchyma with no body cavity. Cestodes lack a digestive tract in contrast to trematodes which are endowed with one. Cestodes have calcareous corpuscles which are basophilic clear corpuscles of unknown function. Trematodes are devoid of calcareous corpuscles.[5] These features can help to delineate these two similar appearing parasites.

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