The Armed Forces Institute of Pathology
Department of Veterinary Pathology

Conference Coordinator:
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Conference Moderator:

Dr. James Raymond, DVM, MS, DACVP

CASE I – S0704434(AFIP 3071894)

Signalment: Female adult Muscovy duck (*Cairina moschata*)

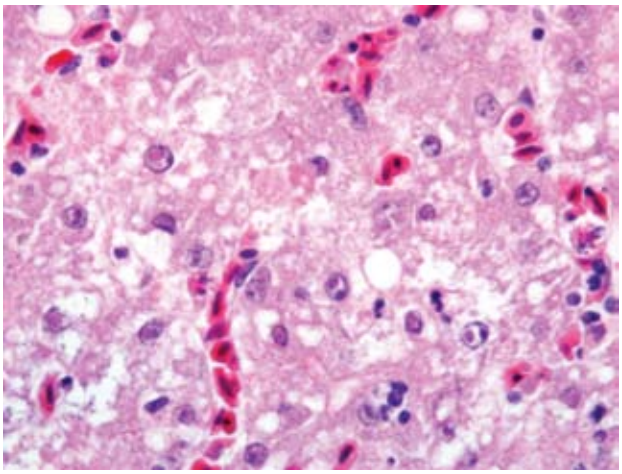
History: The duck was one of 2 birds to die from a total of 75 ducks. Submitted for necropsy. No clinical history was given.

Gross Pathology: The bird was in good flesh and tissues were in a good state of postmortem preservation. The liver was uniformly pale and there was generalized congestion of other visceral organs. Matured ovules of the ovary were markedly hemorrhagic. No other lesions were noted grossly.

Laboratory Results: Mixed flora was isolated from the lung, liver, ovule and intestines; duck enteritis virus (DVE) was detected by PCR.

Histopathologic Description: Liver: At the subgross level there is multifocal pallor or clear areas interpreted as loss of hepatic architecture. On close examination there is edema around most blood vessels and multifocal necrosis. There is dissociation of hepatocytes and some hepatocytes are swollen, disrupted or ruptured. There is abundance of eosinophilic intranuclear inclusion bodies with thinly marginated chromatin **(Fig. 1-1)**. There are no appreciable inflammatory cell infiltrates.

Contributor's Morphologic Diagnosis: Liver: Multifocal necrosis and degeneration, multifocal, acute, with perivascular edema and abundant eosinophilic intranuclear inclusion bodies.



1-1. Liver, duck. Multifocally within hepatocytes are brightly eosinophilic 5-8 micron in diameter intranuclear inclusions which frequently marginate nuclear chromatin. (HE 1000X)

***Sponsored by the American Veterinary Medical Association, the American College of Veterinary Pathologists, and the C. L. Davis Foundation.**

Contributor's Comment: DVE is an acute, contagious herpesvirus infection of ducks, geese, and swans, characterized by vascular damage, tissue hemorrhages, digestive mucosal eruptions, lesions of lymphoid organs, and degenerative changes in parenchymatous organs.[6] Although there is no mention of clinical signs in this bird, the disease has an incubation period of 3 to 7 days. Once overt signs appear, death usually follows within 1 to 5 days. Naturally occurring infection has been observed in ages ranging from 7-day-old ducklings to mature breeder ducks.[6] DVE was first diagnosed in the United States in 1967 in a commercial Pekin duck-producing area of Long Island, NY, where it caused serious economic losses.[2,4] Mature ducks die in good flesh. Typically clinical signs consisted of prolapse of the penis in male matured breeders, and a marked drop in egg production in laying flocks. As DVE progresses within a flock more signs are observed: Photophobia, inappetence, thirst, nasal discharges, diarrhea, weakness, tremors of the head and neck. Mortality may range from 5 to 100%.[6] Based on the histopathologic findings the disease is considered non inflammatory and retrograde in nature; and the distribution of the inclusion bodies suggests that the virus is reticulo-endotheliotropic.[6]

In this bird, in addition to the liver lesions, mucosal lesions were noted in the digestive tract (esophagus, proventriculus), characterized by erosions and ulcerations with rare eosinophilic intranuclear inclusion bodies in few remaining epithelial cells.

Typically, lesions of DVE are those of vascular damage, eruptions of specific locations on mucosal surface of the gastrointestinal tract, lesions of lymphoid organs, and degenerative sequelae in parenchymatous organs. When these lesions are collectively present, are diagnostic of DVE.[6] In the present case the diagnosis was confirmed by PCR on tissue pools.

Differential diagnosis requires consideration of other diseases producing hemorrhagic and necrotic lesions in anseriforms. Such diseases include duck virus hepatitis, pateurellosis, necrotic enteritis, coccidiosis, and specific intoxications.[6]

To prevent this disease from spreading, strict decontamination and depopulation should be carried out whenever possible.[3] A vaccine is also available for prevention of DVE but approval by animal health authorities is required before it can be used.[7]

AFIP Diagnosis: Liver: Hepatitis, necrotizing, acute, random, moderate with eosinophilic intranuclear inclusion bodies, etiology consistent with herpesvirus

Conference Comment: Duck viral enteritis, also known as duck plague, is in the family *Herpesviridae*, and subfamily *Alphaherpesvirinae*. Two other extremely important herpes viruses in this subfamily are gallid herpesvirus 1 also known as infectious laryngotracheitis virus, and gallid herpesvirus 2 also known as Marek's disease.[5]

The pathogenicity of this virus depends on the particular species of duck infected. The blue-winged teal (*Anas discors*) is most susceptible to DVE, while the pintail duck (*Anser acuta*) is the least susceptible.[2] Muscovy ducks, as seen in this case, are highly susceptible to this particular virus. This virus is a major concern because migratory birds can spread this virus to naïve populations leading to massive outbreaks with high mortality.[4]

Dr. Raymond briefly integrated some general pathology topics as they relate to this particular case, and he focused on mechanisms by which free radicals cause damage to cells.

Free radicals are molecules with an unpaired electron making them highly reactive. These molecules are generated as by-products of normal cell function via oxidative metabolism or are a by-product of exposure to radiation, toxins, drugs, or other chemicals. Free radicals are also produced by neutrophils and macrophages in inflammation. Free radicals can damage cells by 1) peroxidation of cellular phospholipid membranes; 2) DNA injury; 3) oxidative modification of proteins.[1]

Free radical damage is minimized by several different antioxidants within the body. These antioxidants include enzymatic and nonenzymatic antioxidants. Enzymatic antioxidants include superoxide dismutase, catalase, and glutathione peroxidase. Nonenzymatic antioxidants include transport proteins transferrin, ferritin, lactoferrin, and ceruloplasmin. Vitamins A, C and E, and other dietary compounds such as lycopenes, flavonoids, genistein, and reserpines help to minimize the damage done by free radicals.[1]

Contributing Institution: California Animal Health and Food Safety Laboratory, UC Davis
http:/cahfs.ucdavis.edu

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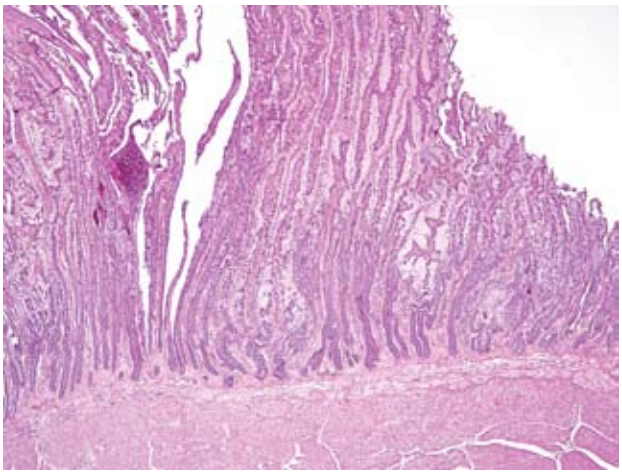
CASE II – N2008-0451 (AFIP 3103941)

Signalment: Adult, female Northern bobwhite (*Colinus virginianus* [no subsp.])

History: Found dead with no premonitory signs

Gross Pathology: At gross necropsy the animal was extremely thin with no visible fat stores and severe pectoral muscle atrophy. The proventriculus was markedly distended and filled with a large amount of thick, off-white, mucoid material. The proventricular walls were severely thickened, particularly the mucosa, which was 3 mm thick, yellow white and gelatinous. Numerous short, white, coiled nematode parasites were present within and adhered to the mucosal lining of the proventriculus and the proximal ventriculus. There was a 0.7 cm diameter region of the proventricular mucosa that was thinner than the adjacent mucosa, and dark red.

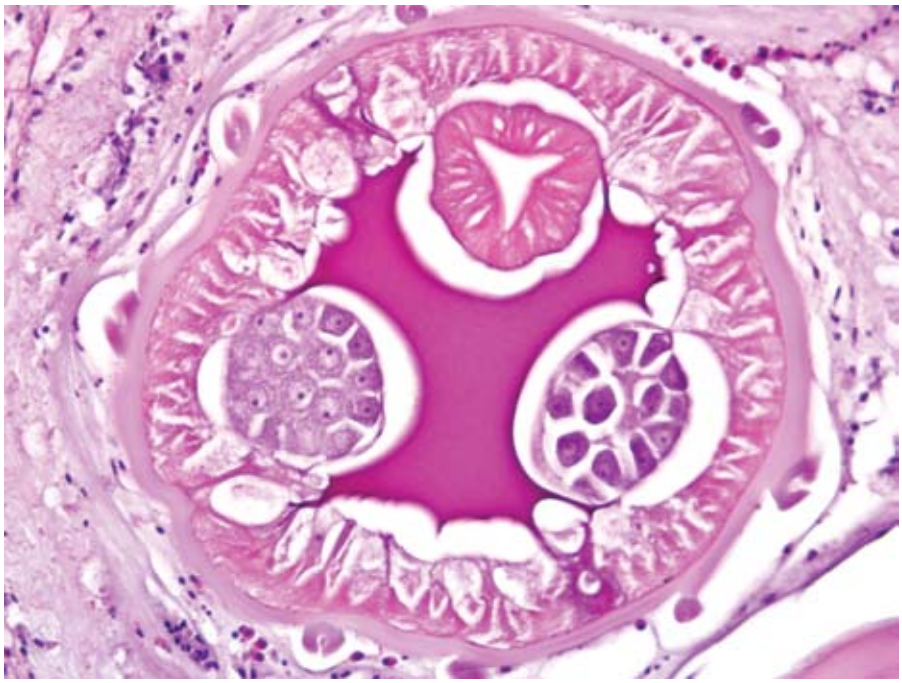
Laboratory Results: Multiple nematodes were collected and examined microscopically. The nematodes were white, between 4 and 7 mm long, less than 1 mm in diameter and typically coiled. On microscopy 4 characteristic wavy cuticular ornaments (cordons) extended from the base of the lips posteriorly. (Figure 1) Myriad 20 x 40 micron embryonated eggs were scattered throughout the background. (Figure 2) The parasite was identified as Dispharynx nasuta at the time of necropsy and later confirmed by the Cornell University, College of Veterinary Medicine, Animal Health Diagnostic Center.



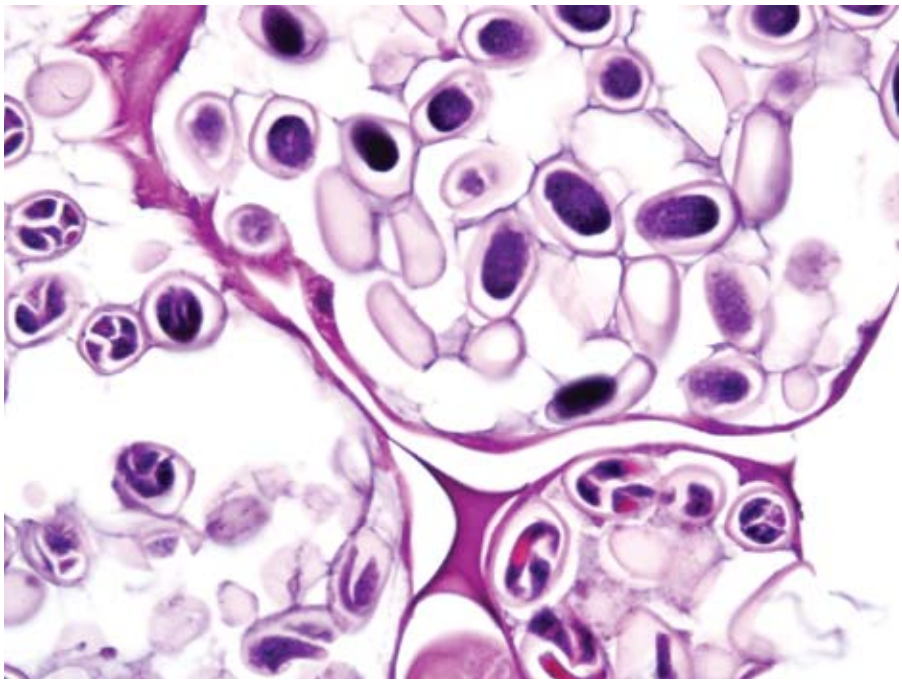
2-1. Proventriculus, quail. Marked proliferation of the mucosa. (HE 200X)

Histopathologic Description: The section submitted is from the proventriculus near the junction with the esophagus. The superficial mucosa varies from thickened and hyperplastic **(Fig. 2-1)** in some sections to severely eroded in other sections. In areas of erosion there is severe necrosis of the superficial portion of the glands with fibrin deposition and hemorrhage. Both the superficial and deep portions of the proventricular glands are distended by a mixture of mucus and numerous Spirurid nematode parasites. In cross section, the nematodes vary from 200 to 600 um in diameter. All have an approximately 5 to 15 um thick cuticle, which varies from smooth to ridged, a thick layer of coelomyarian musculature, a predominantly glandular esophagus, and a prominent intestinal tract composed of cuboidal to columnar cells with a brush border. The coelom contains a small amount of eosinophilic fluid **(Fig. 2-2)**. Female nematodes typically demonstrate a distended uterus containing numerous small (20 by 40 um), thick shelled, embryonated eggs **(Fig. 2-3)**. Within the lamina propria there is a diffuse infiltrate of eosinophils with smaller numbers of lymphocytes and plasma cells. Dilated glands have an attenuated epithelial lining and when free of nematodes contain a large amount of mucus and sloughed necrotic epithelial cells. A thick layer of mucus admixed with similar quantities of epithelial cells and eosinophils covers the mucosal surface. Scattered colonies of mixed bacteria are present within the mucus.

Contributor's Morphologic Diagnosis: Proventriculus: Proventriculitis, erosive to proliferative, eosinophilic, lymphocytic, plasmacytic, diffuse,



2-2. Proventriculus, quail. Cross section of a spirurid nematode with characteristic polymyarian-coelomyarian musculature, abundant eosinophilic material within the pseudocoelom, and numerous external cuticular cordons. (HE 400X)



2-3. Proventriculus, quail. Adult female nematodes contain prominent paired gonads which contain numerous small, oval, thick-shelled embyonated eggs. (HE 400X)

moderate to severe with intralesional Spirurid nematodes (*Dispharynx nasuta*)

Contributor's Comment: *Dispharynx nasuta* (syn. *Dispharynx (Acuaria) spiralis*), also known as the 'proventricular worm', is a spirurid nematode parasite of the proventriculus of many passerine, columbiform and free ranging gallinaceous birds.[5,11] It has been identified in a variety of game birds (ruffed grouse, blue grouse, American woodcock) including the bobwhite quail.[5,7,8] There is a single case report of infection in a captive princess parrot.[11] The highest prevalence and levels of infection are seen in juvenile birds, with infection occurring by 3 to 4 days of age in regions of high incidence.[5,7]

The life cycle of *Dispharynx* is indirect utilizing an

intermediate terrestrial isopod host. Adult female *D. nasuta* pass embryonated eggs into the lumen of the proventriculus, which are later shed in the feces.[11] The eggs are consumed by the intermediate host, wood lice (*Armadillidium vulgare*) or sow bugs (*Porcellio scaber*), in which the larvae subsequently hatch and penetrate the host tissues. First stage larvae (L1) develop into the infective third stage (L3) within 26 days. The third stage larvae (L3) can survive in the infected intermediate host for up to 6 months. Once the intermediate host is consumed by a susceptible bird the third stage larvae (L3) further develop, reaching sexual maturity in 27 days.[7]

Sexually mature adults primarily infect the proventriculus, though in severe cases they can also infect the adjacent esophagus and ventriculus. The nematodes attach by their anterior end to the mucosal epithelial cells initially causing ulceration at the site of attachment. In most avian species these worms cause only a mild nodular reaction in the mucosa and a small amount of inflammation. However in some species (American wood cock, ruffed grouse, blue grouse) *D. nasuta* acts as a primary pathogen.[5,7] When present in large numbers (10 or more), the infection causes severe hyperplasia of the proventricular glands. Associated with the glandular proliferation is an increase in mucus production, as well as excessive sloughing of mucosal epithelial cells. As a result, the lumen of the proventriculus becomes distended by a thick, white, coagulum of mucus and sloughed cells which creates a functional obstruction of the proventriculus and secondary starvation.[5,7]

Diagnosis in this case was made based on the characteristic proventricular lesion, egg and adult worm identification. As stated previously, the significance of this parasite is variable. In most birds this nematode is an incidental finding. However in some species, particularly the ruffed grouse and blue grouse, these organisms are believed to be a significant cause of mortality and decline in wild populations. While not typically considered a primary pathogen of free ranging bobwhites, significant mortality has been seen in cage-reared animals.[5,7,8]

AFIP Diagnosis: 1. Proventriculus: Proventriculitis, proliferative and heterophilic, diffuse, marked with glandular ectasia and adult spirurids
2. Serosa, adipose tissue: Atrophy, diffuse, moderate

Conference Comment: Spirurid nematodes often have several distinguishing characteristics in histologic section. Several types of spirurids have cuticular ornamentations around the buccal cavity varying from spines to cords to collars. Eosinophilic fluid is found in the body cavity and is a distinguishing characteristic of this group of nematodes. Lateral cords can be extremely large in size and are often vacuolated. Most adult females in this group also produce thick shelled, embryonated eggs. Other examples of spirurids in domestic animals include: *Physaloptera* sp., *Gonnglyonema* sp., *Draschia* sp., *Spirocerca* sp., *Thelazia* sp.[4]

In a retrospective study done by Drs. Raymond, Miller, and Garner it was found that in two cases infection with *D. nasuta*, also known as adenomatous proliferative proventriculitis (APP), the lesions progressed to proventricular adenocarcinoma.[9] In humans and domestic animals, several parasites have been associated with subsequent neoplasia. A brief, non-comprehensive list is included.

Parasite	**Associated Neoplasm**
Spirocerca lupi	Esophageal sarcoma in dogs
Opisthorchid flukes	Cholangiocarcinoma in cats and man
Cysticercus fasciolaris	Hepatic sarcoma in rats
Clonorchis sinensis	Cholangiocarcinoma in cats and man
Schistosoma haematobium	Carcinoma in bladder of humans
Trichosomoides crassicauda	Papillomas in urothelium in rats

2,3,6,10

Contributing Institution: Wildlife Conservation Society (http: //www.wcs.org)

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CASE III – 122103-07 (AFIP 3107607)

Signalment: Adult house sparrow (*Passer domesticus*)

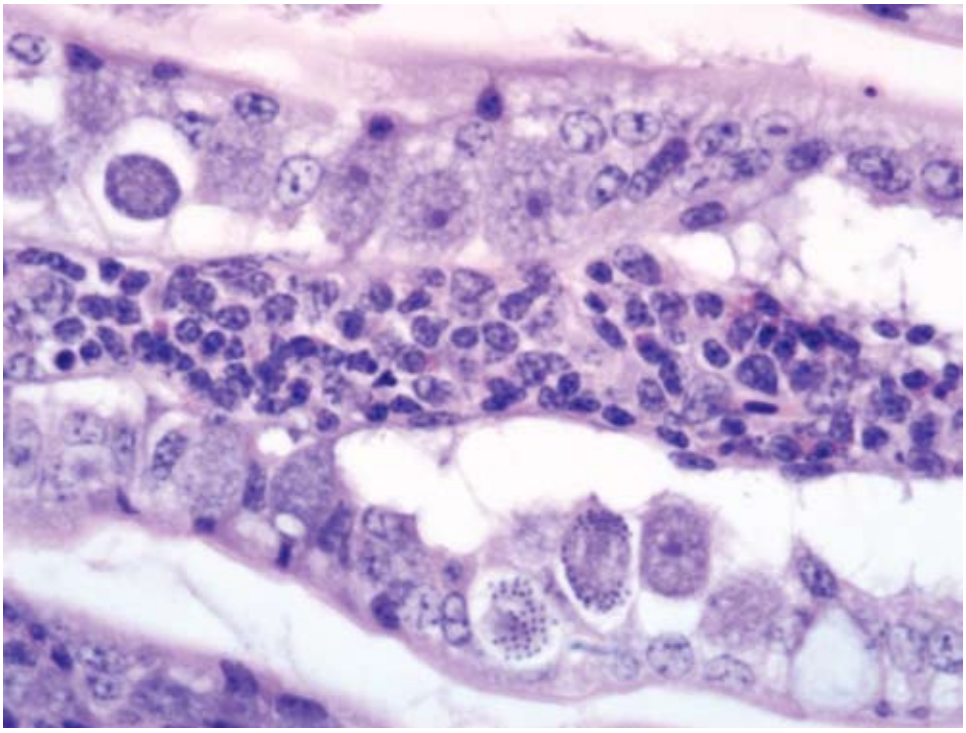
History: Examined is a house sparrow from a wild caught research colony that was submitted for necropsy after the colony began to experience high mortalities. An adjacent colony of American goldfinches (*Carduelis tristis*) experienced high mortalities with the same clinical signs prior to the sparrows. Clinical signs within both colonies consisted of muscle wasting and diarrhea.

Gross Pathology: Within the sampled population of sparrows and goldfinches (16 birds in total) the most severely affected birds exhibited marked wasting of the pectoral muscles, poor coelomic adipose stores, severe thickening of the proximal small intestine that gradually tapered toward the ileum and small, variably sized, irregular foci of pallor on the liver that extended into the parenchyma on section. Most birds had no significant findings and one bird was severely autolyzed.

Laboratory Results: Fecal floatation was run on one bird and *Isospora* spp. were identified but not speciated. CD3 immunohistochemistry was carried out on the intestines and liver to determine if the lymphoblasts were of B cell or T cell lineage. Diffusely the infiltrates exhibited intense membranous staining for CD3 indicating T-cell lineage. The intestinal staining varied between birds with strong immunoreactivity in the basal lamina propria and variable staining in the plical folds and villi. Electron microscopy was performed to rule out viral causes for the lymphocytic infiltrate; no viral particles were identified.

Histopathologic Description: Small intestine: Diffusely infiltrating and severely expanding the lamina propria is a marked mixed inflammatory infiltrate composed predominantly of lymphoblasts, with fewer histiocytes, plasma cells and eosinophils. Multifocally this inflammation extends transmurally with mild accumulations on the serosal surface. Within the luminal epithelial cells there are abundant approximately 20x15µm coccidian parasites at various stages of development **(Fig. 3-1)**. There are abundant macrogamonts characterized by a peripheral ring of large eosinophilic granules; fewer microgamonts that are smaller (approximately 15x10µm) and have more densely packed basophilic cytoplasmic granules; rare schizonts that contain abundant 1-2µm merozoites and occasional unsporulated oocysts that have a thin capsule and a homogenous finely granular amphophilic cytoplasm.

Liver: Infiltrating and partially effacing the peri-portal hepatocytes is a mild to moderate mononuclear inflammatory infiltrate composed predominantly of lymphoblasts with fewer histiocytes and plasma cells. The cytoplasm of scattered lymphoblasts contain 1-5, small, oval, 1-2µm diameter basophilic organisms within a thin, clear, well demarcated parasitophorous vacuole. Rarely these organisms peripheralize and indent the nucleus of the infected cells. Small numbers of the basophilic organisms are free amongst the inflammatory infiltrate. Small aggregates of the lymphoblasts are scattered within the sinusoids.



3-1. Small intestine, house sparrow. Multifocally within intestinal epithelium are numerous protozoal schizonts, microgametocytes, macrogametocytes, and sporulated oocyts. (HE 400X, HE 1000X)

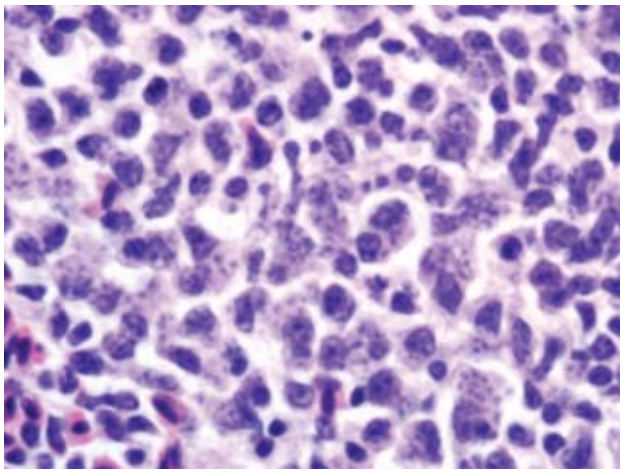
Spleen (not in every section): Diffusely the peri-arteriolar lymphoid sheaths are moderately expanded with variable numbers of small, oval, 1-2µm diameter basophilic organisms with a thin, clear, well demarcated parasitophorous vacuole within the cytoplasm of occasional lymphocytes **(Fig. 3-2)**. Rarely these organisms are present in the sinusoidal histiocytes.

Contributor's Morphologic Diagnosis: Small intestine: Diffuse, severe, chronic, lymphoplasmacytic, histiocytic and eosinophilic enteritis with intraepithelial coccidian parasites.
Liver: Moderate, diffuse, lymphocytic peri-portal hepatitis with intralesional merozoites consistent with *Atoxoplasma* spp.
Spleen: Moderate lymphoid hyperplasia with intralesional merozoites consistent with *Atoxoplasma* spp.

Contributor's Comment: At low magnification the trans-mural intestinal infiltrate and the peri-portal hepatic infiltrate resemble lymphoma. At higher magnification and with the aid of immunohistochemistry it is apparent that the intestinal infiltrate is composed of a mixed cellular infiltrate, with T lymphocytes predominating. One case documents intestinal lymphoma with metastasis and an incidental intestinal coccidian infection that was attributed to immunosuppression from the tumor.[8] No connection between the coccidian parasites and the severe lymphoproliferative infiltrate was made in that case.

Other potential causes of lymphoproliferative disorders and lymphoma in avian species include Marek's disease virus, an alpha-herpesvirus, and Avian leucosis virus, a retrovirus, which can cause lymphoma-like lesions in multiple organs in chickens and many avian species respectively. As the immunohistochemistry results were overwhelmingly positive for CD3, a T cell antigen, a retroviral-induced lesion was determined to be less likely as these viruses typically induce B-cell proliferation. Transmission electron microscopy was performed on the inflammatory infiltrate to rule out viral induced lymphoma. No viral particles were identified in the examined tissues; Atoxoplasma merozoites were not evaluated.

Atoxoplasma spp are host specific apicomplexian parasites that are infective to birds within the family Passeriformes. This is a large family encompassing many different species of birds. Within this family it is the finches, sparrows, thrushes and other small songbirds that are most commonly reported to be affected by this parasite. In the wild these parasites are endemic in low levels [3, 14] however it is likely that the levels are underestimated due to the difficulty in identifying the circulating merozoites.[5,7,8] Atoxoplasma has become a major cause of mortality in zoos and research facilities that utilize wild caught colonies of passeriform birds.[5,6] The stress from being in captivity and poor sanitary conditions have been identified as contributing causes for the high morbidity in these facilities.[6,7,12] Interestingly Atoxoplasma has been



3-2. Spleen, house sparrow. Multifocally within the red pulp are many leukocytes which contain numerous 1-2 micron intracytoplasmic protozoal merozoites. (HE 1000X)

reported in raptors.[10] However, as so few cases exist, the significance of the parasite outside of the passerine birds is unknown.

Affected birds will exhibit a loss of appetite with subsequent weight loss and reduction of the pectoral musculature, which is termed "going light". The animal can become depressed, have ruffled feathers, a distended abdomen, and develop diarrhea, dehydration and a loss of balance.[2,11] Subclinically infected birds will show no outward signs of infection but will shed infective oocysts into the environment. In infected colonies morbidity can approach 100% with a significant mortality rate. Grossly, there are varying degrees of pectoral musculature atrophy, coelomic adipose depletion and enteritis.

Since the discovery of this parasite there has been considerable debate as to how it should be classified. In 1950 Garnham proposed the name Atoxoplasma [12] as it was felt this name addressed the fact that the parasite resembled Toxoplasma but was not Toxoplasma. Since that time it was determined that these parasites belong to the family Eimeriidae and closely resemble *Isospora* spp. Both Atoxoplasma and Isospora oocysts contain two sporocysts each containing four sporozoites. Both parasites follow the basic coccidian life cycle where the sporozoites will excyst in the intestinal tract and invade the enterocytes, undergo merogony, develop into microgamonts or macrogamonts within the enterocytes and undergo sexual reproduction to form unsporulated oocysts. The oocysts are passed in the feces and sporulate in the environment where they will be ingested by the next host.

Atoxoplasma spp differ from *Isospora* spp during merogony. After ingestion of the Atoxoplasma oocysts the sporozoites and merozoites can directly infect mononuclear cells that have been recruited into the intestine in response to the coccidian parasites.[1,7,9,11,12] The merozoites enter into the circulation where they continue to undergo merogony within the mononuclear inflammatory cells. Through a currently unknown mechanism this parasite initiates a marked lymphoproliferative response. Perivascular accumulations of mononuclear inflammatory cells develop systemically and often contain intracytoplasmic merozoites. Depending on the severity of the infection the inflammatory infiltrates can resemble mild lymphohistiocytic inflammatory response to widespread lymphoma.

As previously mentioned the Atoxoplasma merozoites were not evaluated under the electron microscope. The reported ultrastructural characteristics of this parasite include a pellicle consisting of an outer plasma membrane and an inner complex of two membranes, subpellicular microtubules, a conoid, rhoptries, micronemes, mitochondria, and a micropore all within a parasitophorous vacuole.[2,9,11]

In summary, when evaluating tissue from a passerine bird and a coccidial infection with severe lymphoproliferative lesions, Atoxoplasma should be high on the differential list. Close evaluation of the lymphocytic infiltrates for intracellular merozoites is warranted in these cases.

AFIP Diagnosis: 1. Small intestine: Enteritis, lymphoblastic, transmural, diffuse, severe, with crypt loss, intraleukocytic apicomplexan merozoites, and intraepithelial gamonts and schizonts.
2. Liver: Hepatitis, portal, lymphoblastic, diffuse, marked, with intracytoplasmic apicomplexan merozoites.

Conference Comment: The contributor does an outstanding job of covering all aspects of atoxoplasmosis in their case submission report. The moderator mentioned two other very important diseases in birds that result in lymphoproliferative disorders. Marek's disease (gallid herpesvirus 2) is common in chickens and manifests as mononuclear cellular infiltrates in the skin, peripheral nerves, and the iris.[15] Viruses within the avian leukosis/sarcoma group (family Retroviridae) cause many benign and malignant neoplasms.[4]

Contributing Institution: http://www.vet.cornell.edu/biosci/pathology/

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CASE IV – UNL/VDC 2301-06 (AFIP 3075580)

Signalment: Fifteen-week-old mixed breed pig (*Sus scrofa domesticus*)

History: Diarrhea was reported in which there were flecks of undigested blood in a group of pigs. Approximately 10% of the pigs had diarrhea and 5% were markedly emaciated.

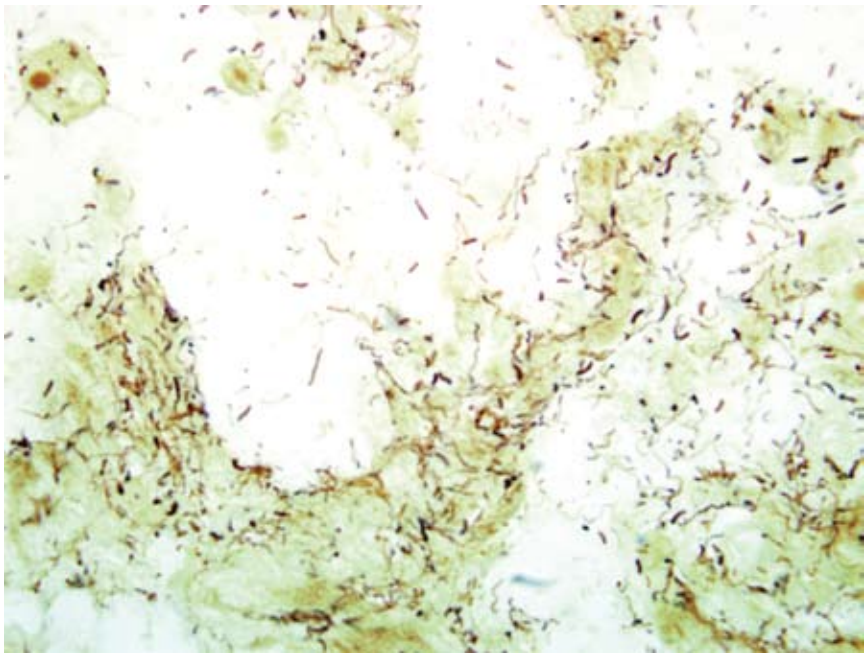
Gross Pathology: The carcass of the euthanized pig was gaunt and the hair coat roughened. Stomach and small intestine contained scant amounts of ingesta. There was a moderate amount of semi-liquid feces in the spiral colon. Fresh blood and fibrin was mixed with the colon contents. Colonic mucosa was glistening and rough in appearance.

Laboratory Results: Polymerase chain reaction testing for *Brachyspira hyodysenteriae* on bacterial colonies from anaerobic cultures were positive.

Histopathologic Description: These sections of large intestine are characterized by multiple large mucosal erosions. Luminal surface is covered by a thick layer of neutrophilic debris mixed with mucus and occasionally extravasated erythrocytes. Rare large protozoa with a morphology consistent with *Balantidium coli* are seen associated with the eroded areas. Mixed bacteria (including myriad long slender spirochetes) are seen in the luminal debris, eroded areas, and in superficial areas of mucosal crypts. Superficial mucosal crypts are mildly dilated with mucous "plugs". Crypt epithelium is mildly hyperplastic and there are reduced numbers of goblet cells.

Contributor's Morphologic Diagnosis: Mild to moderate acute focally extensive erosive and catarrhal colitis

Contributor's Comment: This is a classic case of swine dysentery (SD) caused by *Brachyspira hyodysenteriae*.[3] Along with diarrhea, other signs seen with SD include fever, anorexia and dehydration. Diarrhea is the result of malabsorption of fluids and electrolytes in the large intestine.[1] Death is usually the result of dehydration, acidosis, and hyperkalemia.[3] Pathogenesis of SD is poorly understood, but in gnotobiotic pigs, there is a synergistic effect with other anaerobic bacteria.[5] *B. hyodysenteriae* colonizes the mucus on the surface of the mucosa and in crypts and invades surface epithelial cells.[1] The main virulence factor shown to be associated with severity of disease is a hemolysin.[4]



4-1. Colon, pig. Diffusely adherent to epithelium and free within the lumen are many 0.5 x 4.0 micron argyrophilic spirochete bacteria. (Warthin-Starry 1000X)

AFIP Diagnosis: Colon: Colitis, erosive, multifocal, moderate, with necrosis, luminal mucin accumulation and argyrophilic spiral bacteria **(Fig. 4-1)**

Conference Comment: Swine dysentery (SD) generally affects pigs during the grow-finish phase of production. It causes a mucohemorrhagic diarrhea of the colon, and it can cause serious economic setbacks in a herd of pigs. SD is caused by *Brachyspira hyodysenteriae*, a gram-negative, beta-hemolytic anaerobic spirochete. This organism was also formerly known as *Treponema hyodysenteriae*, and then was reclassified as *Serpulina hyodysenteriae*, and it is now known as *Brachyspira hyodysenteriae*.[2] At least 4 other intestinal spirochetes have been identified in swine with *Brachyspira pilosicoli* being the only other one to cause clinical disease. *B. pilosicoli* can cause a mild colitis in pigs.[2]

Gross lesions for SD are normally present only in the large intestine. The ileocecal junction is normally the boundary for lesions caused by SD. Lesions often begin as hyperemic and edematous mucosal lesions and progress to a mucosa covered by copious amounts of mucus and fibrin admixed with blood. These lesions often form a pseudomembrane. There also may be multifocal erosions of the surface mucosa, and ulcerative lesions are not generally seen.[2]

Microscopically acute lesions consist of mucosal congestion, edema, and neutrophilic infiltrates in superficial lamina propria near blood vessels. Spirochetes may be seen in colonic crypts, and hyperplasia of goblet cells is common. These lesions progress with the development of superficial mucosal erosions and a characteristic mat of fibrin, mucous, hemorrhage, and cellular debris forms on the mucosal surface. Silver stains can be used to identify these spirochetes in tissue section.[2]

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