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Conference Moderator:
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Chris H. Gardiner, PhD

CASE I: NIAH2010-1 (AFIP 3164221).

Signalment: Adult, gender undetermined, whooper swan, avian (*Cygnus cygnus*).

History: The whooper swan was found dead around the lake in Ibaraki Prefecture, Japan. The swan was submitted to our laboratory for postmortem examination in March 2006. Whooper swans move to the lakes and ponds of Ibaraki Prefecture from Siberia for their wintering period (October to March).

Gross Pathology: There were no significant gross lesions in the swan.

Laboratory Results: Neither viruses nor significant bacteria were detected by virologic and bacteriologic examination.

Histopathologic Description: Small intestine, multiple sections: Vascular lesions and schistosome infection were seen in the swan. Marked hypertrophy of the venous walls was characterized in the mesentery, serosa, and muscular layer of the intestines (duodenum, small intestine, ceca, and rectum). Venous lesions were also seen in the capsule of spleen, kidney, adrenal gland, and pancreas, as well as in the serosa of air sacs, pleura, and gallbladder; the connective tissue around the aorta; and the capsule and interlobular connective tissue of the liver. Schistosome flukes were detected in the veins but not in the arteries. In mild lesions, nodular proliferation of smooth muscle fibers was observed in the media of mesentery and serosal veins. In moderate lesions, venous hypertrophy became more marked and the venous lumens were narrowed or occluded by proliferated smooth muscle fibers. The medial smooth muscle fibers of the veins between the muscular layers of the intestines, as well as the mesenteric veins and serosal veins, exhibited nodular or circular hypertrophy. The intestinal muscular layers were depressed and atrophied by hypertrophied veins. In severe lesions, venous hypertrophy increased in severity and distribution. The proliferation of medial smooth muscle fibers under collagen fibers was evident in the medium-sized veins of the livers. There was mild to moderate perivascular lymphocyte infiltration around the hypertrophied veins. The proliferated medial fibers were stained red in azan stain, and positively stained by immunohistochemical staining of alpha smooth muscle actin. Schistosome flukes were detected in the veins. The schistosomes had a cuticle layer, a digestive tract, a sucker, and reproductive organs.

There were oval eggs (about 40 to 70 μm in size) in the liver and lung. The eggs had small projections on one side. Miracidia hatched from some eggs in the liver. There were granulomatous reactions around these eggs in the liver and lung. Bile pigment stained positively using Hall stain deposited in the liver of the eight whooper swans with vascular lesions and schistosomiasis. There was mild to severe deposition of hemosiderin (Berlin-blue-positive) in the liver and spleen.

Contributor's Morphologic Diagnosis: Small intestine: Hypertrophy, medial, venous, severe, with intravenous schistosomes, whooper swans (*Cygnus cygnus*)

Contributor's Comment: Avian schistosomes are a specialized group of trematodes that develop as adults within the circulatory system or nasal tissue of their avian host. They include nine genera: *Allobilharzia, Austrobilharzia, Bilharziella, Dendritobilharzia, Gigantobilharzia, Jilimobilharzia, Macrobilharzia, Ornithobilharzia,* and *Trichobilharzia*[2]. *Trichobilharzia* is the largest genus within the family *Schistosomatidae*, covering roughly 40 species. *Schistosomatidae* is the only group of trematodes that has separate sexes, with most trematodes being hermaphroditic. The blood flukes found in our study appear to be avian schistosomes judging by their morphological characteristics, especially the sucker and gonochorism (separation of the sexes in different individuals).

There is a close relationship between venous hypertrophy and venous parasitism of avian schistosomes. The mechanism of venous hypertrophy is not well known. Two mechanical hypotheses for the pathogenesis are put forward[1]. In one it is due to direct irritation of the schistosomes on the venous walls, and in the other it is due to an immunological (allergic) reaction against the schistosomes. The pathological condition of veins referred to as "obliterative endophlebitis"[3] is similar to the lesions observed in the present case. The venous media consists of smooth muscle fibers, but the venous intima of veins has no smooth muscle fibers. This suggests that the pathological condition may not be "endophlebitis" but may in fact be hypertrophy of smooth muscle fibers in the media of veins.

There have been several reports of avian schistosomiasis in whooper swans, mute swans, black swans, Atlantic Brant geese, green-winged teal, blue-winged teal, and northern pintail. However, vascular lesions characterized by venous medial hypertrophy have never been described in these reports. Obstruction of venous return in the mesenteric, intestinal, and portal veins may have contributed to the emaciation and mortality of the affected whooper swans in the present study.

Deposition of hemosiderin and bile pigment with copper was present in the livers of the whooper swans in the present case. Bile pigment was observed in six of the eight whooper swans infected with avian schistosomes, and adult flukes and/or their eggs were detected in the livers. Therefore, cholestatic jaundice may be caused by avian schistosomiasis.

AFIP Diagnosis: Small intestine, tunica muscularis, serosa, and mesentery, blood vessels: Vasculitis, proliferative and lymphoplasmacytic, multifocal, severe, with intravenous adult schistosomes.

Conference Comment: Much of the conference discussion centered on whether the proliferative vascular lesions were smooth muscle or myofibroblastic in origin. The Masson's trichrome stain demonstrated intense red staining of the vascular walls consistent with smooth muscle proliferation. This finding is similar to the vascular changes seen in humans with hepatic schistosomiasis.

Conference participants also discussed the possibility that the observed lesions might be age-related, as the age of the swans is not known. Severe hepatic infections in humans with *Schistosoma mansoni* or *S. japonicum* typically result in multiple granulomas, marked fibrosis and enlargement of portal areas resulting in the colloquial lesion of "pipe-stem fibrosis." Portal veins are frequently obliterated in such cases, and portal arteries often show muscular hypertrophy and intimal sclerosis. Schistosome eggs may travel to the lungs, producing granulomatous pulmonary arteritis, intimal hyperplasia, and progressive arterial obstruction. Histologic lesions in the arteries include granulomatous and fibrosing disruption of the elastic layer.

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CASE II: 09-14541 (AFIP 3164906).

Signalment: 3-year-old, male intact, quarterhorse, equine (*Equus caballus*).

History: The reported history included neurological signs of one week duration. The owner noticed drooling from the mouth, staggering and reddened eyes. The horse was euthanized at the veterinary teaching hospital.

Gross Pathology: The horse was in good body condition, with well developed musculature and adequate adipose tissue stores. The right cranial and middle lung lobes were dark red, firm and heavy. Moderate amounts of serosanguineous fluid were exuded on the cut surfaces of these lobes. The trachea and major bronchi were filled with large amounts of stable white foam. Mild cerebellar coning was observed, and the leptomeninges were moderately congested.

Histopathologic Description: Brain stem: The brain stem contains multifocal areas of necrosis in the white and grey matter. Many foamy macrophages and lesser numbers of lymphocytes and plasma cells infiltrate affected areas. The cytoplasm of many neurons, astroglial cells and Gitter cells often contain free zoites or protozoal cysts with faint walls (schizonts) approximately 20-30 micron in size and filled with many elongated 2 x 5 µm basophilic merozoites. These each contain a small apically located nucleus. Affected neurons are swollen, hypereosinophilic and often pyknotic, with diffuse chromatolysis. White matter contains randomly scattered dilated axon sheaths and spheroids (Wallerian degeneration). Many degenerate axons are invaded by Gitter cells (digestion chambers). Astrocytes multifocally are increased in numbers and appear swollen, with enlarged amount of cytoplasm (reactive astrocytosis). Glial nodules composed of microglial cells are also present. Blood vessels are severely congested. Perivascular spaces are invaded by many lymphocytes, plasma cells and macrophages forming 5-8 cell layer thick perivascular cuffs. Rare multinucleated giant cells are randomly scattered in the neuropil.

Lung (not included): Alveolar spaces, bronchioles and bronchi are flooded with many degenerate neutrophils mixed with fewer macrophages, lymphocytes and cellular debris, fibrin and extravasated erythrocytes. Interlobular septa and pleura are markedly expanded by clear spaces (edema). Diffusely alveolar spaces are filled with pale proteinaceous fluid material. Blood vessels are moderately congested, and lymphatics are markedly dilated. Coccoid bacterial colonies mixed with small amounts of plant material are scattered randomly in the parenchyma.

Contributor's Morphologic Diagnosis: 1. Brain stem: Encephalitis, necrotizing, granulomatous and lymphoplasmacytic, with gliosis and intralesional protozoal organisms (*Sarcocystis* spp.).
2. Right lung lobes (not submitted): Bronchopneumonia, suppurative, severe, lobar.

Contributor's Comment: Immunohistochemistry for *Sarcocystis neurona* demonstrated positive staining in many neurons, astroglial cells and macrophages within the brain stem. Gomori's methenamine silver stain failed to demonstrate the presence of fungal microflora within sections. *Sarcocystis neurona* is a cause of equine protozoal myeloencephalitis (EPM) in horses; it belongs to cyst-forming coccidia (Apicomplexa: Sarcocystidae). Identical disease is possible due to *Neospora caninum* and *Neospora hughesi*. Horses are considered to be aberrant dead-end hosts of *S. neurona*, whereas armadillos, sea otters, raccoons, skunks and cats are natural intermediate hosts. They become infected when they consume food or water contaminated with protozoal sporocysts. Opossums (*Didelphis* spp.) are believed to be the only definitive host capable of shedding infective sporocysts. Horses of all ages are susceptible to the disease, especially in the summer and fall. Sexual reproduction occurs in the intestinal epithelium of the definitive hosts; infective oocysts, each with two sporocysts, are shed in feces. After ingestion by the intermediate host, sporozoites are released and penetrate intestinal wall and initiate asexual reproduction (schizogony). The forms of asexual reproduction are schizonts and merozoites. The ultimate stage of the asexual reproduction is formation of sarcocysts, usually in the skeletal muscles. Very often, infective sarcocysts (700 µm long and 40 µm wide, with a 1-2 µm thick cyst wall and 5 µm long bradyzoites) can be found in the tongue and other skeletal muscles.

AFIP Diagnosis: Brainstem: Encephalitis, necrotizing, focally extensive, severe, with apicomplexan protozoa.

Conference Comment: Dr. Fabio Del Piero, who attended this conference as a participant, commented he typically observes two distinct clinicopathologic presentations of equine sarcocystosis at his diagnostic laboratory. The first syndrome, much like this case, is fulminant and usually occurs in untreated animals in which low numbers of eosinophils are frequently observed histologically in the affected areas of the central nervous system. The second form typically occurs in treated animals, and the histopathologic presentation is usually that of mild lymphocytic encephalitis resembling viral infections. These latter cases, in which definitive etiologic diagnosis is more difficult, are becoming increasingly more common in his experience.

The ultrastructural characteristics of *Sarcocystis* spp. and *Toxoplasma* spp. are distinctive, allowing differentiation between the two organisms. *Sarcocystis* spp. schizonts and merozoites reside within the host cell cytoplasm; no parasitophorous vacuole is present. *Sarcocystis* spp. merozoites have micronemes, a conoid, apical rings and polar rings, but lack rhoptries. *Toxoplasma* spp. tachyzoites reside within an intracellular parasitophorous vacuole and have an inner and outer membrane (pellicle); merozoites possess an anterior apical complex containing a polar ring characterized by a slight thickening continuous with the inner membrane surrounding the anterior opening; an anterior conoid inside the polar ring; rhoptries; and micronemes.

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CASE III: 07-974 (AFIP 3166496).

Signalment: 5-year-old, male, miniature pinscher, canine (*Canis familiaris*).

History: This dog was presented 1½ weeks prior to necropsy with tetraparesis (worse on left side), non-weight bearing on left forelimb, and decreased withdrawal and hopping reflexes in left forelimb and eventually left hindlimb. Cervical pain was also observed. It was treated with dexamethasone and the condition continued to deteriorate. Horner's syndrome was observed on the left side. Due to poor prognosis the dog was euthanized.

Gross Pathology: A complete gross examination, including the spinal nerves, brachial and lumbosacral plexuses and skeletal muscles was done. No significant changes were noted.

Laboratory Results: Immunoperoxidase on spinal cord sections was performed for *Neospora caninum* and *Toxoplasma gondii*.

Histopathologic Description: Spinal cord, cervical (per contributor): The submitted section is a transverse section of the spinal cord at the C4 level (stained with hematoxylin, phloxin, eosin and saffron). There is a severe, chronic necrotizing myelitis affecting mainly the peripheral portion of most funiculi (white matter), with associated non-suppurative leptomeningitis; the gray matter is only mildly and focally involved in most sections. The lesion is asymmetrical, being more extensive on the left side. It is characterized by vacuolization and loss of neuropil, with numerous gitter cells and variable numbers of neutrophils and perivascular lymphocytes and plasma cells. The leptomeningeal inflammation is mainly lymphoplasmacytic. In some areas there is leptomeningeal fibrosis that extends into the subjacent parenchyma (mostly perivascular), with gliosis. In the ventral horns, there is variable inflammation and occasional neurons demonstrate diffuse chromatolysis. The ventral nerve roots have some Wallerian degeneration and mild inflammation. In the affected white matter, there are several to numerous, round to ovoid, protozoal organisms, roughly 5 μm in diameter, that occur as either within thin-walled cysts (bradyzoites) or individual tachyzoites.

Contributor's Morphologic Diagnosis: Severe and extensive chronic necrotizing myelitis with intralesional protozoal organisms.

Contributor's Comment: Immunoperoxidase identified the protozoal organisms as *Neospora caninum*. The spinal lesions extended from C3 to T4; similar lesions were observed in the cerebellar gray matter and medulla. Lesions were not found in other organs examined.

Neospora caninum is a coccidian parasite of worldwide distribution that has a wide host range. It is very similar in structure and life cycle to *Toxoplasma gondii*, and was misdiagnosed as *T. gondii* until 1988, when Dubey *et al*. described and named this new genus and species.[2] Neosporosis was first described in 1984 in a litter of boxer dogs in Norway. It is a polysystemic protozoal disease that has been reported in several mammalian species;[2,3] in domestic animals, it is a cause of abortion mainly in cattle (also in sheep and goats), a serious disease of dogs and one of the causes of equine protozoal encephalomyelitis. Seropositivity has been observed in humans, but has not been associated with clinical disease; the parasite has never been detected in human tissues, and thus no zoonotic potential has been shown so far (in contrast to toxoplasmosis).[2] *Neospora caninum* has three known infectious stages: tachyzoites, bradyzoites (tissue cysts) and oocysts. Animals with neosporosis act as intermediate hosts in which tachyzoites and bradyzoites are formed by asexual reproduction. Only dogs and coyotes are known definitive hosts, i.e. in which fecal excretion of oocysts occurs.[2] Horizontal and vertical transmission has been demonstrated. Herbivores are thought to become infected by ingestion of oocysts. Although dogs (and other carnivores) can become infected by ingesting bradyzoite-containing tissues, transplacental infection is believed to be the most common route of transmission.[2,3] Tissue cysts are found mainly in the CNS but also may be found in extraneural tissues, especially skeletal muscles.[2]

Canine neosporosis has been reported in dogs of all ages, but the most severe cases have been reported in dogs less than six months of age. Although lesions have been reported in a variety of organs/tissues, e.g. heart, lung, liver, pancreas and skin, the nervous system and skeletal muscles are most consistently involved.[1,2,3] In puppies, ascending hindlimb paresis/paralysis with muscular atrophy and often arthrogryposis is the most frequent clinical presentation and is mostly due to polyradiculoneuritis and myositis; several littermates can be affected.[1,3] Systemic involvement may be present; myocarditis can cause sudden death. In adults, neurological signs usually reflect widespread CNS involvement, and include paresis/paralysis, ataxia, head tilt and seizures which are associated with a multifocal encephalomyelitis. A recent paper shows that neosporosis is a significant cause of progressive cerebellar ataxia in adult dogs.[3] *Neospora caninum* is considered a primary pathogen, but disease may be exacerbated by corticosteroid therapy.[1] Encephalomyelitis in canine neosporosis is nonsuppurative with varying degrees of necrosis and gliosis; tachyzoites and bradyzoites are seen in neurons and neuropil (intracellular). Although *Neospora* has a thicker cyst wall than *Toxoplasma*, they cannot be reliably differentiated by light microscopy, and immunohistochemistry is required; they can also be differentiated with transmission electron microscopy.

AFIP Diagnosis: Spinal cord and spinal nerve roots: Myelitis and polyradiculoneuritis (radiculitis), necrotizing and neutrophilic, multifocal to coalescing, severe, with nonsuppurative meningitis and many protozoal cysts.

Conference Comment: In addition to the various manifestations in dogs mentioned by the contributor, conference participants also discussed the syndrome of bovine abortion due to *Neospora caninum*. Infection in bovids results from consuming oocysts in the feces of canids. Infection does not affect fertilization, and pregnant animals typically abort between the 5th and 6th month of gestation. Interestingly, immunity does not prevent transplacental infection; therefore, infected cows can abort up to three consecutive pregnancies. Fetuses can be expelled in various states of tissue preservation with minimal gross lesions. When present, gross lesions in the fetus consist of random, yellow-tan foci in the brain, brainstem, heart and skeletal muscle. Grossly in the placenta the cotyledons are necrotic with sparing of the intercotyledonary areas. Histologically in the central nervous system of the fetus there is necrosis of the neuropil with mononuclear inflammatory infiltrates and occasional mineralization; protozoal cysts are more commonly found within the spinal cord rather than in the brain. In addition to the brain and spinal cord, intracellular zoites may be found in both cardiomyocytes and Purkinje fibers in the heart.[5]

Rarely, dogs, especially immunocompromised adults, can exhibit cutaneous neosporosis which produces multifocal ulcerative and nodular dermatitis. Dermatitis is often pyogranulomatous and eosinophilic, or necrotizing and hemorrhagic. Although tissue cysts are absent in the skin in the cutaneous form of the disease, zoites can be found in histiocytes, keratinocytes, neutrophils, endothelial cells and fibroblasts.[4]

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CASE IV: HSRL-425 ZC09-447 (AFIP 3167231).

Signalment: Adult, female, California sea lion, pinniped (*Zalophus californianus*).

History: Found stranded on the beach, with repeated seizuring episodes. During the rehabilitation, the animal was becoming progressively weaker in spite of treatment and died 7 days later. No seizures were reported during rehabilitation.

Gross Pathology: At postmortem the stomach showed several raised and thick crater-like ulcers containing several nested round pale yellowish (2-5 cm long, 0.1 to 0.2 cm in diameter) worms. There were numerous round worms mixed with digested blood and mucus in the gastric lumen. The gastric and mesenteric lymph nodes were severely enlarged and blackened.

Other findings in the postmortem examination included marked perianal and rear limb edema. The thoracic cavity was filled with 3 - 4 liters of thick, hemorrhagic fluid. The abdominal cavity contained approximately 200 milliliters of serosanguineous fluid.

Laboratory Results: Leukocytosis and anemia.

Histopathologic Description: Stomach, glandular: The gastric mucosa has a locally extensive ulcerated area with extensive necrosis and inflammation. Within the inflammatory process, there are several sections of degenerate cuticular fragments of nematode parasites. The cuticle shows regularly spaced ridges. The parasite is characterized by a body cavity lined by coelomyarian muscles with prominent hypodermis with lateral cords. The inflammatory cells are predominantly degenerative neutrophils (nuclear streaming), macrophages, and occasional eosinophils. Beneath the ulcerative lesion there is a thick layer of granulation tissue with extensive fibrosis and neovascularization that replaces the submucosa and extends to the tunica muscularis. There are multifocal aggregates and clusters of mainly lymphocytes and plasma cells mixed with moderate numbers of macrophages in between the collagen bundles and fibroblasts. The surrounding, less affected mucosa of the glandular stomach is severely congested and contains moderate amounts of superficial hemorrhage. Scattered throughout the mucosa there is a superficial layer of necrotic tissue mixed with fibrillar acellular eosinophilic material (interpreted as fibrin), mixed with a large number of degenerative neutrophils, eosinophils and red blood cells. In some slides submucosal blood vessels show transmural focal areas of mineralization.

(Note: not all of the slides have cross sections of the parasite).

Contributor's Morphologic Diagnosis: Stomach: Chronic severe locally extensive ulcerative gastritis with intralesional nematodes and marked granulation tissue.

Contributor's Comment: Gastric ulcers are common necropsy findings in California sea lions (*Zalophus californianus*). The ulcerative lesions are mainly associated with nematodes. The majority of these roundworm

infestations are from the family *Anisakidae* (large roundworms). Pinnipeds are typically dead-end hosts for most stomach worms, acquiring them from other aquatic animals (copepods or fish).[3] Animals typically present with severe weight loss, anorexia and dark feces (melena). This case came from a stranded sea lion necropsied at a local marine mammal rehabilitation center and presented with typical gross ulcerative lesions.

Nematodes (roundworms) are the most diverse group of parasites found throughout the gastrointestinal tract of pinnipeds. Common nematode species that cause ulcerative lesions in the stomach of California sea lions include *Anisakis* spp. (larval stages), *Contracaecum* spp., *Phocascaris* spp. and *Pseudoterranova* spp. (adults).[1,2,5] Large numbers of California sea lions necropsied at the Center show macroscopically ulcerative gastritis that ranges from acute to chronic, moderate to severe, and contain various amounts of edema and hemorrhage.[6] Gross pathological findings in the stomach include well defined single or multiple superficial, transmural to volcanic (crater-like) ulcers in different stages of development that vary in size from 0.5 to 3.5 cm in diameter. Various amounts of digested blood mixed with mucus are found in the lumen. In many cases the most prominent finding in the stomach is the presence of one to several volcanic ulcers, with variable numbers of adult and larval stages of stomach worms nested and embedded in the center of the lesions. They frequently appear as chronic nodular ulcerative lesions with extensive granulation tissue with no parasites. Microscopic lesions include large areas of necrosis and hemorrhage in the center of the lesion surrounding cross sections of the parasite wall with coelomyarian musculature with prominent hypodermis with lateral cords.[4,6] The inflammatory component is composed of moderate numbers of eosinophils, neutrophils and macrophages, mixed with lymphoplasmacytic infiltrate.

In general, metazoan parasites in large numbers or in immunocompromised marine mammals contribute to the severity of illness or complicate an underlying disease. Secondary bacterial infections are often associated with parasite migration and tissue damage. Starvation, trauma, stress or toxic insults might predispose pinnipeds to the development of gastric ulcers. Stomach worms of *Anisakidae* family seem well adapted to the marine environment and able to colonize the gastric mucosa of different marine mammals as definite hosts.

AFIP Diagnosis: Stomach: Gastritis, necrotizing and ulcerative, multifocal, moderate, with granulation tissue, fibrosis, and adult anisakid nematodes.

Conference Comment: In addition to gastritis, several conference participants also noted the presence of mineralization of the tunica media in the submucosal vessels; the presence of this finding varied between slides.

One of the moderators (Dr. Chris Gardiner) commented that the presence of brightly eosinophilic homogenous material embedded in the gastric mucosa is seen frequently in cases of gastric anisakiasis in sea lions. While many pathologists interpret the material to be the cuticle of the parasite, Dr. Gardiner believes this material is not parasite *per se*; rather, it is a cast of the head of the worm composed of Splendore-Hoeppli material and represents the nematode's attachment site.

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