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CASE I – 02N2678 (AFIP 2890694)

Signalment: 1-year-old, female spayed Gordon Setter.

History: This animal had a history of being a poor grower and general poor thrift (per owner). The animal presented to the referring veterinarian with a three-week history of limping on the right pelvic limb, swelling of the right pelvic limb paw, enlarged peripheral lymph nodes and a draining tract lesion of the left thoracic limb paw. Abdominal ultrasound revealed nodules within liver, spleen and kidneys and the owners elected euthanasia due to poor prognosis.

Gross Pathology: The body was in poor nutritional condition with generalized atrophy of body fat stores and skeletal muscle. Abundant numbers of coalescing granulomas and pyogranulomas were scattered throughout the peripheral and internal lymph nodes, liver, spleen, kidneys, pancreas, diaphragm and heart.

Laboratory Results:

CBC

WBC	13,900/ul
Bands	417/ul slight toxicity
Neut	10,564/ul slight toxicity
Lymph	1390/ul
Mono	1112/ul
Eos	417

Urinalysis (cystocentesis)

Turbidity: Cloudy	Ketones: Neg
Color: Yellow/red	Hemoprotein: 3+
SG: 1.012	WBC: 4-6/hpf
pH: 5.0	RBC: >100/hpf
Protein: 1+	Bilirubin: Neg
Glucose: Neg	

Blood Chemistry Panel

CK: 2.5 mg/dL (0.5 - 1.6)	Total Protein: 7.6 g/dL (5.4 – 7.4)
BUN: 56 mg/dL (8 – 31)	Albumin: 2.4 g/dL (2.9 – 4.2)
Chloride: 97 mm/L (105 – 116)	Globulin: 5.2 g/dL (2.3 – 4.4)
Phosphorus: 7.0 mg/dL (3.0 – 6.2)	

Fungal cultures

Pseudallescheria boydii and *Scedosporium apiospermum* cultured from lymph nodes and draining tract exudate.

Contributor's Morphologic Diagnosis: Heart: Severe, multifocal to coalescing, pyogranulomatous and necrotizing myocarditis with intralesional fungal hyphae and chlamydospores.

Contributor's Comment: Multiple, coalescing, intensely cellular, nodular foci of inflammation and necrosis expand and efface the normal myocardial architecture. (Fig. 1) These foci are characterized by a central accumulation of degenerate neutrophils and necrotic cellular debris surrounded by large numbers of epithelioid macrophages, and lesser numbers of Langhans and foreign body-type multinucleated giant cells, lymphocytes and plasma cells. (Figs. 2,3) Within regions of intense inflammation there is extensive myocardial necrosis characterized by loss of myocyte cross striation, cellular hypereosinophilia, and fragmented, hyperchromatic nuclei. Abundant numbers of fungal elements including parallel walled, septate, dichotomously branching hyphae, and round, chlamydospores up to 50um in diameter are present in areas of inflammation. Chlamydospores have a clear central space, occasionally containing an eosinophilic granular material, surrounded by a basophilic, 2-4um thick spore wall. (Fig. 4) Often these spores are surrounded by epithelioid macrophages and are occasionally noted within the cytoplasm of macrophages and multinucleated giant cells. Within the interstitium of regions of intense inflammation there are large numbers of plump reactive fibroblasts and there are scattered foci of hemorrhage throughout the section.

Pseudallescheria boydii, and its asexual form *Scedosporium apiospermum*, is a ubiquitous, saprophytic, filamentous fungus belonging to the family *Microascaceae*. The organism is most commonly isolated from soil, vegetation, fresh water and sewage.[1] In recent years this fungal organism has arisen as one of the more

important emerging opportunistic fungal infections of immunocompromised people, especially post-organ transplant recipients and AIDS patients.[2-4] In humans the infection typically results in subcutaneous mycetoma formation, or a more serious condition referred to as pseudallescheriasis. Pseudallescheriasis encompasses a wide variety of diseases including infections of the upper respiratory tract and lungs, sinuses, soft tissues, arthritis, osteomyelitis, myocarditis, ophthalmologic disease and infection of the central nervous system. Infection is most often a result of penetrating trauma or surgical incision; however, primary respiratory infection is also possible. The organism, much like *Aspergillus*, is highly angioinvasive and dissemination may take place through blood vessels or lymphatics.[1]

In animals, infections with *P. boydii* or *S. apiospermum* have been described in the horse, cattle, dogs and a stranded northern elephant seal. The spectrum of disease in animals is quite similar to those seen in humans and includes cutaneous lesions and systemic infections. In the horse and dog, the organism has been reported as a cause of subcutaneous and abdominal mycetoma formation, septic arthritis, osteomyelitis, rhinitis, sinusitis, pneumonia and systemic infection.[5-13] Mycotic onychomycosis caused by *P. boydii* or *S. apiospermum* has been described in seven horses, and keratomycosis has been described in a single dog.[14,15] Infection with *P. boydii* has been implicated as a cause of placentitis and abortion in cattle and horses, and the organism has been reported as a cause of granulomatous pneumonia in a single calf.[16,17] In a single report involving a stranded northern elephant seal, infection resulted in fungal granulomas in multiple organ systems.[18]

In tissues the organism is virtually indistinguishable from other fungi with branching, septate hyphae such as *Aspergillus* and therefore, fungal cultures are necessary to make a definitive diagnosis. Proper identification of *P. boydii* is crucial, as the organism is resistant to many of the more common antifungal drugs. In humans, treatment usually requires both surgical and antimicrobial therapy.[1,2]

AFIP Diagnosis: Heart: Myocarditis, necrotizing, granulomatous, multifocal, severe, with numerous fungal hyphae with intercalary swellings[1], Gordon Setter, canine.

Conference Comment: The contributor gives a thorough review of *Pseudallescheria boydii* infection. As the contributor notes, this organism is difficult to distinguish from *Aspergillus* sp. If present, the identification of filamentous hyphae with terminal and intercalary vesicles (chlamydoconidia) may aid in differentiating these two organisms.[13]

Conference attendees noted rare protozoal cysts within myocytes in some sections. These cysts were not associated with inflammation. Our differential diagnosis included *Toxoplasma gondii* and *Neospora caninum*, but we were unable to further characterize this organism.

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CASE II – E03-263 (AFIP 2895810)

Signalment: One-year-old, castrated male, Holstein *Bos taurus* steer.

History: Holstein (*Bos taurus*) steer ear tag 44 was less than 10 months of age and weighed between 500-600 pounds and necropsy was performed on 8/21 (7 DPI). Inoculated (1 ml) of Rinderpest virus was administered subcutaneously over the right prescapular lymph node on 8/14.
Daily temperature data is as follows:

DPI:	Rectal temp (°F):	
0	Not done	
1	101.1	
2	102.5	
3	105.8	
4	106.2	
5	104.9	
6	105.3	
7	103.2	euthanasia and necropsy that day

Gross Pathology: Scattered vesicular oral lesions, some of them covered with necrotic debris. In the large intestine there are large areas of necrosis affecting

most of the lymphoid areas. These areas are covered by a thick layer of necrotic debris mixed with fibrin.

Laboratory Results: None reported.

Contributor's Morphologic Diagnosis: Colon: Severe diffuse subacute necrosuppurative colitis with intracytoplasmic inclusion bodies and occasional syncytial cells.

Contributor's Comment: Colon: Diffusely, there is a severe, acute, necrotizing and suppurative colitis with lymphangitis. On the mucosal surface, there are thick accumulations of fibrin mixed with large numbers of degenerate neutrophils, necrotic cellular debris, and large colonies of bacterial rods. There is necrosis of the mucosal epithelium and a locally-extensive area of full-thickness mucosal ulceration. The submucosa is markedly expanded by edema and contains moderate numbers of neutrophils and fewer macrophages. There are numerous variable sized (up to 25um) bright eosinophilic intracytoplasmic inclusion bodies. There are few scattered syncytial cells. Submucosal lymphatics are dilated (up to 1 mm) and contain large numbers of degenerate neutrophils mixed with necrotic cellular debris. There is diffuse hyperemia and endothelial cells are reactive.

Rinderpest, also called cattle plague, is an acute to subacute contagious viral disease of cattle with high morbidity rate and high mortality. Clinically it is characterized by fever, necrotic stomatitis and gastroenteritis. It is caused by a virus from the family Paramyxoviridae, genus Morbillivirus.

Rinderpest virus infects a variety of hosts including cattle, zebus, water buffalo and many species of wild animals: African buffalo, eland, kudu, wildebeest, various antelope, bushpig, warthog, giraffe, as well as domestic sheep and goats are susceptible. Asian pigs seem more susceptible than African and European pigs. Rinderpest is rare among camelidae. Rabbits, hamsters, mice, giant rats, ferrets and susliks have been infected only experimentally. There is no age- or sex-linked predisposition.

The disease is characterized by erosions and necrosis in the gastrointestinal tract. These erosions and necrotic areas are the classic vesicular lesions in the mouth and the intestinal wall.

Differential diagnosis include:
Bovine viral diarrhea (BVD) virus (pestivirus)
Foot-and-mouth disease (picornavirus)
Infectious bovine rhinotracheitis (alpha herpesvirus)
Malignant catarrhal fever (gamma herpesvirus)

Vesicular stomatitis (rhabdovirus)
Peste des petits ruminants (small ruminants; paramyxovirus)
Bluetongue (small ruminants; reovirus)

The Global Rinderpest Eradication Programme (GREP) is an international effort, whose Secretariat is at the Food and Agriculture Organization of the United Nations, to eradicate one of the world's most devastating livestock diseases. This effort has been an international partnership with valuable input from the Office International des Epizooties, European Union, African Union-Inter African Bureau for Animal Resources, USAID and other donors in Europe, numerous non-governmental organizations and most importantly the countries and villagers themselves. Currently, Rinderpest is believed to be limited to a focus of infection in east Africa (Somali ecosystem), but further field work is required to ensure that foci of virus activity in southern Pakistan or southern Sudan have been truly removed. GREP is designed to coordinate responses and develop guidelines to address rinderpest issues, including outbreak and surveillance strategies in order to reach the goal of complete global eradication by the year 2010 with the following time table:

2003 - declaration of worldwide provisional freedom from rinderpest.

2006 - freedom from disease for whole world established.

2008 - freedom from sub-clinical infection established.

2010 - Global Declaration of complete freedom from rinderpest.

http://www.fao.org/news/1996/960803-e.htm
http://www.fao.org/news/2000/000607-e.htm

http://www.fao.org/ag/AGA/AGAH/EMPRES/grep/e_rinder1.htm

AFIP Diagnosis: Colon: Colitis, necroulcerative, subacute, diffuse, severe, with lymphoid necrosis, crypt herniation, diphtheritic membrane, transmural edema, and eosinophilic intracytoplasmic inclusion bodies, Holstein, bovine.

Conference Comment: Rinderpest is transmitted directly or indirectly through infectious secretions and, in confined areas, by aerosol droplets. The rinderpest virus is inhaled or ingested and localizes in the palatine tonsils and regional lymph nodes where it replicates. The incubation period usually lasts 4-5 days, after which a 2-3 day period of viremia occurs that coincides with the onset of fever and the clinical syndrome. After the viremic stage, the virus replicates in lymphoid tissues,

bone marrow, and the mucosa of the upper respiratory tract and gastrointestinal tract.[2,3,4]

Typical gross findings include erosions in the upper gastrointestinal and respiratory tracts; edema, hemorrhage, and necrosis of Peyer's patches; and hemorrhage and congestion that run transversely across the colonic mucosa to produce a "zebra striped" appearance. Since the virus is tropic for lymphoid tissues, diffuse lymphoid necrosis is characteristic. Mucosal epithelium of the upper gastrointestinal tract and crypt epithelium become necrotic, and syncytial cells may form. Eosinophilic intracytoplasmic and intranuclear inclusion bodies may be evident in infected cells.[2,3,4]

The contributor gives an important differential diagnosis list for rinderpest. In addition to bovine pestivirus and rinderpest, salmonellosis should be considered as a cause of Peyer's patch necrosis.[3]

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CASE III – 02-2069 (AFIP 2890853)

Signalment: 9 year-old spayed female Labrador Retriever dog (*Canis domesticus*).

History: The dog had a 3-year history of a liver mass that was non-progressive in size, glomerulonephritis, and had been treated for hypothyroidism with Soloxine for an unspecified period of time. Liver enzyme activity was persistently increased. Progressive pulmonary radiodensities were seen on thoracic radiographs. There had been a recent history of *E. coli* sepsis on two occasions along with severe

diarrhea with profound melena and weight loss. Megabacteria and fungal overgrowth were noted in the feces.

Gross Pathology:

External examination: Diffuse, severe muscle wasting
Pendulous abdomen with fluid wave

Integument/Subcutis: Subcutaneous mass (3x2x1.5cm) on the dorsal midline, cranial to the scapulae.

Peritoneal cavity: 1L serosanguineous effusion

Liver: Hepatomegaly, marked
Hepatic mass (hepatocellular carcinoma)

Digestive system: Stomach: Trichobezoar
Multifocal acute and chronic ulcers
Pancreas: Multifocal 1-3 mm raised white firm foci within the parenchyma
Small intestine: Multifocal 1-3 mm raised white firm foci within the mucosa and submucosa.

Respiratory system: Diffuse consolidation with gritty, hard parenchyma on cut surface

Urinary system: Multifocal renal cortical cysts

Cardiovascular system: Mild left AV valve endocardiosis

Laboratory Results: None reported.

Contributor's Morphologic Diagnoses:

Pancreas: 1) Granulomatous pancreatitis, marked, multifocal with intralesional trematode eggs.
2) Interstitial fibrosis, mild to moderate, diffuse.
3) Nodular hyperplasia, mild, multifocal.

Contributor's Comment: The diagnosis of *Heterobilharzia americana* infection was made by recognition of spherical to oval ova containing miracidia in the pancreas and the intestine. Adult schistosomes live in the vascular system of the definitive host after migration through parenchymal organs. Ova pass through the mesenteric vessels, penetrate the intestinal mucosa and pass into the intestinal lumen. Miracidia are released into water and penetrate snails from the Lymnaeidae

family and then from sporocysts. After they develop into cercariae, they leave the snail and are released into water to penetrate the skin of the main hosts, raccoons, dogs or nutria to complete the life cycle. Lesions can be found along the portal vein flow, affecting intestines, pancreas and the liver.[1] There was marked pulmonary interstitial mineralization detected at postmortem examination. Schistosomiasis in canines has been associated with hypercalcemia, but not mineralization of organs. It is unlikely that schistosomiasis was responsible for pulmonary mineralization in this case since there was no evidence of hypercalcemia in this dog. Renal disease in this geriatric canine is the most likely cause for a transiently elevated the Ca:P ratio, indicating that this lung mineralization was most likely attributable to renal rather than parasitic causes. Interestingly, schistosomiasis in humans has been associated with hepatic neoplasia, but there was no evidence of hepatic involvement with schistosomes in this case.

AFIP Diagnosis: Pancreas: Pancreatitis, granulomatous, multifocal, moderate, with nodular regeneration, interstitial fibrosis, and trematode eggs, Labrador Retriever, canine.

Conference Comment: The Schistosomatidae family, or blood flukes of mammals and birds, contains three genera of veterinary importance in mammals: *Schistosoma*, *Heterobilharzia*, and *Orientobilharzia*. These are important parasites in Africa, Asia, the southern United States, and tropical or subtropical regions in which the intermediate snail host is found.[4]

Schistosoma bovis and *S. japonicum* are the most pathogenic of these flukes in cattle and sheep, and are found in the mesenteric veins. The main definitive hosts of *Heterobilharzia americana* are the dog, raccoon (*Procyon lotor*), and nutria (*Myocastor coypus*), although other hosts have been identified. *Schistosoma mansoni* is endemic in humans in Africa, portions of the Middle East, and Central and South America. A link between hepatocellular carcinoma and schistosomiasis in humans has been proposed, but frequent concomitant viral hepatitis complicates understanding the parasite's role. Woodchucks (*Marmota mona*x) can be experimentally infected with *S. mansoni*, which makes them a potentially useful animal model to study the outcomes of chronic concurrent schistosomal and viral hepatitis.[4,5,6,7]

A characteristic feature of these flukes is that the male and female are permanently coupled. The male is shorter than the female and has a distinctive gynecophoric canal, which encloses the more slender female. These trematodes live in veins and their eggs circulate and lodge in tissues, which cause the most severe lesions: microgranulomas (or "pseudotubercles") surrounding schistosome

eggs in the liver, spleen, brain, gastrointestinal tract, urinary bladder, and other organs. The adult schistosomes can cause an eosinophilic endophlebitis, with intimal proliferation and thrombosis.[4,5]

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CASE IV – P03-9694 (AFIP 2908331)

Signalment: 11-year-old, Male intact, Cairn terrier, canine.

History: Bilateral glaucoma.

Gross Pathology: Two eyes (OS and OD). One shows lateral flattening and a local projection within the posterior limbal area, both eyes do not show a trabecular aspect of the ligamentum pectinatum but a solid ridge instead. Both eyes show asteroid hyalosis.

Microscopic Description:
Diffuse proliferation of cells containing large amounts of moderately coarse melanin granules in the iris, ciliary body, iridocorneal angle and to a lesser extent in the

choroid, sclera, cornea and episcleral tissue. Occlusion of the corneoscleral meshwork with focal synechia anterior. Focal extensive retinal atrophy. Pigmentation and slight squamous metaplasia of corneal epithelium (epidermidalization) and marked vascularization of corneal stroma with a mixed inflammatory infiltrate. Moderate lymphoplasmacytic inflammation of conjunctiva.

Laboratory Results: None reported.

Contributor's Morphologic Diagnoses:
MD: eye:
1. Uveal melanosis, severe, diffuse, bilateral, consistent with melanocytic glaucoma in the cairn terrier.
2. Secondary keratitis and retinal atrophy, anterior synechia

Contributor's Comment: The pathological changes in the eye of this Cairn terrier were bilateral. This condition, named melanocytic glaucoma, formerly pigmentary glaucoma, is known to occur occasionally in older cairn terriers (from the age of 10 and older). It has been described in the USA in 1984 by Covitz and others, in the UK in 1991 by Peterson-Jones.

The origin of the proliferating pigmented cells is not yet clear; they are described as being melanocytes or melanophores. The cells seem to show infiltrative behavior. However, metastasis has never been observed. The prognosis is poor, mainly because usually both eyes are affected and sooner or later glaucoma develops due to occlusion of the corneoscleral network.

Formerly the condition was named pigmentary glaucoma, referring to the disease in humans now known as pigment dispersion syndrome. This syndrome occurs rarely in young (20-40 years) myopic individuals, and is characterized by intensive pigment deposition on the posterior lens, iris surface, trabeculas, retina, depigmentation of the iris, Krukenberg's spindle (deposition of liberated melanin pigment on the posterior cornea in a vertical line) and radial thinning of the iris. Most of these features are not present in the dogs, suggesting a different condition with different pathogenesis.

On other slides of this dog (not provided), there are multiple round pale eosinophilic, crystalline birefringent structures in the corpus vitreum (asteroid hyalosis). This is seen as a reactive process in many eye pathologies.

AFIP Diagnosis: Eye: Melanosis, uveal, diffuse, moderate, with peripheral anterior synechiae, retinal atrophy and detachment, and chronic keratoconjunctivitis (melanocytic glaucoma), Cairn terrier, canine.

Conference Comment: Canine glaucoma is classified as either primary, secondary, or congenital, based on possible cause. Congenital glaucoma is seen at birth or shortly thereafter, and is associated with an anterior segment anomaly. Primary glaucoma develops without concurrent ocular disease and is hereditary in some breeds. It may result from defective development of the iridocorneal angle (narrow-angle glaucoma) or a functional abnormality in the filtration angle (open-angle glaucoma).[5] Secondary glaucoma is associated with some antecedent or concurrent ocular disease that causes increased intraocular pressure. Melanocytic glaucoma of the Cairn terrier is an unusual form of secondary glaucoma. It affects middle-aged to older Cairn terriers and may be unilateral or bilateral. Melanocytes or melanomacrophages proliferate and are located in the filtration angle, episcleral and subconjunctival tissues, tapetal ocular fundus, and the meninges around the optic nerve head. Although the cause of this melanocytic proliferation is unknown, the disease process is similar to benign iris melanoma. The pattern of melanocytic glaucoma differs from that of neoplasia, however, in that it is diffuse rather than nodular.[3,4,5]

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