

AAVLD 2020 Diagnostic Pathology Slide Seminar

63rd AAVLD Diagnostic Pathology Slide Seminar



American Association of Veterinary Laboratory
Diagnosticians

Thursday, October 8, 2020

4:00-6:30 PM

63rd AAVLD Diagnostic Pathology Slide Seminar

Case	Species	Tissue	Diagnosis
1*	Cat	Nasal tissue, skin	Protothecal rhinocellulitis
2*	Dog	Esophagus	<i>S. lupi</i> esophagitis
3*	Horse	Brain	<i>S. neurona</i> myeloencephalitis
4*	Turkey	Kidney	Round cell tumor
5*	Bearded dragon	Great vessels	Microsporidial arteritis, aneurysm
6	Pig	Heart	Endocardial actinobacillosis
7	Dog	Brain	Canine distemper
8*	Sheep	Nasal tissue	Enzootic nasal tumor
9*	Ox	Heart	<i>S. cruzi</i> myocarditis
10*	Chicken	Tendon	Reoviral tendonitis
11*	Cat	Brain	Bromethalin toxicosis
12*	Rat snake	Esophagogastric wash	<i>Strongyloides</i> sp. infection (see USB)
13*	Camel	Bone	Fibrous osteodystrophy
14*	Pig	Skin	Erysipelothricosis
15*	Ox	Heart	Monensin toxicosis
16*	Ox	Lymph node	Tuberculosis

*Presenter eligible for the 2020 Diagnostic Pathology Slide Seminar Resident/Graduate Student Award.

Underlining denotes the presenting author.

Case 1

Nasal dermatitis, cellulitis, and rhinitis in a domestic shorthair cat caused by *Prototheca cutis*: a case report*

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An adult, neutered male, indoor/outdoor, domestic short-haired cat presented to its primary veterinarian for sneezing, wheezing, congestion, and rhinitis in June 2019. Over seven months, the nasal planum became progressively rounded and disfigured. A biopsy submitted to a private diagnostic laboratory yielded a diagnosis of fungal rhinitis with organisms suggestive of *Cryptococcus* spp. Due to concerns over the zoonotic potential of *Cryptococcus* spp., the animal was euthanized and submitted to the Athens Veterinary Diagnostic Laboratory for postmortem examination with culture and speciation of the presumptive *Cryptococcus* spp.

Grossly, the dorsal nasal planum and nasal bridge was irregularly rounded, enlarged, and bulging. A locally extensive area of connective tissue and musculature overlying approximately 70% of the nasal bridge and dorsal nasal planum was diffusely soft, variably tan to light orange, and mildly gelatinous. The subjacent nasal turbinates were mildly swollen, red, and soft.

Histopathologic examination revealed dense, granulomatous to pyogranulomatous inflammatory infiltrates in the skin, subcutis, bone, and nasal turbinates. Disseminated throughout the turbinates were numerous, extracellular and intrahistiocytic, round to oval, 8-20 µm diameter algal sporangia with a clear, 2-4 µm thick wall and contained either a central basophilic nucleus or multiple (2-8) wedge-shaped, radially arranged endospores. Algal cell walls were strongly PAS and GMS positive.

Fungal culture was successful, but no genus/species ID was obtained from MALDI-TOF and GEN III Microbial ID. Targeted, partial sequencing of the internal transcribed spacer (ITS) region and the D1/D2 region of the 28S rRNA gene were performed, which yielded sequences 96% and 100% homologous to those from *Prototheca cutis* available in BLAST and CBS-KNAW fungal databases.

This is the first report of *P. cutis* isolated from a veterinary species and only the second description of the agent since its initial isolation from an ulcerative skin lesion in a human. While nasal cryptococcosis is more frequently recognized and diagnosed in cats, protothecosis remains an important differential diagnosis for nasal dermatitis, cellulitis, and rhinitis.

Case 2

Spirocerca lupi* Esophagitis in a German Shepherd Dog

M. Mainenti, A.J. Van Wettere, T. Baldwin
Utah Veterinary Diagnostic Laboratory, Logan, Utah

Signalment: A 15-month-old, 27.0 kg, male, German Shepherd dog.

History: The dog was submitted for necropsy following unexpected death during playing activity.

Gross necropsy findings: Severe hemothorax and pulmonary atelectasis were present. A 9 x 5 x 4 cm, poorly demarcated, firm, dark red mass expanded the middle mediastinum, and adhered to the aortic and esophageal adventitia. The aorta had a focal, 6 x 1 mm, full-thickness tear at the site of the mass. Multiple, up to 2 x 2 cm, firm, white nodules severely thickened the wall of the caudal esophagus. Numerous, 1 x 3 x 20 mm, red, coiled nematodes were within the esophageal nodules and mediastinal mass and protruded through the esophageal mucosa.

Histopathologic findings: Coalescing fibrotic nodules severely expand and efface the esophageal submucosa and muscularis and contain numerous, up to 1 mm wide, cross sections of adult nematodes. Nematodes have a 6 µm thick, hyaline, smooth cuticle, coelomyarian-polymyarian musculature, large lateral cords, and a pseudocoelom with amorphous eosinophilic material, intestine lined by uninucleate epithelial cells with a prominent tall brush border, and male or female reproductive organs. Numerous, 10 x 30 µm, thick-shelled, embryonated eggs with vermiform larvae are within nematode uteri. Numerous eosinophils, fewer neutrophils, and macrophages, degenerate collagen fibers, and necrotic debris surround the nematodes.

Final diagnosis: Spirocercosis with esophagitis, mediastinitis, aortic rupture, hemothorax, and pulmonary atelectasis.

Comments: Diagnosis of spirocercosis was made upon detection of nematodes grossly and histologically consistent with *Spirocerca lupi* within the esophagus and mediastinum. Death was attributed to hypovolemic shock and pulmonary atelectasis consequent to hemothorax caused by rupture of the thoracic aorta secondary to *Spirocerca lupi* infection. *Spirocerca lupi* is a spirurid nematode of canids. Infection occurs by ingestion of L3 larvae in intermediate or paratenic hosts, followed by perforation of the gastric mucosa and migration along visceral arteries to the aorta. After 2 to 4 months, L4 larvae migrate to the wall of the caudal esophagus where they develop to adulthood and shed eggs into the lumen. Aberrant migration may occur. Gross lesions include large, intramural, esophageal nodules which may extend into the mediastinum, aortic aneurysm, rupture, and thromboembolism, esophageal sarcomas, spondylitis, and hypertrophic osteopathy.

Case 3

An unnerving case of *Sarcocystis neurona**

C.G. Hendricks, N. Helgert
Breathitt Veterinary Center, Hopkinsville, Kentucky

Signalment and History: A 13-year-old castrated Quarterhorse was presented for necropsy after 7-days of progressive neurologic disease.

Gross Findings: The neuropil of the rostral thalamus to caudal midbrain was replaced by a 3cm diameter area of soft-firm to viscous material that displaced the midline and was rimmed by a band of white tissue.

Microscopic Findings: The lesion was characterized by a well-circumscribed area of neuropil loss with gitter cells, multinucleated giant cells, lymphocytes, plasma cells, and scattered eosinophils admixed with necrotic cellular debris and fibrin. Surrounding Virchow-Robin spaces were expanded by clear space and up to 10 layers of mixed inflammatory cells. Throughout the surrounding neuropil were many necrotic neurons and gemistocytic astrocytes. There was compression and distortion of the third ventricle.

Additional Findings: Formalin-fixed, paraffin embedded tissues were submitted for PCR and were positive for *Sarcocystis neurona*. Immunohistochemistry for *S. neurona* demonstrated scattered macrophages, glia and endothelial cells contained intracytoplasmic schizonts and merozoites.

Final Diagnosis: Equine protozoal myeloencephalitis

Comment: Equine protozoal myeloencephalitis (EPM) is a necrotizing and inflammatory condition of the brain and spinal cord. The cause, *Sarcocystis neurona*, is an obligate intracellular parasite within the phylum Apicomplexa. Horses are an accidental intermediate or dead-end host, opossum are the definitive host. Transmission occurs when horses ingest opossum feces containing *S. neurona* sporocysts. Once in the intestines, the sporocysts hatch and sporozoites are phagocytosed by macrophages, eventually forming schizonts in endothelial cells. Infected endothelial cells in the CNS rupture and release merozoites into the neuropil resulting in microglial cell activation, vasculitis, thrombosis and hemorrhage. Neurologic signs include depression, cranial nerve paralysis, urinary incontinence, and muscle atrophy. Antemortem diagnosis should integrate neurologic evaluation, radiography, and examination of CSF for intrathecal antibodies. Biomarkers such as decreased nitric oxide in the cerebrospinal fluid and increased serum levels of phosphorylated neurofilament have been recently associated with infection with *S. neurona*.

Case 4

Metastatic round cell tumor in an eastern wild turkey (*Meleagris gallopavo*)*

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SIGNALMENT: Adult, female wild turkey (*Meleagris gallopavo*)

HISTORY: The turkey was lethargic and later found dead by a residential pond.

GROSS: The carcass was in adequate nutritional condition. A 22-cm long, tri-lobed, tan, soft mass was attached to the crop serosa and extended caudally to the heart base. The mass was divided into three discrete segments connected by thin, fibrous strands; the majority of the mass was amorphous, soft and varied from uniformly tan to mottled tan, gray and pink. The cranial portion contained a blind-ended sac that contained greenish-black material and the middle and caudal portions had central, friable material (interpreted as necrotic tissue). The adventitia of an artery adhered to the ventral surface of the mass. Similar, smaller masses were on the serosa or capsular surfaces of the proventriculus, kidneys, and ovarian follicles.

HISTOPATHOLOGY: Approximately 75% of renal architecture is effaced by an expansive, non-encapsulated, poorly demarcated, densely cellular neoplasm composed of sheets of tightly packed round cells over a scant, fibrovascular stroma. The round cells have scant to moderate, weakly eosinophilic, sparsely granular cytoplasm, distinct cell margins, and 1-4, round, deeply basophilic, central to eccentric nuclei with clumped chromatin and no visible nucleolus. There is mild anisocytosis and anisokaryosis. There is one mitotic figure/2.4 mm² (equivalent to 10 FN22/40X fields). The remainder of the mass has few, remnant, tubular epithelial cells, irregular bands of fibrous connective tissue and necrotic foci.

ANCILLIARY DIAGNOSTICS: There was no immunohistochemical labeling of neoplastic cells with antibody to CD3, and rare labeling of neoplastic cells with PAX5. A sample of bone marrow was positive for lymphoproliferative disease virus (LPDV) DNA, while a sample of tumor was negative for LPDV and reticuloendothelial virus (REV) DNA, all by polymerase chain reaction test.

FINAL DIAGNOSIS: Metastatic round cell neoplasia

COMMENTS: This turkey had a disseminated round cell neoplasm that replaced and invaded numerous organs. Although cell morphology was suggestive of lymphocytes or plasma cells, lack of widespread or predominant immunohistochemical labeling of neoplastic cells for CD3 and PAX5 suggested these are not likely the cell of origin. LPDV is a commonly detected oncogenic retrovirus in wild turkeys; REV is also detected, albeit less commonly, and both can cause similar lesions as the present turkey.

Case 5

Microsporidium-associated arteritis and aneurysm in an adult bearded dragon*

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Blacksburg, Virginia

Signalment and Clinical History: An adult, intact male bearded dragon was submitted for necropsy after a brief history of respiratory distress; death occurred spontaneously before veterinary care could be administered. Major postmortem findings included significant hemopericardium and a small blood clot adhered to the adventitia of a great vessel of the heart.

Microscopic Description: Heart, pulmonary artery and aortic arch. The walls of both great vessels had multifocal, transmural, severe arteritis characterized by an infiltration of many foamy macrophages, heterophils, lymphocytes and plasma cells; rare macrophages infiltrated along the epicardial surface of the left ventricle. Occasional macrophages within these inflammatory foci contained many lightly basophilic, elongate microorganisms up to 2.0 μm long. There was a large, focal disruption of the tunica intima of both great vessels; a mixture of hemorrhage, fibrin and inflammatory cells expanded the vessel wall and bulged into the lumen and there was a pocket of hemorrhage at the corresponding adventitial surface (aneurysm).

Histochemistry: The organisms of the heart and great vessels stained positive with Giemsa and PAS and were GMS, acid-fast and gram negative.

PCR: PCR based on ribosomal RNA is pending at the time of abstract submission.

Diagnosis: Pulmonary artery and aortic arch. Arteritis: severe, transmural, granulomatous, with focal aneurysm, fibrinoid necrosis of the vascular wall and intracytoplasmic protozoal-like microorganisms

Comments: Cause of death in this patient was attributed to cardiac tamponade; microorganisms within the artery wall likely initiated arteritis, leading to a weakened vessel wall, aneurysm, and subsequent hemopericardium. In addition, individual macrophages with similar intracellular microorganisms were randomly distributed within the interstitium of the epididymis and adjacent peripheral nerve. A recent case series of two bearded dragons identified similar lesions and characterized the intracytoplasmic organism as the microsporidia *Encephalitozoon pogonae*. The morphology, histochemistry, and angioinvasiveness of the organisms seen in our case are suggestive of the same organism. Research on the source and route of infection is limited; potential routes include diet-induced and transovarial transmission.

Case 6

Endocardial actinobacillosis in a finisher pig

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Veterinary Diagnostic Laboratory, Iowa State University, Ames, Iowa

Signalment: A 12-week-old, finisher pig

History: A barking cough in 50% of the population with 100 reported acute mortalities.

Gross Necropsy Findings: Multifocally lining the cuspal margins of the right atrioventricular valve were multiple, variably sized, pale tan, friable vegetations. Submitted lung sections were distended and dark red with multifocal strands of fibrin adhered to the pleura.

Histopathology Findings: Lining the valvular leaflets are numerous, variably sized, exophytic masses comprised of densely packed accumulations of fibrin and degenerative neutrophils, which surround colonies of bacteria. Densely packed, 2-4 µm in length, extracellular, gram-negative coccobacilli form intricate, arborizing colonies along the fibrinous margin. The underlying valvular leaflets are infiltrated with abundant neutrophils and mononuclear cells. Organized fibrous stroma and fibroblasts multifocally replace the myxomatous matrix of the valve and adjacent endocardium. There are abundant neutrophils and macrophages infiltrating the subjacent interstitium and adjacent myofibers are thin and wavy.

Final Diagnosis: Vegetative valvular endocarditis with bacterial colonies, fibrinosuppurative, chronic, multifocal, severe

Ancillary Diagnostics: Moderate to high growth of *Actinobacillus equuli* was isolated from the heart valve and spleen. *Streptococcus suis* and *Pasteurella multocida* type D were isolated from the lung. Lung tissue was positive for influenza A (H1N1) virus and porcine circovirus type 3 was detected in splenic tissue by PCR. Paraffin embedded heart valve tissue was negative for *A. pleuropneumoniae* and *A. suis* by PCR.

Comments: *A. equuli* is a common cause of embolic nephritis, arthritis, and septicemia in neonatal foals. Two subspecies exist; *A. equuli* subsp. *haemolyticus* and *A. equuli* subsp. *equuli*, with the latter most commonly isolated in equines. *A. suis* represents the most commonly isolated *Actinobacillus* spp. in swine at the ISU VDL with lesions commonly consisting of pleuropneumonia and sepsis. *A. pleuropneumoniae* is another commonly isolated organism, which causes similar disease in finishing pigs. *A. equuli* has been reported sporadically in sows and gilts, consisting of generalized septicemia with case reports of valvular endocarditis reported. This case highlights the detection of *A. equuli* in finishing pigs as a cause of sporadic systemic disease with an incompletely understood ecology within swine production systems.

Case 7

Canine Distemper Virus Encephalomyelitis in a Vaccinated 4-year-old Greyhound

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A properly vaccinated 4-year-old female spayed blue greyhound was euthanized after a period of chronic inappetence and an acute onset of fever, diarrhea, and neurologic signs. The gross findings of the diagnostic necropsy included mild mitral endocardiosis, mild acute multifocal esophageal ulcerations, and moderate multifocal splenic congestion.

Microscopically throughout the brainstem, cerebellum, and spinal cord, there are multifocal aggregates of macrophages and lymphocytes often forming thick perivascular cuffs and large multifocal to coalescing foci of severely vacuolated white matter with gitter cells. The astrocytes within the areas of inflammation contain large (2-7 μ m), round to rectangular, pale eosinophilic intranuclear and less frequently intracytoplasmic inclusion bodies which marginate the chromatin. There are also occasional syncytial cells which also frequently contain pale eosinophilic intracytoplasmic inclusions.

Canine distemper virus (CDV) PCR and immunohistochemistry were both positive resulting in a final diagnosis of CDV encephalomyelitis. CDV is a morbillivirus that causes respiratory, neurologic, and gastrointestinal disease in canids and other species. In older dogs, infections often result in neurologic signs without systemic manifestations. Approximately 30% of reported CDV encephalitis cases occur in immunized dogs as was the case in this dog. While it is likely that vaccine failure accounts for some of these cases, mutation of the wildtype CDV is an emerging phenomenon. New genetic variants of the virus may be responsible for infections in fully vaccinated dogs, as the new variants may not express the antigens targeted by the vaccines.

Case 8

Enzootic nasal tumor in a Suffolk ram*

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A two-year-old Suffolk ram presented with a six week history of labored breathing. The animal was initially responsive to antibiotics but would quickly become refractive to treatment and continued to decline, and euthanasia was elected. Necropsy revealed a moderately well-defined, intra-nasal, white, soft mass that obliterated the nasal turbinates and extended into the pharynx. There was also abundant thick, yellow exudate within the nasal cavity. Histological examination of the mass revealed abundant papillary projections of well-differentiated epithelial cells on a fine fibrovascular stroma. Throughout the mass were free and distinct clusters of neutrophils often associated with lakes of eosinophilic fluid. Rare remnant, entrapped bone spicules from the effaced nasal turbinates are scattered throughout examined sections. Polymerase chain reaction was performed from a sample of the mass and was positive for enzootic nasal tumor virus-1 (ENTV-1). These findings are a classic representation of 'enzootic nasal tumor' in a sheep.

Infection with beta-retroviruses ENVT-1 and ENTV-2 is associated with nasal adenomas/adenocarcinomas in sheep and goats, respectively. The disease is most common in adult animals, but lambs as young as six months have been reported to develop the disease. While experimentally lesions can develop in as little as three months, natural cases typically take one to three years to develop. The histologic appearance is consistent with that of a low grade adenocarcinoma or adenoma, the former of which is justified when there is evidence of bone invasion. Regardless, metastatic disease has not been reported. While the virus may be present in flocks without overt disease, the detection of the virus by PCR in conjunction with characteristic lesions are consistent with retroviral-associated disease; however, electron microscopy or immunohistochemistry are required to confirm viral-associated vs spontaneous development of the neoplasm.

References:

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De las Heras M, Ortín A, Cousens C, Minguijón E, Sharp J.M. 2003. Enzootic Nasal Adenocarcinoma of Sheep and Goats. In: Jaagsiekte Sheep Retrovirus and Lung Cancer. Fan H (editor). Current Topics in Microbiology and Immunology, Vol. 275. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-642-55638-8_8

Case 9

Fatal *Sarcocystis cruzi*-induced eosinophilic myocarditis in a Hereford heifer*

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Clinical history: A 2.3-year-old Hereford heifer presented with sudden death.

Gross findings: The carcass was in good body condition with adequate adipose stores. Both ventricular free walls of the heart, the ventricular septum and to a lesser extent the atria were expanded up to 5 cm thickness and were green-tinged, dull, and semi-friable. Scattered within the affected regions were dozens of 0.2-0.5 cm diameter tan-white, firm, occasionally bulging nodules. Both jugular veins were markedly firm and engorged by abundant clotted blood.

Histopathology: Approximately 80% of the myocardium is obliterated by eosinophilic necrotic debris and large numbers of eosinophils admixed with occasional 120-160 micron in diameter degenerate protozoan cysts containing myriad bradyzoites (*Sarcocystis* sp.). The inflammation centers around degenerate sarcocysts and individual zoites sequestered by multinucleated giant cells, macrophages, abundant degranulated eosinophils and mineralization. Similar eosinophilic granulomatous infiltrations surrounding degenerate sarcocysts were observed in the skeletal muscle of the esophagus. Intact sarcocysts scattered in the myocardium are present external to sites of inflammation.

Ancillary testing: Immunohistochemistry with a non-commercial antibody for *Sarcocystis cruzi* immunolabeled the myocardial sarcocysts and the degraded *S. cruzi* antigens within macrophages and necrotic areas. The sections were immunonegative for *S. falcatula*, *S. neurona* and *Neospora* sp.

Comments: The pathologic and immunohistochemical assessment confirmed the diagnosis of fatal *S. cruzi*- induced eosinophilic granulomatous myocarditis. *Sarcocystis* sp. are very common incidental microscopic findings in many species including cattle, described as 100% frequency per some studies. Nevertheless, ruptured sarcocysts can cause myositis and for cattle the green discolored muscle (bovine eosinophilic myositis; BEM) is a significant cause of condemnation in abattoirs. While BEM is a subclinical disease, it has been fatal in rare cases- attributed to hypersensitivity in some animals. Up to now, the vast majority of fatal and chronic eosinophilic myocarditis have been reported in Hereford or Hereford-cross heifers hypothesized to produce exaggerated levels of IgE against *Sarcocystis* sp. This is the fourth reported case supporting the possible breed predisposition.

References:

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Tessele B, et al. 2013. Pesq Vet Bras, 33: 1345-1348

Case 10

Gastrocnemius Rupture in Broilers with Avian Reovirus Infection*

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Fifteen 8-week-old broiler chickens from 3 farms of the same poultry company (5 chickens per farm) were submitted to the Poultry Diagnostic and Research Center Diagnostic Laboratory for lameness evaluation. Necropsy revealed unilateral or bilateral shank thickening with edematous gastrocnemius and digital flexor tendons in 12 animals and gastrocnemius rupture in 2 animals. Microscopic lesions were typical of Avian Reoviral (ARV) infection, showcasing synovial hypertrophy with villous projections, subepithelial lymphoplasmacytic aggregates with germinal centers, and tendon replacement fibrosis with granulation tissue. The presence of extravasated erythrocytes and hemosiderophages surrounding areas of tendon and tendon sheath fusion indicate repeated fibroplasia with subsequent microtears. The altered composition of the tendon, combined with its impaired movement within the sheath, ultimately culminated in the grossly observed tendon rupture. ARV infection was confirmed via successful virus isolation in five additional, younger flocks within the same company. The significant welfare and economic damage caused by ARV infection is a growing concern in broiler flocks throughout the United States, due to variant serotypes falling outside the protection afforded by available commercial vaccines. Therapeutic intervention is equally inaccessible due to the relatively slow, subclinical progression of the disease preceding clinical manifestation and catastrophic failure of the tendon as animals approach market weight.

Case 11

Bromethalin Toxicosis in a Cat*

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A 3-year old, female spayed domestic shorthair was submitted for necropsy with a 12-hour history of lateral recumbency. MRI was performed postmortem, revealing a diffuse hyperintensity exclusive to the white matter. No significant gross findings were evident. Histopathologic findings revealed moderate to marked, diffuse vacuolation of the white matter within the following locations: internal capsule, corpus callosum, corona radiata, fimbria of the hippocampus, cerebellar white matter, and lateral and ventral funiculi of the spinal cord. Abdominal fat was submitted to the California Animal Health and Food Safety Laboratory (CAHFS) for desbromethalin, which came back positive. Bromethalin is a potent neurotoxin found in rodenticides, in which cats are highly sensitive to. Typical clinical signs include development of hindlimb ataxia, paresis, paralysis, CNS depression, and focal and generalized seizures. Once neurologic signs set in clinically, the prognosis is poor.

Case 12

Ova of a novel *Strongyloides* species identified in the esophagus and stomach of a hundred flower ratsnake (*Elaphe moellendorffi*)*

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A 6-year-old, female flower rat snake was presented for evaluation of open mouth breathing, stertor, and hypersalivation. The animal was fed two live rat pups once a week. Other husbandry information was not provided. In addition to the reported sialorrhea, dyspnea was observed on the physical examination but was otherwise unremarkable. The bacterial culture of a laryngeal swab resulted in no growth. The animal was prescribed ceftazidime and was discharged. The snake re-presented 22 days later without improvement of the clinical signs. A complete blood count yielded no significant findings. Radiographs revealed a possible esophageal gas dilation cranial to the heart and an increased soft tissue opacity with irregular margins caudal to the heart, possibly an esophageal wall thickening or mass. Esophageal and stomach washes were submitted for cytologic evaluation. Both fluids were similar, consisting of low numbers of poorly preserved inflammatory cells (approximately 70% granulocytes, 25% macrophages, and 5% lymphocytes), numerous unremarkable ciliated columnar cells, and moderate numbers of erythrocytes. Low numbers of thin-walled, blue, oval structures (25-35 x 45-50 μm), occasionally with a deeply basophilic granular content, suspicious for larvated ova, were identified. DNA was retrieved from the remaining stained slides for sequencing, revealing the presence of a novel *Strongyloides* species, family *Strongyloididae*. The snake was discharged with anthelmintics but euthanized the next day; neither necropsy nor fecal evaluation was performed. Two parasites of snakes produce larvated ova: *Rhabdias* spp. and *Strongyloides* spp. The first resides within the lungs and can cause mild to severe symptoms including pneumonia. The latter resides in the small intestine and causes enteritis, though larval migration can cause respiratory distress. The presence of *Strongyloides* spp. eggs in the esophagus and stomach of this snake was an unusual finding. Since *Strongyloides* spp. can be seen in the lungs of lizards, it is possible that this newly identified nematode can parasitize the lungs of snakes, although, in our case, there was no evidence of pneumonia on radiographs. The esophageal soft tissue opacity observed on radiographs could also represent a nidus of helminth infection. Future surveillance of this serpentarium, including fecal flotation and necropsy of other animals that may be affected, is necessary to further evaluate the etiopathogenesis of this parasite.

Case 13

Pathological Findings of Fibrous Osteodystrophy in a Dromedary Camel*

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A 6-y-old female dromedary camel (*Camelus dromedarius* L.) was presented for assessment of firm, bilateral swellings cranial, and ventral to the eyes. Serum biochemistry revealed hyperglycemia (28.5 mmol/L), hypocalcemia (1.27 mmol/L), hyperphosphatemia (3.39 mmol/L), hypoproteinemia (total protein 50 g/L), and hypoalbuminemia (20 g/L). Based on the poor prognosis associated with the presumptive diagnosis of fibrous osteodystrophy, the camel was euthanized. Grossly, upon sectioning, the maxilla and mandible were largely replaced by symmetrical proliferative fibrous tissue and the teeth were loose. There was also a bilateral increase in parathyroid glands of about double the expected size. Microscopically, there was loose proliferative fibrous tissue with widely scattered, regularly spaced, small spicules of mineralized bone in the maxilla, with rare osteoclasts indicating minimal ongoing bone reabsorption. The parathyroid glands were prominent bilaterally; the internal and external parathyroid glands were composed of plump cells with abundant pale basophilic cytoplasm and open nuclei. A more complex superficial duct was noted, with occasionally keratinized squamous lining and lymphocytes and macrophages infiltration, which corresponds with a diagnosis of chronic diffuse parathyroid hyperplasia. The clinical and pathologic findings are consistent with a diagnosis of nutritional secondary hyperparathyroidism, leading to fibrous osteodystrophy. The camel's diet consisted of grass hay, sweet feed, and alfalfa pellets, and treats of cookies. However, it was not specifically balanced for a camel, and feed analysis was not available. Nutritional secondary hyperparathyroidism leading to fibrous osteodystrophy is a well-documented condition in domestic species. It is characterized by increased PTH and active vitamin D in response to an imbalanced dietary calcium-to-phosphorus ratio or excessive consumption of calcium-binding oxalates in the feed. In response to increased PTH and vitamin D, calcium and phosphorus are reabsorbed from the gastrointestinal tract and bones, replacing the bone with proliferative fibrous tissue. This is the second time this condition has been documented in this species in North America.

Case 14

Acute erysipeloethricosis in a 4-week-old pot belly pig*

S. Darling, A. Choi

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Clinical history: A 4-week-old pot belly boar piglet presented with a < 24 hour history of progressive purple discoloration of the skin (cyanosis). On physical examination, the piglet was also obtunded with cold extremities.

Gross Findings: The carcass is in good body condition with small amounts of adipose stores. The skin covering the tip of the ears, neck, snout, left front distal limb, prepuce, and perianal region is variably red and mildly to moderately swollen. The subcutaneous tissue is expanded by edema. The abdominal cavity contains ~5 ml of fibrinous effusion. The kidneys are subjectively large.

Histopathology: Haired skin: Small capillaries within the superficial dermis are predominately congested and often are occluded with fibrin thrombi. The walls of these capillaries are often smudgy and fragmented and the endothelium is reactive and plump. Segmentally, the superficial dermis and overlying epidermis are hyper- eosinophilic, with loss of tinctorial change and cellular detail (ischemic necrosis). Frequently, blood vessels in the submucosa contain fibrin thrombi.

Kidneys: Almost all glomerular tufts are often asymmetrically expanded by hyper-eosinophilic, amorphous material (fibrin). A lymphohistiocytic inflammatory population and fibrin, fill the vascular lumina of the renal vasculature and extend into the interstitium. Occasionally, small capillaries are occluded with fibrin thrombi.

Ancillary Testing: Aerobic culture of the spleen grew *Erysipelothrix rhusiopathiae*. On PTAH stain, the material expanding the glomeruli stains dark purple to blue (fibrin thrombi).

Comments: The constellation of clinical signs, histopathology and aerobic bacterial culture confirm the diagnosis of erysipeloethricosis. Of the three forms of swine erysipelas (acute, subacute, and chronic), this piglet's disease is characteristic for the acute form which, is attributable to septicemia. Erysipelothricosis most commonly affects growing, finishing or mature swine (~3 months - 3 years old). This case is unusual in that this piglet falls into the weaner category. Piglets at this age normally have protective maternal antibodies via colostrum ingestion. Failure of passive transfer may have played a role in this piglet's increased susceptibility to bacterial infection.

References:

Jubb, Kennedy and Palmer's *Pathology of Domestic Animals*, 6th edition. 2016. M. Grant Maxie, ed. Elsevier Mosby, St. Louis, MO. Volume 1. Chapter 6 Integumentary System pp. 645-646

Case 15

Rumensin Toxicosis in a Herd of Feedlot Cattle*

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Signalment: 3-month-old Holstein heifer

History: This heifer's tissues were submitted to the Michigan State University Veterinary Diagnostic Laboratory to investigate acute high mortality in 2-4-month-old calves with respiratory distress and poor response to therapy in a feedlot of 130 head of cattle.

Gross Necropsy Findings: Necropsy of this heifer was performed by the primary veterinarian. The lungs were reportedly wet with severe septal edema. There were no other gross findings.

Histopathologic Findings: Throughout the myocardium, there are widespread, multifocal areas of interstitial fibrosis which surround, separate and replace necrotic and degenerate cardiomyocytes. In affected regions, remaining cardiomyocytes vary in size and staining affinity, have loss of cross striations, and exhibit variation in nuclear size and shape.

Final Diagnosis: A diagnosis of monensin (Rumensin) toxicosis was made after it was discovered that roughly 4 to 5 times the recommended dose of Rumensin was fed to the cattle, in addition to the histologic findings in the heart associated with ionophore toxicosis.

Comments: Ionophores are compounds frequently used as coccidiostats and growth promoters in animal production. Ionophores function by altering the transfer of cations across biologic membranes, which results in organelle damage in coccidia. Excessive ingestion of ionophores results in necrosis and degeneration of cardiac and/or skeletal muscle in a number of species, including horses, cattle, poultry and dogs. Susceptibility to ionophore toxicosis varies among species, with horses being the most sensitive. Toxicosis in cattle occurs most commonly due to mixing error, ingestion of concentrated products, or ingestion of poultry litter. In this case, affected cattle were exposed to approximately 4 to 5 times the recommended Rumensin dose due to a mixing error. Of the 130 cattle in the feedlot exposed to this toxic dose, an estimated 20% have died thus far. Differential diagnoses include ingestion of cardiotoxic plants such as gossypol from cottonseed, yew and oleander among others.

References:

1. Cooper BJ and Valentine BA. Muscle and Tendon. In: Maxie MG, ed. Jubb, Kennedy & Palmer's *Pathology of Domestic Animals*. 6th ed., St. Louis, MO: Elsevier; 2016: Vol. 1 219
2. Roder JD. Ionophore Toxicity and Tolerance. In: Osweiler GD. *Ruminant Toxicology*. *Veterinary Clinics: Food Animal Practice*. Philadelphia, PA: Elsevier; 2011: Vol. 27(2) 305-314

Case 16

Bovine tuberculosis in a dairy herd*

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Signalment: Adult Holstein dairy cows from a Texas herd with a known history of infection with the bacterium *Mycobacterium bovis*. As part of the US TB eradication program, the herd underwent regulatory ante-mortem testing for bovine tuberculosis. The submitted tissues originated from cattle in this herd that were either suspects or reactors on the comparative cervical tuberculin skin test for bovine tuberculosis, and additionally tested positive on an interferon-gamma release assay. Following necropsy, tissues from these cattle were submitted for histopathology and mycobacterial culture.

Histopathology: Within the lymph nodes are multiple to coalescing granulomas measuring up to 1.5 cm in diameter that are composed of single to multiple foci of caseous necrosis, often accompanied by central mineralization. The necrotic foci are rimmed by enlarged macrophages and multinucleated giant cells that are admixed with lymphocytes, and a few plasma cells and neutrophils. Further surrounding the granulomas are fibroblasts and encircling strands of collagen. More peripherally there are often clusters of epithelioid macrophages (satellite granulomas). Within multinucleated giant cells and areas of necrosis are rare (<1/200x field) rod-shaped bacteria, approximately 5-7µm in length, visible when stained with a modified Ziehl-Neelsen acid-fast stain or an Acridine Orange Auramine-O fluorescent stain.

Discussion: Tuberculosis in humans and animals is caused by infection with a bacterium in the *Mycobacterium tuberculosis* complex group, which includes *M. tuberculosis*, *M. bovis*, and a few others. Within North America, *M. bovis* is the species that is most often isolated from tuberculous cattle. *M. bovis* is zoonotic to humans, and an eradication program for bovine tuberculosis has been in place since 1917. Although the US TB eradication program has been extremely successful in reducing livestock infection, sporadic cases are still detected, often through surveillance for granulomas at slaughter. Gross differentials for granulomatous lesions include infections with nontuberculous mycobacteria, *Rhodococcus equi*, *Nocardia*, *Actinobacillus*, *Actinomyces*, fungi, other pathogens, and some neoplasms.