

AAVLD 2016 Slide Seminar

# **59<sup>th</sup> AAVLD Diagnostic Pathology Slide Seminar**



American Association of Veterinary Laboratory  
Diagnosticians  
Greensboro, North Carolina  
Saturday, October 15, 2016  
3:30-6:00 PM

## 59<sup>th</sup> AAVLD Diagnostic Pathology Slide Seminar

Case #	Species	Organ	Diagnosis
1	Calf	Abomasum	Necrohemorrhagic abomasitis
2	Dog	Small intestine	Clostridial hemorrhagic gastroenteritis
3	Pig	Heart, kidney	<i>Toxoplasma</i> abortion
4	Horse	Liver	<i>Clostridium novyi</i> hepatitis
5	Dog	Liver, lung	Herpesviral hepatitis and pneumonia
6	Steer	Heart	<i>Histophilus somni</i> myocarditis
7	Cat	Spleen	Systemic mastocytosis
8	Owl	Liver, spleen	Herpesvirus inclusion body disease
9	Horse	Lung	<i>Acanthamoeba</i> pneumonia
10	Dog	Brain	Oligoastrocytoma
11	Sheep	Thyroid, placenta	Iodine deficiency associated conditions
12	Calf	Skin	Hemotrophic <i>Mycoplasma</i> dermatitis and vasculitis
13	Dog	Liver	Teratoma
14	Horse	Colon	<i>Rhodococcus equi</i> colitis
15	Fox	Liver, lymph node	<i>Francisella tularensis</i> hepatitis and lymphadenitis
16	Horse	Eye	Ischemic optic neuropathy
17	Deer	Intestine	<i>Mycobacterium avium</i> subsp. <i>hominissuis</i> enteritis

**Case 1**  
**Abomasal Rupture in a Holstein Calf**

**Lani Bower, Shelley Newman**

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**Signalment and History:** A 19 day-old male intact Holstein calf developed watery diarrhea (scours) and was treated with antibiotics. Two days later he was laterally recumbent and then died.

**Gross Findings:** Within the peritoneal cavity there was approximately 300 ml of free malodorous flocculent light brown liquid with an intermixed small clump of grass and oats. The right lateral aspect of the abomasum was ruptured with a 14cm in diameter irregularly marginated hole along the greater curvature. The rupture was bi-layered with the serosa being separated from the underlying mucosa. Around the perforation there was a dark red to purple rim of discoloration. There were two, 2cm diameter transmural perforations in the abomasum adjacent to the larger perforation. The rugal folds were friable, dark purple and expanded up to 15mm. The abomasum contained brown flocculent liquid with some oats similar to what was free within the peritoneal cavity. Along the ventral caudal abdomen and extending down the flanks the subcutaneous soft tissues were expanded by clear gelatinous to liquid fluid (edema). Other gross findings were considered incidental.

**Microscopic Findings:** Within the abomasum there is focally extensive congestion as well as extravascular erythrocytes throughout the lamina propria, dissecting down into the submucosa and multifocally extending into the muscularis. Within the lumen and along the villi extending down between glands there are large multifocal mixed bacterial colonies intermixed with sloughed necrotic epithelial cells and karyorrhectic debris. Bacterial colonies include gram positive cocci, rods, gram negative rods and abundant 2-3micrometer round gram positive bacteria arranged in tetrads. The lamina propria and mucosa are expanded by clear space and there is loss of superficial parietal cells. There are dilated clear spaces within the submucosa that are occasionally lined by endothelial cells (lymphatics and emphysema).

**Additional Findings:** Culture of the abomasal wall grew mixed enteric bacteria and *Lactobacillus*.

**Final Diagnosis:** Marked acute necrohemorrhagic abomasitis with edema and abundant gram positive cuboidal (consistent with *Sarcina sp.*) and mixed bacterial colonies

**Comment:** The gross and histologic finding of acute transmural abomasal hemorrhage with the presence of gram positive bacteria with a cuboidal arrangement is consistent with *Sarcina* associated abomasitis. *Sarcina* species are gram positive facultative anaerobes that are difficult to culture. Light microscopy can be readily used to identify these bacteria. The cause of this bacterial overgrowth is unknown and may be associated with dietary changes. It is suspected that the mixture of hay with grain at a young age contributed to this overgrowth. *Sarcina* overgrowth is often reported in conjunction with Clostridial species, which were not isolated in this patient though numerous mixed bacterial species were noted with gram staining by bacteriology and histology. *Sarcina* is most commonly reported to cause emphysematous abomasitis in lambs, less commonly in kids and rarely in calves. Calves with partial or complete failure of passive transfer are at higher risk of being affected by bacterial infections. Insufficient immunity may have contributed to *sarcina* overgrowth.

**References:**

1. Clostridial abomasitis in calves: Case report and review of the literature. *Anaerobe* 11:290-294, 2005.
2. Emphysematous abomasitis in a lamb by bacteria of the *Sarcina* genus in Southern Brazil. *Ciencia Rural* 46(2): 200, 2016.
3. *Sarcina*-like bacteria associated with bloat in young lambs and calves. *Veterinary Record* 163:391, 2008.

**Case 2**  
**Histologic Features of Canine Hemorrhagic Gastroenteritis**

**Scott D. Fitzgerald**

Diagnostic Center for Population and Animal Health, Michigan State University, Lansing, MI

**Signalment:** This case was an 11 year old, female-spayed Miniature Schnauzer.

**Clinical History:** This dog presented with vomiting and diarrhea, which rapidly progressed to frank bloody stools. In less than 24 hours she became moribund, hypothermic, developed respiratory distress and died. On necropsy, the intestinal tract was dark purple discolored, with numerous serosal hemorrhages, and hemorrhagic fluid contents.

**Microscopic Description:** Histologically the small intestines had severe superficial mucosal necrosis and hemorrhage, with lining of the necrotic mucosa by large numbers of stout Gram-positive bacilli. The submucosa was markedly expanded by edema and hemorrhage. Hemorrhages extended transmurally to the serosal surface. The stomach had a focal area of mucosal necrosis, hemorrhage, and bacterial colonization, while the remainder to of the mucosa was morphologically normal with no associated bacterial colonization. No evidence of crypt drop-out/necrosis was found.

**Diagnosis:** Acute hemorrhagic and necrotizing gastroenteritis with Clostridial colonization

**Microbiology:** Bacterial cultures revealed moderate numbers of mixed coliforms, but no growth of *Yersinia* or *Salmonella*. Anaerobic cultures revealed high numbers of *Clostridium perfringens* type A. PCR for Canine Parvovirus was negative.

**Comments:** Hemorrhagic Gastroenteritis (HGE) is a syndrome predominantly seen in young adult, small and toy breed dogs. It is most commonly attributed to overgrowth of *Clostridium perfringens*, but this remains difficult to confirm. Clinical findings generally include acute onset of vomiting, profuse hemorrhagic diarrhea, hypovolemic shock and a short clinical course of 12-48 hours with moderate mortality rate. This case has particularly well preserved mucosa, with marked bacterial colonization by Gram-positive bacilli. The focal nature of the stomach lesion correlates well with the presence of the bacteria histologically. Recent research has demonstrated that some canine cases of HGE are associated with *Cl. perfringens* type A which possesses a novel pore-forming Leukocidin/Hemolysin toxin gene, which helps to explain both the mucosal necrosis and hemorrhage which commonly accompanies this syndrome.

**References:**

Gohari IM, Parreira VR, Nopwell VJ, et al.: 2015, A novel pore-forming toxin in type A *Clostridium perfringens* is associated with both fatal canine hemorrhagic gastroenteritis and fatal foal necrotizing enterocolitis. PLOS One DOI: 10.1371/journal.pone.0122684  
Schlegel BJ, Van Dreumel T, Slavic D, and JF Prescott: 2012, *Clostridium perfringens* type A fatal acute hemorrhagic gastroenteritis in a dog. Can Vet J 53:555-557.

**Case 3**  
**Porcine Abortion Case**

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**SIGNALMENT:** Porcine fetuses and neonates

**History:** Premature farrowing in outdoor-housed, crossbred swine. Herd is not PRRSV vaccinated.

**Gross Necropsy Findings:** Three bodies are submitted, one with advanced autolysis, one appearing near-term, and the third with partially inflated (float in formalin) lungs.

**Histologic Findings:** Heart (1 of 3): There are multifocal areas of necrosis characterized by hypereosinophilia with granular mineralization of cytoplasm and loss or pyknosis of nuclei. Multifocally along the edge of these lesions are variable numbers of protozoal organisms. Organisms are 2-3 microns, round to slightly oval with a deeply basophilic nucleus and occur as few, scattered zoites or clusters of zoites within myocytes.

Kidney (1 of 3): There are multifocal protozoal zoites similar to those described in the heart within the medulla. There are low numbers of lymphocytes, plasma cells, and macrophages scattered throughout the medulla.

Lung and liver: Unremarkable.

**Laboratory Findings:** No significant bacterial growth from fetal tissues. Fetal tissues negative by PCR for *Leptospira*, Porcine Circovirus-2 (PCV-2), Porcine Parvovirus (PPV), Porcine Reproductive and Respiratory Syndrome Virus (PRRSV), and *Neospora*. Fetal tissues positive for *Toxoplasma* by immunohistochemistry and PCR (qualitative, pooled tissue). *Neospora* IHC inconclusive. Fetal thoracic fluid seronegative for *Toxoplasma* or *Neospora*. Oral fluid from sows positive for PRRSV (Ct 36.1).

**Diagnosis:** 1. Myocarditis, necrotizing, multifocal, with intralesional protozoa  
2. Nephritis, lymphoplasmacytic, multifocal, with intralesional protozoa

**Comments:** Samples from subsequent stillbirths/abortions from this farm did not reveal any etiologic agents or significant lesions in fetal tissue. Sow sera was PCR negative for PRRSV, but ELISA positive. Sow seropositivity to *Toxoplasma* (including one dam with normal birth and one that aborted) and *Neospora* varied, but all sows tested had a positive titer for PRRSV. While not expected to be a PRRSV negative herd, no controlled exposure or vaccine measure was in place. Given the unlikelihood of an outbreak of toxoplasmosis in swine and failure to identify lesions or other agents, PRRSV was suspected to be the primary cause of reproductive disorder in this herd. Whether PRRSV predisposes toward clinical manifestation of toxoplasmosis is unknown, as very few cases of *Toxoplasma* have been reported in swine and current commonly used husbandry practices throughout North America drastically reduce the potential for exposure to oocysts. Regardless, it was recommended to reduce potential exposure to small carnivore feces, in particular felines.

**Case 4**  
**Equine clostridial hepatitis**

**Akinyi Nyaoke<sup>1</sup>, Janet Moore<sup>1</sup>, Mauricio Navarro<sup>1</sup>, and Thomas Brauer<sup>2</sup>**

1 California Animal Health and Food Safety Laboratory, UC Davis, San Bernardino, CA 92408, and 2 Chino Hills, CA 91709

**SIGNALMENT:** 14 year-old, neutered male Quarter horse

**History:** The horse had a three day history of progressive neurologic signs and fever ranging from 102 to 105°F, followed by death.

**Gross Necropsy Findings:** Ocular sclera, fat [subcutaneous, pericardial, and abdominal], and articular surfaces [hip and shoulder] were icteric. Ecchymotic to suffusive hemorrhages were present in the subcutis, along the ventral aspect of the vertebral column, multifocally in musculature, epicardium, endocardium, on serosal surfaces of most viscera, and in cerebral cortex. Heart contained deep red-black blood. Abdominal cavity contained approximately 5 liters of serosanguineous fluid with abundant fibrin that coated serosal surfaces of viscera and the peritoneal lining of the diaphragm. The liver was markedly enlarged, and the right and quadrate lobes were orange brown and friable. The left liver lobe was firm and deep red-black with numerous subcapsular and parenchymal emphysematous bullae. Cut surface had large, irregular pale foci surrounded by a hemorrhagic to black rim.

**Histopathology Findings:** The hepatic capsule is lined by fibrin and cellular debris, and there are multiple, variably sized subcapsular and parenchymal emphysematous bullae. There is multifocal to focally extensive coagulative necrosis mainly of centrilobular to midzonal hepatocytes often extending to involve entire lobules, with hemorrhage, fibrin, cellular debris, and large aggregates of gram-positive bacterial rods in necrotic foci. Large numbers of viable and degenerate neutrophils border necrotic foci, and are present in surrounding sinusoids. There is bile stasis, focal hemorrhages, vascular fibrinoid necrosis and thrombosis.

**Laboratory findings:** Liver impression smear was positive for *Clostridium novyi* by FA, and negative for *C. chauvoei*, *C. septicum* and *C. sordelli* by FA. Cultures were negative for *C. perfringens* and *C. difficile*.

**Final diagnosis:** *Clostridium novyi* hepatitis

**Comments:** The diagnosis of *Clostridium novyi* hepatitis was made based on the typical gross and histologic findings, and positive FA on impression smears. Infectious necrotic hepatitis [black disease] is an acute lethal disease of ruminants, and is rarely seen in horses and dogs.

**References:**

1. Navarro M, and Uzal, FA. Infectious necrotic hepatitis. In: Clostridial diseases of Animals. [eds.] Uzal FA, Songer JG, Prescott JF, Popoff MR. 1<sup>st</sup> edition, 2016, pp. 275-279, Wiley & Sons Inc.
2. Whitfield, L.K., et al. Necrotic hepatitis associated with *Clostridium novyi* infection (black disease) in a horse in New Zealand. New Zeal. Vet. J., 2015. 63: 177–179.
3. Stalker, M.J., et al. Liver and biliary system. In: Maxie, M.G. (eds.) Jubb, Kennedy and Palmer's Pathology of Domestic Animals, 5th edition, Vol. 2, 2007. pp. 297–388. Elsevier, Philadelphia

**Case 5**  
**Canid herpesvirus infection in an 8-week-old bulldog puppy**

**Drew Magstadt**, Greg Stevenson, Eric Burrough  
Veterinary Diagnostic Laboratory, Department of Veterinary Diagnostic and Production Animal  
Medicine, Iowa State University, Ames, IA

**Clinical History:** An 8-week-old bulldog puppy was sold by a pet store in northeast Nebraska. The animal died suddenly the day it was sent home with no reported signs of illness prior to death. The entire animal was refrigerated and submitted for necropsy 5 days later at the ISU-VDL, after its littermate presented with serous nasal discharge.

**Gross Pathology:** External examination of the animal was unremarkable. On necropsy, the lungs were diffusely firm and non-collapsing and diffuse meningeal vessels on the surface of the cerebral hemispheres were mildly congested. The remainder of the necropsy was unremarkable.

**Microscopic Description:**

**Liver:** Random areas of coagulative and lytic hepatocellular necrosis with associated hemorrhage are present throughout examined sections. Hepatocytes at the periphery of these foci are often swollen and frequently contain a round to polygonal, eosinophilic intranuclear inclusion body that marginate the chromatin.

**Lung:** Random areas of alveolar septal necrosis are present throughout the parenchyma, characterized by aggregates of cellular debris admixed with moderate amounts of fibrin, macrophages, and few neutrophils. Occasional alveolar epithelial cells within/near affected areas and rare bronchiolar epithelial cells contain eosinophilic intranuclear inclusion bodies. There is moderate perivascular edema and variable alveolar hemorrhage and edema.

**Virology:**

- Canine herpesvirus PCR: **Positive**
- Canine adenovirus 1 and 2 PCR: Negative
- Canine circovirus PCR: Negative

**Diagnosis:** Necrotizing hepatitis and fibrinonecrotic pneumonia, acute, random, severe, with intralesional intranuclear inclusion bodies consistent with herpesvirus

**Comments:** Canid herpesvirus 1 (CHV-1) infection in neonatal puppies less than 4 weeks of age results in foci of acute random necrosis in multiple organs with a high rate of mortality.<sup>1</sup> Older puppies and adult dogs infected with the virus generally present with upper respiratory disease.<sup>2</sup> A recent report described similar microscopic lesions in a 9-year-old female dog infected with CHV-1; however, the dog in that case displayed clinical signs of systemic disease for 5 days and had significant gross lesions on necropsy.<sup>3</sup> In contrast, the puppy in this case died acutely without clinical signs and lacked characteristic gross lesions. Very little background information was provided regarding the medical history or exposure to other animals in this case. Attempts to isolate and sequence the detected virus are in progress as of the writing of this report. Variation in strain virulence and/or host-specific risk factors such as immunosuppression or stress may contribute to mortality associated with CHV-1 infection in older dogs.

**References:**

1. Carmichael L.E., Squire R.A., Krook L. Clinical and pathologic features of a fatal viral disease of newborn pups, *Am. J. Vet.Res.* (1965) 26:803–814.
2. Appel M.J., Menegus M., Parsonson I.M., Carmichael L.E. Pathogenesis of canine herpesvirus in specific-pathogen-free dogs: 5- to 12-week-old pups, *Am. J. Vet. Res.* (1969) 30:2067–2073.
3. Gadsden BJ, Maes RK, Wise AG, Kiupel M, Langohr IM. Fatal Canid herpesvirus 1 infection in an adult dog. *J. Vet. Diag. Inv.* (2012) 24 (4): 604-607.

**Case 6**

***Histophilus somni*-associated vasculitis and necrotizing myocarditis in a Shorthorn steer**

**Colleen Monahan** and Dodd Sledge

Diagnostic Center for Population and Animal Health, Michigan State University, Lansing, MI

**Signalment and History:** An 8-month-old Shorthorn steer was submitted to DCPAH for postmortem examination. He was last seen alive and appeared fine in the barn the evening prior to being found dead in the morning. There were no signs of struggling in the area around the carcass.

**Gross Findings:** Within the left ventricular wall, there were multifocal and coalescing, sharply demarcated regions of non-raised pale pink to tan discoloration which were often bordered by thin dark red rims. The largest of such regions was roughly rhomboid, 9x3 cm on the epicardial surface, and extended 2 cm into the myocardium on cut section. Other regions were round to ovoid, and 0.5-1 cm in diameter.

**Histopathologic Findings:** In the grossly pale areas of the left ventricular free wall of the heart, there were extensive regions of myocardial necrosis of variable chronicity associated extensive fibrinonecrotizing vasculitis and thrombosis. Within areas of necrosis, myofibers were hypereosinophilic and angular, and had variable loss of cross striations, clumping of sarcoplasmic constituents, stippled sarcoplasmic mineralization, and nuclear hypertrophy, pyknosis, or karyolysis. Areas of necrosis were often surrounded by a rim of degenerate neutrophils and thick bands of granulation tissue. Walls of many vessels within and surrounding areas of necrosis were obscured by fibrin, necrotic debris, and degenerate neutrophils, and lumens of these vessels were occluded by dense, variably organized fibrin thrombi. Scattered throughout the remaining myocardium there were marked perivascular and interstitial edema, regionally extensive areas of hemorrhage, replacement of myofibers by dense fibrosis, and dense perivascular and scattered interstitial infiltrates of lymphocytes, neutrophils, and histiocytes.

**Additional Testing:** Aerobic culture of the grossly affected heart yielded *Histophilus somni*

**Diagnosis:** Heart: Severe chronic necrotizing myocarditis with multifocal fibrinonecrotizing vasculitis and thrombosis, extensive fibrosis, and regionally extensive sequestration

**Comments:** *H. somni* is known to cause bronchopneumonia, septicemia, otitis media, abortion, arthritis, thrombotic meningoencephalitis (TME), and myocarditis in cattle. Virulence factors of *H. somni* include but are not limited to immunoglobulin binding proteins (IgBP) and lipooligosaccharide (LOS), which can undergo antigenic variation including the variable incorporation of phosphorylcholine (ChoP). IgBP have been shown to contribute to *H. somni* biofilm formation and act as heparin binding proteins to aid adherence to endothelial cells. Expression of ChoP in LOS contributes to adhesion to respiratory epithelial cells, while strains of *H. somni* that lack of ChoP expression are more likely to be isolated from systemic infections. As such, variation in expression of virulence factors likely contributes to the variance in affected sites within individual animals infected with *H. somni*.

**References:**

1. Elswaifi SF, Scarratt WK, Inzana TJ. The role of lipooligosaccharide phosphorylcholine in colonization and pathogenesis of *Histophilus somni* in cattle. Vet Res. 2012. 2012;43:49.



**Case 7**

**Systemic mastocytosis in a domestic short hair cat (*Felis catus*)**

**Moges Woldemeskel** and Ian Hawkins.

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**Signalment, history and gross findings:** An eight-year-old, female, spayed, domestic short hair cat (*Felis catus*) had a clinical history of intermittent vomiting, decreased appetite and loss of weight. On clinical examination, the mucus membranes were pale. Rare mast cells were observed on cytological evaluation of peripheral blood smears. On CBC moderate lymphocytosis and normochromic, normocytic anemia were recorded. Mast cell tumor was suspected and the cat was treated with antibiotics and prednisone. The cat failed to respond to treatment and was euthanized. The carcass was examined at Tifton Veterinary Diagnostic and Investigational Laboratory, University of Georgia. On gross examination the spleen and liver were markedly and moderately enlarged, respectively.

**Histopathology:** On microscopic examination, round cells diffusely infiltrated the spleen, liver, hepatic sinusoids and veins, pancreatic vessels, and the mesenteric, pre-scapular and mandibular lymph nodes. Mitotic cells were very rare. Abundant cytoplasmic metachromatic granules were observed in the infiltrative round cells, in additional sections of spleen, liver and lymph nodes stained with Toluidine blue stain. Mast cells were not observed in multiple sections of the bone marrow.

**Morphological diagnosis:** systemic mastocytosis associated with splenic mast cell tumor,

**Comments:** Feline systemic mastocytosis is a very rare finding in cats. The clinical signs are nonspecific rendering diagnosis of the disease a clinical challenge. This report describes a cytological, gross and microscopic findings of an interesting, rare case of feline systemic mastocytosis that involved multiple tissues and organs. Feline systemic mastocytosis is usually associated with visceral MCT. It is different from that in dogs, in which mastocytosis is not pathognomonic for MCT, but can occur in association with various other conditions such as hypersensitivity, tissue injury, necrosis and severe regenerative anemia. The report emphasizes the significance of cytological evaluations of peripheral blood smears and fine needle aspirates of affected (enlarged) organs, and histopathological examinations of fine needle biopsies and/or tissue specimens in the diagnosis of feline mast cell-associated systemic mastocytosis to institute an immediate appropriate treatment and save patient's life.

**References**

1. Skeldon NC1, Gerber KL, Wilson RJ, Cunningham SJ. Mastocytosis in cats: prevalence, detection and quantification methods, haematological associations and potential implications in 30 cats with mast cell tumours. *J Feline Med Surg.* 2010; 12:960-966.
2. Allan R, Halsey TR, Thompson KG. Splenic mast cell tumour and mastocytosis in a cat: case study and literature review. *N Z Vet J.* 2000; 48:117-121.
3. Antognoni MT, Spaterna A, Lepri E, Fruganti A, Laus F. Characteristic Clinical, Haematological and Histopathological Findings in Feline Mastocytoma. *Vet Res Commun.* 2003; 27 Suppl. 1. 727-730.
4. Piviani M, Walton RM, Patel RT. Significance of mastocytosis in cats. *Vet Clin Pathol.* 2013; 42:4-10.
5. Isotani M, Tamura K, Yagihara H, et al. Identification of a c-kit exon 8 internal tandem duplication in a feline mast cell tumor case and its favorable response to the tyrosine kinase inhibitor imatinib mesylate. *Vet Immunol Immunopathol.* 2006; 114:168-172.

**Case 8**

**Herpesvirus inclusion body disease in two great horned owls (*Bubo virginianus*)**

**Rahul B Dange<sup>1</sup>, Scott D Fitzgerald<sup>1</sup>, Thomas Cooley<sup>2</sup>**

<sup>1</sup>Diagnostic Center for Population and Animal Health, Michigan State University, Lansing, MI

<sup>2</sup>Wildlife Disease Laboratory, Michigan Department of Natural Resources, Lansing, MI

**Signalment**

A pair of Great Horned Owls, (*Bubo virginianus*).

**Clinical History**

According to the history provided, a pair of Great Horned Owls was found dead approx. 200 yards apart.

**Gross examination**

Liver and spleen contained numerous, small, pinpoint, flat to slightly raised, round to irregular, tan white foci of necrosis on their surfaces that extended deep in to the parenchyma on cut surface.

**Microscopic Description**

The liver parenchyma was characterized by random, multifocal to coalescing areas of coagulative necrosis. Surrounding the foci of necrosis, there was mild to moderate inflammation composed of lymphocytes and few histiocytes. Occasionally, the hepatocytes contained large nuclei and a large eosinophilic intranuclear viral inclusion body and margination of chromatin. The spleen contained similar multifocal coagulative necrosis lesions. Occasional histiocytic and splenic cells contained enlarged nuclei with marginated chromatin, and eosinophilic, intranuclear viral inclusions. Sections of lung were diffusely congested and edematous. Sections of brain, heart and kidney were morphologically unremarkable.

**PCR for Herpesvirus:** Positive, DNA sequencing in progress

**Diagnosis:**

Multifocal necrotizing hepatitis and splenitis with intranuclear, eosinophilic, inclusion bodies

**Comments:**

The great horned owl (*Bubo virginianus*) is the most common North American large bird of prey. Recently, sequencing has confirmed that columbid Herpesvirus (CoHV-1, an alpha-herpesvirus) is a common cause of the herpesvirus inclusion body disease in pigeons, owls, and falcons. Raptors occasionally are infected by CoHV-1 when they feed on infected pigeons. A Cooper's Hawk in Michigan (2008) and Great Horned Owls from Canada (2012) were reported to have been affected by this disease. Multifocal necrotizing hepatosplenitis with intranuclear inclusion bodies in the parenchymal or inflammatory cells are consistent histologic features observed in the affected birds. In this case, it is possible these two owls shared the same sick pigeon if they were pair-bonded; or maybe this area of the woods simply has a lot of infected pigeons and they were separately exposed.

**References:**

Rose et al: Columbid Herpesvirus-1 mortality in great horned owls from Calgary, Alberta. *Canadian Veterinary Journal*, 2012.53:265-268.

Katherine L. Gailbreath and J. Lindsay Oaks: Herpesviral Inclusion Body Disease in Owls and Falcons is caused by the Pigeon Herpesvirus (Columbid herpesvirus 1). *Journal of Wildlife Diseases*, 2008. 44 (2): 427-433

**Case 9**  
**Embolic pneumonia due to *Acanthamoeba sp.* in a horse.**

**Karina C. Fresneda**, Francisco R. Carvallo.

California Animal Health and Food Safety Laboratory, San Bernardino branch. University of California,  
Davis.

**Signalment and History:** A 1-year-old Quarter horse filly with history of 10 day lethargy. Owner was suspicious of a rattlesnake bite.

**Gross examination:** All lung lobes had numerous white firm multifocal nodules of approximately 0.8 to 1.3 cm in diameter, surrounded by a dark red halo. The rest of the parenchyma was dark red and soft. Similar nodules were also observed in kidneys and mesentery.

**Microscopic Findings:** Lung: the parenchyma is multifocally and randomly infiltrated by large numbers of viable and degenerated neutrophils and fewer histiocytes, admixed with cellular debris, fibrin and several rounded eosinophilic protozoal structures (trophozoites) which measured approximately 15 to 20 µm in diameter and have an eccentric single round nucleus. In addition, there are numerous fibrin thrombi and segments of alveolar necrosis. A few blood vessels display transmural necrosis, with fibrinoid degeneration and the presence of pyknotic debris. The remaining parenchyma shows moderate congestion, occasional small hemorrhages and multiple focal areas of intraalveolar edema. Gram stain: no bacteria was observed. *Acanthamoeba* Immunohistochemistry: Positive. *Balamuthia* Immunohistochemistry: Negative. *Naegleria* Immunohistochemistry: Negative

**Diagnosis: Lung:** Embolic pneumonia, necrosuppurative, severe, multifocal, with numerous intralesional protozoal trophozoites, etiology: *Acanthamoeba sp.*

**Comment:** *Acanthamoeba sp.* are ubiquitous free-living amoebae widely distributed in the environment. Amoebic infection in horses is very rare and they have been previously reported in Southern California, USA, in cases of pneumonia, meningoencephalitis and/or systemic infections, and in placentitis in the region of New South Wales, Australia. Amoebas can be easily missed in histology sections because of the cryptic morphology of their trophozoites, which resemble macrophages. Reports of human infection with *Acanthamoeba sp.* have traditionally been limited to cases of systemic disease in the immunocompromised, granulomatous encephalitis, keratitis, cutaneous and sinus lesions.

**References:**

1. Begg A., Todhunter, K., Donahoe, S, Krockenberger, M., Slapeta J. Severe amoebic placentitis in a horse caused by an *Acanthamoeba hatchetti* isolate identified using next generation sequencing. Journal of clinical microbiology. 3101-3104. 2014
2. Bradbury R S, French L.P., Blizzard L. Prevalence of *Acanthamoeba spp* in Tasmanian intensive care clinical specimen. Journal of Hospital Infection 86, 178-181. 2014.
3. Kinde, H., Read D.H., Daft, B., Manzer, M., Nordhausen R., Kelly D., Fuerst P.A., Booton G., Visvesvara G.S. Infections caused by pathogenic free-living amebas (*Balamuthia mandrillaris* and *Acanthamoeba sp.*) in horses. J Vet Diagn Invest 19:317-322. 2007.
4. Siddiqui R and Khan N A. Biology and pathogenesis of *Acanthamoeba*. Review. Parasites and vectors. 5:6. 2012.

**Case 10**  
**Oligoastrocytoma in a dog**

**Lorelei L. Clarke<sup>1</sup>**, Renee Barber<sup>2</sup>, Elizabeth W. Howerth<sup>1</sup>

<sup>1</sup>University of Georgia, College of Veterinary Medicine, Department of Pathology, Athens, GA 30602

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**Signalment:** 7-year-old male neutered mixed breed dog

**History:** The dog was presented to the UGA Neurology service a month prior to necropsy for a three-week history of circling toward the left and altered mentation. On neurologic exam, the dog had inappropriate, dull mentation and a left head turn. He was ambulatory with no weakness or ataxia but circled to the left with right-sided postural reaction deficits in the thoracic and pelvic limbs. There was an inconsistent menace response on the right side. His neuroanatomic localization was to left prosencephalon. Magnetic resonance imaging (MRI) revealed a T2-weighted homogeneously hyperintense, non-contrast enhancing mass lesion within the left thalamus and midbrain with secondary obstructive hydrocephalus, characterized by distension of the lateral and third ventricles. The dog was started on corticosteroid therapy, but gradually declined until owners elected to euthanize.

**Gross Necropsy Findings:** Upon opening the cranium, the brain was mildly edematous and there was thinning of the cerebral cortex bilaterally with distension of the lateral ventricles. Serial sectioning revealed a well circumscribed, well demarcated, pale, gelatinous mass on the left side effacing the mesencephalon with compression of the third ventricle and mesencephalic aqueduct.

**Histopathology Findings:** Extending from the thalamus caudally to the cerebellar peduncles caudally and expanding the mesencephalon with compression of the lateral ventricles and mesencephalic aqueduct was a well demarcated, unencapsulated, expansile, moderately cellular mass. There were two morphologically distinct cellular populations within the mass: bipolar, spindle cells with small ovoid nuclei and long thin wispy cytoplasmic processes, and proliferative, small, round, individualized cells with round to indented, condensed nuclei and scant, granular cytoplasm. Most of the large, bipolar cells and their associated spindle processes were strongly positive for GFAP on immunohistochemistry. The nuclei of the smaller round cells stained positively for Olig2, but did not stain with GFAP. Cells were embedded within an abundant loose myxoid matrix that was positive on Alcian blue pH2.5. Throughout the mass, there were scattered proliferations of hypertrophied endothelial cells, frequently forming glomeruloid capillary loops. There was pallor and mild vacuolation of the surrounding neuroparenchyma.

**Final Diagnosis:** Oligoastrocytoma, thalamus to brain stem, left side with bilateral hydrocephalus

**Comments:** There is a diffuse mixture of GFAP-positive and Olig2-positive cells within the mass. The Olig2-positive cells, considered to be oligodendrocytes, are too proliferative to be considered reactive cells and therefore are likely neoplastic. There is also proliferation of GFAP-positive astrocytic cells, but it is less clear whether these are reactive or neoplastic. Astrocytes will commonly proliferate into and around oligodendrogliomas in response to the neuroparenchymal destruction<sup>1</sup>. This reactive response should comprise no more than 30% of the mass, which is far less than the proportion of astrocytic, GFAP-positive cells observed in this mass, which led us to the diagnosis of oligoastrocytoma. Oligoastrocytomas, or mixed gliomas, have been described previously in dogs, where both glial lineages have neoplastic features. These two populations are often separated and distinguishable within the mass, but more heterogeneous and mixed masses are reported<sup>1,2</sup>. In recent reports<sup>3</sup>, it has also been proposed that gliomas originating from more primitive cell lines may be dually positive for astrocytic and oligodendroglial markers. In this case, however, there appear to be two distinct populations.

**References:**

<sup>1</sup>Summers BA, et al. Tumors of the central nervous system. In: *Veterinary Neuropathology*. St. Louis, MO: Mosby, 1995:370-373

<sup>2</sup>Withrow SJ, et al. Tumors of the nervous system. In: *Small Animal Clinical Oncology*. St Louis, MO: Saunders Elsevier, 2007:659-670

<sup>3</sup>Ide T, et al. Immunohistochemical characterization of canine neuroepithelial tumors. *Vet Pathol* 2010; 47(4):741-50

### Case 11

## Iodine deficiency-associated premature placental separation, goiter and failure to thrive in a Suffolk lamb

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**History and Gross Findings:** Over the spring of 2016, nearly half of lambs (7 of 19) in a Michigan flock of Suffolk sheep were born weak and died within a couple of days of birth. Pregnant ewes were fed alfalfa hay, supplemented with a mineral salt block and grain after lambing. One male lamb which was born alive and died within hours of birth, and the corresponding placenta were submitted for examination. On gross necropsy of the lamb, the lungs were poorly aerated, there was no ingesta in the abomasum, and eponychium remained over the hooves, but there was mild hemorrhage around the umbilicus. Such findings are consistent the animal having been born alive, but having failed to thrive. Both thyroid glands were symmetrically enlarged, being 4.5x2x2 cm. No significant lesions were grossly obvious in the placenta.

**Histopathologic Findings:** In the thyroid glands, there was diffuse thyroid follicular cell hyperplasia and hypertrophy as characterized by piling of tall columnar cells with foamy eosinophilic cytoplasm around central follicular areas. Central follicular areas were often collapsed and contained scant, lightly eosinophilic, foamy colloid and few foamy histiocytes. In the cotyledonary areas of the placenta, trophoblasts of the arcade regions and spaces between placental villi often contained golden brown, globular material consistent with hemosiderin, hematoidin, and free and phagocytosized erythrocytes.

#### **Morphologic diagnoses:**

Thyroid gland: Diffuse follicular cell hyperplasia and follicular collapse (hyperplastic goiter)

Placenta: Intratrophoblastic hemosiderin accumulation consistent with bleeding associated with premature placental separation

**Additional Testing:** The thyroid iodine concentration was critically deficient at 27.7 µg/g on a dry tissue basis (DT) (reference range [RR]1100-1800 µg/g DT). In mineral analysis of the liver, there were mild decreased concentrations of selenium (0.44 µg/g DT; RR 0.9-6.1 µg/g DT), copper (47.6 µg/g DT; RR 72-475 µg/g DT) and zinc (36.88 µg/g DT; RR 80-460 µg/g DT). PCR on pooled liver, lung, heart, kidney, spleen, thymus, and placenta for pestivirus, Cache Valley virus, bluetongue virus and enzootic hemorrhagic disease virus was negative.

**Comments:** On further investigation with the lamb owner, the trace mineral block that was used to supplement pregnant ewes did not contain iodine. The deficient selenium concentrations may have further exacerbated the iodine deficiency as selenium is required by the enzyme iodothyronine deiodinase which converts T4 to active T3 and aids in regulation of iodine homeostasis.<sup>3</sup> In humans, women with subclinical hypothyroidism during pregnancy had a 3 times greater likelihood of complications due to premature placental separation, or abruption, than euthyroid women.<sup>1</sup> Related to this, it has been hypothesized that thyroid hormone is essential for normal placental development and that lack of normal development is associated with abruption.<sup>2</sup> This case illuminates the importance of proper iodine supplementation during gestation because its deficiency can be associated with negative pregnancy outcomes such as abruption, neonatal hypothyroidism, and subsequent neonatal death.

#### **References:**

1. Casey BM, Dashe JS, Wells CE, McIntire DD, Byrd W, Leveno KJ, Cunningham FG. Subclinical hypothyroidism and pregnancy outcomes. *Obstet Gynecol* 2005; 105(2):239-45.
2. Dommissse J, Tiltman AJ. Placental bed biopsies in placental abruption. *Br J Obstet Gynaecol* 1992;99:651-4.
3. Schneider MJ, Fiering SN, Thai B, et al. Targeted disruption of the type 1 selenodeiodinase gene (Dio1) results in marked changes in thyroid economy in mice. *Endocrinology* 2006;147(1):580-89.

**Case 12**  
**Lymphohistiocytic dermatitis and vasculitis in calves associated with hemotropic**  
***Mycoplasma* spp**

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**Signalment and History:** Several Jersey-Holstein cross calves in one barn of approximately 250, 300lbs calves acutely developed swelling of the distal hind limbs and temperatures of 104F. Within two days, 16 more calves had moderate limb edema and 40% of all calves in the barn had erythema and edema around the dew claws. Affected calves had no lameness, but swollen areas were sore on palpation. The next morning, four animals developed signs of pneumonia and fevers of 104-106F, and few others had mild coughs. One animal was sacrificed for a field necropsy, during which only limb edema was observed.

**Histopathologic Findings:** The submitted slide represents skin from the pastern region. The superficial, mid, and deep dermal plexuses are surrounded by moderate aggregates of plump histiocytes, lymphocytes, few plasma cells, eosinophils, and, primarily surrounding superficial vessels, neutrophils. The deep dermal stroma is moderately expanded by nonstaining edema fluid, and there is occasional extravasation of red blood cells into the superficial and mid dermal stroma and small perivascular accumulations of extracellular karyorrhectic debris. Vessel walls of all dermal plexuses are frequently poorly defined and partially to fully obscured by smudgy amphophilic to eosinophilic material or are lined by plump, reactive endothelial cells. The epidermis is moderately hyperplastic and lined by marked laminar orthokeratotic to multifocally parakeratotic hyperkeratosis and multifocal crusts comprised of serum and dense aggregates of degenerate neutrophils. The epidermis is focally ulcerated and replaced by similar crusts.

**Diagnosis:** Severe lymphohistiocytic perivascular dermatitis and vasculitis; focal epidermal ulceration, moderate epidermal hyperplasia, hyperkeratosis, and crusts

**Additional Findings:** PCR on formalin fixed, paraffin embedded tissue was positive for 16s rRNA gene of *Mycoplasma* spp, and sequencing of a 597 bp amplicon from the same gene was consistent with 'Candidatus *Mycoplasma haemobos*'

**Comment:** There are several species of hemotropic mycoplasmas of veterinary importance, including *Mycoplasma wenyonii* (formerly *Eperythrozoon wenyonii*) and 'Candidatus *Mycoplasma haemobos*', both of which affect cattle and have been previously reported in Michigan dairy herds.<sup>1</sup> The exact pathogenic potential of these eperythrozoon parasites in cattle is unclear. While the majority of infected cattle are subclinical, development of severe clinical disease has been reported previously, particularly in immunosuppressed or stressed animals, or cattle with concurrent infections. Few cases with similar histologic changes in the skin of cattle with severe hind limb edema and concurrent hemotropic *Mycoplasma* spp infection have been described.<sup>2</sup> However, the exact pathogenesis of these changes is not fully understood. Other differentials for the perivascular dermatitis and vasculitis observed in this case included septicemia, viral-induced vasculitis, and Hairy Vetch toxicosis. There was no histologic or laboratory evidence to support any of these causes in this and all calves had been restricted to the barn with no exposure to pasture.

**References:**

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**Case 13**  
**Primary Hepatic Teratoma in an Adult, Spayed Female Cairn Terrier**

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**Clinical History:** This 13 year-old, spayed female Cairn terrier presented to the referring veterinarian for anorexia, vomiting, and lethargy. Abdominal radiographs revealed a moderately enlarged liver. Following a month of conservative treatment, the patient presented to the VMCVM Veterinary Teaching Hospital on referral for hepatomegaly and elevated liver enzymes [ALT 869 U/L (16-75), AST 185 U/L (13-48), ALP 1291 U/L (8-70)]. An abdominal ultrasound revealed a complex structure containing multifocal, thin-walled echogenic cysts. A computed tomography scan revealed a heterogeneous mass within the left lateral liver lobe containing attenuating foci of mineralization and multifocal cysts as well as multifocal, enlarged, heterogeneous hepatic and mesenteric lymph nodes.

**Microscopic Description:**

Left lateral liver lobe: An indistinct, densely cellular, expansile and infiltrative, multilobular and cystic mass compresses hepatic lobules to the periphery. The mass consists of large populations of both epithelial and mesenchymal cells replacing and expanding hepatic lobules. The spindle shaped cells are arranged in sheets and streams and multifocally produce osteoid matrix or surround spicules of lamellar bone. Alternate aggregates of spindle cells are accompanied by abundant mucinous matrix, while some resemble primitive neuroblastic cells and are surrounded by matrix resembling neuropil. The majority of the epithelial cells are squamous to cuboidal and line cystic structures containing laminar or matrical keratin. In other areas, the epithelial cells are ciliated, columnar, and pseudostratified, lining cystic structures that contain variable amounts of eosinophilic, homogeneous to fibrillar material admixed with few degenerate neutrophils. In more solid areas, neoplastic cells have scant to moderate eosinophilic cytoplasm and indistinct cell borders. Nuclei are round to oval, variably sized, and vesicular with prominent nucleoli. The mitotic index is 32. Small nests of tumor cells infiltrate the adjacent, more normal liver. In lobules adjacent to the mass, there is severe biliary hyperplasia and bile ducts and periportal hepatocytes are infiltrated by large numbers of neutrophils. In other lobules, periportal and midzonal hepatocytes are moderately swollen and contain numerous indistinct, clear intracytoplasmic vacuoles.

Left medial, right medial, and caudate liver lobes: Periportal and midzonal hepatocytes are often swollen and contain numerous, indistinct, intracytoplasmic vacuoles separate by eosinophilic, protoplasmic material. A few, small hyperplastic nodules are scattered throughout the lobules. In these nodules, the hepatocytes are similarly vacuolated. Multifocal small aggregates of erythroid and myeloid precursor cells widen the sinusoids and surround occasional central veins.

**Diagnosis:**

Left lateral liver lobe: Malignant teratoma with severe biliary hyperplasia, and periportal and midzonal vacuolar hepatopathy

Left medial, right medial, and caudate liver lobes: Periportal and midzonal vacuolar hepatopathy with nodular hyperplasia and extramedullary hematopoiesis

**Comment:**

Teratomas are uncommon in domestic animals. Most often, they occur in the gonads, specifically the ovary of a bitch. They are often benign and composed of well-differentiated, mature tissue, however, any tissue within a teratoma has potential to be malignant. The high mitotic rate and extension of the tumor cells into the adjacent liver suggest malignancy in this case. No primary hepatic teratomas have been described in dogs; however, a few case reports describe these in humans.

**Case 14**

***Rhodococcus equi* colitis in a foal**

**Francisco A. Uzal**

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**Clinical history:** two-month-old Thoroughbred colt with a history of high temperature of 2-3 day duration.

**Gross findings:** In the large colon there were many multifocal, irregular, elevated and crateriform nodules with central ulcers, measuring between 1 and 2 mm diameter. There was mild, diffuse congestion and edema of the wall of the large colon. The mesenteric and colonic lymph nodes were moderately enlarged (up to 1 cm diameter) by caseo-purulent exudate that obliterated the structure of the nodes. Affecting all areas of both lungs, there were multifocal to coalescing, variable size (up to several cm diameter) pyogranulomas.

**Histopathology:** Colon: There are multifocal erosions and ulcers of the superficial epithelium, and moderate accumulation of macrophages, neutrophils and occasional multinucleated giant cells in the lamina propria. The macrophages contain intracytoplasmic aggregates of coccobacilli, which stain positive with Gram. There is also necrosis of lymphoid follicles, with infiltration of macrophages, neutrophils and occasional multinucleated giant cells.

**Morphologic diagnosis:** colitis, pyogranulomatous, multifocal to coalescing, with intracytoplasmic cocco-bacilli.

**Comments:** *R. equi* is a facultative, intracellular, pleomorphic, Gram-positive coccobacillus which survives within macrophages and causes mainly pyogranulomatous pneumonia in foals between 1 and 6 months of age; cases in older animals may also occur. Abdominal lesions are found in approximately 50% of foals with *R. equi* pneumonia that are submitted for post-mortem examination. The abdominal lesions include one or more of the following: pyogranulomatous enterotyphlocolitis, lymphadenitis of mesenteric or colonic lymph nodes, abscesses and peritonitis. The pathogenesis of intestinal lesions has not been fully elucidated, although occurrence of these lesions seems to be dose-related. In natural disease, repeated exposure to *R. equi* in swallowed respiratory exudate is thought to be the main source of infection in animals with pneumonia. Enteric lesions in foals without pneumonic lesions may also occur, albeit rarely. Other lesions occasionally associated with *R. equi* infection are polysynovitis and/or arthritis and osteomyelitis. Rarely, abortion may also occur. The disease has higher incidence during summer in dry and dusty environments. The gross and microscopic lesions provide a presumptive diagnosis of acceptable certainty. Confirmation of the diagnosis should be based on culture followed by PCR to confirm that the strains isolated are virulent.



**Case 15**

**Necrotizing hepatitis and mesenteric lymphadenitis in a gray fox (*Urocyon cinereoargenteus*), associated with isolation of *Francisella tularensis***

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**Signalment:** Adult female Gray Fox (*Urocyon cinereoargenteus*)

**Clinical History:** This fox was radio-collared as part of a cause-specific mortality study by the Ohio Department of Natural Resources. It was found dead in Gallia County on 2/29/16. Body was kept refrigerated until 3/1/16 when it was presented to the ADDL for necropsy, rabies testing, and determination of cause of death.

**Gross lesions:** Necropsy findings included multiple fleas (ectoparasitism), marked negative energy balance with generalized muscle wasting, gastroenteritis with mild gastric ulceration, mild mesenteric lymphadenopathy, and mild intestinal parasitism (few nematodes, several *Dipylidium* sp, and *Taenia* sp. cestodes observed).

**Ancillary test results:**

**Rabies testing:** Brain tissue submitted to the Ohio Department of Health was negative for rabies.

**Bacteriology:** *Francisella tularensis* was isolated from liver.

**PCR:** Nucleic acid of *Francisella tularensis* detected in liver.

**Virology:** FA tests equivocal for canine distemper virus and negative for canine parvovirus-2 antigens.

**Immunohistochemistry:** Positive immunoreactivity in necrotic foci of liver and lymph node (ADDL, Purdue University).

**Histopathology:**

**Liver:** Moderate numbers of randomly distributed, variably sized foci of necrosis are present throughout the hepatic parenchyma, necrotic foci ranging from <100x100 to 800x600 microns. These foci contain necrotic hepatocytes with shrunken hypereosinophilic cytoplasm and pyknotic nuclei dissociated from hepatic cords that are admixed with degenerate neutrophils, macrophages, few lymphocytes, necrotic round cells with pyknotic nuclei, and nuclear debris. Eosinophilic intracytoplasmic and intranuclear inclusions are present in bile duct epithelium. Mesenteric lymph node: Multiple extensive focal regions of eosinophilic amorphous to fibrillar material admixed with necrotic neutrophils, pyknotic and karyorrhectic debris are present throughout the cortex, effacing normal architecture of the node. Nuclear debris is admixed with increased numbers of plasma cells in medullary cords and there is moderate sinus histiocytosis.

**Morphologic Diagnoses:**

**Liver:** 1. Mild to moderate, random multifocal to coalescent, necrotizing hepatitis, with eosinophilic intracytoplasmic and rare intranuclear inclusions in bile duct epithelium

**Lymph node, mesenteric:** Marked, acute, multifocal and coalescing fibrinopurulent to necrotizing lymphadenitis

**Discussion:** Histologic lesions in liver and mesenteric lymph node are consistent with tularemia. Bacterial colonies tentatively identified as *F. tularensis* were isolated from the liver at the ADDL by bacterial culture on sheep blood and chocolate agar, and characterized by PCR assay. The isolate was confirmed as *F. tularensis* by the National Veterinary Services Laboratories. Canine distemper virus can infect a wide range of canids, mustelids, and procyonids. Gray foxes are more susceptible to canine distemper virus infection than are red foxes.

**References:**

1. Schlotthauer CF, Thompson L, Olson Jr. C. 1935. Tularemia in Wild Gray Foxes: Report of an Epizootic. The Journal of Infectious Diseases, 56:1, p 28-30.
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**Case 16**  
**Optic neuropathy associated with optic tract elongation and compression**

**Alan T. Loynachan**

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**Signalment:** 10-day-old Thoroughbred colt

**History:** The foal developed exophthalmos of the right eye approximately 24-30 h after birth. On the second day of life, an equine ophthalmologist examined the foal. The right globe was prominent, did not retropulse, and exhibited diffuse hyphema that obstructed visualization of the pupil. The fundus of the left eye was within-normal-limits, but the eye was dilated and lacked a pupillary light reflex and menace response; the foal was thought to be blind in this eye. Radiographs suggested a possible fracture at the base of the calvarium. The foal was reevaluated 1 week later. Observations of the right eye included: improved retropulsion with decreased exophthalmos, ventral deviation of the globe, optic nerve pallor, and retinal detachment. The left eye had not improved. A presumptive diagnosis of head trauma with orbital fracture, hematoma, and optic nerve trauma was made. The foal was euthanized due to a poor prognosis.

**Gross Necropsy Findings:** The right eye exhibited mild exophthalmos. A 3.5 cm redundant loop of enlarged right optic tract was located proximal to the optic chiasm. The chiasm and elongated optic tract were entrapped and constricted by meninges, middle cerebral artery, and internal ophthalmic artery. The right optic disc was cavitated and surrounded by a hemorrhagic border. More than 2.5 cm of retrobulbar optic nerve was centrally cavitated and contained vitreous-like material.

**Histopathology Findings:** Right eye: The retina was detached and absent, and the subjacent retinal pigmented epithelial cells were multifocally hypertrophied and hyperpigmented. Diffusely, the choroid was loosely arranged. The optic disc and nerve were cavitated and lacked a large majority of the nerve fibers; a small focus of gliotic neuropil was evident at the periphery adjacent to the lamina cribrosa region. The cavitation contained erythrocytes and acidophilic proteinaceous material, and the meninges were thickened by fibrous connective tissue. Periocular myofibers were multifocally atrophied, degenerative, necrotic, and regenerative. Fibrous connective tissue multifocally separated the myofibers.

**Morphologic diagnosis:**

Optic neuropathy with optic tract elongation and optic tract and chiasm compression

**Comments:**

Progressive unilateral and bilateral vision loss can develop secondary to compression of the anterior visual pathway (eye, optic nerve, optic chiasm, and optic tract). Reported causes of compression include: aneurysm, dolichoectatic vasculature, inflammation, neoplasia, and trauma. The pathogenesis of the case reported here is not definitively known but likely involved a complex relationship of congenital malformation and meningeal and vascular compression associated with elongated optic tract and the optic chiasm. It has been speculated that compression of the optic chiasm and nerve have deleterious effects on the vasa nervorum and subsequently result in optic nerve ischemia.

**References:**

Bacon TS, TT Lamki M Ammirati, DK Hirsh, and CF Kirsch. Optic Neuropathy Secondary to Multifocal Nerve Compression by dolichoectatic Vasculature. *J Clin Exp Ophthalmol*. 2003. 4(5).

**Case 17**

**Johne's-like disease in a white-tailed deer fawn associated with *Mycobacterium avium* subsp. *hominissuis***

**Heather Fenton**<sup>1</sup>, Charles Ruth,<sup>2</sup> Sualee Robbe-Austerman<sup>3</sup>

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<sup>2</sup>South Carolina Department of Natural Resources, Columbia, SC, <sup>3</sup>National Veterinary Services Laboratories, USDA-APHIS, Ames, IA

**Signalment:** One-year-old, male, white-tailed deer fawn

**History:** This deer fawn was submitted to a wildlife rehabilitation center due to injuries associated with a dog attack in July of 2014. The deer recovered from the original injury and had intermittent diarrhea and weight loss during its nine month stay at the rehabilitation center. Over the course of its stay, it was given Panacur (antihelminthic), fluids, selenium injections, dexamethasone, vitamin B injections, and a variety of antibiotics including spectinomycin, penicillin, and enrofloxacin at different times. This deer was found dead in its pen in May of 2015, and was delivered to SCWDS for necropsy.

**Gross Necropsy Findings:** The fawn was in extremely poor body condition with evidence of muscle wasting and serous atrophy of fat in the bone marrow. A large amount of dried fecal matter covered the perineum and underside of the tail. The hair coat was unkempt. Mesenteric lymph nodes were diffusely enlarged, firm on palpation, and contained white caseous to purulent material on cross section. The lymphatics were prominent, the small intestinal contents were watery and light brown, and the colonic wall was slightly thickened.

**Histology (Slide W15-402):** The mucosa, submucosa, and lamina propria of the ileum are expanded by large infiltrates of foamy macrophages with intracytoplasmic and extracytoplasmic Acid fast positive coccobacilli. Villi are often blunted and fused with sloughing of epithelial cells into the lumen. The lymphatics draining the ileum are dilated and the lumen is replaced by fibrin, cellular debris, and aggregates mineral. Lymphatics are surrounded by fibrous tissue that is infiltrated by small numbers of lymphocytes, plasma cells, and large numbers of foamy macrophages with intracytoplasmic and extracytoplasmic Acid fast positive coccobacilli.

**Diagnosis:** Ileum and adjacent mesentery: granulomatous enteritis and lymphangitis; segmental, moderate, chronic with intracytoplasmic bacteria.

**Ancillary Testing:** Acid fast bacteria were recovered from the mesenteric lymph node and ileum by the National Veterinary Laboratory in Ames, Iowa, following bacterial culture. The lymph node isolate was sequenced at the 16S rRNA and the rpoB regions and matched the reference strain *Mycobacterium avium* 104, which has been previously classified as *Mycobacterium avium* subsp. *hominissuis* at 100.0% sequence similarity. Because the media used also supports *M. avium* ssp. *paratuberculosis* an IS900 PCR was performed and confirmed negative.

**Discussion:** *Mycobacterium avium* subsp. *hominissuis* is an opportunistic, environmental pathogen that can cause disease in domestic swine, humans, horses, dogs, captive and free-ranging red deer. Differentiation from *M. subsp. paratuberculosis* was important in terms of larger-scale implications.

**References:**

- Mijs, et al. 2002. Molecular evidence to support a proposal to reserve the designation *Mycobacterium avium* subsp. *avium* for bird-type isolates and '*M. avium* subsp. *hominissuis*' for the human/porcine type of *M. avium*. Int J Syst and Evol Microbiol 52: 1505–1518.
- Glawischnig et al. 2006. Infections caused by *Mycobacterium avium* subspecies *avium*, *hominissuis* and *paratuberculosis* in free-ranging red deer (*Cervus elaphus hippelaphus*) in Austria, 2001-2004. Journal of Wildlife Diseases 42: 724-731.