56th AAVLD Diagnostic Pathology Slide Session

American Association of Veterinary Laboratory Diagnosticians
San Diego, California
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56th AAVLD Diagnostic Pathology Slide Session
October 19, 2013 San Diego, California

2013 AAVLD Diagnostic Pathology Slide Seminar Presenters

Case #	Presenter	Species	Institution
1	Marcia R. S. Ilha	Canine	TVDIL-UGA
2	Alison Tucker	Swine	NCVDLS
3	Scott D. Fitzgerald	Raccoon	DCPAH-MSU
4	Bailey L. Wilberts	Bovine	ISU
5	Kelly Hughes	Canine	OSU-VDL
6	Dodd Sledge	Chicken	DCPAH-MSU
7	Tuddow Thaiwong	Canine	DCPAH-MSU
8	Kelli Almes	Feline	KSVDL-KSU
9	Chanran Ganta	Swine	KSVDL-KSU
10	Mahogany Caesar	Equine	NCVDLS
11	Panayiotis Loukopoulos	Pacific gopher snake	CAHFS-UCDavis
12	Andrew Brooks	Canine	AHL-UofG
13	Donal O'Toole	Canine	WSVL-UW
14	Sandra Scholes	Bovine	AHVLA
15	Leslie W. Woods	Pacific fisher	CAHFS-UCDavis
16	Francisco A. Uzal	Bovine	CAHFS-UCDavis
17	Jeffrey R. Hayes	Swine	Ohio ADDL
18	Tim Cushing	Feline	CVDC

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2013 AAVLD Diagnostic Pathology Slide Seminar Diagnoses

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Case #1

Extra-adrenal retroperitoneal paraganglioma in a dog

Marcia R. S. Ilha

University of Georgia, Tifton Veterinary Diagnostic and Investigational Laboratory (TVDIL), 43 Brighton Road, Tifton, GA 31793

Signalment: 10.5-year-old, male, Boxer dog.

Clinical history and gross findings: The dog had a short clinical history of sudden collapse and death. At postmortem, lesions included hemoabdomen, a heart base tumor, retroperitoneal mass, cortical adrenal nodules, thyroid gland nodules, and testicular tumors.

Histopathology: The retroperitoneal mass was composed of lobules of polyhedral cells separated by fine fibrovascular stroma. Neoplastic cells had moderate amounts of eosinophilic granular to vacuolated cytoplasm, and round to oval nuclei with predominantly single central nucleoli and coarsely stippled chromatin. Cells with karyomegalic and bizarre nuclei were frequently observed. Mitoses were 17 per 10 HPFs (400x). Histological sections of the heart base mass revealed a neoplasm with similar histological features of the retroperitoneal mass.

Immunohistochemistry and electron microscopy: Intracytoplasmic expression of chromogranin A and synaptophysin was observed in neoplastic cells of the retroperitoneal mass and heart base tumor. Neoplastic cells of the retroperitoneal mass contained numerous electron dense neurosecretory granules.

Morphologic diagnosis (retroperitoneal mass): Paraganglioma

Comments: The heart base mass was consistent with an aortic body tumor (paraganglioma, chemodectoma) and the retroperitoneal mass was called an extra-adrenal paraganglioma. Other lesions observed were multiple thyroid gland adenomas, multiple testicular interstitial cell tumors, bilateral adrenal cortical nodular hyperplasia and parathyroid gland hyperplasia suggesting possible multiple endocrine neoplasia syndrome in this dog[2]. Based on the fact that 1) a large percentage of paragangliomas in brachicephalic dogs involve multicentric origin[4], 2) retroperitoneal location is not a common metastatic site of paragangliomas in dogs[4], 3) metastases in other organs were not observed in this dog, 4) a previous report described a para-adrenal paraganglioma in a dog[1], and 5) paraganglionic cells are found in the retroperitoneum of dogs[3], it is plausible to believe that the extra-adrenal retroperitoneal paraganglioma observed in this dog may represent a local primary tumor and this is a case of multicentric origin of paragangliomas rather than a metastatic neoplasm.

References:

1. Dorn A, et al. Exp Pathol 27:99-104. 1985.
2. Feldman EC, Nelson RW. Canine and feline endocrinology and reproduction. 2004.
3. Mascorro JA, Yates RD. Tissue Cell 9:447-460. 1977.
4. Maxie MG. Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. 2007.

Case #2

Seneca Valley Virus and Idiopathic Vesicular Disease of Swine

Alison Tucker, Richard Mock

North Carolina Veterinary Diagnostic Laboratory System

History: According to the history provided, approximately 10% of the sows in this 2500-sow farrow to weaning operation developed vesicular lesions on the nose. The sows were afebrile. There were no lesions in the oral cavity or on the feet. Appetite was decreased and a drop in milk production in lactating sows was attributed to this transient decreased appetite. Samples were submitted for testing at the Foreign Animal Disease Diagnostics Laboratory (FADDL) at Plum Island, New York. Tissue samples and serum were submitted to the NCVDLS.

Histopathology: Sections of nasal mucosa from 10 adult, mixed breed female pigs are examined. In all sections there is epithelial necrosis with separation from the epidermal basal layer and dermis consistent with vesicle and bulla formation. The base of the vesicles is not present in all sections. There is loss of cell to cell adhesion and rounding of epithelial cells affecting the basal and spinous layer. Few sections have full thickness epithelial necrosis without acantholysis. Where the epithelium is intact there is acanthosis. The stratum corneum is expanded by edema in severely affected areas. A plaque of fibrin with neutrophils and eosinophils coats the exposed necrotic epithelium with variable and mixed rod and cocci bacterial colonization. Acute edema and neutrophilic and eosinophilic inflammation expand the superficial dermis where intact dermis is present in the sections.

Diagnosis: Idiopathic Vesicular Disease of Swine

Comment: Foreign animal diseases and domestic vesicular diseases were ruled out at FADDL by their routine testing. Virus isolation using tissue and vesicular fluid from 5 animals did result in cytopathic effect on Vero cells. PCR on this isolate was positive for Seneca Valley Virus nucleic acid. Seneca Valley virus is an oncolytic picornavirus that was initially isolated from cell culture media as a presumed contaminant of fetal bovine serum. Neutralizing antibodies have been identified in pigs and cattle. The virus has been associated with idiopathic vesicular disease in swine but disease has not been reproduced by inoculating animals; however, serologic conversion can be obtained. The association was first reported in pigs shipped from Manitoba to a harvest facility in Minnesota and this association has been repeated in cases in multiple states. Seasonal occurrence (spring-fall) is typical. Idiopathic vesicular disease is not debilitating but resembles other vesicular diseases and therefore practitioners and diagnostic laboratories should consider this as a potential foreign animal disease until confirmed otherwise.

References:

1. Pasma T, Davidson S and Shaw S. Idiopathic vesicular disease in swine in Manitoba, Can Vet J 2008; 49:84-85.
2. Adams SF, Schneider JL, Miller CA, et al. Idiopathic vesicular disease in a swine herd in Indiana. J Swine Health Prod. 2004; 12:192-196.
3. Yang M, van Bruggen R, Xu W. Generation and diagnostic application of monoclonal antibodies against Seneca Valley virus. J Vet Diagn Invest. 2012; 24:42-50.
4. Singh K, Corner S, Clark SG, et al. Seneca Valley Virus and Vesicular Lesions in a Pig with Idiopathic Vesicular Disease. J Vet Sci Technol 2012; 3:123-125.

Case #3

Concurrent Infection with Canine Distemper and Infectious Canine Hepatitis in a Wild Raccoon

Scott D. Fitzgerald[1], Thomas Cooley[2], Roger Maes[1] and Matti Kiupel[1]

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

[2]Wildlife Disease Laboratory, MI Department of Natural Resources, Lansing, MI

Clinical history and gross findings: A hunter noticed a wild raccoon (*Procyon lotor*) acting weak, ataxic, with nasal discharge and labored breathing. The raccoon was observed alive on the first day, but died during the night, and was collected and submitted to the Wildlife Laboratory for examination as canine distemper was suspected. The raccoon was in good nutritional condition, but the sclera and mucous membranes were icteric. The animal was a young adult female weighing 9 lbs. Internal lesions included congested lungs, the right middle lung lobe was consolidated, and purulent exudate was present in the major airways. The liver was irregularly mottled.

Histopathology: Histopathology revealed aspiration pneumonia characterized by necrosis of airways and adjacent alveoli, with necrotic debris, degenerate neutrophils, and mixed bacterial colonies. Some intact bronchiolar epithelial cells contained multiple 2-4 micron diameter cytoplasmic, eosinophilic viral inclusions, consistent with canine distemper inclusions. Sections of liver had marked multifocal to coalescing parenchymal necrosis, with numerous large eosinophilic intranuclear inclusion bodies, consistent with adenoviral inclusions. In addition, bile duct epithelial cells contained eosinophilic cytoplasmic inclusions, similar to those seen in the lungs.

Immunohistochemistry: Canine distemper was confirmed by both immunohistochemical staining and PCR; while canine adenovirus was positively stained by immunohistochemistry.

Comments: While canine distemper is known to be a common infectious condition in raccoons, infectious canine hepatitis (IHC) is much more rarely reported. Those cases of ICH previously reported were either experimentally inoculated, or found in raccoons suffering from concurrent diseases including candidasis and listeriosis.

We believe the progression of disease in this individual started with canine distemper, which resulted in immune suppression allowing secondary infectious canine hepatitis. ICH resulted in liver necrosis, vomiting, and aspiration of gastrointestinal contents, leading to this raccoon's fatal aspiration pneumonia. It appears that raccoon's are relatively resistant to ICH, and that concurrent infections or immunocompromise is necessary for fulminant disease and mortality to result.

References:

1. Hamir, AN, DE Mattson, RJ Sonn et al. 2000. Concurrent candidiasis, listeriosis and adenoviral infections in a raccoon (*Procyon lotor*). Vet. Rec. 146: 320-322.
2. Woods, LW. 2001. Adenoviral Diseases. *In:* Williams, ES and IK Barker (eds.). Infectious Disease of Wild Mammals. Iowa State University Press, Ames, Iowa. Pp. 202-212.

Case #4

Combined Pneumonic Mannheimiosis and Adenoviral Infection in a Holstein

Bailey L. Wilberts,[1] Jianqiang Zhang,[2] Eric R. Burrough[2]

Depts. of Veterinary Pathology[1] and Diagnostic and Production Animal Medicine[2], ISU, Ames, IA

Clinical History: Multiple calves falling behind; the owner was suspicious of *Salmonella*. Sections of lung from a 6-month-old Holstein with large coalescing areas of consolidation, hemorrhage, and necrosis were received at the VDL. A 4 μm, H&E stained slide with section of lung is included in the slide set.

Microscopic Description:

Lung: There are extensive areas of coagulation necrosis in a mosaic pattern affecting roughly 50% of evaluated sections. These areas are often bordered by a thick band of degenerate neutrophils with streaming nuclei (oat cells). Affected alveoli and associated conducting airways often contain accumulations of fibrin with variable numbers of enmeshed neutrophils, macrophages, and red blood cells. Occasionally, remaining alveolar septa are expanded by low numbers of lymphocytes, macrophages, and neutrophils with hyperplasia of adjacent type II pneumocytes. Interlobular septa are variably expanded by fibrin and edema. Endothelial cells in vessels adjacent to the large necrotic foci are often hypertrophied and frequently contain large eosinophilic to amphophilic intranuclear inclusion bodies which marginate the chromatin.

Bacteriology:

- Routine culture: Heavy growth of *Mannheimia haemolytica* from the lung tissue.

Virology:

- IHC for IBRV, BRSV, and BVDV: negative
- PCR using available pan-adenovirus primers: **positive**

Electron microscopy:

- Endothelial intranuclear 70-80 nm icosahedral viral particles forming paracrystalline arrays

Diagnosis: *Mannheimia haemolytica* bronchopneumonia with endothelial intranuclear inclusions consistent with adenovirus

Comment: Vascular inclusion bodies, similar to those identified in the case of this report, have been described in the lung, kidney, liver, abomasum, small intestine and colon of calves infected with BAV type 10;[1,2] however, it is uncertain if BAV type 10 was involved in the case reported here as adenoviral infection was confirmed using pan-adenovirus PCR. Attempts to further type and to isolate the detected virus remain underway as of the writing of this report. This calf also had lesions of pneumonic mannheimiosis which was confirmed by culture. *Mannheimia haemolytica* can colonize the nasopharynx of normal calves and often shifts from commensal to pathogenic following stress or immunosuppression associated with viral infections such as bovine viral diarrhea virus, infectious bovine rhinotracheitis virus, and bovine respiratory syncytial virus.[3] None of these primary viral pathogens were detected in the present case, and it is possible that the adenovirus detected may have compromised the immune system thereby allowing *M. haemolytica* to proliferate.

References:

1. Lehmkuhl HD, Utlip RC, DeBey BM. Isolation of a bovine adenovirus serotype 10 from a calf in the United States.*J Vet Diagn Invest*. 1999; 11: 485-490.

2. Smyth JA, Benkö M, Moffett DA, et al. Bovine Adenovirus Type 10 identified in fatal cases of adenovirus-associated enteric disease in cattle by *in situ* hybridization. *J Clin Microbiol*. 1996; 34: 1270-1274.

3. Caswell JL, Williams KJ. Respiratory system. In: Maxie MG, ed. *Jubb, Kennedy, and Palmer's pathology of domestic animals*. Vol 2. 5th ed. Edinburgh: Saunders Elsevier, 2007; 602.

Case #5

Canine meningeal polyarteritis (steroid responsive meningitis-arteritis, beagle pain syndrome, necrotizing meningeal arteritis, canine juvenile polyarteritis syndrome)

Kelly Hughes[1], Beth Valentine[1], Susanne M. Stieger-Vanegas[2]

Oregon State University Veterinary Diagnostic Laboratory[1], Department of Clinical Sciences[2],

Oregon State University, Corvallis, OR

Clinical findings: A 6-month-old, intact, female coonhound initially was referred to the Oregon State University Veterinary Teaching hospital for evaluation of progressive cervical pain. At presentation she had a normal TPR, with dull mentation and neurologic examination revealed ataxia, decreased proprioception in the left fore and hind limbs, increased patellar reflexes in both hind limbs and bilateral cervical pain more severe on the left. A computed tomography exam of the cervical spine before and after intravenous iodinated contrast agent injection was performed during which the patient developed ventricular premature contractions unresponsive to lidocaine. The contrast-enhanced CT was followed by a myelo CT. Before the myelogram was started, during spinal tap of the atlanto-occcipital region, blood was identified. CT with and without contrast was highly suggestive of hypertension of the right cerebrum with prolapse of the cerebellum. Post CT myelogram revealed multifocal marked dilation of the cranial spinal cord central canal along C2-C4, suspicious for spinal cord necrosis. There was also a mild dorsal deviation of the ventral contrast column over C2-C3, suggestive of extradural compression of the spinal cord. Recovery from anesthesia post CT was poor and the dog subsequently had a cardiac arrest.

Gross findings: On gross examination, approximately 75% of the right cerebral hemisphere was covered by submeningeal clotted blood and there was coning of the caudal cerebellum. There was extensive meningeal hemorrhage of the cranial 23 cm of the spinal cord often surrounding the spinal cord and there was an irregular zone of central canal hemorrhage in the area of C3.

Histopathology: Microscopically, the meningeal space of the cervical spinal cord is markedly thickened by hemorrhage and fine fibrillar material surrounded by neutrophils, macrophages, and fewer lymphoplasmacytic cells. There is also a focal artery with fibrinoid necrosis. In the ovary there is a cluster of three arteries with necrosis with amorphous eosinophilic deposition (fibrinoid necrosis).

Diagnosis: Spinal cord, brain, ovary: polyarteritis with fibrinoid necrosis consistent with Canine meningeal polyarteritis

Comment: Canine meningeal polyarteritis is a disease occurring in young dogs, with a breed-predisposition noted in Beagles and large breeds. The classic clinical signs include sudden onset of fever, anorexia, hunched stance with remission of signs for 1-3 weeks followed by syndrome recurrence.[1, 2] In a pathological study of 18 Beagle dogs with this syndrome, systemic vasculitis and perivasculitis involving small to medium-sized arteries in the heart, cranial mediastinum and cervical spinal meninges was found in all animals with individual variation in the stages of inflammation.[3] Diagnosis is based on laboratory results which include increased IgA levels in the serum and CSF, neurologic examination, and clinical findings.[1] Characteristic lesions include fibrinoid arteritis, leptomeningeal inflammation, and extensive leptomeningeal hemorrhages may be grossly visible.[4] In this case there was extensive meningeal hemorrhage with affected medium sized vessels in the cerebrum, cerebellum, and most prominently, cervical spinal cord. There were also hemorrhages in the thymus and skeletal muscle. Interestingly, in this case, fibrinoid necrosis was found in the ovary which has not previously been described. The gross and histologic findings and signalment were a classic presentation for this uncommon entity.

References:

1. de Lahunta A, Glass E. Veterinary Neuroanatomy and Clinical Neurology, 3rd ed. Elsevier: St. Louis 2009, pp. 281-282
2. Hayes, T.J., G.K. Roberts, and W.H. Halliwell, *An idiopathic febrile necrotizing arteritis syndrome in the dog: beagle pain syndrome.* Toxicol Pathol, 1989. **17**(1 Pt 2): p. 129-37.
3. Snyder, P.W., et al., *Pathologic features of naturally occurring juvenile polyarteritis in beagle dogs.* Vet Pathol, 1995. **32**(4): p. 337-45.
4. Tipold, A. and S.J. Schatzberg, *An update on steroid responsive meningitis-arteritis.* J Small Anim Pract, 2010. **51**(3): p. 150-4.

Case #6

Infectious Laryngotracheitis Virus-Associated Conjunctivitis in Laying Hens

Dodd Sledge, R. Mick Fulton

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Signalment and History: A flock of 8-month-old brown laying chickens experienced a sudden outbreak of upper respiratory track disease and conjunctivitis shortly after introducing new hens. The eyes of these animals were swollen and squeezing the eyes resulted in exudation of large amounts of caseous material. Some affected animals also appeared to be gasping for air. Ten birds died over a two-day period. Two live and four dead brown laying hens were submitted for necropsy.

Gross Findings: The two live chickens both had yellow fibrinous and inflammatory exudate on the edges of their eyelids. The infraorbital sinuses of one of the live and two of the dead chickens were expanded by similar material.

Histopathologic Findings: The submitted slide represents an eye and associated eyelids from one of the chickens that were submitted live. The conjunctival epithelium is extensively lost, and there are marked infiltrates of heterophils in the subepithelial stroma and exudation of fibrin from the ulcerated mucosal surface. Rare remaining conjunctival epithelial cells are attenuated. Fibrin, proteinaceous fluid, heterophils, sloughed necrotic epithelial cells, and occasional multinucleated syncytial cells expand the conjunctival sac. Occasionally, the nuclei of sloughed epithelial cells and syncytial cells contain eosinophilic hyaline inclusions that peripheralize chromatin. The superficial corneal epithelium is irregularly eroded and there are mild perivascular infiltrates of heterophils, lymphocytes, and plasma cells in the anterior uvea.

Diagnosis: Severe acute fibrinonecrotizing and heterophilic conjunctivitis with syncytial cells and intranuclear viral inclusion bodies consistent with infectious laryngotracheitis (ILT) virus infection

Additional Findings: In addition to the lesions described above, there was similar necrosis of mucosal epithelium associated with marked exudation of fibrin and heterophils, formation of syncytial cells, and intranuclear inclusions in the oropharynx, larynx, trachea, and sinuses of the head (affected portions of the infraorbital sinus are present in some submitted sections). The presence of ILT virus was confirmed by PCR on tracheal swabs. On subsequent sequencing at the Poultry Disease Research Center, Athens, GA, the strain was most consistent with a chicken embryo origin (CEO) vaccine strain-like virus.

Comment: Vaccination with modified live viruses is effective in preventing infections with virulent strains of ILT virus; however, vaccination can create latently infected carrier chickens that can pass virus to other birds. Induction of clinical disease by vaccine strains of ILT has been well described with the majority of outbreaks in the US being associated with vaccine strains. [2,3] CEO vaccines in particular have been shown to increase in virulence with multiple bird-to-bird passages.[1] Such infection of naïve resident chickens by a vaccine strain from latently infected, recently introduced chickens is suspected in this case.

References:

1: Guy JS, Barnes HJ, Smith L. Increased virulence of modified-live infectious laryngotracheitis vaccine virus following bird-to-bird passage. Avian Dis. 1991 Apr-Jun;35(2):348-55.

2: Oldoni I, Rodríguez-Avila A, Riblet S, García M. Characterization of infectious laryngotracheitis virus (ILTV) isolates from commercial poultry by polymerase chain reaction and restriction fragment length polymorphism (PCR-RFLP). Avian Dis. 2008 Mar;52(1):59-63.

3: Oldoni I, García M. Characterization of infectious laryngotracheitis virus isolates from the US by polymerase chain reaction and restriction fragment length polymorphism of multiple genome regions. Avian Pathol. 2007 Apr;36(2):167-76.

Case #7

Leptospirosis in a Doberman Pinscher

Tuddow Thaiwong[1], Dodd Sledge[1], and Alan Ralph[2]

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2College of Veterinary Medicine, Michigan State University, East Lansing, MI

Singalment and History: A 5 year-old, intact male, Doberman Pinscher from Detroit presented to MSU for management of anuric renal failure and liver failure through hemodialysis and supportive care; however, cardiovascular dysfunction and disseminated intravascular coagulation developed and the animal died. Fixed samples of liver, lung, and kidney were submitted to the DCPAH for evaluation.

Gross Findings: In the kidney, there were radiating pale white streaks at the corticomedullary junction that were arranged perpendicular to the capsular surface and fewer less distinct pinpoint pale foci distributed throughout the remainder of the cortex. On cut section of the liver, there was an enhanced centrilobular pattern. In the lung, there were sharply demarcated regions of dark red to brown discoloration randomly distributed throughout the pulmonary parenchyma.

Histopathologic Findings: Sections of kidney were affected by chronic interstitial nephritis characterized by expansion of the renal cortical interstitium by fibrosis, edema, and scattered infiltrates of lymphocytes and plasma cells. Renal tubular epithelial cells were focally necrotic or contained hemosiderin. Granular casts often obliterated proximal tubules, and medullary tubules and collecting ducts often contained proteinaceous material or cellular debris. Glomerular changes were mild. In the liver, there was marked cholestasis, centrilobular congestion, and centrilobular atrophy. Centrilobular areas were often hypercellular due to infiltrates of lymphocytes, plasma cells, and histiocytes. Rare hepatocytes were dissociated from hepatic cords and necrotic. In the lung, there was fibrinoid necrosis of alveolar septa associated with intra-alveolar accumulation of fibrin, proteinaceous edema fluid, and hemorrhage.

Additional Testing: Serologic testing by microscopic agglutination (MA) test revealed a high titer (800) to *Leptospira* serovar *icterohemorrhagica*. Immunohistochemistry confirmed the presence of Leptospiral organisms at the apical surface of tubular epithelium, in tubular lumens, and within the renal interstitium.

Comments: This case demonstrates a range of lesions that can be seen in cases of leptospirosis. Infection results in acute leptospiremia and consequently nephritis, hepatitis, and hemolysis may occur (1). Interstitial nephritis is common as organisms persist in proximal convoluted tubules following the development of a humoral immune response. The marked cholestasis observed in this case is likely related to hemolysis, hepatocellular injury and/or centrilobular ischemia. The lesions in the lung are most consistent with acute alveolar damage as would be expected with acute respiratory distress syndrome. Endotoxemia, septicemia, uremia, shock, and/or disseminated intravascular coagulation likely contributed to such alveolar damage (2). Leptospirosis is a reportable animal and human disease in Michigan (3). Outbreaks of leptospirosis have been reported in Detroit-area dogs in 1980 and 2011 (4). Investigation suggested that such outbreaks occurred mainly through direct contact with water or soil contaminated with the urine of infected rats in abandoned properties in metro Detroit. Such outbreaks of Leptospirosis represent a great example of the health related consequences of urban decay.

References:

1. André-Fontaine G. Canine leptospirosis--do we have a problem? Vet Microbiol. 2006 Oct 5;117(1):19-24.
2. Goldstein RE. Canine leptospirosis. Vet Clin North Am Small Anim Pract. 2010 Nov;40(6):1091-101.
3. Langston CE, Heuter KJ. Leptospirosis. A re-emerging zoonotic disease. Vet Clin North Am Small Anim Pract. 2003 Jul;33(4):791-807.
4. Thiermann AB. Canine leptospirosis in Detroit. Am J Vet Res. 1980 Oct;41(10):1659-61.

Case #8

Tularemia in a Three Cat Household

Loni Schumacher[1], **Kelli Almes[1]**, Sarah Mills[2]

1. Kansas State Veterinary Diagnostic Laboratory, Kansas State University, Manhattan, KS

2. Mills Veterinary Service, LLC, Chapman, KS

Signalment: Three approximately 7 month old domestic short-haired cats.

History: One week prior to presentation to the rDVM the owners had discovered what they assumed to be rabbit remains on their garage floor at their rural home. All 3 cats became ill within a 1 week time period. One cat recovered and the other 2 were presented with extreme dehydration, icterus, hyperthermia, and enlarged submandibular lymph nodes. Both cats were euthanized and a necropsy examination was started on one cat by the rDVM. Coalescing white nodules on the spleen as a possible gross lesion associated with tularemia were recognized, therefore necropsy was suspended and the animals were delivered to the Kansas State Veterinary Diagnostic Laboratory the following morning.

Gross Findings: Both cats had similar gross necropsy findings, which included multifocal to coalescing white nodules (necrosis) throughout the spleen, pinpoint to coalescing white foci throughout the liver and lungs, systemic marked lymphadenopathy, and multifocal glossal ulcerations (one cat). One cat also had a markedly thickened ileum with a pseudomembrane and diffuse necrosis of Peyer's patches.

Laboratory Findings: The spleens of both cats were cultured and the growth was suggestive of *Francisella tularensis*. The tissues were shipped to Fort Collins CDC where a confirmatory culture was positive for *Francisella tularensis*.

Histopathology Findings: The spleen and all lymph nodes contained multifocal to coalescing areas of massive necrosis with large numbers of degenerative neutrophils admixed with macrophages, fibrin and cellular debris. The liver and lungs contained scattered multifocal areas of similar necrosis. There was diffuse necrosis of Peyer's patches within the ileum, which were markedly expanded. The mucosal lamina propria was also infiltrated by large numbers of neutrophils and there was necrosis and loss of villi. Immunohistochemistry was strongly positive for *F. tularensis* on multiple tissues.

Final Diagnosis: A diagnosis of tularemia was confirmed with cultures at the CDC which was consistent with clinical, gross, histologic, and immunohistochemical findings.

Comments: Tularemia is an important zoonotic disease still seen on a regular basis in our companion animal population. The disease is nearly always fatal in our domestic feline population and contact with these animals can lead to disease in humans. Cases are seen each year on a seasonal basis at the KSVDL and this case represents a typical diagnostic work-up with confirmatory IHC and culture. The histologic lesions were quite severe in these two cats and the fact that the other cat in the household recovered is quite unique.

References:

1. Debey, et al.: 2002, Immunohistochemical Demonstration of *Francisella Tularensis* in Lesions of Cats with Tularemia
2. Centers for Disease Control Tularemia website (www.cdc.gov/tularemia/)

Case #9

Acute Pulmonary Edema Associated with Porcine Circovirus Type2 Infection

Chanran Ganta, Madhu Goravanahally, Ada G. Cino-Ozuna, Loni Schumacher, Jerome Nietfeld, Steve Dritz, Richard Hesse and Jamie Henningson.

Kansas State Veterinary Diagnostic Laboratory, Department of Diagnostic Medicine and Pathobiology, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66506

Signalment: Five 45-day old piglets were submitted for necropsy.

History: Acute deaths in young pigs.

Gross Necropsy findings: Lungs did not collapse after the thoracic cavity was opened with rib impressions on the pleural surface. Diffusely all lungs lobes were edematous and moderately firm. Interlobular septa were severely expanded by edema, on cut section oozed moderate amounts of clear fluid. The mesenteric lymph nodes and tracheobronchial lymph nodes were moderately enlarged and congested. The thymus was diffusely and markedly reduced in size.

Histopathology Findings: Lungs. Interlobular septa and the pleura were markedly expanded by edema and few mixed inflammatory cells. A few vessels contain fibrin thrombi and in some the tunica media was expanded by degenerate neutrophils, edema and fibrin (vasculitis). Diffusely alveolar septa were expanded by mixed inflammatory cells admixed with severe edema and fibrin. Occasional bronchioles contain degenerate cellular debris, neutrophils and macrophages with mucosal epithelial hyperplasia.

Lymphoid organs: Within the spleen, extending from the serosal margins towards the center, there were irregular sharp delineated multifocal to coalescing areas of necrosis that were rimmed by congestion. Occasional vessels contain fibrin thrombi with infiltrates of degenerate neutrophils in the tunica intima and media admixed with cellular debris (vasculitis). Periarteriolar lymphoid sheets were diffusely absent and replaced by karyorrhectic debris. There was marked lymphoid depletion and necrosis and occasional vessels had similar changes as in the spleen. There were infiltrates of large numbers of histiocytes and multinucleated giant cells in the cortical region of the lymph nodes. There was marked thymic lobular atrophy with depletion of lymphocytes.

Laboratory Findings: The PCR on pooled tissues from all the piglets was positive (Ct=12) for PCV2 infection. IHC is strongly positive for PCV2 in the lung, spleen, and lymph nodes and negative for Porcine reproductive and respiratory syndrome (PRRS) virus. The bacterial cultures were not significant.

Final Diagnosis: Lungs had diffuse interstitial pneumonia with marked edema along with systemic vasculitis and marked lymphoid atrophy (spleen and lymph nodes) and necrosis. PCR and IHC results were strongly positive for PCV2 and negative for PRRS.

Comments: Lesions were consistent with acute pulmonary edema (APE) due to PCV-2 infection a syndrome that was recently by Cino et. al. Porcine circovirus-associated disease (PCVAD) encompasses a group of wasting syndromes linked to porcine circovirus type 2. APE unlike other PCVAD syndromes has peracute onset and sudden death.

References:

1. Cino-Ozuna AG, Henry S, Hesse R, Nietfeld JC, Bai J, Scott HM, Rowland RR. Characterization of a new disease syndrome associated with porcine circovirus type 2 in previously vaccinated herds. *J Clin Microbiol. 2011 May;49(5):2012-6. doi: 10.1128/JCM.02543-10.*

Case #10

Equine Cryptococcosis

Mahogany Caesar, Jennifer Haugland

North Carolina Veterinary Diagnostic Laboratory, Raleigh, NC

Signalment and History: Reported in an 11-year-old Thoroughbred gelding were weight loss, muscle wasting and a 1.5 month duration of neurologic deficits, characterized by limited/painful neck movement and bilateral rear limb ataxia with decreased proprioception. Cervical radiographs revealed degenerative arthritis of C4-C7 articular facets. The horse failed to respond to treatment and was euthanized.
Necropsy examination: The principal abnormalities included excessive yellow cerebrospinal fluid with many air bubbles in the subarachnoid space of the brain and subdural space of the spinal cord extending from C1 to T10 (decreasing number of air bubbles and loss of yellowing distally) and firm white granular material expanding the choroid plexus and the subdural space of C1 and the brainstem. While trimming the brain for histopathology, a 2 x 2 x 1 cm focus of malacia with multifocal yellow gelatinous material was identified in the right internal capsule. A similar 2 x 2.5 x 0.5 cm focus was observed in the left corona radiata.
Histopathology: There was marked cavitation of the cerebral parenchyma by a multifocal to coalescing inflammatory infiltrate containing yeast organisms surrounded by large numbers of macrophages, variable numbers of lymphocytes and plasma cells, and few foreign-body type multinucleated giant cells and neutrophils. Associated with the inflammation were well-differentiated fibroblasts and small-caliber blood vessels lined by hypertrophic endothelium (granulation tissue). The extracellular yeasts were 10-40 um in diameter and round with a 5-8 um thick amphophilic mucinous capsule. The meninges of the cerebrum, cerebellum and spinal cord were frequently expanded by a similar inflammatory infiltrate admixed with multinucleated giant cells, hypereosinophilic and karyorrhectic debris (necrosis), and extracellular yeast organisms. Vascular congestion, fibrin, hemorrhage and edema were also identified.
Morphologic diagnoses: Brain; meningoencephalitis, granulomatous, multifocal to coalescing, chronic, severe with intralesional fungi consistent with *Cryptococcus* sp. (similar lesions also noted in the spinal cord)
Mycology: Yeast identified as *Cryptococcus neoformans* were isolated in the spinal cord.
Comments: Cryptococcosis is a systemic mycosis caused by a dimorphic fungus with an infective yeast phase. *Cryptococcus neoformans* is a saprophytic yeast that is found primarily in pigeon feces acquired mainly via inhalation where it may establish itself in the upper respiratory tract or in the alveoli to form granulomas. In tissues, the organism forms a thick protective mucopolysaccharide capsule that prevents it from being cleared by the host's immune system. Routes of CNS transmission include: (1) direct extension to the leptomeninges and subarachnoid space through the cribriform plate following sinus/nasal infection, and (2) hematogenous spread and leukocytic trafficking from other infection sites. The infection is uncommon in horses, however sporadic cases have been linked with granulomatous pneumonia, nasal granuloma, osteomyelitis, intestinal polypoid granulomas, endometritis and placentitis with neonatal cryptococcal pneumonia, mesenteric lymph node abscess, and abortion. Cryptococcus with CNS involvement has been reported in dogs, cats, cattle and horses.
References:

1. Riley CB, Bolton JR, Mills JN, Thomas JB. Cryptococcosis in seven horses. Australian Veterinary Journal 1992;69:135-139
2. Zachary JF. Nervous system: fungi and algae. In: McGavin MD, Zachary JF, editors. Pathologic basis of veterinary disease, 5th ed. St.Louis, MO: Mosby; 2007. Pages 642-644
3. Del Fava, Claudia, Levy FL, Schannapieco EM. Cryptococcal pneumonia and meningitis in a horse. Journal of Equine Veterinary Science 2011;31:693-695

Case #11

Gastric cryptosporidiosis in a captive Pacific gopher snake (*Pituophis catenifer*)

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History/signalment: A 20 year old, 1.70 kg, 1.88 cm long, female Pacific gopher snake (*Pituophis catenifer*) was presented to CAHFS for a full diagnostic work-up. The snake had been housed at a non-for-profit museum in California. It had a history of recurrent mid-coelomic swelling. Cryptosporidia were detected in the feces and it was euthanized due to epizootic concerns.

Gross pathology: The gastric wall was diffusely thickened with the mucosal surface diffusely rugose and multifocally reddened. The liver had multifocal, random, pinpoint, golden foci. The intestines were moderately dilated by moderate amounts of mucinous material admixed with ingesta. The rectum contained large amounts of semisolid viscous feces.

Histopathology: Stomach: the gastric wall was markedly thickened. The mucosa was expanded by multifocal to diffuse, severe hypertrophy and hyperplasia of the mucus neck cells that often replaced the granular cells of gastric glands. Lining the mucosal surface, the luminal glandular epithelium, and free within the lumen of the gastric glands, were myriad 3-7 μm diameter, round, pale amphophilic protozoa with basophilic nuclei, consistent with *Cryptosporidium sp.* Multifocally the lamina propria, submucosa and tunica muscularis contained few lymphocytes and plasma cells. Within the lumen and on the surface of the gastric mucosa there were few sloughed epithelial cells. The mucosa was multifocally mildly eroded. Liver: Diffusely hepatocytes showed severe, macro- and micro- vacuolation; most hepatocytes contained many fine brownish Perl's blue (iron) positive hemosiderin granules. Kidney: Many tubular epithelial cells contained hemosiderin granules.

Other results: Cryptosporidia were detected in Ziehl-Neelsen stained smears from the gastric mucosa and the feces. Both fecal and stomach cryptosporidia were identified as *Cryptosporidium serpentis* by PCR and direct sequencing[1]. Liver, lung, small intestinal samples were negative for *Salmonella sp* by PCR. No other parasites were detected in the feces. Liver and lung culture yielded no growth after 48h. Liver iron levels were elevated (6000 ppm).

Morphologic diagnoses: 1. Gastric hypertrophy and hyperplasia, chronic, diffuse, severe, with myriad free and apically attached protozoa, consistent with *Cryptosporidium* sp. 2. Hepatocellular hemosiderosis, diffuse, marked with diffuse fatty lipidosis. 3. Renal hemosiderosis, diffuse, marked.

Etiologic diagnosis (etiology): Gastric cryptosporidiosis *(Cryptosporidium serpentis)*.

Comments: Gastric hypertrophy/hyperplasia, although not pathognomonic, is a lesion often associated with Cryptosporidium infection in snakes[2]. *C. serpentis* is a snake specific species, rather than a species associated with consumed infected prey or other reptile species, and was possibly introduced to the collection by a new acquisition. The diagnosis allowed appropriate health management decisions to be taken for the rest of the reptile collection, and alleviated epizoonotic and public health concerns. The etiology of hemosiderosis in the present case is unknown, although it may result from congestion, hemolysis, overexposure to iron, iron storage abnormalities or starvation, and has been associated with halofuginone treatment of cryptosporidiosis in snakes[3] (not used here).

References:

[1] Richter B et al (2011) J Vet Diagn Invest 23 (3): 430.
[2] Valentin A et al (1998) Tierarztl Praxis (Kleintiere) 26: 55.
[3] Graczyk TK et al (1996) Parasitol Res 82(2): 143.

Case #12

Hepatic alveolar echinococcosis in a dog in Ontario, Canada

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History: A 2-year old neutered male Boxer dog presented to a small animal clinic in southern Ontario with a 12-hour history of anorexia and lethargy. Laparotomy revealed a hemorrhagic, destructive, liver mass. The patient was euthanized.

Histopathology: In and attached to the liver there is a multiloculated cystic structure. This structure contains several coalescing cystic spaces that contain eosinophilic fragmented hyaline membranes, necrotic material, mineralized granular material and prominent chronic inflammation. The inflammation is eosinophilic and granulomatous with discrete granulomas and a mixture of eosinophils, plasma cells, lymphocytes, macrophages, multinucleated cells and neutrophils. The cystic structure is compressing adjacent liver. In the adjacent liver tissue there is mild fibrosis of portal areas and around terminal hepatic veins, mild vascular and biliary proliferation, mild inflammation with a few eosinophils and mononuclear cells, hepatocellular loss and lobular atrophy, and occasional bile plugs.

Interim diagnosis and comment: Cystic liver lesion with eosinophilic granulomatous inflammation, necrosis and mineralization - suspect parasitic cyst, likely of cestode origin

Molecular testing (Institut fur Parasitologie, Bern, Switzerland): PCR performed on DNA extracted from formalin-fixed tissue. Sequencing of PCR-generated fragment of the mitochondrial 12S rRNA gene, and RFLP analysis of PCR-generated fragments of mitochondrial 12S rRNA and NADH dehydrogenase 1 genes confirmed the diagnosis of hepatic metacestode of *Echinococcus multilocularis*.

Final diagnosis: Hepatic metacestode of *Echinococcus multilocularis*

Comment: Hepatic alveolar echinococcosis (HAE) in dogs is an extremely rare diagnosis in North America. One case has previously been reported in British Columbia (1). This is the first reported case in Ontario. It is important that veterinarians recognize that dogs can act as intermediate and definitive hosts for *E. multilocularis*, and that this tapeworm poses a serious zoonotic threat (2). To date, thirteen people that came in contact with this dog, or its littermates, have tested negative for antibody to *E. multilocularis*.

References:

1. Peregrine AS, et al., Alveolar hydatid disease (*Echinococcus multilocularis*) in the liver of a Canadian dog in British Columbia, a newly endemic region. Can Vet J 2012;53:870-874.
2. Deplazes P, Eckert J. Veterinary aspects of alveolar echinococcosis – a zoonosis of public health significance. Vet Parasitol 2001;98:65-8.

Case #13

Cerebellar herniation (Chiari-like malformation) in a small-breed dog

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Signalment: Female 14 week old Cavalier King Charles spaniel pup.

Clinical History: The pup was presented in acute respiratory shock. Earlier in the day the pup had been snapped at by another dog. The pup avoided the bite with a sudden movement of the head, and immediately became 'stiff.' Hematology revealed lymphocytosis with neutrophilia. Other abnormalities were elevated phosphate, glucose and serum alkaline phosphatase activity, and low serum potassium and total protein. Radiography revealed moderate pulmonary edema and pericardial effusion; skull and neck were not examined radiologically. The pup did not respond to oxygen, antibiotics or the benediction of glucocorticoids. Acute progressive neurologic signs developed, progressing to brain death after several hours. Total clinical time from onset to death: 6 hours.

Gross examination: A well-nourished 3.28 kg carcass was presented. There were two principal changes: severe acute multifocal pulmonary hemorrhage and hemorrhagic necrosis of cerebellar vermis herniated into foramen magnum. Occipital condyles were dorsoventrally flattened.

Microscopic description:

- Lung: Moderate acute hemorrhagic exudative pneumonia with intra-bronchial bacteria
- Cerebellum-medulla oblongata: Severe acute hemorrhage with necrosis of ventral cerebellar vermis and cerebellar roof nuclei, with multifocal hemorrhage of posterior medulla oblongata and associated leptomeninges.

Diagnosis: Fatal Chiari-like malformation of Cavalier King Charles spaniel with hypoplasia of posterior cranial fossa, and peracute aspiration pneumonia

Comment: Small-breed dogs are prone to craniocervical junction abnormalities such as Chiari-like malformation, atlanto-occipital overlap, and dorsal constriction of C1-C2 vertebral junction. 'Craniocervical junction abnormalities' is a useful umbrella term since the abnormalities can occur in combination. Chiari-like malformation has the remarkable estimated prevalence of 92 – 100% in this breed. It is likely to have a genetic component. The cause is impaired occipital bone development, leading to a short posterior cerebral fossa with crowding of medulla oblongata and cerebellum. The result is cerebellar herniation (as here) and/or syringomyelia affecting cervical, thoracic and lumbar portions of the spinal cord. Unfortunately I neglected to take spinal cord in this case. Clinically affected dogs present with cervical pain and apparent pruritus of head, neck and shoulder, leading to phantom scratching. Neurological defects include cerebello-vestibular dysfunction and seizures. The owners of this dog reported that the pup did scratch its neck, but they interpreted the behavior as a normal response to having a collar. There are few morphological reports of fatal Chiari-like malformation in small breed dogs due to the current widespread use of MRI. Affected dogs are treated medically or surgically, the latter involving foramen magnum decompression with craniotomy using titanium mesh and polymethylmetharylate. The development of pneumonia, presumably from aspiration, is an unusual outcome for this syndrome, based on published reviews of clinical signs in affected spaniels.

References:

1. Dewey CW, Marino DJ, Loughin CA: 2013, Craniocervical junction abnormalities in dogs. N Zealand Vet J 61(4): 202-211.

Case #14

Unusual congenital cerebrocortical lesions in a calf

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Clinical history: A three-day-old 42kg male Devon calf was submitted for necropsy to AHVLA Luddington. The calf was reported as being 'not right' since birth, with incoordination, head pressing, abnormal head posture and "absence of menace reflex". The calf had been fed colostrum. The calf had kyphosis, otherwise significant gross findings were confined to the CNS. The whole brain and cerebellum weighed 170.5g and 19.2g respectively. Cut surfaces of brain revealed multiple small gyri, bilateral focal translucency of cerebral white matter, particularly in the frontal cortices, and cavum septum pellucidum.
Histopathology and laboratory findings: Histological examination revealed multiple areas of narrowing of cerebrocortical grey matter where the number of differentiated neurons was moderately to severely reduced. The latter areas contained atypical glial nuclei in intensely GFAP-positive loose delicate fibrillary neuropil with occasional radial glial processes. In adjacent areas and to a lesser extent elsewhere in the cortex there were near-monomorphic populations of cells with small dark nuclei (consistent with primitive neuroglia) arranged in clusters, more diffusely or rarely in linear arrays perpendicular to the pial surface, mainly in areas corresponding to lamina 2, to a lesser extent in deeper laminae and occasionally closely associated with the glia limitans. Increased cellularity including presence of probable lymphocytes in overlying leptomeninges was a variable feature. Areas of less prominent thinning of cortex with apparent reduction in the number of laminae and a narrow linear cavity in white matter in the occipital cortex were also present. There was an impression of widening of ventral fissure in the anterior cervical spinal cord. Schmallenberg virus (SBV) or Bovine virus diarrhoea virus (BVDV) RNA was not detected by PCR in samples of cerebral cortex and spleen respectively. Antibodies to SBV and BVDV were detected in post-colostral serum.
Comments: The total brain to body weight ratio (0.4%) was below the reference range for neonatal calves [0.5–0.6%], consistent with disproportionate microencephaly. The cerebellum to whole brain ratio (11.3%) was above the laboratory reference range [9-10%], suggesting that the cerebrum was disproportionately small, however the cerebellar weight was also low compared with neonatal calves of similar body weight. The lesions of multifocal cerebral dysgenesis with dyslamination (compromised tangential and / or radial organisation of cortical architecture) and hypoplasia without a 6 layered structure are similar to those of human Focal Cortical Dysplasia (FCD) type IIId, which can be associated with a large spectrum of lesions acquired during early life – trauma, ischaemia, inflammatory or infectious diseases (Blümcke et al 2011). The presence of neuroglial rest-like clusters in abnormal positions suggests interference with neuronal migration/differentiation, as does the persistence of radial glial processes detected by GFAP immunohistochemistry. The dense meshwork of glial processes and unusual prominence of radial glial processes have been observed in cases of SBV-associated cerebellar dysgenesis in lambs. Whilst overt myelodysgenesis was not detected, the apparent widening of the ventral fissure raises the possibility of a degree of reduction in size of the ventral funiculi, and this type of pathology has been observed in SBV-induced lesions although it is not considered specific for this. Failure to detect viral RNA does not exclude the possibility of *in utero* infection, as viral RNA may no longer be detectable in calves with malformations at term. Taking into consideration the clinical findings and laboratory results, it is possible that this case may represent a previously unrecorded manifestation of *in utero* teratogenic viral infection in cattle, however *in utero* exposure to SBV or BVDV could not be assessed by antibody analysis, as pre-colostral serum was not available.
Reference:
The clinicopathologic spectrum of focal cortical dysplasias: a consensus classification proposed by an ad hoc Task Force of the ILAE Diagnostic Methods Commission. Blümcke I, *et al* Epilepsia. 2011 52 (1):158-74.

Case #15

Perianal celluitis associated with a novel trematode in Pacific fishers (*Martes pennanti*)

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Clinical history: The Pacific fisher (*Martes pennani*), a rare mustelid carnivore that lives in mixed coniferous forest in the Pacific northwest, is currently a candidate for listing under the Endangered Species Act. As part of a collaborative project to translocate the animal from northwestern California back to historic range in northeastern California, fishers being considered for relocation were captured, given health examinations and tested for disease. In 2010, several captured fishers had perianal swelling with multifocal subcutaneous abscesses and were therefore biopsied.

Histopathology: Histopathology of the affected region revealed severe diffuse necrosis and pleocellular (eosinophilic, plasmacytic and histiocytic) inflammation with intralesional trematode eggs and rarely trematode adults. Additionally, over a three year period from 2010-2012, 14/65 fishers necropsied at the California Animal Health and Food Safety Laboratory in Davis had perianal cellulitis associated with trematode eggs.

Comments: Trematode parasites were recovered from animals in Humboldt county and submitted for identification. The trematode has been identified as a novel trematode in the Pacific fisher. Current studies to identify the species, intermediate host(s) and geographic distribution, prevalence, and potential impact on the fisher population are ongoing.

Case #16

Focal symmetrical encephalomalacia in a calf

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Introduction: Focal symmetrical encephalomalacia (FSE) is the hallmark of chronic and sometimes sub-acute forms of *C. perfringens* type D enterotoxemia in sheep and it has also observed, albeit rarely, in goats with this disease. FSE has been described in cattle but no causal relationship between this lesion and *C. perfringens* type D has been established.

Clinical history: A 9 month old calf was inoculated intraduodenally with washed cells of *C. perfringens* type D. This animal showed moderate central nervous system clinical signs that started a few hours after inoculation and lasted for 8 days, until the animal was euthanized. The clinical signs included ataxia with wide base stance followed by recumbency, paddling, opisthotonous, hyperesthesia, blindness with loss of pupillary reflex, strabismus, bruxism, decreased muscular tone, hypersalivation and convulsions.

Gross findings: Grossly, this animal showed multi-focal, bilateral and symmetrical cavitation and softening of the internal capsule within the corpus striatum.

Histopathology: Histologically there was severe, focal necrosis of corpus striatum, cerebellar peduncles and thalamus. In these areas there was severe vacuolation of the white matter, with dilated myelin sheaths, dilated axons, neuronal necrosis, perivascular and interstitial edema, diffuse gliosis and presence of large, vacuolated macrophages (Gitter cells). Surrounding the area of degeneration and necrosis, the astrocytes were hypertrophic, with large, vesicular nuclei, and there was also hypertrophy of endothelial cells in both veins and arteries.

Comments: These results show that experimental enterotoxemia by *C. perfringens* type D in cattle has similar clinical and pathological characteristics to the natural and experimental disease in sheep.

Case #17

Porcine epidemic diarrhea virus in three neonatal piglets

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Signalment: Three neonatal crossbred piglets

Clinical History: Acute diarrhea and vomiting was reported in sows and newborn pigs starting on June 13, 2013. Sows were reportedly off feed. Differential diagnosis included porcine epidemic diarrhea virus (PEDV) and transmissible gastroenteritis virus (TGEV). Three dead neonatal piglets and five fecal samples were submitted to the ADDL on June 14, 2013.

Gross lesions: All three piglets were markedly dehydrated. The stomach of each piglet was partially filled with casein curd. The small intestines, cecum and spiral colon were thin walled and were distended with gas and thin, clear, pale green fluid.

Histopathology: Small intestines: There is marked villous atrophy in sections of jejunum and ileum, with occasional fusing of villi. Superficial villous enterocytes are markedly attenuated in affected villi, cell morphology ranging from low cuboidal to flattened. Low numbers of flattened villous enterocytes appear to have hypereosinophilic cytoplasm and hyperchromatic to pyknotic nuclei (degeneration and/or necrosis). The villous:crypt ratio is appreciably decreased, estimated at 1.5-2:1 in many areas. Epithelial cells in a small number of crypts exhibit increased mitotic figures and irregular piling in multiple layers (crypt hyperplasia).

Ancillary test results: Nucleic acid of porcine epidemic diarrhea virus was detected in cecal and colon contents by reverse transcriptase polymerase chain reaction (RT-PCR).

Morphologic Diagnosis:
Small intestines: Marked segmental to diffuse villous atrophy with attenuation of villous enterocytes

Discussion: The USDA National Veterinary Services Laboratories first confirmed the diagnosis of porcine epidemic diarrhea virus (PEDV) in the United States on May 17, 2013 in Iowa. PED virus is a single-stranded, positive-sense RNA group 1 coronavirus of the family *Coronaviridae* that can only be distinguished from TGE, a closely related coronavirus, by laboratory tests including reverse transcriptase polymerase chain reaction (RT-PCR). Virus particles have a diameter which averages 130 nm and contain club-shaped projections of 18 to 23 nm that are encoded by a 28-kb portion of the genome (structural "spike" or "S" gene) that encodes a multifunctional virulence factor (Li et al., 2012). Clinical signs consist of thin watery feces and vomiting, with secondary signs of dehydration and metabolic acidosis as a result of malabsorptive diarrhea. Morbidity is very high in young pigs, and mortality may approach 50-80% in suckling pigs, but often decreases with age to 1-3% in grower pigs. As of June 17, 2013, the NAHLN reported PEDV positive cases in at least 12 states. PED is not a listed disease for either the World Organization for Animal Health (OIE) or the USDA at this time.

References:

1. Porcine Epidemic Diarrhea (PED) Technical Notes. United States Department of Agriculture. http://www.aphis.usda.gov/animal_health/animal_dis_spec/swine/ped_info.shtml. Accessed July 1, 2013.
2. Saif LJ, Pensaert MB, Sestak K, Yeo S-G, Jung K. Coronaviruses. In: Diseases of Swine, 10th edition, ed. Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ and Stevenson GW. P. 514-517.Wiley-Blackwell, Ames, Iowa. 2012.

Case #18

Feline osteochondroma

Tim Cushing
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Signalment: 11 year old, male castrated, domestic short hair cat.
History: Owner described a several month history of decreased control of tail function and increased difficulty jumping onto shelves where this cat normally rested. At the rDVM a moderate neutrophilia ($26x10^3$/µl) with a left shift was observed on blood work and a large proliferative and somewhat lytic mass was found on the wing of the right ilium via radiographs. A core biopsy of the mass was taken but was found to be non-diagnostic. The cat returned to the rDVM 3 months later after developing fecal incontinence. During evaluation it was noted that the ilial mass had increased in size. The animal was euthanasized due to a poor prognosis related to the fecal incontinence and the ilial mass.

Gross and histologic findings: A 3x3.5x4cm, hard, white-tan to slightly blue-grey, proliferative mass was observed encompassing the wing of the right ilium and the transverse processes of the adjacent lumbar vertebrae (L5-6). Circumferentially expanding the periosteum are confluent boney proliferations composed of boney trabeculae that are perpendicular to the intact cortex of the ilium. Multiple foci of cartilage are present within the proliferative mass; mostly toward the periphery of the mass (cartilage caps). The cartilage is disorganized and frequently exhibits necrosis which is characterized by marked pallor of the cartilaginous matrix, chondrocyte atrophy with nuclear pyknosis and chondrocyte loss. Between the boney trabeculae is a moderate amount of adipose, small amounts of fibrous connective tissue and occasional small islands of hematopoietic cells. Within the marrow cavity of the ilium and vertebra there is variable myeloid hyperplasia. Myocytes adjacent to the boney proliferations exhibit mild to moderate atrophy.

Histologic diagnoses: Ilium and vertebrae: Osteochondroma

Comments: Osteochondromas are benign masses that have been reported in many species, with the dog, cat and horse represented most frequently. Differences in osteochondromas are present between these species as masses in dogs and horses occur along the metaphyseal region of long bones in young animals and cease growing upon maturation of the bone, while these masses in cats occur mostly on the flat bones of older animals and exhibit continuous growth.[1,2] This continuous growth is considered a feature of a true tumor, while the growth pattern in dogs and horses is believed to be more accurately described as a physeal/skeletal dysplasia. In cats, type C- retroviral particles consistent with feline leukemia virus or feline sarcoma virus have been found in chondrocytes within these masses, suggesting a viral etiology[1,2]; however, causation has not been proven.

References:

1. Thompson KG and Pool RR. Feline Osteochondromatosis In*:* Mueten DJ, ed. *Tumors of Domestic Animals* 4th ed. Ames IA. Blackwell Publishing, 2002: 258-259.
2. Thompson K. Osteochondroma In*:* Maxie MG, ed. *Jubb, Kennedy, and Palmer's pathology of domestic animals*. 5th ed. Philadeplphia, PA. Elsiver, 2007:119