



62nd Annual AAVLD Meeting
Diagnostic Pathology Slide Seminar
October 26, 2019
Providence, RI

Case #	Species	Organ	Diagnosis
1	White-tailed deer	Skin	Follicular dysplasia
2	Sheep	Rumen	Halogeton intoxication
3	Ox	Lung	Atypical interstitial pneumonia
4	Ox	Bone	Ricketts
5	Pig	Liver	African swine fever cholangiohepatitis (See disc)
6	Budgerigar	Head	Pituitary carcinoma
7	Eastern gray squirrel	Lung, skin	Disseminated squirrel fibromatosis
8	Dog	Lymph node	Mycobacterial lymphadenitis (<i>M. avium</i> ssp <i>hominissuis</i>)
9	Raccoon	Intestine	Canine distemper and cryptosporidiosis
10	Horse	Intestine	<i>Clostridium sordellii</i> -associated colitis
11	Horse	Intestine	NSAID-associated colitis (right dorsal colitis)
12	Red-footed tortoise	Pleura	Amoebic pleuritis (<i>Entamoeba invadens</i>)
13	Saki monkey	Brain	Listeriosis
14	Ball python	Lung	Nidoviral pneumonia
15	Ox	Larynx	Calf diphtheria
16	Ox	Placenta	Fungal placentitis (<i>Candida parapsilosis</i>)

Case # 1

Follicular dysplasia in an adult white-tailed deer (*Odocoileus virginianus*)

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Signalment: Skin from a hunter-harvested 2.5-year-old (6-point) male white-tailed deer.

History: The hunter had previously observed the deer with hair loss on a trail camera.

Gross Necropsy Findings: There was severe alopecia over the trunk, lateral thighs, and distal limbs with sparing of the head and neck.

Histopathology Findings: Hair follicles and adnexa are present in relatively normal numbers. The epidermis has hyperkeratosis extending into the follicular ostia that also markedly dilates the lumina (infundibular hyperkeratosis). Hair follicles have variably irregular contours and are typically empty or contain fragmented and irregular hair shafts. Sebaceous glands have ectatic central ducts and contain fragments of keratin; and apocrine glands are moderately ectatic and contain eosinophilic, acellular amorphous material. Hair bulbs are subjectively atrophied. In a few areas, the superficial dermis exhibits minimal pigmentary incontinence as does the perifollicular dermis. The dermis, superficially, has multiple perifollicular and periglandular aggregates of small numbers of lymphocytes and plasma cells. Within the stratum corneum are low numbers of intracorneal pustules with multiple foci of superficial serocellular crusts. Some regions have mild hyperpigmentation of the basal epithelium. No infectious organisms were identified with Gram or Grocott's methenamine silver stains.

Final Diagnosis: Follicular dysplasia with follicular distortion, infundibular hyperkeratosis, and multifocal mild lymphoplasmacytic dermatitis (toothpaste hair disease).

Comments: In white-tailed deer, documentation and characterization of so-called toothpaste hair disease is limited, although the condition is well-known. There is a report of a similarly affected 4-month-old fawn from South Dakota, which was diagnosed as hypotrichosis⁵. In our case, a diagnosis of follicular dysplasia was favored based on the adequate number of hair follicles, follicular distortion, and infundibular hyperkeratosis. In the existing literature regarding follicular diseases, the distinction between congenital hypotrichosis and follicular dysplasia ranges from murky to nonexistent.^{1,2} Follicular dysplasia is generally defined as hair follicles with an irregular contour and thin hair shafts¹. In described cases of both follicular dysplasia and hypotrichosis, the epidermis and dermis are spared of inflammation.¹ Infundibular hyperkeratosis, follicle atrophy or a telogen-state of follicles, and variable distortion of the follicle are more reliably described findings in follicular dysplasia.^{2,3} Pigmentary incontinence is variable.¹ Hypotrichosis can be genetic or have non-genetic causes, including iodine deficiency, adeno-hypophyseal hypoplasia and pestiviral infections such as bovine viral diarrhea virus.² Congenital hypotrichosis is characterized by lack of hair or hair that is lost in the first month of life.¹ In hairless breeds of dogs and cattle, it can also be associated with other defects such as brachygnathism, anodontia, and thymic and genital abnormalities.^{1,2} The lack of reports of similar defects in white-tailed deer is likely influenced by the viability of affected animals in the wild.

References:

1. Gross, T. L., Ihrke, P. J., Walder, E. J., & Affolter, V. K. (2008). Skin diseases of the dog and cat: clinical and histopathologic diagnosis. John Wiley & Sons.
2. Mauldin, E. A., & Peters-Kennedy, J. (2016). Integumentary system. In Jubb, Kennedy & Palmer's Pathology of Domestic Animals: Volume 1 (pp. 509-736). WB Saunders.
3. Mecklenburg, L. (2006). An overview on congenital alopecia in domestic animals. *Veterinary Dermatology*, 17(6), 393-410.
4. Mecklenburg, L., Linek, M., & Tobin, D. J. (Eds.). (2009). Hair loss disorders in domestic animals. John Wiley & Sons.
5. Zimmerman, T. J., Jenks, J. A., Holler, L. D., Jacques, C. N., & Morlock, W. W. (2004). Congenital hypotrichosis in a white-tailed deer fawn from South Dakota. *Journal of Wildlife Diseases*, 40(1), 145-149.

Case # 2

Halogeton Poisoning in a Sheep Flock

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Signalment: Two, 3-year-old, Merino ewes.

History: The ewes were submitted to the Utah Veterinary Diagnostic Laboratory for necropsy to investigate mortality in a flock grazing in central Utah in late February 2019. The owner suspected toxic plant exposure and submitted plant samples from the pasture for identification.

Gross Necropsy Findings: Both ewes were in good body condition and each was pregnant with a single fetus. Plants found in the pasture and in the rumen content had red to purple tinged stems branching from the base, and green, fleshy, tubular leaves with round edges and single spines on the tips. The lungs were congested.

Histopathologic Findings: Both ewes had a diffuse suppurative rumenitis with vacuolation of the epithelial cells. Numerous, 5 to 10 um in diameter, rhomboid, angular, or prismatic, birefringent crystals compatible with oxalate crystals were within the ruminal epithelium and submucosa, and renal tubules.

Final Diagnosis: A diagnosis of halogeton poisoning was made upon identification of the plant in the pasture and in the rumen content as *Halogeton glomeratus*, and presence of numerous oxalate crystals in histological sections of rumen and kidneys.

Comments: *Halogeton glomeratus* is an invasive, noxious, and poisonous weed native to Russia and China found across the western United States. This plant typically invades disturbed sites such as around railroad loading sites, trail heads, stock trails, and water holes, especially where native plant cover is thin. The toxic compounds are sodium and potassium oxalates. Upon ingestion, soluble oxalates are absorbed in the rumen and bind to circulating calcium to create insoluble calcium oxalate crystals that precipitate in tissues, blood, and renal tubules. Death can result from a combination of rapid hypocalcemia, disruption of the Krebs cycle, and acute renal failure. Ingestion of as little as 300 g of the plant can cause death in an adult sheep. Deaths usually occur in fall and early winter when the plant's oxalate content is higher and the plant is more palatable. Underfed sheep are more susceptible as they are more likely to ingest halogeton and lower amounts of oxalates are lethal compared to well-fed sheep grazing nutrient forage. Poisoning can also occur in cattle after periods of prolonged food deprivation. Although infrequent, halogeton poisoning can cause severe outbreaks with reports of up to 1200 sheep and dozens of cattle poisoned in a single event. This case provides an example of halogeton toxicosis with suppurative rumenitis and numerous characteristic oxalate crystals. Differential diagnoses should include ingestion of other high oxalate-containing plants (e.g. *Rheum rhaponticum*, *Sarcobatus vermiculatus*, *Rumex spp.*, etc.), and feedstuffs contaminated with *Aspergillus niger* and *A. flavus*. Collection of a thorough history and identification of the ingested plant are important for a definitive diagnosis.

References:

1. Panter KE, Welch KD, Gardner DR, Lee ST, Green BT, Pfister JA, Cook D, Davis TZ, Stegelmeier BL. Poisonous Plants of the United States. In: Gupta RC, ed. *Veterinary Toxicology*. 3rd ed. London UK: Academic Press, Elsevier; 2018: 837-889.
2. Rood KA, Panter KE, Gardner DR, Stegelmeier BL, Hall JO. Halogeton (*H. glomeratus*) Poisoning in Cattle: Case Report. *Int. J. Pharmacognosy and Phytochem. Res.* 2014; 3: 23-25.
3. Cianciolo RE and Mohr FC. Urinary System. In: Maxie MG, ed. Jubb, Kennedy & Palmer's *Pathology of Domestic Animals*. 6th ed., St. Louis, MO: Elsevier; 2016: Vol. 2 425-426.

Case #3

Atypical Interstitial Pneumonia in Weaned Calves*

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Signalment: Spring weaned calf (approximately 6 months old)

History: Calves weaned and vaccinated (Pyramid 5 plus Prespense and Vision somnus) 1 month prior to acute onset of illness with 3 head dead. The animals were treated with tulathromycin and flunixin meglumine. Formalin fixed lung was submitted for histologic evaluation.

Histopathologic Findings: Affecting 100% of the examined section of lung there is alveolar septal necrosis with replacement by thick hyaline membranes, which often surround increased numbers of alveolar macrophages, occasional multinucleate cells, cellular debris, and multifocal hemorrhage. There is frequent loss of type I pneumocytes and hyperplasia of type II pneumocytes throughout the tissue. Interlobular septae are expanded by dilated lymphatics and edema. Bronchi and bronchioles contain a small number of neutrophils.

Morphologic Diagnosis: Lung: Interstitial pneumonia, fibrinonecrotizing, diffuse with hyaline membrane formation and type II pneumocyte hyperplasia, subacute.

Ancillary Testing: Bovine Respiratory PCR Panel.

Agent:	BVDV	BHV-1		BRSV	M bovis	Bov. Corona V
Result:	Neg	Neg		CT: 27.64	Neg	Neg

Agent:	Influ. D Virus	M. haemolytica		P. multocida	H. somni	B. trehalosi
Result:	CT: 36.11	CT: 34.71		Neg	Neg	Neg

Comments: The histologic lesions present are consistent with the condition known as atypical interstitial pneumonia. This condition is also referred to as acute respiratory disease syndrome because the etiologic agent is typically unknown. Factors associated with this condition include dietary changes, environment, and concurrent infections (bacterial or viral). The positive BRSV PCR (CT of 27.64) supports a BRSV infection as opposed to vaccine interference, but the examined sections lacked characteristic lesions and IHC for BRSV was negative. Bacterial pathogens were not visualized on histopathology, but PCR revealed a weak positive for *M. haemolytica*. The history accompanying the samples indicated antimicrobial therapy was utilized making the interpretation of microbial data challenging. Influenza D Virus (IDV) causes a mild rhinotracheitis in cattle and was also a weak positive on PCR. The inflammatory response associated with IDV could potentially enable a secondary infection with an opportunistic pathogen.

References:

1. Bingham HR, et al. Synergistic effects of concurrent challenge with bovine respiratory syncytial virus and 3-methylindole in calves. American Journal of Veterinary Research 1999;60:563-570.
2. Caswell JL, et al. Respiratory System. In Maxie MG, ed. Jubb, Kennedy and Palmer's Pathology of Domestic Animals. 6th ed. Vol 2. St. Louis, MO: Elsevier, 2016:465-591.
3. Doster AR. Bovine Atypical Interstitial Pneumonia. Veterinary Clinics of North America-Food Animal Practice 2010;26:395-407.
4. Ferguson L, et al. Pathogenesis of Influenza D Virus in Cattle. Journal of Virology 2016;90:5636-5642.
5. Woolums AR, et al. Microbiologic findings in feedlot cattle with acute interstitial pneumonia. American Journal of Veterinary Research 2004;65:1525-1532.

Case # 4

Rickets in a Calf

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Signalment: Four-month-old Holstein steer.

History: Three calves suddenly were down and could not get up. The calves would still eat and drink while laying down.

Gross Necropsy Findings: The cortices of the proximal and distal portions of long bones were markedly thinned to approximately 1mm at the narrowest point, and there were decreased numbers of secondary trabeculae throughout the metaphyses and diaphyses of these bones. There was a spiral fracture of the right femur, a comminuted fracture of the left femur, and an incomplete fracture on the medial side of the left humerus with impaction of the cortical bone laterally and proximally into the metaphyseal trabecular bone. All fractures were surrounded by hemorrhage. The ribs were pliable, and there was flaring of the costochondral junctions. The largest costochondral junction was approximately 3cm x 3cm x 3cm. On sagittal section of one rib, there was a tongue of cartilage extending into the metaphysis.

Histopathologic Findings: In a section of rib at the costochondral junction, the zone of hypertrophy of the physis is markedly expanded causing flaring of the cartilage at the edges of the physis. There was regional elongation of a broad cartilaginous tongue into the metaphysis. The chondrocytes in this zone are arranged in haphazard cords that are occasionally separated by large clear cavitations that are lined by hypereosinophilic necrotic debris and occasionally contain myxomatous material. There is marked variation in the size of chondrocytes and lacunae within this zone. Within the metaphyseal bone just distal to the physis, there is a moderate amount of hemorrhage, and trabeculae are markedly decreased in number and surrounded by fibrosis. The trabeculae often have a moth-eaten appearance. There are rare osteoblasts around trabeculae in this region. Numerous trabeculae have a central core of cartilage extending into the metaphysis. Multifocally the trabeculae are collapsed together and fragmented (microfractures).

Laboratory Findings: Serum Calcium: 6.3 [8.8-10.4] mg/dL

Final Diagnosis: Osteopenia and failure of endochondral ossification consistent with rickets

Comments: Rickets is characterized by abnormal endochondral ossification at growth plates in young growing animals. Deficiencies in vitamin D or phosphorus are common causes in cattle. This farm and a neighboring farm both had several symptomatic calves. This farmer was not working with a nutritionist, so dietary imbalances and lack of vitamin D due to indoor housing are suspected. Enlargement of costochondral junctions is a commonly observed gross lesion known as a "rachitic rosary." Histologically, the zone of hypertrophy in the physis will be expanded resulting in an irregularly thickened physis with tongues of cartilage extending into the metaphysis.

References:

1. Dittmer KE, Thompson KG. Vitamin D metabolism and rickets in domestic animals: A review. *Vet Pathol.* 2011; 48:389-407.
2. Craig LE, Dittmer KE, Thompson KG. Bones and Joints in *Jubb & Kennedy's Pathology of Domestic Animals, Volume 1.* 6th Ed. St Louis, Missouri. Elsevier. 2016. 16-163.

Case # 5

Acute African swine fever virus cholangiohepatitis in domestic swine

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Signalment: Eight-week-old pig

History: An 8-week-old pig died of acute fever, malaise, and inappetence. Multiple pigs were sick or dead.

Gross Necropsy Findings: Gross lesions included mild to severe pulmonary edema, hemorrhage and edema of lymph nodes, and marked splenic enlargement. The liver was grossly swollen and markedly congested with edematous to hemorrhagic cholangiohepatitis which extended to involve the entire surface of the gall bladder, resulting in marked thickening of the wall with edema and hemorrhage.

Histopathologic Findings: Liver (Scanned Slide#66). Hepatic lesions were quite variable and ranged from low number of mononuclear infiltrates in the portal tracts to severe eosinophilic to non-suppurative periportal hepatitis with edema, congestion, hemorrhage and necrosis. Portal triads were commonly disrupted and infiltrated with edema, extravasated erythrocytes, and necrotic cellular debris. There is necrosis and loss of the bile duct epithelium and thrombi occur in necrotic vessels (fibrin necrotic vasculitis). Severe hepatic congestion was accompanied by moderate mid-zonal hepatocyte necrosis characterized by cytoplasmic swelling, hypereosinophilia and fragmentation accompanied by nuclear karyorrhexis. Small lakes of coccoid bacteria were present free within sinusoids and the lumen of small vessels. Histological lesions observed in lymphoid organs include mild to marked lympholysis, fibrinonecrotic vasculitis, and hemorrhage and edema within the lymph nodes, tonsil, and spleen. Histologically, the lung had varying degrees of pulmonary edema, congestion and hemorrhage accompanied by fibrinous to histiocytic alveolitis and interstitial pneumonia.

Ancillary Testing: Whole blood and fresh tissues were tested with qPCR for ASFv p72 gene(1). A protocol was developed and validated to testing of formalin fixed and formalin fixed and paraffin embedded tissues. All PCR testing demonstrated high levels of ASFv in the blood, lymph nodes, liver and other tissues from this animal and others within the group.

Diagnosis: Moderate non-suppurative, eosinophilic hemorrhagic cholangiohepatitis with vasculitis and lymphoid necrosis (mandibular, hepatic, renal, and mesenteric lymph nodes, spleen and tonsil)(2) consistent with acute African Swine Fever.

Comments: This pig was one of 10 pigs experimentally infected with a moderate dose of 360 HA units of ASFv genotype 2 virus (Armenia 2007 strain) as part of an experimental study to assess the virulence of ASFv in US domestic swine and to establish a challenge model for ASFv vaccine efficacy studies. The challenge virus strain is the one that escaped out of Africa in 2007 and had spread through Transcaucasia, the Russian Republic, and into Eastern Europe and China where it is now endemic. This study was performed in BSL3ag facilities at the Biosecurity Research Institute, Manhattan Kansas. Although hemorrhagic lymph nodes are well known in ASFv infection, their appearance can be quite variable dependent on duration of infection stain and dose. Hepatic lesions are less well described and can be commonly overlooked particularly due to the presence of eosinophils and secondary bacterial septicemia. Differential diagnoses include other viral (CSFv) or bacterial septicemia.

References:

1. Zsak L, Borca MV, Risatti GR, Zsak A, French RA, Lu Z, Kutish GF, Neilan JG, Callahan JD, Nelson WM, Rock DL. 2005. Preclinical Diagnosis of African Swine Fever in Contact-Exposed Swine by a Real-Time PCR Assay. 43:112-119.
2. Sunwoo S-Y, Pérez-Núñez D, Morozov I, Sánchez EG, Gaudreault NN, Trujillo JD, Mur L, Nogal M, Madden D, Urbaniak K, Kim IJ, Ma W, Revilla Y, Richt JA. 2019. DNA-Protein Vaccination Strategy Does Not Protect from Challenge with African Swine Fever Virus Armenia 2007 Strain. Vaccines 7:12.

Case # 6

Pituitary tumor in a Budgerigar

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Signalment: Two year-old adult male budgerigar.

History: The budgerigar was euthanized after being found on the ground in an exhibit at Zoo Knoxville, dyspneic, and in poor body condition (2/5).

Gross Necropsy Findings: Within the ventral right peribulbar area, hypothalamus, and facial muscles is a multinodular mass ranging from 3x2x2mm to 6mm diameter that is homogenously pale tan to white, soft, and compresses the surrounding glands, musculature, and neuropil.

Histopathologic Findings: Infiltrating the cranial vault, bone medullary cavities, cerebral neuropil and surrounding musculature is a multinodular, unencapsulated, moderately well-demarcated, compressive neoplasm composed of vague packets and sheets of round to polygonal cells within a moderate fibrovascular stroma. Neoplastic cells have variably distinct cell borders, moderate amounts of pale eosinophilic to clear cytoplasm, a round nucleus with finely stippled chromatin and indistinct nucleoli. There is one mitotic figure per ten 400x fields. Anisocytosis and anisokaryosis are marked, with up to 8x size variations in neoplastic cells. There are frequent intranuclear cytoplasmic invaginations within the karyomegalic cells. Within the tumor are multifocal areas of eosinophilic and karyorrhectic debris (necrosis), and fibrin. The surrounding neuropil is mildly vacuolated. Bone medullary cavities are foamy macrophages and pale eosinophilic fluid. An island of neoplastic cells is within a vascular lumen.

Additional Findings: Immunohistochemistry was performed with a rabbit polyclonal anti-growth hormone antibody. Positive canine and horse controls and negative budgerigar controls stained appropriately. Approximately 75% of neoplastic cells have strong cytoplasmic granular staining.

Final Diagnosis: Pituitary carcinoma with intramedullary and intravascular invasion.

Comment: Pituitary tumors are common intracranial neoplasms reported in budgerigars that frequently stain immunohistochemically positive for growth hormone (somatotroph), similar to cats. The neoplastic cells often have marked anisocytosis and anisokaryosis with intranuclear cytoplasmic invaginations and invasion into the surrounding bone medullary cavities and neuropil, as seen in this case. Additionally, metastasis to the liver has been reported. Criteria to differentiate malignant and benign pituitary neoplasms have not been described in budgerigars, but in mammals metastasis is the only differentiating factor to determine malignancy. Since there is extensive local invasion and neoplastic cells within the vasculature, this is consistent with a pituitary carcinoma. In a recent survey at the University of Tennessee of 15 budgerigars determined to have died from other causes, 4 had undiagnosed pituitary tumors. Therefore, trimming budgerigar heads is important to assess all contributing factors to death.

References:

Langohr, I. M., M. M. Garner, and M. Kiupel. "Somatotroph pituitary tumors in budgerigars (*Melopsittacus undulatus*)." *Veterinary pathology* 49.3 (2012): 503-507.

Case # 7

Disseminated squirrel fibromatosis

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SIGNALMENT: Reportedly eight-week-old, suspect eastern gray squirrel (*Sciurus carolinensis*)

History: Found abandoned by the side of the road, the squirrel died while being cared for at a rehabilitation facility and was submitted to the Athens Veterinary Diagnostic Laboratory (AVDL) for necropsy.

Gross Necropsy Findings: Externally, coalescing along the ventral flanks, distal limbs, periocular regions, face, and prepuce were pale tan, firm, non-haired cutaneous nodules. Cutaneous nodules were multifocally covered in thick, dark brown crusts, with occasional thick, pale yellow discharge. Along the chin were areas of alopecia with dark brown crusts which oozed a red, serosanguinous fluid. Disseminated throughout all lung lobes were multifocal, 0.3 cm³ – 0.5 cm³, pale tan, soft, coalescing nodules that extended into the parenchyma.

Histopathologic Findings: The visceral pleura of the lungs was elevated, and the parenchyma replaced by densely cellular, well-demarcated, nodules, which multifocally replaced the parenchyma. Nodules consisted of a moderately cellular mesenchymal neoplastic population. Neoplastic cells had indistinct cell borders and contain moderate, eosinophilic, granular cytoplasm with a round to oval nucleus with finely stippled chromatin and one prominent nucleolus. Anisocytosis and anisokaryosis were mild to moderate. Mitoses were infrequent with less than one mitotic figure per single, 400X HPF. Within the mesenchymal population there were large, 5-15 um in diameter, discrete, eosinophilic, intracytoplasmic inclusion bodies. Surrounding neoplastic nodules, type II pneumocytes were moderately to markedly increased, lining alveoli and characterized by abundant, eosinophilic cytoplasm. Adjacent alveoli were filled with edema, foamy macrophages, and few erythrocytes. Similar mesenchymal nodules expanded the skin, kidney, testicle, lymph nodes, and spleen.

Final Diagnosis: Mesenchymal proliferation with intracytoplasmic inclusion bodies, pulmonary edema, and type II pneumocyte hyperplasia

Comments: Squirrel fibromatosis is caused by a poxvirus, genus *Leporipoxvirus*, known as squirrel fibroma virus. Squirrel fibroma virus and rabbit fibroma virus (Shope fibroma virus) are closely related with immunological cross-reactivity and similar ultrastructural characteristics. This viral disease is known to affect western gray squirrels (*Sciurus griseus*), eastern gray squirrels (*Sciurus carolinensis*), and American red squirrels (*Tamiasciurus hudsonius*), with presumptive infections in fox squirrels (*Sciurus niger*). Squirrel fibromatosis most commonly develops in a cutaneous form, however it can progress to a fatal disseminated form with visceral nodules. Squirrel flea (*Orchopeus howardi*) and mosquitoes (*Aedes aegypti* and *Anopheles quadrimaculatus*) are suspected to be vectors capable of transmitting disease. The pulmonary form of this disease may be due to intratracheal inoculation or viremia.

Case # 8

Disseminated infection with *Mycobacterium avium* subsp. *hominissuis* in a dog

Jeff Hayes, Beverly Byrum

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Signalment 3.5-year old spayed female Chihuahua cross bred dog.

History: The dog presented for boarding, and the owner requested examination of a lump on the neck. A fine needle aspirate of the right submandibular lymph node was submitted for cytologic examination, which was reported as marked macrophage inflammation with myriad intra- and extracellular non-staining bacteria supportive of *Mycobacterium* sp. infection. The dog bit a clinic employee, and was then euthanized at the request of the owners. The referring veterinarian submitted the head for rabies testing, and also submitted chilled and formalin-fixed tissues to the ADDL.

Gross Necropsy Findings: The mandibular and mesenteric lymph nodes were enlarged and firm. The spleen was enlarged, pale pink, had a dense meaty texture, and contained mottled pale yellow areas.

Histopathologic Findings: Submandibular and mesenteric lymph node: All six sections contain sheets of vast numbers of large epithelioid macrophages with abundant eosinophilic cytoplasm and eccentrically placed round to oval vesicular nuclei efface the normal architecture of the nodes such that remnants of scattered lymphoid follicles are the only recognizable remaining architectural features. Acid-fast staining of all lymph node sections reveals very large numbers of slender acid-fast bacilli within the cytoplasm of virtually all macrophages, not only of those within the parenchyma but also in macrophages extending into mesenteric adipose tissue adjacent to mesenteric lymph node sections. Occasionally extra-cytoplasmic acid-fast bacilli are noted. Similar findings are observed in spleen sections, and two focal accumulations of epithelioid macrophages admixed with lymphocytes are present within and focally expand subpleural connective tissue in a section of lung. Acid-fast bacilli are present in macrophages in spleen and lung.

Final Diagnosis: Lymph node, mesenteric: 1) Severe diffuse granulomatous lymphadenitis with myriad intralésional acid-fast bacilli; 2) Focally severe granulomatous peritonitis, with intralésional acid-fast bacilli

Virology: Brain tissue submitted to the Ohio Department of Health was found to be negative for rabies.

Bacteriology: *Mycobacterium avium* was isolated from a composite of spleen, mesenteric and mandibular lymph node by the National Veterinary Services Laboratories. The isolate was sequenced at the 16S rRNA and the rpoB regions of the genome and results were blasted against GenBank. Based on rpoB sequence, the isolate matched the 104 type strain of *M. avium* with 100% identity.

Comments: The reference strain *Mycobacterium avium* 104 has been previously classified as *Mycobacterium avium* subsp. *hominissuis* (Mijs et al., 2002). This organism is an opportunistic, environmental pathogen that can cause disease in domestic dogs, swine, humans, horses, captive and free-ranging red deer. *Mycobacterium avium* as a genus has been recovered from the environment, including soil, water, dust, and air, as well as from different host species and body sites. Based on differences in genotypic and growth characteristics, pathogenicity, and host range, *M. avium* species is subdivided into four subspecies, namely, *M. a. avium*, *M. a. hominissuis*, *M. a. silvaticum*, and *M. a. paratuberculosis*.

References:

1. 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.

2. 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.

Case # 9

Cryptosporidiosis and Distemper in a raccoon

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Signalment: Juvenile raccoon

History: Juvenile raccoon with history of upper respiratory signs, hyporexia and weight loss.

Gross Necropsy Findings: The carcass was in fair nutritional condition, with a small amount of fat reserves, but still well fleshed. Bilaterally, the lungs were diffusely, moderately wet, firm white to cream-colored. The lumen of the distal portion of the trachea and main bronchi contained soft to friable, yellow material. The perianal region fur was matted with light yellow fecal material. Minimal content was found in the small intestine, and the large intestine contained small amounts of yellow-green pasty content.

Histopathologic Findings: Diffusely, the lamina propria of the small intestine is infiltrated by moderate numbers of eosinophils, and lesser numbers of lymphocytes and plasma cells. Intestinal villi are markedly shortened (villus blunting). Lining the apical border of the intestinal epithelium are numerous multiple, 5-10 um round, basophilic structures (morphology compatible with *Cryptosporidium* sp.). The crypt epithelium is either attenuated or necrotic, and in the crypt lumen there are dead cells and cell debris (crypt necrosis).

Final Diagnosis: Enteritis, eosinophilic, mild to moderate, diffuse, with moderate villous atrophy and severe crypt necrosis, with intralesional *Cryptosporidium* sp.

Comments: *Cryptosporidium* sp. was detected by PCR targeting the 18S rRNA gene on mucosal scrapes of the small intestine of this raccoon. The sequenced amplicon clustered with the *Cryptosporidium* skunk genotype. Fecal smears stained with modified acid fast showed rare numbers of *Cryptosporidium* spp. ELISA on small intestine content was positive for *Cryptosporidium* spp. Transmission electron microscopy demonstrated small rounded protozoa compatible with *Cryptosporidium* spp. No parasites or parasite eggs were detected by fecal float. Immunohistochemistry for canine distemper virus (CDV) revealed strong immunolabelling of the crypt epithelium. Canine parvovirus (CPV) immunohistochemistry was negative. It is possible that these changes were produced by *Cryptosporidium* infection as previously reported, or that the crypt changes were associated with concomitant distemper virus infection. CDV is known to be endemic in some North American free-ranging raccoon populations, causing regular epidemics. Juvenile animals are particularly susceptible to CDV infection, and may develop lifelong immunity if overcoming the infection. It is possible that the profound immunosuppression caused by CDV makes raccoons (especially young individuals) vulnerable to be colonized by moderate to large numbers of cryptosporidia, exacerbating gastrointestinal signs.

Case # 10

Necrotizing colitis in a horse

Mauricio A. Navarro; Akinyi C. Nyaoke; Francisco A. Uzal

California Animal Health and Food Safety laboratory, UC Davis, San Bernardino Branch, California.

Signalment: 7-year-old Mustang horse

History: acute colic and fever of unknown origin. The animal was given IV fluids but went into cardiac arrest and died.

Gross Necropsy Findings: The carcass was in good nutritional condition, well-muscled, with adequate amount of fat reserves and moderately dehydrated. The distal half of the small intestine had a diffusely dark red serosa, while the proximal half presented irregular, large multi-segmental areas of red serosa. Throughout the whole length of the small intestine there was transmural hemorrhage and the wall was severely thickened. The mucosa of the whole small intestine was grey, dull and velvety, and presented multifocal areas covered by a thin fibrinous pseudomembrane. There was a moderate amount of dark red fluid content in the lumen of the small intestine. The cecum and the right and left ventral large colon presented diffusely and severely edematous mucosa and many randomly distributed small (~1-2 cm) foci of mucosal necrosis. The content of cecum, colon and rectum was watery, greenish and translucent. There were several sub-endocardial ecchymoses within both ventricles of the heart. These lesions were most severe over the papillary muscles of the left side.

Histopathologic Findings: Colon; diffusely, the intestinal wall is edematous and hemorrhagic. The mucosa is diffusely necrotic, with loss of superficial epithelium and lamina propria, and it is infiltrated by lymphocytes and plasma cells which separate the colonic crypts. There is luminal pseudomembrane formation composed of fibrin, sloughed and necrotic epithelium, red blood cells, leukocytes and myriad rods. The mucosal and sub-mucosal blood vessels present fibrin thrombi and multifocal areas of hemorrhages. The sub-mucosa is markedly expanded by homogeneous eosinophilic edema.

Final diagnosis: Colon: colitis, severe, diffuse, acute, necrotizing and pseudomembranous with fibrin thrombosis and severe submucosal edema and hemorrhages.

Comments: Culture of colon content for *Clostridium difficile* was negative. Salmonella culture and PCR were negative on colon tissue. ELISAs for toxins A and B of *C. difficile* and for alpha, beta and epsilon toxins of *Clostridium perfringens*, respectively, were negative. Coronavirus immunohistochemistry on colon tissue was negative. Aerobic and anaerobic culture of colon tissue and content grew large mixed flora, and large numbers of *Clostridium sordellii* were isolated. Large numbers of rods were positively stained for *C. sordellii* immunohistochemistry in sections of affected colon. By PCR, the recovered isolate tested positive for the sordellilysin (*sdl*) gene, but negative for the lethal (*tsl*) and hemorrhagic (*tcsh*) toxin genes of *C. sordellii*.

Given the morphologic diagnosis, location, exclusion of *C. difficile*, *C. perfringens*, *Salmonella spp.*, coronavirus, and the isolation and identification of *C. sordellii* in colonic tissue, a presumptive diagnosis of *C. sordellii*-associated colitis was established. *C. sordellii* is a gram-positive, sporulating rod that may be found in soil and in the gastrointestinal tract of humans and animals. This microorganism has been mainly associated with cases of toxic shock syndrome in humans, secondary to gas gangrene or gynecologic events (e.g. childbirth and abortion), as a small proportion of women may carry this bacterium in the vagina. In animals, *C. sordellii* has been associated with gas gangrene in ruminants, pigs and horses, and omphalitis in foals. A few reports of necrotic enteritis, and abomasitis, have been documented in birds and sheep, respectively. To this date, however, the role of *C. sordellii* in causing intestinal disease of horses is not fully understood, and the disease has not been replicated in animal models. Nevertheless, we suggest adding *C. sordellii* to the list of differential diagnosis of equine colitis, particularly when the most common causes have been excluded.

Case # 11

Right dorsal colitis in a horse Francisco A. Uzal; Patricia Gaffney

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Signalment: Horse

History: Colic of unknown duration which could not be controlled with non-steroidal anti-inflammatory drugs (banamine and others) and was euthanized.

Gross Necropsy Findings: The carcass was in good nutritional condition, well-muscled and with adequate fat reserves. The abdomen contained approximately 1 liter of red, thin, opaque fluid. Affecting three quarters of the right dorsal colon, were the following changes: the serosa was diffusely red to dark red; the wall was transmurally thickened up to 2 cm and dark red and wet; the mucosa was mottled red to dark red to brown with a diffusely corrugated appearance; and the ingesta was semi-liquid green plant material that was red-tinged. There was a sharp line of demarcation between this region and the distal one quarter of the right dorsal colon, which had a slightly less thickened wall and diffusely tan serosa and mucosa. There was extensive petechiation of the epicardial surfaces of the atria and ventricles of the heart. No other significant gross abnormalities were observed in the rest of the carcass.

Histopathologic Findings: Colon; diffusely, there is transmural congestion, edema and hemorrhage. The whole thickness of the mucosa is necrotic and presents lymphoplasmacytic and neutrophilic infiltration. The mucosal and sub-mucosal vasculature presents fibrin thrombi and thrombotic vessels show fibrinoid necrosis and infiltration with viable and degenerate neutrophils. These vessels are frequently surrounded by hemorrhage and viable and degenerate leukocytes. The sub-mucosa is greatly expanded by homogeneous eosinophilic edema and diffuse infiltration of lymphocytes, plasma cells, macrophages and neutrophils. The lymphatic vessels are dilated and contain large amounts of fibrin, red blood cells and neutrophils, both viable and degenerate. Large numbers of mix bacteria admixed with fibrin and cell debris cover the denudated superficial mucosa. The serosal blood vessels are also thrombotic and present fibrinoid necrosis.

Final Diagnosis: Colon, right dorsal: colitis, severe, diffuse, acute, necrotizing with severe fibrinonecrotizing vasculitis, fibrin thrombosis and massive submucosal edema and congestion

Comments: Aerobic and anaerobic culture of colon tissue and content grew large mixed flora; culture of colon content for *Clostridium difficile* was negative. ELISAs for toxins A and B of *C. difficile* and for alpha, beta and epsilon toxins of *Clostridium perfringens*, respectively were negative. Salmonella culture and PCR were negative on colon tissue. Aerobic culture of liver and lung grew small numbers of *Streptococcus equi* ssp. *zooepidemicus* and mixed flora.

Microscopic examination confirmed severe necrotizing right dorsal colitis with extensive vasculitis and many fibrin thrombi. Given the morphologic diagnosis, location, exclusion of *C. difficile*, *C. perfringens* and *Salmonella* spp., and history of banamine administration, this is likely consequence of nonsteroidal anti-inflammatory drugs (NSAIDs) toxicity. NSAIDs cause ulceration of the small intestine and colon in horses and other animals. Because the right dorsal colon is preferentially involved, the condition is usually named “right dorsal colitis”; however, lesions in other parts of the colon and in the small intestine may also occur. The pathogenesis of this condition is associated with decreased production of prostaglandin E₂ and nitric oxide. Decreased prostaglandin is due to NSAID inhibition of cyclooxygenase 2 (COX-2). Morphologically, right dorsal colitis cannot be differentiated from some of the most common infectious colitis of horses (e.g. *C. difficile*, *C. perfringens* and *Salmonella* spp. infections). There are no specific tests to confirm the diagnosis of NSAID’s toxicity, and the diagnosis should therefore be based on a history of NSAIDs administration and ruling out infectious causes of colitis.

Case # 12

Amoebic infection with pleural involvement in a red-footed tortoise (*Chelonoidis carbonarius*)

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Signalment: Red-footed tortoise (*Chelonoidis carbonarius*), female, unknown age.

History: The tortoise was part of a research colony. Several animals from the colony died and ancillary testing (*Mycoplasma* spp., intranuclear coccidia and ranavirus) was negative. The animal was found dead with blood pooling from the nares without previous evidence of clinical disease.

Gross Necropsy Findings: The coelomic cavity had a moderate amount of red-tinged fluid. The left liver lobe had a focally extensive, poorly demarcated, tan to white, friable area coated by a thin layer of fibrin. The spleen was enlarged and friable. The colonic mucosa was focally coated by a thin layer of fibrin. The lungs were diffusely reddened, and the left lung was coated by a thick layer of fibrin.

Histopathologic Findings: The visceral pleura of the left lung was effaced by extensive liquefactive necrosis characterized by abundant hypereosinophilic and karyorrhectic cell debris and fibrin exudation mixed with numerous degranulating heterophils and delimited by abundant histiocytes and multinucleated giant cells. The affected areas contained numerous intralesional colonies of rod-shaped bacteria and amoebic organisms, which measured approximately 20-30 µm in diameter and had a basophilic cytoplasm with occasional clear vacuoles and either a single nucleus (trophozoites) or multiple condensed nuclei (cysts). The cyst walls were highlighted by the PAS reaction. The hepatic parenchyma had multifocal to coalescing areas of coagulative necrosis with abundant fibrin exudation and numerous intralesional amoebic organisms and was delimited by abundant degranulating heterophils and multinucleated giant cells. The remaining parenchyma and ducts contained numerous amoebic organisms, and the hepatic capsule was partially coated by a thick layer of fibrin and degranulating heterophils (not represented in the submitted sections). The superficial colonic mucosa was focally effaced by liquefactive necrosis with occasional amoebic organisms and delimited by numerous degranulating heterophils and multinucleated giant cells (not represented in the submitted sections).

Final Diagnosis: Pleura; pleuritis, fibrinonecrotic and granulomatous, extensive, marked, subacute to chronic, with intralesional amoebic organisms consistent with *Entamoeba invadens*.

Laboratory Results: Infection with *E. invadens* was confirmed by PCR and Sanger sequencing.

Comments: *Entamoeba invadens* is a well-recognized protozoal organism associated with amoebiasis in numerous reptile species, particularly in chelonians. Poor quarantine and hygienic practices constitute an important risk factor, and the infection can spread rapidly within the colony. No zoonotic potential has been recognized thus far for *E. invadens*. Lesions mainly consist of fibrinonecrotic colitis and hepatitis following dissemination of organisms through the portal vasculature or common bile duct. The organisms are readily apparent on H&E stained sections. Trophozoites are characterized by an eccentric nucleus, basophilic cytoplasm and multiple cytoplasmic vacuoles while cyst forms are quadrinucleated and its wall can be highlighted via PAS reaction. *E. invadens* has a direct life cycle and transmission occurs through the fecal-oral route. Following cyst ingestion, invasive trophozoites develop and induce the formation of a fibrinonecrotic pseudomembrane in the colon. Encystation occurs and the infective cysts are shed in the feces. Protozoal (and secondary bacterial) dissemination occurs to the liver (with induction of necrotizing hepatitis) as well as other organs. In this case, the dissemination of trophozoites to the pleura constitutes a less frequent finding.

Case # 13

Listerial Meningoencephalitis and Choroid Plexitis in a Saki Monkey (*Simia pithecia*)

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Signalment: A 27-year-old, intact female, saki monkey.

History: The saki monkey was examined at the Phoenix Zoo for acute lethargy and hyporexia, and was found to be thin, dehydrated, and sensitive on cranial abdominal palpation. Abdominal radiographs revealed mild gas distension in the small and large intestines. Supportive treatments were administered. The day after, she was found deceased after an episode of vomiting and ptyalism.

Gross Necropsy Findings: Postmortem exam detected gas distension of the alimentary tract, aortic arteriosclerosis, a focal, 0.5 cm, firm superficial nodule in the left hemisphere's frontal lobe (fibrosis attributed to previous vascular accident), and the cerebral ventricles contained opaque viscous material.

Histopathologic Findings: Histopathology revealed suppurative ventriculitis, choroid plexitis, periventricular encephalitis, and meningitis with intralesional Gram-positive coccobacilli and diploid rods. There was lytic suppurative hepatitis and mild suppurative enterocolitis with crypt necrosis.

Final Diagnoses:

1. Brain, ventricles, meninges: Ventriculitis, periventricular encephalitis, and meningitis, suppurative, multifocal, severe, acute with intralesional gram-positive bacterial diploid rods.
2. Cervical spinal cord: Meningitis, suppurative, multifocal, marked, acute.
3. Liver: Hepatitis, necrosuppurative, random multifocal, moderate, acute.
4. Colon: Colitis, suppurative, multifocal, mild, acute with crypt necrosis.

Ancillary Diagnostics:

1. PCR positive for *Listeria monocytogenes* on brain
2. Bacteriology on liver and cervical spinal cord yielded moderate to heavy growth of *Listeria monocytogenes*

Comments: This geriatric monkey suffered from fatal hematogenous listerial meningoencephalitis thought to have an intestinal origin. In nonhuman primates, the most common cause of bacterial meningoencephalitis is *Streptococcus pneumoniae*, and less commonly, *Klebsiella pneumoniae*, *Pasteurella multocida*, *Haemophilus influenzae*, and *Neisseria meningitidis*.¹ *Listeria monocytogenes* infections in nonhuman primates are infrequent and mostly involve experimental infections of pregnant dams that cause reproductive failure without clinical signs.^{2,3} Although immunocompromised people are at an increased risk of septic listeriosis, hematogenous listeriosis has been reported in an immunocompetent macaque.² This saki monkey likely ingested environmental listeria that caused disease potentially facilitated by preexisting co-morbidities.

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Case # 14

Nidoviral Pneumonia in a Ball Python (*Python regius*)

Mary Drozd, Sarah Schneider

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Signalment: A 4-year-old, adult, female ball python (*Python regius*).

History: The python was part of a breeding colony used to supply a reptile specialty pet shop. The owner had recently purchased new breeding stock and subsequently approximately 20% of his snakes died following progressive upper respiratory signs.

Gross Necropsy Findings: The snake was in good post-mortem condition and had abundant fat stores and good body musculing. Both eyes had a small focus of congestion on the dorsal iris. The oral cavity contained approximately 0.5 mL of thick, opaque, tan, mucinous fluid.

The carina and main stem bronchi contained approximately 0.3 mL of mucoid fluid similar to the oral cavity. The lungs were wet and diffusely mottled red to dark red. The pericardial sac contained 0.3 mL of serosanguineous fluid. The cranial one-third of the esophagus contained a small amount of mucinous, opaque, tan fluid.

The liver was moderately pale, friable, and mottled tan and red with rounded peripheral margins and multifocal fissures on the dorsal-costal surface (up to 1.4 cm long). The stomach contained a small amount of tan, mucoid fluid. The jejunal mucosa was severely and diffusely thickened and edematous.

Histopathologic Findings: The bronchial epithelium has frequent irregular piling of epithelial cells (hyperplasia) as well as multifocal erosions of the bronchi epithelium. The foveolar epithelium has extensive erosion and ulceration; the lumen is frequently occluded by a fibrinous exudate containing clumps of bacteria, heterophils and cellular debris. The air capillary interstitium is moderately to severely expanded by heterophils, histiocytes, edema, fibrin, and degenerate lymphocytes and plasma cells.

Final Diagnosis: Lungs: Proliferative, interstitial pneumonia with necrosuppurative exudate containing mixed bacteria.

Comments: Three RNA viruses most commonly cause proliferative, interstitial pneumonia in snakes: nidovirus, ophidian paramyxovirus and reptilian orthoreoviruses. Reoviral pneumonia and Paramyxovirus may be associated with respiratory epithelial synechia, which were not observed in this case. Paramyxovirus and nidovirus are commonly associated with necroexudative airway occlusion. Both nidovirus and paramyxovirus cause proliferative and ulcerative upper respiratory disease as well as proliferative enteritis. In some cases, paramyxoviruses can be differentiated by viral inclusion in infected neurons, but lack of inclusions (such as in this case) does not rule out paramyxovirus infection. Rt-PCR testing of lung tissue performed at the University of Florida diagnostic lab was positive for nidovirus and negative for ophidian paramyxovirus.

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Case # 15

Calf Diphtheria: A Fresh Look at a Classic Disease

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Signalment: This case was a 6-week old, Holstein steer

History: This calf presented with open mouth breathing, honking noises on inspiration, and fever. At the Michigan State University Veterinary Teaching Hospital it had malodorous purulent discharge in its trachea, and a severely edematous larynx.

Gross Necropsy Findings: Tonsils were bilaterally enlarged and dark red. The laryngeal mucosa was edematous and hyperemic, with a white fibrinous coating. Caseous material was present within the lateral vocal folds, and the tracheal lumen contained white fibrinous material. Cranioventral lungs were reddened, edematous and consolidated.

Histopathologic Findings: Sections of larynx had erosion of the superficial epithelial covering, and the skeletal muscle and cartilage were infiltrated by granulation tissue, and dense mixed inflammatory infiltrates. The arytenoid cartilage was deformed, necrotic and sequestered while surrounded by caseogranulomatous inflammation.

Final Diagnosis: Chronic-active caseogranulomatous laryngitis with fibrosis, cartilage necrosis and sequestration, consistent with calf diphtheria

Microbiology: Anaerobic bacterial cultures revealed numerous *Fusobacterium necrophorum*, numerous *Porphyromonas levii*, and moderate *Prevotella* species. Aerobic isolates included moderate *Trueperella pyogenes*, and moderate *Helcococcus* species.

Comments: In my experience calf diphtheria or necrotic laryngitis is not as common as it was decades ago. But this case manifests many of the features of advanced chronic infection with *Fusobacterium necrophorum*. This organism is commonly present in the oral cavity, gastrointestinal tract, and the environment, and opportunistically invades oral and laryngeal tissues when an ulcerative lesion occurs. A variety of toxins including endotoxin, leukotoxin, hemolysin and adhesin have been implicated as virulence factors for this organism. In addition, it is most commonly encountered as part of mixed anaerobic and aerobic bacterial infections. In this case, infection by *Porphyromonas*, *Prevotella* and *Trueperella* are contributing organisms, which often co-infect cattle oral and foot lesions in association with *F. necrophorum*. Other manifestations in cattle associated with these mixed infections include hepatic abscesses, periodontitis and foot rot.

References:

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Case # 16

Bovine Abortion Caused by *Candida parapsilosis*

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Signalment: A 6-month-old female Holstein cow fetus.

History: The fetus and its placenta were autopsied after being aborted from a multiparous cow. Gross changes were detected only in the intercotyledonary areas, which were tan, opaque, and thickened.

Histopathologic Findings: There are clusters of fibrin and necrotic cellular debris were scattered throughout the intercotyledonary stroma, and less frequently at the base of caruncles. Intralesional clusters of 3-5 µm diameter, periodic acid-Schiff- and Gomori-methenamine-silver-positive pseudohyphae were present extracellularly or within the cytoplasm of binucleated trophoblasts. The chorion was edematous and had low numbers of macrophages and neutrophils dispersed throughout. Necrotic cellular debris, meconium, and moderate numbers of pseudohyphae were loosely adhered to both the chorionic and amniotic surfaces.

Final Diagnosis: Placentitis, necrotizing with intralesional fungal hyphae.

Laboratory Testing: Fresh samples of lung, liver, and placenta were negative after submitted to a bovine abortion PCR panel (*Neospora caninum*, *Tritrichomonas foetus*, *Brucella* spp., *Campylobacter fetus*, *Campylobacter jejuni*, *Chlamydia* spp., *Leptospira* spp., bovine viral diarrhea virus type 1 and 2, and bovine herpesvirus). Aerobic bacterial culture of abomasal contents yielded light to moderate growth of *Candida parapsilosis*.

Comments: Although not nearly as common as *Aspergillus fumigatus* and other zygomycetes, a small number of bovine abortions are associated with *C. parapsilosis*. The route of infection is unknown but lesions in the placentomes suggest a hematogenous mechanism. In cases with mild placentitis, the typical fetal cutaneous lesions may be absent, but fungal organisms are often isolated from gastric contents, as in the current case. This case highlights a rare cause of bovine mycotic abortion and the importance of a multimodal approach to diagnosing abortion in ruminants.