51st AAVLD Diagnostic Pathology Slide Session

American Association of Veterinary Laboratory Diagnosticians

Greensboro, North Carolina
Saturday, October 25, 2008
3:30-6:00 PM

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Session Coordinators: Jose Antonio Ramos-Vara, Purdue, and Gayle C. Johnson, University of Missouri

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Case/Page #	Presenter	Species	Diagnosis
1	Uneeda Bryant	equine	Interventricular septal mass
2	A. Stern	snake	Metastatic carcinoma
3	Gayle Johnson	bovine	Osteopetrosis
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5	Dale Miskimins	mink	Staphylococcal granuloma
6.	S. D. Cramer	feline	Tularemia
7	Rob Bildfell	cervid	Deerpox infection
8	Grant Burcham	bovine	Hypotrichosis and frostbite
9	Scott D. Fitzgerald	avian	Ovarian teratoma
10	A. Stern	canine	Testicular blastomycosis
11	Matti Kiupel	canine	Lymphosarcoma
12	Francisco Uzal	caprine	Clostridial colitis
13	Stephen Smith	canine	Protothecal cystitis
14	Floyd Wilson	avian	Bacterial meningoencephalitis
15	S.F.E. Scholes	porcine	Cerebellar hemorrhage
16	Alexander Loretti	avian	Schistosomal encephalitis
17	H. L. Shivaprasad	avian	*Hemoproteus* myositis
18	H. L. Shivaprasad	avian	Hepatic necrosis
19	Jose A. Ramos-Vara	equine	Multifocal pulmonary fibrosis

Anomalous Interventricular Septal Defect in a Foal

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A full-term, female, Thoroughbred foal from central Kentucky was delivered on the expected due date and presented with respiratory distress immediately after foaling, which was reported to have been uneventful. The foal took a few breaths independently and foam began exuding from the nostrils. Shortly thereafter the foal went into respiratory arrest and died despite implementation of resuscitation. The foal was submitted to the University of Kentucky Livestock Disease Diagnostic Center for necropsy examination. On gross examination the heart was markedly enlarged and rounded. There was a large (approximately 16.5 cm in diameter) bulging, fluctuant structure located in the region of the interventricular septum impinging on the lumens of the left and right ventricles. The structure was cystic, smooth, and appeared to be formed by myocardial tissue. A small (.1mm diameter) opening in the wall of the structure had smooth, rounded, edges and exuded streams of blood within the left ventricular chamber. The cystic structure was completely occluded by clotted and non-clotted blood. The blood was removed and numerous rudimentary papillary muscles, chordae tendineae, trabeculae, and thin irregular valve-like structures were discovered along the inner lining (resembling a ventricular chamber). This cystic structure was devoid of any blood outflow tract. Histologic sections of the wall of the cystic structure were characterized by well-differentiated cardiomyocytes with an inner and outer lining of fibrocollagenous tissue. The fibrocollagenous tissue lining the outer portion of the structure was moderately thickened. Entrapped within the fibrocollagenous tissue lining both surfaces were multiple variably-sized bundles of purkinje fibers which were more developed and frequently observed on the ventricular surface of the cystic structure. Purkinje fibers observed along the inner surface of the cystic structure were fairly rudimentary yet discernible. Gross and histologic characteristics of the cystic structure were suggestive of a non-functional anomalous ventricular chamber. To the author's knowledge this cardiac anomaly has not been previously described.

Metastatic Ductular Adenocarcinoma in a Western Hognose Snake

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A 16 year-old Western Hognose Snake presented to the referring veterinarian with a one week history of a mid-body swelling and regurgitation. Surgical removal of the mass was performed and histology revealed a ductular adenocarcinoma. Four months later, the original mass recurred and a second more caudal mass were observed. The animal was euthanized and was submitted for necropsy examination. Grossly, there were multiple, firm, expansile, well-demarcated, and infiltrative masses observed within the right kidney, large intestine, and liver. Histologically, the normal architecture of the liver, kidney, and large intestine were effaced by the tubules of columnar to flattened neoplastic cells with abundant stroma, consistent with an adenocarcinoma. This is the first report of metastatic ductular adenocarcinoma in a snake. Ductular adenocarcinomas are reported in various snake species but have not been reported to metastasize.

Osteopetrosis in a Herd of Commercial Angus Cattle

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Two badly scavenged calf carcasses presented for necropsy had malformed heads, impacted teeth, and thick, heavy leg bones. The owner related that he had a herd 0f 240 pregnant commercial Angus cattle, most of them 3 years old. Ten animals had already calved and 3 of the calves had been born alive but had failed to stand and nurse. The two present had died 2 and 7 days after birth.

The owner indicated that he had a few calves born with similar signs during the previous calving season. All had undershot jaws. A single red Angus bull had been used as the sole herd semen donor for several years and for natural cover. The vaccination status of the cattle was uncertain, but none of the dams were ill and no abortions had been recorded.

The gross examination of the two calves revealed a similar phenotype in both. There was moderate attenuation of the maxilla, with bowing of the nose, but more severe attenuation of the mandible. The top of the cranium was less domed than normal, and posterior cerebellar compression and herniation was present in both calves. A discontinuity of the ethymoid bone further reduced the anterior cranial capacity. The dental arcade was shorter than normal, and teeth were stacked on top of one another in the mandible. The long bones had less marrow than expected and marrow cavities were reduced.

Histologically, osteoblasts were irregularly present in bones, but the primary spongiosa was retained, containing cartilage cores. Minimal compacted cortical bone was present. Growth plates were normal. Bone modeling was deficient, and osteoclasts were very rare. The marrow cavity was minimal, and space between boney trabeculae contained spindle cells and clusters of plasma cells.

Based on these findings, a diagnosis of osteopetrosis was made. As an inherited disease of cattle, osteopetrosis is autosomal recessive, and is usually lethal. The defect is in osteoclastic remodeling, with a dearth of osteoclasts. Although bones of osteopetrosis patients are thick, they are also brittle and fracture easily. Several types of osteopetrosis in other species are categorized as those with an absence of or malfunction of normal numbers of osteoclasts. The exact defect remains uncertain in Angus cattle.

Other potential causes of osteopetrosis in calves include BVD infection, in which it is attributed to virus-induced osteoclast dysfunction and lead poisoning. In acquired disease, impaired bone remodeling is localized to a band beneath the growth plate.

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Pancreatic Acinar Atrophy in a Golden Retriever

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A reportedly 11-year-old, spayed female, Golden Retriever dog was submitted to Purdue University Animal Disease Diagnostic Laboratory for necropsy. According to the history provided, this dog had lost 3.3 kg (17% body weight) in the previous 7 months. There was no reported history of pancreatitis, diarrhea, or steatorrhea. At necropsy, the dog was emaciated, with prominent boney protuberances including the ribs, ischial tuberosities, dorsal vertebral processes, and the spines of the scapulae. Skin over the ventral abdomen, flanks, perineum, and caudal thighs was alopecic. The dog had adequate subcutaneous and abdominal fat, but marked muscle wasting. Skeletal muscles were thin and pale red-tan with linear white streaks. The pancreas was grossly inapparent. Histologically, there was complete loss of pancreatic acini and replacement by adipose tissue. Remaining pancreatic ducts were aggregated and surrounded by narrow rims of dense fibrous connective tissue. Few islets of Langerhans, ganglia, and lymphocytic aggregates were scattered throughout the adipose tissue. In sections of skeletal muscle, there was moderate variation in myofiber diameter with many small, angular myofibers. The perimysium was expanded by adipose tissue and moderate collagen. Based on the marked loss of pancreatic acini, replacement by adipose tissue rather than fibrosis, and historical absence of pancreatitis, pancreatic lesions were most consistent with pancreatic acinar atrophy. Pancreatic acinar atrophy is most common in juvenile German Shepherds and Rough-Coated Collies. Clinical signs include diarrhea, steatorrhea, progressive weight loss, muscle wasting, and poor hair quality. Early histologic lesions consist of CD8+ T-lymphocyte infiltration, and acinar degeneration and necrosis, suggesting cell-mediated autoimmunity. This case is unique in the late onset of clinical disease, non-characteristic breed, and reported lack of steatorrhea.

Staphylococcal Granuloma of Mink

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Staphylococcal granuloma, or botryomycosis, is seen in numerous animal species, including mice, horses, pigs, and humans. The condition has not been described in mink. Six adult, female mink were submitted for examination with ulcerated lesions measuring 3-7 mm in diameter on the dorsal and ventral surfaces of front and rear paws. Microscopic examination of the lesions revealed ulceration of overlying epidermis and multiple pyogranulomas in the dermis. Bacterial grains (Splendore-Hoeppli effect) were at the center of the lesions surrounded first by a mixture of neutrophils and macrophages, and then by a collar of fibrous connective tissue. Gram positive cocci could be observed in most lesions. Bacterial culture revealed *Staphylococcus intermedius* and / or *Staphylococcus aureus* in affected tissue. Virus isolation was negative on a skin sample from one mink. The owner of the mink reports antibiotic treatment of affected mink may temporarily improve the skin lesions; however, the lesions recur or worsen following cessation of treatment.

Francisella tularensis infection in two cats

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Two dead juvenile domestic shorthair cats were presented for necropsy. The owner reported a history of 7 to 10 out of 30 feral barn cats dying following consumption of a dead rabbit. On gross examination of both cats, the mesenteric lymph nodes were diffusely markedly enlarged, nodular, and mottled white and tan. Within the spleen, there were a dozen, 3 mm diameter, round, red, raised areas that extended into the parenchyma on section. Scattered throughout the liver were dozens of pinpoint, soft, white and red areas. Histologically, there were multifocal to coalescing areas of inflammation and liquefactive necrosis within the lymph nodes, spleen, and liver. These areas were characterized by loss of architecture and replacement by eosinophilic amorphous debris surrounded by small numbers of neutrophils, and macrophages. Differentials based on the histologic changes included infection with *Francisella tularensis* and *Yersinia pestis*. *Francisella tularensis* was detected by immunohistochemistry in the spleen and lymph node, confirming Tularemia in both cats.

Tularemia is a zoonotic disease caused by the Gram negative coccobacillus *Francisella tularensis.* Tularemia has gained attention recently due to its potential use as a bioterrorism agent[3]. It is a United States category A agent and is therefore considered one of the highest security threats to public health[1]. The number of tularemia cases in the United States has decreased over the past century, and most cases currently originate in the southwest[2]. The findings in these cases will be used as a platform to discuss Tularemia infection in general, with emphasis on public health concerns.

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Deerpox Infection in Black-tailed Deer (*Odocoileus hemionis columbianus*)

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This 2 – 3 month old female black tailed deer was group housed with 3 other similarly aged fawns as part of an experimental study. The fawn was collected as an "orphan" from landowner's premises in western Oregon and was in very poor body condition with multifocal areas of alopecia over the trunk when admitted to the study.

Cutaneous lesions became visible on the ventral neck at d12 post-exposure to the group. Over the next 2 days the fawn developed more widespread skin lesions, as well as mucocutaneous and oral lesions. By d14 this animal was depressed and reluctant to eat and was euthanized the next day. Other than plaques and ulcers in the oral cavity, the only internal abnormality detected at necropsy was a 1 cm circular ulcer in the reticular mucosa.

Microscopic changes are typical of poxviral infections, including epithelial cell hyperplasia with hydropic change and the formation of pustules and ulcers. Dermal infiltrates include large numbers of neutrophils but also many lymphohistiocytic cells. Destruction of upper aspect of follicles is common. Vascular changes in the superficial dermis include congestion, hemorrhage, endothelial cell hypertrophy and occasionally intraluminal fibrin thrombi. Intracytoplasmic inclusions are poorly defined in epidermal cells but a few oral keratinocytes contain more condensed eosinophilic inclusions. Deerpox viral infection was confirmed via virus isolation, PCR and serum neutralization tests.

Other fawns in this pen had been successfully infected with deerpox virus (DPV) via intracutaneous (2 fawns) and intravenous (1 fawn) routes and had active lesions at the time this fawn was added to the study. This commingled animal developed the most severe disease but 2 of the others developed locally extensive ulcerative lesions at the site of inoculation, possibly as a consequence of furunculosis and secondary bacterial infections.

The single previous report of DPV-associated disease involved two Wyoming mule deer fawns in which orofacial lesions and keratoconjunctivitis predominated. Unpublished cases of infection in black-tailed deer in California and a single mule deer fawn in Oregon suggest that this virus can also precipitate locally extensive damage to the skin of the neck, without obvious keratoconjuncitivitis or orofacial lesions. The natural mode of transmission of DPV is unknown at this time.

The genome of DPV has been published and phylogenetic analysis suggests it is in a distinct genus, most closely related to Suipox and Capripox viruses. The DPV genome includes many genes capable of influencing the host immune and inflammatory responses.

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Congenital hypotrichosis and frostbite in a neonatal beef calf

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A 2-week-old red Angus heifer calf was submitted to the Animal Disease Diagnostic Laboratory for necropsy. Two calves on the farm, including the one submitted, were recently born without hair. This calf died after a recent bout of freezing temperature.

Grossly, thick, curly hair covered the ventrum, the caudal aspect of the hindlimbs, and the ventral neck. A thin strip of hair covered the dorsal midline, and extended the length of the tail. The tail switch was normal. Hair covered the internal surface of the pinnae and surrounded the calf's eyes. Remaining skin was completely devoid of hair, and was diffusely pink to red. Subcutaneous tissue at the level of the distal hindlimbs was diffusely mottled dark red to black, and expanded with red gelatinous fluid. Areas of discoloration extended 10 cm dorsally from the coronary bands, and were well-demarcated from more normal subcutaneous tissue.

In microscopic sections of hairless skin, hair bulbs were absent. Hair follicles were few, and those present were rudimentary and hypoplastic. Hypoplastic follicles were comprised of shallow epidermial invaginations, lined by keratin, and did not contain hair shafts. A few aggregates of epidermal cells with hyperchromatic nuclei formed small bulb-like structures deep to hypoplastic follicles. Adnexal structures, such as sebaceous and apocrine glands, were present in normal numbers throughout the dermis. Apocrine sweat glands were dilated. In those areas of hairless skin that were grossly hemorrhagic, a thin layer of erythrocytes and fibrin separated epidermis and superficial dermis. Superficial subcutaneous tissue was expanded by clear edema. Extensive foci of hemorrhage dissected fascial planes in the deep dermis and superficial subcutis. In sections of haired skin, hair bulbs and mature hair follicles with hair shafts were evenly-spaced throughout the dermis.

Histologic features were consistent with congenital hypotrichosis, as well as acute changes associated with frostbite. Frostbite produces vasoconstriction to the distal extremities and thrombosis, which results in epidermal-dermal necrosis, separation, and hemorrhage.

Causes of congenital hyotrichosis in cattle include *in utero* exposure to BVD virus, iodine deficiency and congenital goiter, adenohypophyseal dysplasia, or genetic causes. A syndrome of hypotrichosis and adontia has been recognized in male mixed Maine-Anjou-Normandy cross calves, and a hypotrichosis and incisor adontia syndrome has been recognized in Holstein cross calves. Hereford cattle have a congential hypotrichosis, and "rat-tail syndrome," in which tail hair is abnormal, occurs in some Continental breeds crossed with British breeds. Many of these genetic abnormalities are attributed to autosomal recessive traits; however, hypotrichosis in Hereford calves is a dominant trait. Congenital zinc deficiency (lethal trait A 46), in which calves cannot absorb zinc from their diet, causes loss of hair and hypotrichosis.

In this case, BVD virus and any endocrinopathies were ruled out via virology and

histopathology, respectively. Thus, a genetic defect was deemed the most likely cause. This herd had one young bull, originating from the same herd, which had bred a small number of cows this season and, potentially, last season.

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Ovarian Teratoma in a Juvenile Great Horned Owl (*Bubo virginanus*)

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A juvenile female free-ranging great horned owl (*Bubo virginianus*) was observed on a farm, and subsequently found dead the next day. The bird was presented in poor nutritional condition with no internal fat stores visible. A large irregular mass measuring 12 x 10 x 7 cm was present within the cranial abdomen, pressing on the liver cranially and the ventriculus to the left, and appeared to originate in the area of the right ovary which could not be identified. The mass contained a blood-filled cavity, and was mottled white, yellow and red**,** with variable consistency on cut section and contained mineralized foci. The liver was roughly half normal size, and the ventriculus was shrunken and devoid of ingesta.

Histologically, the tumor mass consisted of multiple well differentiated tissues which included: fibrous connective tissue, smooth muscle, well differentiated nodules of cartilage some of which were associated with osteoid, adipose tissue, vessels, stratified squamous epithelial nests filled with keratinized pearls, and other stratified epithelial glands which contained mucinous material. A diagnosis of teratoma, most likely arising from the right ovary was made based on morphology.

Immunohistochemical staining was applied for vimentin, pancytokeratin and S-100. This staining confirmed that multiple cell types were represented from 2 of 3 embryonic layers (mesoderm, ectoderm, and endoderm), making this a teratoma. Teratomas have been reported in birds in the past, primarily domestic chickens and ducks, although scattered reports include wild birds such as a great blue heron and a black headed gull. However, no reports in owls or raptors were found. Sites commonly affected include testes, ovaries, or intra-abdominal; less common sites include air sac, intracranial, kidney, adrenal, spinal cord, and Meckel's diverticulum. Generally these arise spontaneously, however, they have also been associated with implantation of heavy metals such as zinc or mercury within the gonad.

We believe this is the first reported case of a teratoma in a raptor. We feel this tumor most likely arose from ovarian tissue. And that the large tumor mass interfered with the digestive tract function in this bird leading to secondary emaciation and death.

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Testicular Blastomycosis in a Dog

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A 10 year-old Rottweiler dog presented to the referring veterinarian with a two day history of a swollen and painful scrotum. Physical examination revealed a markedly enlarged right testicle. The animal was castrated and the testicles were submitted for histopathology. Histologically, the normal testicular architecture was entirely effaced by pyogranulomatous inflammation which surrounds a large central area of necrosis. Within the central region of necrosis there were small numbers of 30-40 um diameter yeast organisms that had a 15 um diameter eosinophilic center, clear halo, a thin basophilic wall, and exhibited broad-based budding. This slide represents the characteristic histology of fungal orchitis, likely *Blastomyces dermatitidis*.

Bacterial infection is the most common cause of epididymo-orchitis, although this is relatively uncommon. Bacterial causes include *Escherichia coli* and *Brucella canis*. Less commonly, fungi such as *Blastomyces dermatitidis* and *Coccidioides immitis* can cause epididimymo-orchitis, however this typically results with disseminated fungal infections.

Anaplastic Large Cell B-cell Lymphoma in a Dog

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Signalment and clinical history: A surgically excised enlarged inguinal lymph node, measuring 2 by 4 cm, from an 8 year old spayed female mixed breed dog was submitted to a diagnostic biopsy service. At the time of the surgical excision, there was no evidence of metastases, all other palpabable lymph nodes appeared grossly unremarkable and chest radiographs were unremarkable. Based on the initial microsocopic examination, the submitted biopsy was diagnosed as a histiocytic sarcoma. Following referral of the dog to an oncology specialty practice, the original biopsy was referred to the Diagnostic Center for Population and Animal Health for a second opinion and immunohistochemical confirmation of the diagnosis.

Microscopic lesions: Sections of submitted lymph node are almost completely effaced by a large, poorly demarcated, expansile and infiltrative, unencapsulated malignant round cell neoplasm. Cells are arranged in sheets, with intermixed packets of cells separated by fine fibrovascular stroma. Neoplastic cells are large, with abundant eosinophilic cytoplasm, often with perinuclear clearing and sometimes with a finely vacuolated cytoplasm giving them a histiocytoid appearance. These cells contain large, vesicular nuclei with coarsely stippled chromatin and 1-3 variably sized prominent nucleoli. There is marked anisocytosis and anisokaryosis, with many binucleated and multinucleated cells, as well as moderate megalocytosis and megalokaryosis. The mitotic rate is high, with 2-6/HPF, and occasional bizarre mitotic figures. Neoplastic cells extend occasionally through the nodal capsule into adjacent adipose tissue along one of the inked margins. Neoplastic cells were immunohistochemically positive for CD18, CD79a, CD20 and CD45 and negative for CD3, IgG, and MNF116

Morphologic diagnosis: Lymph node, Anaplastic large cell B-cell lymphoma

Discussion: Based on the morphology and immunohistochemical characteristics of the neoplastic cells, a diagnosis of an anaplastic large cell B-cell lymphoma was made. Anaplastic large cell lymphomas (ALCL) are rarely described in dogs and require immunohistochemical differentiation from histiocytic sarcomas. As this case demonstrates, primary cutaneous or nodal masses morphologically resembling histiocytic sarcomas due to their marked cellular anaplasia, abundant cytoplasm and large numbers of multinucleated giant cells may actually represent ALCL of B-or T-cell origin. In human medicine CD30 and anaplastic lymphoma kinase (ALK) are essential for the diagnosis of ALCLs and the vast majority of ALCLs are CD30+ and ALK+. Unfortunately, neither antibody has been validated for formalin fixed canine tissues. CD30 is a transmembrane receptor and a member of the tumor necrosis factor superfamily that is normally expressed by activated T cells. CD30 is not specific for

ALCL and was originally described as a marker relatively specific for Reed-Sternberg and Hodgkin cells of classical Hodgkin lymphoma. However, the staining pattern for CD30 in ALCL is distinctive, being intense with a membranous and paranuclear pattern (target-like appearance). ALK, also known as CD246, is a tyrosine kinase that belongs to the insulin receptor superfamily. ALK is normally expressed by a small subset of cells in the central and peripheral nervous systems of adults. The normal functions of ALK remain unclear.

There is controversy in the literature as to whether anaplastic large-cell lymphoma of B-cell phenotype is related to the T- or null cell lymphoma of the same morphology. The WHO has recently excluded CD30+ B-cell lymphomas from ALCL and grouped them with diffuse large cell lymphomas (DLCL). These B-cell ALCLs are mainly ALK-, but rare ALK+ cases have been described. Based on their clinical behavior and general phenotype there is a current consensus in the literature that ALK- B-cell ALCLs represents a morphological pattern occasionally encountered among diffuse large B-cell lymphomas.

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Clostridium perfringens type D colitis in a goat

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This 3-month old, male, unvaccinated Saanen goat was inoculated into the duodenum with 300 ml of a toxigenic culture of *Clostridium perfringens* type D. At the same time, 200 ml of a 20% starch solution was inoculated into the abomasum. Clinical signs consisting of hemorrhagic diarrhea, respiratory distress and CNS signs, including recumbency, paddling, bleating, convulsions and opisthotonos began 8 hrs after inoculation. The animal was euthanized 1 hr after the onset of clinical signs. Gross post-mortem changes consisted of severe pulmonary edema and colitis, including congestion and hemorrhage of the mucosa, together with adherent whitish pseudo-membranes. The wall of the colon was thickened and a variable degree of edema was observed in the serosa of the colon and adjacent mesentery. The histological changes included degeneration, necrosis and desquamation of the superficial epithelial cells, with formation of an incipient pseudomembrane consisting of cell debris, fibrin, inflammatory cells (mostly neutrophils) and bacilli. Neutrophilic infiltration was observed in the lamina propria. Proteinaceous edema was observed in the lamina propria, submucosa, muscular layers and subserosa. Pseudomembranous colitis is characteristically observed in the sub-acute and chronic forms of caprine enterotoxemia by *C. perfringens* type D. These lesions are usually associated with systemic changes including pulmonary and, rarely, brain edema. Both colonic and systemic changes are believed to be produced by the effects of *C. perfringens* type D epsilon toxin. It is important to stress that intestinal lesions are usually not present in enterotoxemia of sheep, in which epsilon toxin seems to act only systemically producing pulmonary and brain edema.

Protothecal Cystitis in a Dog

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A six year-old female Rhodesian Ridgeback presented to the referring DVM with a history of chemosis, blindness, and pain in her right eye. Retinal detachment was observed multifocally throughout the eye, and the eye was enucleated and submitted for histopathologic examination. Myriad intraocular *Prototheca spp*. were observed within the posterior segment along with a detached retina and pyogranulomatous endophthalmitis. Approximately six months later, the dog presented to the referring DVM with clinical signs of mild depression, disorientation, weight loss, polyuria, and polydipsia. The dog was euthanized, and a complete necropsy was performed.

At necropsy, gross findings were as follows: multiple white foci scattered throughout the myocardium, cloudy vitreous fluid, cloudy urine, and thickened urinary bladder mucosa with a dark red, pebbled surface. Nearly all of the tissues examined histologically showed changes associated with Protothecosis. There was a marked granulomatous myocarditis, severe granulomatous endopthalmitis, marked follicular lymphoplasmacytic cystitis, moderate lymphoplasmacytic nephritis, mild encephalitis, and minimal colitis. The urinary bladder has been submitted for interpretation.

Within the lamina propria of the urinary bladder are multiple, large, inflammatory foci composed of lymphocytes and plasma cells (reactive lymphoid follicles). On the surface of the transitional epithelium, there are a few multifocal aggregates of moderate numbers of plasma cells and lymphocytes. No protothecal organisms are identified within the mucosa or the submucosal lymphoid nodules.

Prototheca spp. are achlorophyllic algae that rarely cause disease in animals or humans.[1] In dogs, infection typically causes intestinal disease with spread from the intestinal tract to the eyes, kidneys, bones, central nervous system, and heart. Urinary tract disease, manifest as polyuria and polydipsia, has been reported in several cases. From some of these cases, *Prototheca spp*. have been visualized on microscopic examination of the urine, and they have been cultured from the urine. However, infections with *Prototheca spp*. are not commonly reported to cause cystitis.[2]

Follicular cystitis, characterized by lymphocytic nodules beneath the urinary epithelium, is common in dogs. In general, cystitis results most commonly from bacterial infection, including *E. coli, Staphylococcus spp., Proteus spp., Klebsiella spp., Enterococcus spp., Streptococcus spp., and Mycoplasma spp*.. Cystitis is less commonly caused by *Blastomyces spp., Aspergillus spp., Candida spp., and Nocardia spp*..[1] This case is unique because it demonstrates a severe follicular cystitis secondary to systemic infection by *Prototheca spp*.. It is presumed that the continued presence of *Prototheca spp*. in the urine resulted in the formation of reactive lymphoid nodules due to chronic antigenic stimulation.

Outbreak of Meningitis [Meningoencephalitis] in Commercial Broiler Chickens

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Signalment:

Increased mortality associated with neurological signs that included torticollis and incoordination was observed in 32-day-old, mixed-sex, Ross cross Mississippi broilers.

Abstract:

An outbreak of elevated mortality associated with clinical neurological signs including torticollis and incoordination was observed in 32-day-old mixed-sex, Ross-Ross cross commercial broiler chickens in Southern Mississippi. Half of the birds submitted to PRDL for evaluation also grossly demonstrated either unilateral or bilateral hypopyon. Histopathology of the brain demonstrated the occurrence of variable pleocelluar or mixed heterophilic and histiolymphocytic meningitis that often was associated with mononuclear perivascular inflammatory cell infiltration or "lymphocytic cuffing" of the brain parenchyma, and sometimes multilfocal gilosis and hemorrhage. *Ornithobacterium rhinotracheale* was isolated from about half of the brains cultured, and several of the eyes and ears of the affected birds. Our findings provide additional data indicating the role of *O. rhinotracheale* as a neural pathogen of chickens (1-3).

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Cerebellar hemorrhage associated with Porcine circovirus 2 in a weaned pig

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Clinical history

3-5% of 400 post weaned pigs affected with weight loss and scour on an indoor unit. 1.7% post weaning mortality. One of 3 thin diarrheic pigs submitted for necropsy. Neurological signs not specified (suspected meningitis) were also reported in this pig prior to euthanasia.

Necropsy observations

A 23kg pig in poor body condition. Significant findings were excess serous peritoneal fluid with fibrin strands, edema of mesocolon and large intestinal mucosa, loose large intestinal content and enlarged mesenteric lymph nodes. Prominent petechial and echymotic hemorrhages on dorsocaudal cerebellar meninges; cut surfaces revealed petechial hemorrhages extending into grey and white matter of caudal lobules of vermis.

Laboratory results

Bacteriology: Single colony of a non-hemolytic *Staphylococcus* isolated from meninges. No bacterial growth from liver or lung. Profuse growth of non-hemolytic coliforms isolated from large intestine.

Virology: PRRSV RNA was not detected in serum by PCR.

Histopathology

Brain:

Portions of parasaggital cerebellum (slide set): in areas corresponding to regions of macroscopic hemorrhage, microscopic lesions include fibrin exudation and patchy mononuclear infiltration in meninges; in grey matter marked red blood cell and fibrinoid extravasation and hyaline protein droplet formation particularly subpial and perivascular, marked capillary endothelial activation, multifocal basophilic stippling of molecular layer (consistent with mineralisation of Purkinje cell dendrites), shrunken Purkinje cells with loss of Nissl substance, necrosis of internal granule cells; in white matter, marked rarefaction, perivascular fibrinoid exudation and ring haemorrhages and presence of hyaline protein droplets in glial cytoplasm. In less severely affected areas, variable meningeal mononuclear cell infiltrate (lymphocytes, macrophages) and patchy subpial rarefaction are present. Elsewhere in brain there is mild perivascular cuffing in meninges and neuroparenchyma, becoming more frequent caudally, mainly in occipital cortex and caudal brainstem. Marked mononuclear cell infiltration and abundant fibrin exudation in choroid plexus of 4th ventricle.

Other tissues: Granulomatous inflammation and lymphoid depletion with intracytoplasmic amphophilic inclusions typical of PCV2 in lymph nodes and gut-associated lymphoid tissue, consistent with postweaning multisystemic wasting syndrome (PMWS).

Immunohistochemistry

PCV2: Cerebellum : Intense labelling of endothelium, both cytoplasmic and nuclear, occasional glia in areas of hemorrhagic necrosis and of macrophage-like cells in overlying meninges.

PRRSV: Cerebellum : No labelling.

COMMENT

Similar CNS lesions have been reported in pigs affected with PMWS in Brazil (Correa and others 2007). The reason for the targeting of cerebellum is unclear: the lobular distribution in vermis differs from lesions occurring secondary to brain swelling (thought to be a consequence of compression of the rostral cerebellar artery). The nature of the lesions suggest a primary vascular event and the location of the PCV2 antigen strongly supports a role for this virus in the pathogenesis of the lesions, although additional factors / pathogens may be involved. Mesocolonic and large intestinal oedema have been reported in a range of conditions including PMWS (Done and others 2001) and Edema disease. The CNS lesions differ in their distribution and histological characteristics from those previously associated with Edema disease.

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Granulomatous encephalitis in a Canada goose caused by schistosome eggs

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Mortality of birds was observed at a water treatment facility in Alameda County, CA, in July 2008; 6 geese, 7 ducks, and 1 pigeon were found dead within 2 days. Clinical signs observed in these birds were weakness and inability to fly. The carcasses of three birds (1 Canada goose and 2 mallards) were sent to our veterinary diagnostic laboratory for postmortem examination. The submitter suspected botulism. Gross lesions consistent with emaciation (atrophy of the pectoral muscles, prominent keel bone, and hydropericardium) were seen at necropsy. Histologically, there was severe, diffuse granulomatous encephalitis in the goose. Multiple granulomas were scattered throughout the neuropil and also under the meninges of the cerebrum and cerebellum; most of these granulomas consisted of undulating, yellow to brown, intravascular or extravascular structures interpreted as remnants of egg shells from trematodes which were surrounded by many foamy, plump macrophages and multinucleate giant cells, some mononuclear cells, and few heterophils; few of these were mineralized. Rarely, viable trematode eggs were found within these foreign body type granulomas. In the small intestine, serosal and mesenteric blood vessels had marked myointimal hyperplasia with stenosis of the vascular lumen, and were surrounded by small to moderate numbers of mononuclear cells (hyperplastic, obliterative, nonsuppurative perivasculitis). Small numbers of identifiable schistosome eggs were found within the lumena of the blood capillaries from the lamina propria of the small intestinal mucosa. Lesions consistent with those of schistosomiasis (obliterative nonsupppurative perivasculitis and egg-induced intraluminal granulomas) were found in the two mallards but no brain lesions were noted in any of these ducks. A mouse bioassay was used for the screening of botulism toxin in the serum of one of the birds (goose) and in the livers of all the 3 birds. No botulinum toxin was detected in any of the livers tested. Signs suggestive of botulism were seen in those mice inoculated with the goose's serum sample, the suspicion of botulism couldn't be not confirmed as there was not enough serum available to perform the neutralization test.

Schistosomes are blood flukes that occur in mammals and birds, and cause schistosomiasis (bilharziasis). *Allobilharzia*, *Bilharziella*, *Dendritobilharzia*, *Gigantobilharzia*, and *Trichobilharzia* are the genera of schistosomes from waterfowl. Adult female worms living within blood vessels (mesenteric veins or arteries) lay eggs which pass through the intestinal blood vessel walls and mucosa and are excreted in the feces. Miracidia hatch from these eggs and penetrate water snails. Cercariae released from these snails actively penetrate the skin of waterfowl and gain the circulatory system. Cercariae of avian schistosomes can invade the skin of humans causing dermatitis (swimmer's itch). *Schistosoma mansoni* and *S. japonicum* are the schistosomes of human

beings which eggs can induce marked granulomatous response in the brain and spinal cord causing severe neurological disease (neuroschistosomiasis). In birds, there are only 3 reports of encephalitis due to schistosomes – two in swans and one in geese, all of these with associated neurological signs. In the case here presented, however, it was not clear if the florid disseminated lesions found in the brain were actually clinically significant as the botulism toxin test was inconclusive, and the other two birds affected had inconclusive, and the other two birds affected had **no** brain lesions. Schistosome-associated vascular lesions in the serosal blood vessels are very common in the waterfowl that have been necropsied in our diagnostic lab, and are considered as incidental microscopic findings.

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Myositis associated with *Haemoproteus* spp in Bobwhite quail (*Colinus virginianus*).

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Several 14-16- week-old Bobwhite quail were submitted for necropsy with a history of anorexia, weakness, lethargy, reluctance to move and increased mortality in the flock. Postmortem examination revealed linear red streaks in the skeletal muscles of the limbs, wings, back and pectorals. Other lesions included enlarged and dark spleens and livers.
Histopathology of the skeletal muscles revealed multifocal degeneration and necrosis of myocytes with hemorrhage and infiltration primarily by mononuclear cells and occasionally heterophils randomly scattered through out. Within the myocytes there were megaloschizonts and cyst forms of protozoa consistent with *Haemoproteus* species.

Comments: *Haemoproteus* spp (*H. lophortyx*?) is an intraerythrocytic protozoan parasite first described in California quail in 1930. Since then there have been many reports of its occurrence in Bobwhite quail particularly during summer and fall in California. The protozoa are transmitted by insects (midges?) and the mortality can range from 5 to 20 %. The clinical signs, pathology and mortality are probably a result of parasitemia, anemia and myositis and generalized inflammation in various organs including the heart, liver, spleen, brain, *etc.* PCR is being performed to identify the species of Haemoproteus that was causing the disease the quail.

Hepatic necrosis associated with West Nile virus in an Eastern Rosella Parakeet (*Platycercus eximis*)

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An 18-week-old Eastern Rosella was presented for necropsy with clinical signs of acute respiratory distress, weakness and death. Two other young and a mature Rosella's had died over a period of two to three months before. The Rosella's were housed outdoors along with cockatiels, love birds, doves and other species of parakeets in separate flight cages. Only Rosella's were affected in the aviary. There were numerous mosquitos in the vicinity. Postmortem findings in the Rosella included mild to moderate emaciation and mild to moderately enlarged liver and spleen.
Histopathology of the liver revealed acute diffuse coagulative necrosis of hepatocytes with minimal inflammation, mild periportal inflammation and mild increased cellularity in the sinusoids. Immunohistochemistry for West Nile virus revealed strong intracytoplasmic positive staining in the hepatocytes, Kupffer cells and monocytes in the blood vessels

Comments: West Nile virus (WNV) infection has been diagnosed in more than 60 psittacines in CAHFS since 2004. Loss of weight is the most common clinical sign described due to WNV in psittacines and pathology includes non suppurative interstitial nephritis, myocarditis, splenitis, hepatitis, enteritis, pancreatitis and occasionally encephalitis and other lesions. But acute hepatic necrosis due to WNV is very rare in psittacines. The strong IHC staining in the liver and other organs of this bird suggests that there was high viremia. No other concurrent diseases were detected in this bird. Rosella's appear to be more susceptible to WNV among psittacines.

Equine Multinodular Pulmonary Fibrosis

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A 13-year-old, Throughbred mare developed progressive exercise intolerance. Radiographs showed an interstitial nodular pattern in all lung fields. Bronchoalveolar lavage (BAL) fluid was positive for EHV2 and EHV5 viruses by PCR. The animal was treated with steroids. Due to progressive worsening of clinical signs, the mare was euthanized. Gross findings were concentrated in the lung. There were multiple 2-10 cm in diameter nodules, visible from the pleural surface. On cut section, these nodules were tan to white, firmer than and fairly well demarcated from the surrounding parenchyma. Microscopically, these nodules were nonencapsulated and consisted of trabeculae of connective tissue rich in collagen and some fibroblasts surrounding spaces lined by a single layer of cuboidal to squamous epithelium. The lumen of these spaces contained variable numbers of leukocytes (neutrophils, and fewer lymphocytes and macrophages). Epithelial cells had a large round vesicular nucleus and nucleolus. Some polyhedral or round cells, interpreted as epithelial cells or macrophages, had enlarged, swollen nuclei with marginated chromatin and amphophilic to eosinophilic inclusions filling the nucleus. Most cells with inclusions were free in the lumen. Binucleated cells were rare. The "alveolar" pattern observed in some nodules was not apparent in others in which the lining epithelium was lost and fibrosis predominated. Airways within these nodules were surrounded by fibrosis and variably affected by epithelial hyperplasia, goblet cell metaplasia, focal epithelial dysplasia, or smooth muscle hyperplasia.

Clinical and microscopic findings in this lung are compatible with a diagnosis of equine multinodular pulmonary fibrosis.[2,3] This recently characterized disease is attributed, at least partially, to infection by EHV5. EHV5 genome was detected in this horse's BAL, supporting this diagnosis. This condition has been reported in about 30 horses in the USA. Clinical signs are mostly the result of pulmonary disease and are progressive. Some animals recover clinically after treatment with corticosteroids and acyclovir. Due to the high cost of acyclovir treatment, only costicosteroids were used in this case, which raises the possibility of reactivation of a latent herpes infection. Fibrosis is a fairly common finding in the lungs of aging horses. However, the nodular pattern of this lesion and its association with EHV5 makes this condition unique. There is an additional report of pulmonary fibrosis and herpesviral infection (by genome identification) in donkeys. Microscopic lesions in this report seem to be different from those in our case, with numerous syncytial cells present.[1]

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