HISTOPATHOLOGY SLIDE SEMINAR

CASE NUMBER	ANIMAL	CASE NUMBER	ANIMAL
1	Owl	10	Sheep
2	Dog	11	Dog
3	Cat	12	Cat
4	Cat	13	Fish
5	Dog	14	Goat
6	Cow	15	Goat
7	Goat	16	Dog
8	Squirrel	17	Alpaca
9	Dog	18	Chicken

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2. Lupoid Dermatosis and Demodicosis in a German Short-Haired Pointer – **J.A. Ramos-Vara**, M.A. Miller
3. Cryptococcoma in the Spinal Cord of a Cat – **M.A. Miller**, N. Williams
4. *Streptococcus pneumoniae* Cellutis and Pneumonia Secondary to Traumatic Injury in an Eight Week Old Domestic Shorthair Kitten – S. Zhang, **F. Wilson**, C. Gustavsen, G. Henson
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6. Sarcocystis Lymphadenitis in a Cow – M.M. Sebastian, R.C. Giles, BC Barr
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15. Proliferative Reaction on the Penis of a Goat with Urolithiasis - **F. Uzal**, P. Ladd, D. Read, B. Daft
16. Acute Carprofen (Rimadyl) Hepatotoxicity in a Bitch – **D. O'Toole**, C. Quist, T. Berry
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18. Bone Marrow Necrosis Associated with Exotic Newcastle Disease In Chickens – **H.L. Shivaprasad**

Histopathology of West Nile Virus Infection in Owls

S.D. Fitzgerald[1*], M. Kiupel[1], J.S. Patterson[1], S.D. Grimes[2], H.A. Simmons[1]

During the summer of 2002, high incidence of neurologic signs and mortality in both captive and free-ranging owls was noted in several Midwestern states. Owls affected were all in the Family Strigidae, including snowy owls, great-horned owls, barred owls and short-eared owls. Diagnosis of West Nile virus (WNV) infection was made by specific RT-PCR assay on frozen heart and kidney, and was supplemented by immunohistochemical staining of a variety of formalin-fixed, paraffin-embedded tissues, using a polyclonal antibody that reacted to group specific antigens in several flaviviruses in the Japanese encephalitis virus group.

The heart was the most frequent tissue to demonstrate microscopic lesions, followed by the brain, liver and kidney. Cardiac lesions varied from moderate to severe, with multifocal necrotizing myocarditis which was associated with mixed infiltrates of lymphocytes, plasma cells, histiocytes, and variable numbers of heterophils. The brain lesions occurred throughout all levels of the brain. There was mild to moderate non-suppurative meningoencephalitis, with perivascular cuffing and gliosis. Heart and kidney were the two most frequent tissues to exhibit positive immunohistochemical staining.

Microscopic lesions of WNV infection were much more prominent in owls than is generally seen in crows collected for surveillance, although both owls and crows tend to exhibit strong immunohistochemical reactivity. It may be that owls are more resistant to WNV than crows, requiring a longer period post-infection for clinical signs, mortality, and histologic lesions to develop. Previous reports of WNV lesions in owls are limited, but we believe owls are relatively susceptible to this newly emerging disease, and it may result in significant decline in wild owl populations.

Reference:

Fitzgerald, S.D. et al.: Clinical and Pathologic Features of West Nile Virus Infection in Native North American Owls (Family Strigidae). Avian Dis. (In press)

[1]Diagnostic Center for Population and Animal Health, College of Veterinary Medicine, Michigan State University, East Lansing, MI
[2]Animal Disease Diagnostic Laboratory, Division of Animal Industry, Ohio Department of Agriculture, Reynoldsburg, OH

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Lupoid dermatosis and demodicosis in a German short-haired pointer

J.A Ramos-Vara, M.A. Miller

An intact male German short-haired pointer dog developed pruritus and seborrhea at the age of 1 year. A littermate had been euthanized for "painful skin" more than one year earlier. On presentation at the VTH it had seborrhea sicca along the trunk and pain upon palpation. Thoracic skin biopsies were characterized as a cell-poor interface dermatitis with occasional epidermal apoptotic keratinocytes, folliculitis and furunculosis. Based on the clinical history and microscopic findings a diagnosis of lupoid dermatosis was made. The dog responded to immunosuppressive doses of prednisone and antibiotics with marked improvement. Eight months later the patient presented again with intensified pruritus, truncal seborrhea sicca, increased dorsal midline hair loss, thinning and decreased elasticity of the caudal abdominal skin and a pot-bellied appearance. Cytology revealed an increased number of *Malassezia spp*. Yeasts and numerous coccoid bacteria. Another eight months later, the animal was humanely euthanized due to profound deterioration and generalized demodicosis.

At necropsy there was marked alopecia, seborrhea, and miliary nodules throughout the skin. Microscopically, the skin had diffuse, mild to moderate epidermal hyperplasia (acanthosis), orthokeratotic hyperkeratosis, hydropic degeneration of the basal cell layer and uncommon apoptotic keratinocytes in the basal cell layer or suprabasally. There was pleocellular lichenoid infiltrate of macrophages, lymphocytes, fewer plasma cells, and rare neutrophils. There was also pigmentary incontinence with numerous melanophages in the superficial dermis. Similar lichenoid infiltrate and pigmentary incontinence was observed in follicular infundibula. Vacuolation of basal keratinocytes and occasional apoptotic cells were also present in the outer root sheath of follicular infundibula. Numerous hair follicles were dilated with abundant keratin or arthropods, consistent with *Demodex spp*. Parasites occupied the lumen of infundibula and isthmus of the hair follicle and sometimes deeper segments. Many follicles were ruptured and infiltrated with mixed leukocytes forming pyogranulomas. The center of these granulomas usually contained hair shafts or degenerated parasites which were surrounded by palisading epithelioid macrophages, neutrophils, lymphocytes, and plasma cells. Parasites were also extruded onto the epidermal surface. In areas of severe inflammation, dermal fibrosis was common.

Immunohistochemically, leukocytes in the lichenoid infiltrate were mainly CD3-positive (T-lymphocytes) and less commonly CD79a-positive (B-lymphocytes). Similar infiltrate surrounded follicular infundibula. Some CD3-positive cells infiltrated the epidermis. Granulomas had central histiocytic antigen-positive cells surrounded by mainly CD3-positive cells. Antibody to CD18 antigen detected lymphocytes and macrophages in all lesions.

Exfoliative cutaneous lupus erythematosus (ECLE), the current name for lupoid dermatosis, is a rare cutaneous condition of German short-haired pointer dogs that appears to have a familial predisposition. Immunopathologic analysis of skin biopsies suggests an immune-mediated response against basal epithelial cells as part of the pathogenesis. The German short-haired pointer is not recognized as a breed predisposed to demodicosis. It has been speculated that predilection for generalized demodicosis is caused by functional deficiency of T cells. In our case, demodicosis was most likely secondary to immunosuppressive therapy to control ECLE. Although ECLE has microscopic features of lupus erythematosus, it does not seem to respond to immunosuppressive treatment. This case shows two unrelated conditions; without a proper history, ECLE might have been overlooked due to the generalized and severe demodicosis.

Animal Disease Diagnostic Laboratory. Purdue University. 406 S. University St. West Lafayette, IN 47907

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Cryptococcoma in the spinal cord of a cat

Margaret A. Miller, Ned Williams,
Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN
Eastern Carolina Vet Referral, Wilmington, NC

Slide 11411-03. A 14-year-old castrated male domestic shorthaired cat presented for development of neurologic signs that the owner had attributed originally to a fall into a bucket of pine oil/detergent mix. (In retrospect, the fall was probably secondary to ataxia resulting from the spinal cord lesion.) The cat was negative for feline leukemia virus and feline immunodeficiency virus. The referring veterinarian had treated the cat with methimazole (5 mg per os bid) and acupuncture. The cat had not responded to treatment and right hind limb paresis had progressed to bilateral hind limb paralysis with no deep pain over the course of 4 months.

Upon neurologic examination, the cat demonstrated bilateral hind limb paralysis with upper motor neuron reflexes to the sciatic, cranial tibial, and patellar regions. Good withdrawal and crossed extensor reflexes were present in both pelvic limbs with no deep pain sensation bilaterally. Neurologic signs indicated a lesion between the third thoracic and third lumbar segments of the spinal cord. There was no clinical evidence of brain or other central nervous system (CNS) disease.

CT scan and CSF analysis were offered to the owners; however, given the poor prognosis, the cat was euthanized. At necropsy, a mass about 5 mm in diameter was detected in the lumbar tumescence of the spinal cord. The mass was composed of light brown homogeneous tissue. Adjacent tissue of the cord was malacic and compressed by the mass. A segment of spinal cord that included the mass was submitted in formalin for histologic examination.

Histologically, the mass was a granuloma within the neuropil. In the center of the mass, inflammation was less severe, but the periphery was a densely cellular mixture of epithelioid macrophages, neutrophils, lymphocytes, and plasma cells. Inflammation extended into the overlying meninges. Numerous intact and degenerate yeasts, usually 5 to 15 um in diameter, were detected in the granuloma in HE sections. Some yeasts had been phagocytized by macrophages. The yeasts were strongly stained by PAS; a few narrow-based buds were noted. Mucin in the capsule stained weakly with mucicarmine. The histologic diagnosis was cryptococcal granuloma.

Cryptococcus has a predilection for the CNS; infection typically results in meningitis. Meningoencephalitis ensues if the fungus invades the brain or spinal cord and typically leads to fulminating disease. In people, *Cryptococcus neoformans* is the most common cause of fungal meningitis and affects up to 10% of AIDS patients. Human immunodeficiency virus (HIV) is a factor in about 50% of the cases of CNS cryptococcosis; fungal infection is attributed to low CD4 lymphocyte count.

This case is an unusual presentation of cryptococcosis in a cat that was seronegative for FIV infection and lacked evidence of systemic, respiratory, or cerebral disease. Cryptococcosis only rarely manifests as a discrete mass in the brain or spinal cord. These cryptococcal granulomas or 'cryptococcomas' cause localized neurologic deficits and resemble neoplasms by MRI, CT, or macroscopic appearance. Cryptococcoma (along with toxoplasmosis, lymphoma, and tuberculoma) is included in the differential diagnosis for masses in the CNS of AIDS patients and should be considered as a cause for tumor-like masses in the CNS of cats, regardless of FIV status.

Streptococcus pneumoniae Cellulitis and Pneumonia Secondary to Traumatic Injury in an Eight Week Old Domestic Shorthair Kitten.

Shuping Zhang, Floyd Wilson [Presenter],
Connie Gustavsen and Gay Henson
MS Veterinary Research & Diagnostic Laboratory,
College of Veterinary Medicine, Mississippi State University

ABSTRACT:

HISTORY: An approximate two month-old intact female domestic shorthair kitten presented to MSVDL with a history of having suffered trauma to the left shoulder region while playing with children and was found dead the following day.

GROSS PATHOLOGY: The animal exhibited palor of the oral mucus membranes and conjunctiva. The left front leg from the shoulder joint to the elbow joint was severely swollen owing in part to extensive gray-brown gelatinous subcutaneous edema. Multifocal echymotic hemorrhage was also apparent in the musculature of the upper leg. The lungs exhibited moderate numbers of irregular red areas present in the anterior and cardiac lobes. The stomach contained a “bolus” of adult round worms and numerous round worms were also present throughout the upper regions of the small intestine.

HISTOPATHOLOGY: Extensive pathology was evidenced in the subcutaneous tissues and skeletal muscles of the left leg. The connective tissues of the muscles from the leg appeared expanded and edematous containing very numerous but often degenerate or necrotic forms of mixed inflammatory cells that consisted largely neutrophilic polymorphs but included numerous histiocytes and lesser numbers of lymphocytes. Fibrin exudation was sometimes evidenced. In addition, massive numbers of small bacterial cocci that sometimes were diplococcal or formed small chains were seen throughout the connective tissues intermixed with the inflammatory elements. Similar inflammatory changes that included massive neutrophilic inflammatory infiltration, edema and abundant bacterial cocci. Were present in the subcutaneous connective tissues. Marked congestion, muscle degeneration and sometimes numerous fibrin thrombi were evidenced in some regions. Other pathological changes included moderate interstitial pneumonia and mild multifocal interstitial myocarditis, lymph node hemorrhage, nematodiasis, mild enteritis and interstitial nephritis.

DIAGNOSIS:

Left Leg: Severe Diffuse Cellulitis & Myositis [Septic]

COMMENTS & DISCUSSION: The severe bacterial cellulitis and myositis; presumably reflecting secondary complications to the traumatic injury to the leg but may also indicate consequences of debilitation in this kitten. *Streptococcus pneumoniae* was recovered from the bacterial cultures of the leg skeletal muscle, lung and kidney. In addition, numerous gram positive cocci often forming short chains were observed microscopically at the leg site. *Streptococcus pneumoniae* is a common cause of pneumonia in humans. However, it rarely has been reported to be pathogenic for animals. One reported case of septicemia and septic arthritis resulting from S. pneumonia was reported in an adult cat. In that report, it was hypothesized that the infection was transmitted from a child to the cat [J Am Vet Med Assoc. 191:703-4, 1987].

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Myocardial necrosis secondary to CNS injury in a dog

A. Kasuske
Animal Disease Research & Diagnostic Laboratory, South Dakota State University, Brookings, SD

A 2-year-old, mixed-breed dog was submitted dead to the Animal Disease Research and Diagnostic Laboratory at South Dakota State University with a history of seizures and vocalization indicating pain. The dog was minimally responsive to unresponsive to supportive therapy. Euthanasia was elected by the owners.

At necropsy, there was marked left perirenal hemorrhage and multifocal ecchymotic hemorrhages throughout the mesentery within the caudal abdomen. The stomach contained approximately 50-70 ml of dark-red liquid (unclotted blood, suspected) and a small amount of grass. The distal small intestine contained minimal amounts of dark-red to black, slightly granular material (digested blood, suspected). Within the left ventricular free wall, there were multifocal pale areas throughout the myocardium. No skeletal fractures or obvious lesions suggesting external trauma were noted.

Microscopically within several sections of heart examined, there was multifocal severe acute myocardial necrosis with edema and minimal inflammatory response present. Additionally, there was multifocal mineralization present. Within several sections of brain examined, there was multifocal marked hemorrhage and liquefactive necrosis of neuropil with gliosis and marked neutrophil infiltration. Adjacent to these areas, there were often areas of leukoencephalomalacia. Additionally, there was multifocal congestion of meningeal vessels and mild hemorrhage.

Myocardial necrosis secondary to central nervous system injury and/or disease is a known phenomenon in several species including dogs, horses, sheep, goats, cattle and pigs.[1] Neurogenic myocardial necrosis is usually not fatal and often only noted as an incidental finding at necropsy. This condition is believed to result from sympathetic overactivity and excess local catecholamine release.[1] The pattern of focal myocardial necrosis interspersed with normal myocardial fibers is thought to coincide with the distribution of the adrenergic nervous supply to the heart.[3] Central nervous system disease resulting in the development of this lesion can manifest as traumatic, infectious and/or space-occupying lesions. A single report in a dog suggested excess catecholamine release due to severe stress was the sole cause of myocardial necrosis.[2]

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SARCOCYSTIS LYMPHADENITIS IN A COW

MM.Sebastian [1], RC Giles and [1] BC Barr [2]

1.LDDC, University of Kentucky, 1429 Newton Pike , Lexington,KY-40511

2.CAHFS-Davis Lab, University of California,Davis ,CA 95616

A 10-year old cow found down and paralysed was submitted for necropsy to the Livestock Disease Diagnostic Center, University of Kentucky. The cow had very less body fat reserves. Fibrinous exudate was adhering to the surface of the uncollapsed lung. The posterior mediastinal lymphnodes and the bronchial lymph nodes were enlarged three to five times its normal size.

Multiple sections of the pulmonary lymph nodes and lungs were examined microscopically. Diffusely altering the architecture, the cortical and medullary lymphocytes are replaced by proliferating immature fibroblasts and mature fibrocytes with scattered histiocytes and erythrocytes. Protozoan organism in various stages are scattered through out the cortex and medulla in a random fashion .In other sections of lymphnode, mutifocal necrosis of the lymphocytes and proliferation of histiocytes are observed. The pleura overlying the lung sections is diffusely thickened and the attached lymph node is diffusely infiltrated by histiocytes and few multinucleate giant cells, which contain various stages of protozoan organism. The attached lung section has diffuse infiltration of macrophages in the alveolar septum. Several Sarcocysts are present in the myocardium. No histopathological lesions were observed in kidney, liver, brain, mammary gland and spleen.

Immunohistochemical staining of the lymphnode was positive for *Sarcocystis cruzi* (polyvalent rabbit antisera) organism and was negative for *Sarcocystis falcatula*, *Toxoplasma gondii* and *Neosporum caninum*.

Chronic sarcocystosis characterized by cachexia, muscle atrophy, hair loss, nystagmus nervousness and death is less well understood than acute sarcocystosis because clinical laboratory findings in chronic cases are usually normal and the inflammation in muscles and neural tissue is less intense than in acute sarcocystosis. Some sarcocysts probably rupture from time to time and thus the antigenic stimulus for antibody production is maintained. A non cytolytic extravascular immunohemolytic mechanism is suggested to be the cause of anemia and subsequent emaciation.

Landsverk T, Gamlem H , Svenkerud R. A Sarcocystosis –like protozoan in a sheep with lymphadenopathy and myocarditis. Vet Pathol 1978, 15: 186-195.

Frelier F, Mayhew IG, Pollock R. Bovine sarcocystosis: Pathological features of naturally occurring infection with *Sarcocystis cruzi*

OSU Case #2003-02106 AAVLD Histopath Slide conference

History: This seven-week-old female Boer goat kid was weak at birth but was unable to stand and walk until 5 weeks of age. Clinical signs progressed from stumbling to recumbency and inability to rise. Neurologic exam suggested upper motor neuron deficits to the hind end with possible cerebellar involvement. The animal was euthanized and submitted for necropsy.

Gross findings: No abnormalities noted.

Histologic findings: Cerebellar organization appears normal. Many of the neuronal changes seen in brainstem nuclei in this section may be due to fixation artifact – shrinkage, hypereosinophilia, etc. However, other neuronal changes such as cell swelling, dissolution of Nissl substance, cytoplasmic vacuolation and granular eosinophilic cytoplasmic inclusions that are visible in some cells are more convincing. Nuclei of these cells are generally unremarkable. The cytoplasmic inclusion/vacuoles do not stain with Luxol fast blue or PAS and unstained sections do ot fluoresce under UV light.
Other changes include similar cytoplasmic alterations in the neurons at various levels of spinal cord, mild gliosis of the lateral cuneate nucleus, and axonal degeneration in one of two peripheral nerves sampled. A focus of denervation atrophy was also identified in skeletal muscle from a hind limb. The neuronal cytoplasmic changes were less common in midbrain and could not be located in cerebral cortex.

Discussion: This was the third kid from the flock with this clinical presentation – born weak, progressive locomotory impairment. All 3 were from the same maternal line. One kid was not necropsied but re-examination of a previous submission of 2 wk-old male kid revealed similar but less extensive neuronal changes to those of the present case. Hepatic analysis of liver from the earlier submission yielded normal values for copper and selenium. Since that time these minerals had been further supplemented to the flock so no mineral analyses were performed on the older kid.

Electron microscopy of the abnormal neurons indicates these cells contain very large numbers of mitochondria. The specimens were formalin-fixed and thus mitochondrial detail is poor. We suspect this is a mitochondrial encephalopathy, similar to Leigh's disease in humans. Such entities are rare in domestic animals but have been reported in the dog and in the mouse.

Refs.

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Maternal inheritance and the evaluation of oxidative phosphorylation diseases. Shoffner, J.M. *Lancet* 1996;348:1283-1288.

Submitted by:

R. Bildfell, J. Heidel and M. Saulez
Departments of Biomedical Sciences and Clinical Sciences
Oregon State University
Box 429
Corvallis, Or 97339-0429

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West Nile Virus infection in an Eastern fox squirrel (Sciurus niger)

M. Kiupel M. Kiupel[1, 2], H. A. Simmons[1, 2], S. D. Fitzgerald[1, 2], A. Wise[1, 3], J.G. Sikarskie[4], T.M. Cooley[5], S.R. Hollamby[4], and R. Maes[1, 3]

[1] Diagnostic Center for Population and Animal Health and [2] Department of Pathobiology and Diagnostic Investigation and [3] Department of Microbiology and Molecular Genetics and [4] Small Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824; [5] Wildlife Disease Laboratory, Michigan Department of Natural Resources, East Lansing, Michigan 48823;

An adult Eastern fox squirrel (*Sciurus niger*) exhibited various signs of neurologic disease including weakness, depression, head tilt, torticollis, lateral recumbency, uncoordinated movements, inability to right itself when pushed over, scratching of its forehead with both feet and tremors. The squirrel was euthanized and no gross lesions were found at necropsy. Microscopic lesions were found in the kidneys, brain, heart and liver. In the kidneys (submitted), there was multifocal, moderate to severe lympho-plasmacytic nephritis characterized by multifocal, randomly scattered aggregates of lymphocytes, plasma cells, and a few histiocytes that expanded the interstitium. In the brain was diffuse, moderate, nonsuppurative meningo-encephalitis with lymphoplasmacytic perivascular cuffing in both the meninges and the neuropil. Scattered individual necrotic neurons that were sometimes surrounded by glial cells (satellitosis) were found in the cerebrum and the hippocampus. In addition, there was focal gliosis and a few glial nodules were scattered throughout the neuropil. Other lesions included a severe multifocal necrotizing myocarditis in the heart, a moderate perivascular lymphohistiocytic hepatitis with bile duct proliferation and mild intracellular cholestasis in the liver and mild lymphohistiocytic perivascular in the lungs. Brain, kidneys, heart, liver and lung stained positively for WNV using IHC. The kidneys exhibited fairly abundant staining in both tubular epithelial cells and interstitial macrophages. There was focal, but strong staining of multiple neurons and glial cells in the cerebrum and hippocampus. Myocardial fibers and interstitial macrophages exhibiting specific staining in the heart. In the liver and lungs, there were single positive macrophages. Formalin fixed renal tissue also tested positive by RT-PCR for WNV RNA.

There were no reports of increased mortality associated with neurological signs in squirrels in Michigan prior to 2002. Most likely squirrels represent a spill-over host, similar to humans and horses, none of which exhibited clinical cases of WNV during 2001 in Michigan. However, we speculate that in 2002 the prevalence and geographic distribution of both WNV-infected mosquitoes and crows had increased to some critical level that allowed significant spill-over infection into non-target host species. Alternatively, WNV may be adapting to new host species over time. Additional studies on both WNV strains and species-specific pathogenesis are needed to answer these questions. In our experience with surveillance of hundreds of free-ranging crows with WNV infection, histologic lesions tend to be mild or absent, while IHC staining confirms the presence of large amounts of viral antigen in various tissues. In contrast, horses tend to exhibit moderate or even severe histologic lesions in the brain and spinal cord, but very minimal WNV antigen, if any, can be detected by IHC. Commonly nested RT-PCR is required to confirm the diagnosis. Interestingly, Eastern fox squirrels had large amounts of WNV antigen associated with moderate to severe microscopic lesions in different tissues. It is possible that squirrels are somewhat more resistant to infection than are crows, but less resistant than horses, which means that infection may persist longer in squirrels than in crows and stimulate a greater inflammatory response prior to illness and death. Experimental inoculation studies with WNV in squirrels are needed to determine their relative susceptibility compared to other mammals. WNV represents a new challenge for North American diagnosticians, wildlife rehabilitators, wildlife biologists, zoological staff and others involved with wild bird and mammalian species. While it is widely recognized that crows and other corvids are highly susceptible to this newly emergent disease, many other species are also exposed. When presented with a squirrel with a history of neurologic disease, and microscopic nonsuppurative lesions in various tissues, particularly the heart, WNV infection should be one of the primary differential diagnoses.

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Poxvirus infection in a prairie dog

I.M. Langohr[1[1]*], L. Thacker[1], and P. Lockard[1]

A 12-week-old female prairie dog was euthanatized after presentation with respiratory distress. It was submitted to the Animal Disease Diagnostic Laboratory at Purdue University to determine the specific cause of the clinical signs since deaths of prairie dogs with similar clinical signs had occurred where this animal was bought. Gross lesions included numerous variably sized ulcers on the tongue and hard palate, dark red consolidation of both cranial and the right middle lobes of the lungs, affecting approximately 40% of the pulmonary parenchyma, and small (<3 mm) white firm slightly raised plaque-like lesions sparsely distributed throughout the wall of the gastrointestinal tract. Microscopic alterations in the lung consisted of multifocal to coalescing severe necrotizing bronchopneumonia, with vasculitis and poorly defined eosinophilic intracytoplasmic inclusions. Multifocal necrotizing lesions, often accompanied by myxoid edema, were also present in the sections of nasal turbinates, trachea, thymus and adjacent brown fat, tracheobronchial lymph nodes, lips, tongue, esophagus, stomach, jejunum, cecum, colon, liver, kidney, adrenal gland, vagina, vestibule, female accessory genital glands, conjunctiva, and cornea. Ultrastructural examination of lung tissue revealed scattered aggregates of mature, non-enveloped, oval or brick-shaped virions measuring 200x250 nm, with an electron-lucent core and two lateral bodies, surrounded by an outer membrane, located within the cytoplasm of degenerating cells. The morphology of the virions was consistent with poxvirus. Specimens of selected tissues were submitted to the Center of Disease Control and Prevention (CDC) to confirm the presumptive diagnosis of monkeypox infection. Laboratory evaluation of these tissues is in progress.

Monkeypox virus, a member of the orthopoxvirus genus, appears to be enzootic among wild mammals in the west and central African rainforest, where the principal reservoirs are thought to be squirrels and other rodents. Despite the name of the virus, primates are infected only accidentally through direct or close contact with infected reservoir hosts. In humans, infection with this virus causes a vesicular and pustular rash similar to but usually milder than smallpox. The incubation period is approximately 12 days, and the death rate among infected humans in Africa has ranged from 1-10%. In primates, monkeypox should be considered in any outbreak of a systemic febrile illness that involves a skin rash. Rashes can be severe and generalized in some species. Dyspnea caused by pneumonia may develop in severe cases. Concurrent bacterial septicemia might be present.

The diagnosis is confirmed by histological or electron microscopic examination of tissues with lesions, by serological tests, and by virus isolation; however, the characteristic lesions on inoculated chicken chorioallantoic membrane and the large eosinophilic intracytoplasmic inclusions typical of poxvirus infections may not be seen with monkeypox virus infection. Immunohistochemistry, Western blot, and polymerase chain reaction (PCR) tests for monkeypox viral antigen detection are now available.

In the United States, the disease was reported in early June 2003 among several residents of the Midwestern region who became ill after having contact with sick pet prairie dogs and, in one case, a rabbit. It is likely that the virus entered the United States via imported rodent species from Africa, with secondary transmission to domestic prairie dogs housed in the same animal-holding facility or pet shop. This outbreak is clear evidence about how introduction of exotic species might pose a serious threat to animals and public health.

[1[1]] Animal Disease Diagnostic Laboratory, West Lafayette, IN
[*] Presenter

IF IT LOOKS LIKE A DUCK, QUACKS LIKE A DUCK, YET DIES LIKE A SHEEP…

IONOPHORE TOXICOSIS IN LAMBS WHERE A FEED COMPANY EXCLUDED IONOPHORE TOXICOSIS

D. O'Toole[1], M. Raisbeck[1], T. Cornish[1], W. Wilson[2]

For six years an experienced sheep producer in Wyoming fed a special order protein supplement containing lasalocid to feeder lambs. In 2003 he fed 600, 20 – 30-kg home-raised Targhee and Finn-Targhee lambs using alfalfa grass hay (~5 bales/day), shell corn, live and tank water, special order pellets (22% protein; 120 g lasalocid/ton feed; target intake 15 – 70 mg/head/day), and free choice salt mineral mix containing lasalocid. The mineral mix contained selenium (>12 ppm) and vitamin E (605 IU/kg). The private label feed was used by three producers in the area. The owner injected lambs with a sodium selenite-vitamin E mixture.

On 11 May the owner fed 200 lambs with one bag from the bottom of the pallet and noticed that pellets were mixed with granular material that he interpreted as fines. The contents were offered to lambs. The following day, two lambs were dead and approximately 50% of pen mates were "down in their pasterns." Two adjacent pens of lambs, fed from other bags on the pallet, were healthy. The owner suspected a problem with the feed. Samples were collected from the feed bunks for analysis. Lambs displayed weakness and difficulty walking over the next three week. Ten lambs died. The remaining 90 affected lambs recovered. Two months later a young ram died acutely; post-mortem revealed subacute-chronic myocardial necrosis. Neither of the other producers using the feed reported a problem.

Observations at necropsy were pale or variegated thigh muscles, full abomasums, blanched or pale cardiac septum, epicardial petechiation and, in lambs recumbent for extended periods, antero-ventral pneumonia. Serum chemistry analysis revealed high AST (5,000 -25,000 IU), CK (91,000 – 427,000 IU) and LDH activities (15,000 - 99,000 IU). Lambs had mild neutrophilia, and granular casts in urine. Tissues from 5 lambs were submitted. Lesions were severe acute or subacute myonecrosis of skeletal muscle (submitted slide), inhalational bacterial pneumonia, and mild multifocal myocardial degeneration. The presumptive diagnosis was ionophore toxicosis related to "fines" present in one bag of feed. A manufacturer-commissioned analysis for lasalocid was performed at a commercial laboratory. It reported 110.7 g/T (pellets) and 9 g/T ("fines"), which were within the label claim (120 g/T). The feed company contended its feed was not the problem and that another explanation should be sought. It declined to pay for additional laboratory testing to resolve the cause of losses.

Hepatic vitamin E (2 ppm) and selenium (0.24 ppm) were adequate. Fines collected from the bunk were analyzed for ionophores by thin layer chromatograph and HPLC-MS. Results were <1 ppm naracin, <1 ppm salinomycin, 90 ppm lasalocid, and 500 ppm monensin in one bunk, and <1 ppm naracin, <1 ppm salinomycin, 80 ppm lasalocid and 150 ppm monensin in a second. The cause of death was monensin contamination, probably carryover from a previous batch of feed, restricted to one bag in the pallet, presumably the first in the production sequence of the owner's special order.

In cases of suspect feed contamination with a fatal outcome, testing for a range of potential contaminants associated with a particular lesion profile (in this case, severe skeletal myonecrosis with mild myocardial necrosis; ionophores) is essential to define the problem. The "targeted" testing by the company was for the ionophores additive, not for all ionophores capable of causing myonecrosis. After results were disclosed to the company, it revealed that it recognized carryover of monensin in recent feed runs.

[1] Wyoming State Veterinary Laboratory, University of Wyoming, Laramie, WY 82070
[2] All Creatures Veterinary Hospital, Riverton, WY 82501

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FELINE DYSAUTONOMIA

T. Cornish[1] and M. Schreiber[2]

Dysautonomia in cats is an idiopathic disease affecting the autonomic nervous system. This syndrome first was recognized in England in 1982, by Key and Gaskell, hence the common name "Key-Gaskell syndrome". During the early 1980s this was a common epidemic disease of cats in the United Kingdom and Ireland. It was subsequently diagnosed elsewhere in Europe and, on rare occasions, in cats in North America. The incidence of the disease has markedly declined and its etiology in cats remains unknown. The purpose of this presentation is to review gross and histological lesions of feline dysautonomia in an affected cat with typical clinical signs.

A previously healthy, 4 year-old, male castrate, domestic shorthaired cat weighing 4.5 kg was presented to a veterinary clinic in northern Wyoming after two weeks of illness. Clinical signs included lethargy, mydriasis with no response to light, tacky mucous membranes and decreased tear production, prolapsed third eyelids, regurgitation of food, and constipation. This was the only cat in the household and was predominantly an indoor animal. For four days the veterinarian administered enemas, intravenous fluids and electrolytes, broad-spectrum antibiotics, and force-fed the cat. There was no response to treatment. The presumptive diagnosis was feline dysautonomia. The cat was euthanized and the carcass submitted to the Wyoming State Veterinary Laboratory for necropsy.

At necropsy the cat was in fair nutritional condition. Significant gross findings included prolapsed third eyelids, megaesophagus with mucosal inflammation and pooled malodorous feed contents, megacolon with dry fecal impaction, and red mottled lungs. Bacterial culture, virus isolation and associated tests, and electron microscopy did not reveal any significant pathogens in tissue or fecal samples. Enteric parasites were identified on fecal flotation (38 EPG *Toxocara cati*), but were considered incidental in this case. There were severe and widespread histological lesions in the autonomic nervous system. These consisted of neuronal degeneration, necrosis, and dropout in celiac and mesenteric ganglia, in submucosal and myenteric plexuses in the gut, and in select (vagal and cranial nerve) nuclei in the brainstem. Other changes were diffuse megaesophagus with erosive/fibrinopurulent esophagitis, fibrinopurulent glossitis, and mild acute bronchopneumonia consistent with aspiration pneumonia.

Confirmatory clinical testing was not done (prompt papillary constriction in response to 0.05% pilocarpine solution; urination after administration of 0.04 mg/kg bethanechol subcutaneously). Diagnosticians should include dysautonomia on their differential list and sample the autonomic nervous system accordingly when cat carcasses are submitted with a history of acute onset lethargy, anorexia, depression, weight loss, vomiting/regurgitation, and dysuria, particularly if the clinician noted one or more of the following: dilated non-responsive pupils, prolapsed third eyelids, decreased tear production, megaesophagus with regurgitation, dry nasal and oral mucosa, dysphagia, constipation, bradycardia, loss of anal tone, or proprioceptive deficits. Approximately 25% of such cats survive with good supportive care, but they remain autonomic cripples. Most affected cats are euthanized within a year of clinical diagnosis.

[1]Wyoming State Veterinary Laboratory, University of Wyoming, Laramie, WY 82070
[2]Moxey Veterinary Hospital, Sheridan, WY 82801

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Severe Protocephalus Infection in Largemouth Bass with Ovarian Damage

S.D. Fitzgerald*, E. Elsayed, M. Munger, M. Faisal

Largemouth bass were collected from eight lakes in southern Michigan for surveillance of disease conditions. Approx. 90 % (247 of 290) of the adult bass collected had widespread abdominal adhesions associated with large numbers of adult and plerocercoid cestodes present free in the peritoneal cavity, and attached to abdominal viscera, as well as infiltrating the abdominal organs. The ovaries of adult female bass were particularly heavily infested with plerocercoid stages.

Parasitism is common in free- living fish. The adult tape worm, Proteocephalus ambloplitis is the common bass tapeworm. Identification of the adult worms from the abdominal cavity was accomplished by dissecting microscopic examination, which revealed typical vestigial apical sucker, dumbbell-shaped eggs, and a cirrus pouch stretching across one third of the proglottid segment. Interestingly, the juvenile plerocerecoid stage was present in high numbers within the ovarian tissue, and
was association with lack of normal maturation of oocytes. In histological sections examined, most oocytes were in the immature or pre-vitellogenic stages, while the remaining later developmental stages were degenerate or atrectic; no normal early or late vitellogenic stage oocytes were present. If a high percentage of female fish in this population were similarly affected, it could have significant impact on reproductive capacity. There are reports in the aquatic literature of fish being " castrated " by severe internal parasitism of the gonads.

Department of Pathology and Diagnostic Investigation, and Diagnostic Center for Population and Animal Health, College of Veterinary Medicine, Michigan State University, East Lansing, MI

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ABORTION STORM IN A HERD OF BOER GOATS

A. W. Layton, Hana VanCampen, Roger Maes and Mark Anderson

Mid to late term abortions; stillbirths; occasional weak neonates and fetal mummification occurred in 30 percent of the does of a backyard herd of 100 Boer/ Boer X goats. The owners designed the herd health program in which record management was based upon faulty recollection. The owner who also served as the prosector submitted an assortment of specimens for diagnostic work-up. Grossly, petechial hemorrhage could be identified on the surfaces of the lung, liver, intestinal serosa and adrenal glands when autolysis was minimal. Histologic lesions varied in severity and were characterized by scattered necrosis with hemorrhage that occurred in the adrenal glands, liver, lung, thymus, spleen and colonic mucosa. Intranuclear eosinophilic inclusions were identified in some sections of lung. Additionally in one section of a placenta, the cytoplasm of trophoblasts was distended by basophilic inclusions. Inflammation was not a feature in the section of placenta.

Caprine Herpesvirus 1 was isolated from pooled tissue of several fetuses. BHV-1 FA test of tissues was equivocal. The cytoplasmic inclusions in trophoblasts were gram negative and stained positive with Macchiavello's stain and Coxiella IHC.

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Proliferative reaction on the penis of a goat with urolithiasis: neoplasia or hyperplasia?

Uzal F.A., Ladd P., Read D., Kinde H., Daft B.

California Animal Health and Food Safety Laboratory, University of California, Davis. 105 W Central Ave, San Bernardino, CA, USA

An aged Anglo-Nubian male goat was presented with a history of acute abdominal pain. The goat originated from a herd in which several cases of urolithiasis and obstruction had been previously diagnosed. A clinical diagnosis of urethral obstruction was made and due to the severity of the clinical signs and the poor prognosis, the animal was euthanised.

A complete post-mortem examination was performed in the field by the submitting veterinarian, who reported that the urinary bladder was distended with clear-yellow urine and its wall was diffusely red and thick. No gross abnormalities were observed externally on the penis, but when this organ was transversally sectioned a large (5 mm diameter), roughly circular dark red area was observed surrounding the urethra; this lesion comprised the distal 2 cm of the penis, including the glands. Sand-like material was found in the distal urethral process. Histologically, the urethra was very dilated and its epithelium was completely necrotic with a few neutrophils infiltrating the lamina propria. Mineralized amorphous material, which in one location appeared to contain some epithelial remnants, filled and distended the penile urethra, which was surrounded entirely by dense collagen. Beyond this, large vessels were congested and contained fibrinous thrombi, and there were frequent hemorrhages into the stroma. The hemorrhages were associated in some areas with small deposits of hemosiderin. Infiltrating diffusely the corpus spongiosum, dorsal fibrous cord and connective tissue underlying the external squamous epithelium, were cells with non- or poorly defined cytoplasmic boundaries, vacuolated basophilic cytoplasm and pleomorphic, fusiform pointed ended or stellate nuclei with prevalent mitotic figures (approximately 4 per high power field). The pleomorphic nuclei in mitoses could sometimes be seen to line blood vessels. These cells also formed large whorls surrounding blood vessels the endothelial cells of which were prominent. Endothelial cells stained positively for factor VIII and CD31. No positive reaction to smooth alpha actin or pan cytokeratin were observed.

Based on the history, clinical signs and post-mortem changes, two differential diagnosis were proposed, i.e. i) ruptured urethra with exuberant fibrosis and vascular proliferation and ii) hemangiosacoma). Hemangiosarcoma was finally ruled out based on the negativity of most proliferating cells (except those lining blood vessels) to endothelial cell markers and because of the more or less differentiated appearance of the blood vessels. This report stresses the importance of how to differentiate a malignant neoplasia from granulation tissue.

16

ACUTE CARPROFEN (RIMADYL) HEPATOTOXICITY IN A BITCH

D. O'Toole[1], C. Quist[1], T. Berry[2]

A 5-year old German shepherd bitch was presented for left pelvic limb lameness. She was treated with a non-steroidal anti-inflammatory drug (carprofen; Rimadyl, Pfizer Animal Health)(87.5 mg BID). After 14 days of treatment the bitch was re-examined and a drawer sign detected in the left stifle. The animal was treated with another COX-2 inhibitor (deracoxib; Deramax, Novartis Animal Health); carprofen was withheld for 4 days. The veterinarian performed surgery for a ruptured anterior cruciate ligament. The bitch was discharged and given a course of carprofen for 5 days BID. The dog was treated with carprofen for a total of 19 days.

At the end of the second course of carprofen, the bitch was presented by the owner. The bitch was unwell, constipated, icteric, and vomiting. A seroma developed at the surgery site. The veterinarian hospitalized the animal and administered amoxycillin, prednisolone and cimetidine. After four days the bitch appeared improved and the veterinarian planned to send her home. At that time she was vomiting but less icteric. The following day icterus was marked and petechial hemorrhages developed in mucous membranes. The veterinarian gave her half a unit of blood, the bitch arrested and was revived, but never regained consciousness.

The presumed cause subacute hepatopathy in this dog is idiosyncratic carprofen-associated hepatocellular toxicosis. The basis of the diagnosis is a temporal association with recent administration of carprofen, and histological lesions consistent with toxic hepatopathy. The dog's breed (Labrador retriever) may be significant. Thirteen of 21 cases of carprofen hepatocellular toxicosis in one report occurred in the Labrador breed.[1]

Histological changes of variable severity were reported in liver in association with carprofen administration.[1] They included multifocal to extensive hepatocellular necrosis, periportal neutrophilic and lymphocytic inflammation, bridging fibrosis, biliary hyperplasia, intracanalicular and hepatocellular bile pigment accumulation, and extramedullary hematopoiesis. Four of 21 dogs in that study died 3 – 5 days after presentation. In addition to hepatocellular necrosis, one of two dogs examined post-mortem had multifocal renal tubular necrosis with regeneration and a perforating jejunal ulcer. Only liver was available from this dog, so the presence or absence of lesions in other tissues could not be documented histologically.

Carprofen (Rimadyl) is a non-steroidal anti-inflammatory drug (NSAID) in the propionic acid class. It is widely used of osteoarthritic and post-operative pain. The compound is a substituted carbazole, 6-chloro-a-methyl-9H-carbazole-2-acetic acid ($C_{15}H_{12}ClNO_2$) that inhibits cycyclooxygenase activity, particularly the inducible cyclooxygenase COX-2. It is rapidly and nearly completely absorbed (>90% bioavailable) when administered orally, with peak blood plasma concentrations 1–3 hours after oral administration. It has a half-life of ~8 hours. It is eliminated primarily by biotransformation in liver followed by rapid excretion of metabolites in feces (70–80%) and urine (10–20%). Some enterohepatic circulation of the drug occurs. The manufacturer (Pfizer) has reported animal safety studies and adverse reactions.[2] The most common adverse clinical reactions are vomiting (4%), diarrhea (4%), changes in appetite (3%), lethargy (1.4%), behavioral changes (1%), and constipation (0.3%). Hepatopathy was not consistently reproduced in safety studies. This dog was also treated with another NSAID, deracoxib. Hepatotoxicity is a rare adverse reaction associated with this drug.[3] The manufactures recommend that deracoxib should not be given at the same time as other NSAIDs, including carprofen. Concurrent administration was not done in this instance.The basis for hepatotoxicity in a small proportion of dogs given carprofen is unknown. The most likely explanation is an idiosyncratic toxic reaction. The drug was sold on the European continent for 10 years before an association was detected in North America between use of the drug and hepatic disease. A genetic feature unique to some Labrador bloodlines in North America might be an explanation, but that possibility was not pursued due to limited pedigree data.

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[1] Wyoming State Veterinary Laboratory, University of Wyoming, Laramie, WY 82070
[2] Buffalo Veterinary Clinic, Buffalo, WY

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Salinomycin Toxicosis in Alpacas

Sheila D. Grimes, John Thilsted, Craig Sarver and David Anderson
Ohio Department of Agriculture Animal Disease Diagnostic Laboratory and The Ohio State University

During the spring of 2003, alpacas on three farms were fed a diet that was accidentally contaminated with salminomycin, an ionophore. Initial clinical signs occurred three days following the ingestion of the contaminated feed. Some deaths occurred the next day. Clinical signs included the following: weak tail tone, incoordination, difficulty urinating, extreme pain, feed refusal, and recumbency. Alpacas continued dying for at least 21 days following the discontinuation of the contaminated feed in the diet. Greater than 100 alpacas died and approximately 500 alpacas had clinical signs of illness.

Twenty-eight alpacas from three premises were submitted to the Animal Disease Diagnostic Laboratory from March 17 to March 31. The most consistent gross lesions noted were pulmonary edema, hydropericardium, hydrothorax and subcutaneous edema. Additional gross lesions included myoglobinuria and an enteritis. Microscopic lesions in initial submissions were minimal. Later cases had varying degrees of a skeletal muscle and cardiac degeneration and fibrosis, and myoregeneration. The alpaca in this case was a later submission with myoregeneration a prominent feature in the skeletal muscle sections. Sarcolemmal nuclear proliferation and nuclear rowing of myonuclei and/or myoblasts was prominent in numerous myofibers. Other myofibers had clumped or fragmented, hypereosinophilic sarcoplasms lacking cross striations. The sarcoplasm and myofibrils were degenerate in some myofibers with intact sarcolemmal lamina, myonuclei and satellite cells remaining.

Ionophore analyses were performed at four laboratories. Salinomycin was detected in the feed at a concentration of approximately 70-90 ppm. An investigation of the feed mill, revealed that medication from a previous batch of broiler feed was inadvertently added to the alpaca feed, possibly the result of a plant computer-hopper malfunction.

Ionophores, such as monensin, lasalocid, salinomycin, narasin and maduramicin, are carboxylic ionophores, which are used an anticoccidial drugs for poultry and as growth promotants for ruminants. Deaths due to salinomycin toxicity in this case were postulated to be caused by three events: 1) direct effects of ionophores on ion channels causing acute myocardial failure, 2) rhabdomyolysis in which massive skeletal muscle damage causes metabolic acidosis and renal failure and 3) myocardial injury.

18. Bone marrow necrosis associated with Exotic Newcastle Disease.

H.L. Shivaprasad, University of California

Two juvenile and one adult game chickens in a group of 21 were euthanized and submitted for laboratory evaluation. These chickens exhibited lethargy, reluctance to move, drooling of mucus from the mouth and down on their legs. Upon post mortem examination two birds exhibited soiled vent and tail feathers. Oral cavity and pharynx had fibrinonecrotic foci (2/3), trachea and larynx had diffuse hemorrhage (3/3), crop had ulcers (1/3), lungs were diffusely congested (2/3), nasal passages had serosanguinous fluid (3/3), thymic lobes were congested (2/2) and the bursa of Fabricius were small (2/2).

Bone Marrow: Histopathology revealed necrosis of both erythroid and myeloid series with fibrin exudation randomly scattered through out.

Other lesions consisted of diffuse necrohemorrhagic laryngotracheitis and rhinitis, ulcerative stomatitis/pharyngitis, glossitis and ingluvitis. Mild to moderate bronchopneumonia, enteritis, conjunctivitis, necrosis of lymphocytes in thymus and bursa of Fabricius, encephalitis, myocarditis and erythrophagocytosis in the spleen nd liver were was also present.

Virology: Avian Paramyxovirus – 1 from pooled tissues (trachea, lung, spleen and cecal tonsils) was isolated and characterized as exotic new castle disease virus.

[1] Animal Disease Diagnostic Laboratory, West Lafayette, IN
* Presenter