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Experimental Pathology Laboratories, Inc.
Sterling, VA 20166

CASE I – N04-188 (AFIP 2937770)

Signalment: 27 year-old, female, mongoose lemur (*Eulemur mongoz)*, non-human primate.

History: The lemur presented with a history of rapidly progressive neurological symptoms, marked polyuria and polyphagia. A neurological examination revealed a wide-based stance, circling, ataxia and falling to the right, as well as a weak grip in the right hand and right foot. Cranial nerve reflexes and anal tone were within normal limits. Additional findings included patchy truncal alopecia and a cataract in the left eye. The very thin and debilitated lemur was euthanized due to poor prognosis and advanced age.

Gross Pathology: The submitting veterinarian reported multifocal to coalescing atheromatous plaques on the intima of the proximal abdominal aorta. On gross examination of the brain after formalin fixation, there were multifocal random (approx. 10), pinpoint to 1 mm, red-brown foci in the gray and white matter of the cerebrum, cerebellum and brain stem.

Laboratory Results: Serial serum chemistry profiles revealed marked persistent hyperglycemia and on the day of euthanasia, the blood glucose was >700 mg/dL and the urine glucose was >500 mg/dL.

Contributor's Morphologic Diagnosis: Brain: 1) Severe, regionally extensive to diffuse, granulomatous meningoencephalitis with intralesional myriad fungal yeast forms (*Cryptococcus* sp.).
2) Mild to moderate, multifocal atherosclerosis with vascular thrombosis and acute and chronic infarcts.

Contributor's Comment: The brain contains a combination of lesions that are associated with persistent diabetes mellitus, namely exacerbation of atherosclerosis with vascular thrombosis and opportunistic fungal infection.[1] The fungal

meningoencephalitis dominates the tissue changes. The yeast forms within these lesions are highlighted with Gomori's methenamine silver (GMS) stain and have morphology consistent with *Cryptococcus*. (Although cultures were not performed, infection with *C. neoformans* is suspected.) The precise mechanisms associated with susceptibility to infection and immune compromise in diabetic patients is unclear, but appear to involve both humoral and cellular mechanisms.[2] In human patients with diabetes mellitus, the common opportunistic fungal infections include sino-orbital aspergillosis, rhinocerebral mucormycosis, and cryptococcal meningitis.[3]

Vascular thromboses and/or infarcts are present in the majority of brain sections; some lesions are prominent and acute, while others are subtle and may be evidenced by focal accumulations of hemosiderin. The cause of vascular disease in diabetes mellitus is due in part to accelerated atherosclerosis, hypertension, and thickening of small vessels (microangiopathy).[1] Vascular thromboses and infarcts in the brain are recognized as a complication of diabetes mellitus; although, there is evidence that cerebral infarction may occur in cases of chronic meningitis.[4,5]

AFIP Diagnoses: 1. Brain, cerebrum: Meningitis, granulomatous, multifocal, moderate, with numerous yeast, etiology consistent with *Cryptococcus neoformans*, mongoose lemur (*Eulemur mongoz)*, primate.
2. Brain, cerebrum: Infarcts, multifocal, acute and chronic.

Conference Comment: *Cryptococcus neoformans* is a saprophytic fungus that causes disease in a wide variety of animals, but most frequently in cats, dogs, horses and humans. Infected animals are often immunosuppressed. Lesions can occur in any organ, but are most common in the central nervous and respiratory systems, followed by the integumentary system and eyes. *Cryptococcus neoformans* is a cause of mastitis in cattle. Cryptococcosis is the most frequent systemic mycoses in cats, and often occurs in the nasal cavity.

Gross lesions of *Cryptococcus neoformans*, are usually gelatinous due to the organism's mucopolysaccharide capsule. The capsule hinders phagocytosis and is a major diagnostic feature of the organism. However, acapsular forms do exist. Histologically, the yeasts are round, 5-20 µm in diameter, reproduce by narrow-based budding and are usually surrounded by a 2-8 µm mucopolysaccharide capsule that stains with mucicarmine and Alcian blue.[6,7] The immune response varies from sparse to granulomatous depending on the presence of a capsule and the host's immune status.
Chronic diabetes mellitus (DM) results in immunosuppression and damage to multiple organ systems, especially the kidneys, eyes, nerves, and blood vessels. In humans, diabetic macrovascular disease is a common complication of DM and is characterized by accelerated atherosclerosis that often leads to infarction. Atherosclerotic vessels were not evident in the sections of brain examined during the conference, which is attributed to variation among slides noted by the contributor.

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CASE II – ND-1 (AFIP 2935562)

Signalment: 1-year-old female domestic shorthair cat (*Felis domesticus*).

History: This animal developed a sinus infection following surgery (procedure not specified). Following antibiotic therapy the cat improved for a week then regressed to prior condition. The owners requested euthanasia.

Gross Pathology: There was marked, bilateral, pulmonary stiffness with mottling of the tissue. Fibrin strands were present on the pleural surface. Mucopurulent exudate was present at the opening of both nares.

Laboratory Results: Aerobic culture of lung resulted in no growth. Fluorescent antibody (FA) examination of frozen sections of lung was positive for feline herpesvirus-1.

Contributor's Morphologic Diagnosis: Pneumonia, necrotizing and fibrinous, diffuse, acute, severe with intranuclear eosinophilic inclusion bodies.

Contributor's Comment: Feline herpesvirus-1 (FHV-1) infections in cats typically result in keratoconjunctivitis, upper respiratory tract disease and abortion. Along with *Chlamydophila psittaci* and feline calicivirus, FHV-1 is part of an upper respiratory disease complex referred to as "feline rhinopneumonitis". Young animals are more severely infected. Transmission is accomplished through direct contact with infectious secretions or excretions. Vaccination does not prevent illness, but will limit clinical signs. The disease tends to be more common where crowding, poor management and carrier animals exist.

Acutely infected animals shed large amounts of virus for several weeks. The virus is typically transmitted horizontally, but can be passed from mother to fetus as well. Most infections are restricted to the upper respiratory tract; however, less common involvement of the trachea and lung can occur. Systemic infections can lead to reproductive tract disease and reproductive failure. Kittens infected *in utero* may be aborted or delivered normally and develop "fading kitten" syndrome. More typical presentations of the disease result in sneezing, oculonasal discharge, rhinitis, conjunctivitis, fever, anorexia, ulceration of the tongue and hard palate and herpetic keratitis. Healthy cats recover in one to two weeks.

Multifocal necrosis typical of herpesvirus infections may be seen microscopically, often leading to secondary bacterial infections. Acidophilic intranuclear inclusion bodies can be detected up to a week after infection. This case is an example of the less common necrotizing pneumonia seen in fulminant viral infections.

AFIP Diagnosis: Lung: Bronchopneumonia, necrotizing, acute, diffuse, severe, with syncytia and epithelial eosinophilic intranuclear inclusion bodies, domestic shorthair, feline.

Conference Comment: The contributor gives a thorough overview of feline herpesvirus-1. Although upper respiratory tract infections are common in cats, pneumonia is uncommon except when there is immunosuppression.[4] Some differentials that conference attendees considered for pneumonia in a cat included feline calicivirus, *Toxoplasma gondii*, aspiration pneumonia, and toxins.

Feline caliciviral disease in cats has clinical and pathological similarities to feline herpesviral infection. Clinical signs include oculonasal discharge, rhinitis, conjunctivitis, and ulcerative stomatitis. Feline calicivirus has an affinity for epithelium, and additional lesions include interstitial pneumonia and necrotizing bronchiolitis. The primary viral infection is transient, but secondary bacterial infections are common.[4]

Toxoplasmosis is a worldwide disease that is often triggered by immunosuppression and affects humans, dogs, cats, and many wild mammals. Pulmonary lesions include severe necrotizing interstitial pneumonia, with prominent proliferation of type II pneumocytes and infiltrates of histiocytes and neutrophils.[4]

Aspiration pneumonia, resulting from vomiting, regurgitation, dysphagia, or anesthetic complication, is a common condition in cats. Lesions may be unilateral or bilateral and most often affect the right cranial lobe. Histologically, the severity of the lesion often depends on the chemical and microbial composition of the aspirated material.[4]

Toxins, such as paraquat, a broad-spectrum herbicide, can cause severe and often fatal interstitial pneumonia in dogs, cats, humans and other species. Gross lesions include interstitial emphysema, bullous emphysema and pneumomediastinum. Histologically, there is extensive necrosis of alveolar epithelial and endothelial cells, interstitial and alveolar edema, intraalveolar hemorrhage and proliferation of type II pneumocytes.[4]

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CASE III – 04-0825 (AFIP 2936447)

Signalment: 14-month-old female Angus, *Bos taurus*, bovine

History: This cow was from a herd of 32 cattle maintained on a partially forested pasture in west-central Georgia. The cattle had been purchased in October 2003 and had shown no clinical signs of disease until April 2004, when two adult cows were found dead without precipitating clinical signs. This animal displayed depression, ataxia, anorexia, and ptyalism. Clinical examination revealed central blindness, tremors, and reduced rumen motility. Therapy included thiamine, vitamin B_{12}, oral electrolytes, and antibiotics. The cow was euthanized for diagnostic purposes. Differential diagnoses included polioencephalomalacia (PEM), thromboembolic meningoencephalomyelitis (TEME), plant toxicosis, infectious bovine rhinotracheitis (IBR), rabies, and lead toxicosis.

Gross Pathology: The body of a 14-month-old female Angus bovine in good body condition weighing 635 kg was presented for necropsy. Excessive saliva was present in the oral cavity. A thick cloudy discharge exuded from the nares. The reticulum and rumen contained approximately 10-15 liters of white to light-green fibrous contents, which incorporated many angular irregularly shaped fragments of metal measuring 5-15 mm. Upon further examination, metal fragments were found not to be attracted to a magnet. Scattered pieces of black plastic were scattered throughout the rumen contents.

Laboratory Results:
Clinical Pathology:
WBC: 14,990 cells/_l
Neutrophils: 5,097 cells/_l (some toxic neutrophils present)
Lymphocytes: 8,095 cells/_l
Basophils: 300 cells/_l

Fibrinogen: 700 mg/dl
Aspartate aminotransferase (AST): 317 U/L
Creatine kinase (CK): 3,631 U/L
Calcium: 8.2 mg/dl
Phosphorus: 11.3 mg/dl
Magnesium: 1.5 mg/dl
Glucose: 80 mg/dl
Iron: 175 mg/dl

Fluorescent antibody testing of brain for rabies virus: Negative
Lead analysis of kidney: 85ppm (wet weight)
Arsenic analysis of kidney: <1ppm (wet weight)
Lead analysis of metallic rumen contents: 17%

Contributor's Morphologic Diagnosis: Neuronal necrosis, acute, locally extensive (laminar), severe, with capillary endothelial hypertrophy and astrocytosis, cerebral cortex.

Contributor's Comment: Lead levels in the kidney of this case (85 ppm) were within the reported toxic range for cattle (5-700 ppm).[1] A diagnosis of acute lead toxicosis was confirmed by the presence of lead-containing metal fragments within the rumen and a laminar pattern of neuronal necrosis and capillary endothelial swelling within the cerebral cortex. Further examination of the pasture revealed the recently destroyed remnants of an automotive battery. Acid-fast intranuclear inclusion bodies were demonstrated within the renal tubular epithelial cells.

Cattle are usually exposed to lead through the ingestion of automotive batteries, petroleum products, roofing felt, or lead-based agricultural products. Clinical signs are indicative of central nervous system dysfunction, and include depression, tremors, ataxia, blindness, seizures, and dementia.[2] Gross lesions are uncommon after acute lead toxicosis. Histologically, lesions include laminar cortical necrosis of the cerebrum with swelling of capillary endothelium, cerebral edema, congestion, and astrogliosis. These lesions are thought to reflect ischemic-anoxic injury, and are therefore not specific for lead toxicity. Ischemia may be induced by capillary endothelial swelling, the pathogenesis of which is not understood. Lead does not accumulate appreciably in CNS tissue. Renal tubular epithelial necrosis is evident in some cases; tubular epithelial cells often contain acid-fast intranuclear inclusion bodies.[3]

Lead is present in the environment in three forms: metallic lead, lead salt, and organic lead.[2] Many man-made products incorporate metallic lead or lead salts; these include automotive batteries, lead weights, lead-based paints, lead shot, various plumbing waste products, computer equipment, and lead arsenate pesticides.[1,2] The industrial process of lead smelting has resulted in livestock exposure through the airborne contamination of pasture. Organic lead (tetraethyl- and tetramethyl-lead) is found primarily in leaded petroleum products.

The toxicity of lead is attributable to multiple mechanisms, including binding and inactivation of enzymatic sulfhydryl groups, competition with calcium ions, and alteration of vitamin D metabolism. Sulfhydryl binding is most evident in enzymes involved in heme synthesis, such as gamma-aminolevulinic acid dehydratase (ALAD) and ferrochelatase; hence, toxicity often results in red blood cell abnormalities. Inhibition of heme synthesis may contribute indirectly to neurological abnormalities through the increased production of serotonin, resulting in aberrant neurotransmission in the brain. Lead competes with calcium ions in bone resulting in the formation of lead precipitates ("lead line") in the long bones of young animals. Lead also competes with calcium at the neuromuscular junction, resulting in tremors and paresis.[2]

The absorption of lead is dependent on its form and the route of exposure. In general, organic lead is better absorbed than lead salts and metallic lead. Ingested lead is most readily absorbed from the acidic environment of the stomach, yet the majority of ingested lead is passed in the feces. Once absorbed, more than 90% of absorbed lead is bound to erythrocytes, making whole blood the tissue of choice for the clinical diagnosis of lead toxicosis. Blood lead levels in excess of 0.6 ppm (60 _g/dl) are diagnostic for lead toxicosis. Other tests include the detection of reduced blood ALAD activity (<50 nmol porphobilinogen/ml erythrocytes/hr in adult cattle) or excessive urinary ALA levels (>500 _g/dl).[2] Upon post-mortem examination, kidney, liver, and bone harbor the highest concentrations of lead. As in this case, liver and kidney levels in excess of 10 ppm (wet weight) are considered diagnostic for lead toxicosis.[1,2]

AFIP Diagnosis: Brain, cerebrum: Neuronal necrosis, laminar, with gliosis and hypertrophic endothelial cells, Angus, bovine.

Conference Comment: The majority of the sections exhibit laminar neuronal necrosis in the middle to deep layers of the cerebral grey matter. Additionally, scattered throughout all layers, there is neuronal necrosis evidenced by shrunken, angulated, hypereosinophilic and often karyorrhectic or pyknotic neurons.

The contributor gives a thorough and complete overview of lead toxicity in cattle. Lead toxicosis, or plumbism, has been reported in many different mammals, birds, and reptiles.[2] Unlike cattle, equine lead poisoning is usually chronic and affected individuals characteristically present with laryngeal and pharyngeal paralysis, and ingestion of large amounts can produce generalized paralysis.[3] Lead toxicity is not uncommon in humans. Humans are exposed to lead by two main routes, occupational and non-occupational. Occupational exposures include inhalation during spray painting, foundry work, mining and extracting lead, and battery burning. Non-occupational exposures, which may be more difficult to track in relation to individual exposure, include ingestion of contaminated water due to lead plumbing, lead solder in food and soft drink cans, paint dust and flakes in homes with interior lead paint, soil contaminated with exterior lead paint, newsprint, automotive exhaust, and illegally produced alcoholic beverages (moonshine).[4]

Similar to cattle, intestinal absorption in humans is enhanced by calcium, iron, or zinc deficiency. Children have a greater absorption capacity than adults and thus are more vulnerable to lead toxicity. Absorbed lead clears rapidly from the blood, but is often deposited in bones where it has a half-life of 30 years. Therefore, high blood lead levels indicate a recent exposure but are not indicative of total body burden.[4]

As with cattle, lead toxicosis is a multisystemic disease in humans, affecting the central and peripheral nervous systems, hematopoiesis, gastrointestinal system, kidneys, and bones. Symptoms include headache, dizziness, memory deficits, decreased nerve conduction velocity, a microcytic hypochromic anemia with characteristic basophilic stippling of erythrocytes, colic, anorexia, and damage to the proximal renal tubules with classic intranuclear lead inclusions. Chronically, lead may cause diffuse interstitial fibrosis, gout and renal failure, in addition to infertility and hypertension.[4]

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CASE IV – 04F36 (AFIP 2935878)

Signalment: Adult 4-6 kg Chinook salmon (*Oncorhynchus tshawytscha*), mixed gender.

History: During June, 2004, mortalities at a salt water net pen facility along the coast of British Columbia increased from 0.5 to 10% over a week span. These fish had previously had episodes of low-grade bacterial kidney disease and sporadic losses due to vibriosis (*Listonella anguillarum*).

Fish initially presented with inappetence and lethargy, which progressed over 1-2 days to listlessness, opercular flaring, swimming high in the water column and increased mortality.

Gross Pathology: The most significant lesions involved the gills. Involving multiple gill arches, randomly throughout the primary, and to a much lesser extent secondary lamellae, there were 2-3 mm diameter, firm, glistening white to grey spherical cysts with scattered foci of acute hemorrhage. Within a small proportion of fish, there were miliary granulomas within the anterior and posterior kidneys as well as scattered foci of acute hemorrhage within the mesentery.

Laboratory Results: Aerobic culture yielded variably mixed isolates of *Vibrio* spp, *Pseudomonas* spp, and *Escherichia spp*. Immunofluorescence of pooled liver, kidney and spleen was positive for *Renibacterium salmoninarum* and polymerase chain reaction of pooled gill tissue was positive for *Loma salmonae*.

Contributor's Morphologic Diagnosis: Gills: Branchitis, moderate, multifocal to coalescing, fibrinohemorrhagic and necrotizing, acute, with telangiectasia, respiratory epithelial hyperplasia and intralesional protozoal xenomas morphologically consistent with *Loma salmonae*.

Contributor's Comment: In British Columbia, because of the emergence of infectious hematopoietic virus (IHNV) and *Kudoa* spp in farmed Atlantic salmon (*Salmo salar*), producers are opting to further domesticate and raise Pacific salmon species (*Oncorhynchus* spp). With expansion and intensification of Pacific salmon aquaculture, there has been an emerging concern about a branchial microsporidian parasitic infection, *Loma salmonae*. This parasite has a narrow host range and is a recognized pathogen of Chinook (*O. tshawytscha*) and to a much lesser extent, Coho salmon (*O. kisutch*). Atlantic salmon, brook trout (*Salvelinus fontinalis*) and Arctic char (*Salvelinus alpinus*) appear refractory to experimental infection.

Parasites are acquired through ingestion of infective stages. The sporoplasm either invades between or through enterocytes, localizing within the lamina propria by 24 hours post infection. In experimental models of rainbow trout (*O. mykiss*) maintained at 15C, the merogonic stages are consistently detected within the heart as early as 2 days post infection and remain for 2 weeks.[1] After 2 weeks, and up to 5 weeks post infection, parasitic xenomas (which have undergone sporogony) are detected within the gills and spleen.

Electron microscopy has revealed uni- and binucleate meronts in sub-intimal host cells of the capillary channels of secondary lamellae and lamellar arteries. Involvement of phagocytic pillar cells has also been reported. Localization to these cells may be due to embolism, receptor mediated internalization, or evasion (by directly transferring from monocytes or lymphocytes). Respiratory distress is attributed to dissolution of the xenoma with inflammation directed against the chitin-rich wall of spores, and necrosis. Horizontal transmission is believed to result from rupture of the xenomas.

Temperature has a profound influence on the kinetics of infection; at 5C or 20C xenoma formation is interrupted, but branchial infections can still occur. At 5C, parasites localize to the heart in 7 days with no detectable xenomas within the gills to 4 weeks post

exposure. At 20C, parasites are detected within the spleen, heart, and gills 3 days post exposure with no apparent xenoma formation. Maintaining fish in ambient water temperatures less than 5 C and greater than 20C appears to interrupt the life cycle of this parasite and stock that recover are resistant to re-infection.[2] Ectoparasitic copepods, such as sea lice (*Lepeophtheirus salmonis*) appear to enhance susceptibility of stock to *Loma salmonae* infection.

At present there is no recognized treatment for *Loma salmonae* infection. Efforts are currently underway for vaccine development and possible management schemes to limit development of clinical disease.

AFIP Diagnosis: Gill: Branchitis, necrotizing and proliferative, multifocal, moderate, with mucus cell metaplasia, and numerous protozoal cysts, Chinook salmon (*Oncorhynchus tshawytscha*), piscine.

Conference Comment: Microsporidians are generally taxonomically specific intracellular parasites with a direct life cycle. After spore ingestion by the host, the sporoplasm is discharged and migrates to the target organ, begins the proliferative phase (merogony), producing large numbers of cells (meronts) by binary and multiple fission. Meronts then give rise to sporonts that undergo sporogony, producing mature spores. These spores are then released from lesions on the body surfaces or after death of the host.[3]

Nodular microsporidial lesions may grossly resemble those of other pathogens including myxozoans, ich, lymphocystis, and dermal metacercariae, bacterial granulomas, or neoplasia. However, these can easily be differentiated histologically. Microsporidial spores are typically 7 µm or less, egg-shaped to elliptical, and contain a posterior vacuole. Myxozoans, except during autogamy (sexual reproduction), all have multinucleated forms that have enveloping (primary) cells that contain enveloped (secondary) cells.[3] Their spores contain two polar capsules which stain intensely blue with Geimsa.[6] Ich, or white spot disease, is caused by *Ichthyophthirius multifilis*. The trophonts are large and characterized by a uniform layer of external cilia and a unique horseshoe-shaped macronucleus.[4] Lymphocystis is caused by a piscine iridovirus that preferentially infects dermal fibroblasts and inhibits mitosis, producing tremendous cellular hypertrophy.[5] Dermal metacercariae of digenean trematodes, result in white to yellow or black raised nodules that contain the parasite.[3]

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