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Department of Veterinary Pathology
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Silver Spring, MD 20910-7500

CASE I – 01-3206 (AFIP 2790822)

Signalment: 7 year old, castrated male, Golden Retriever mix, canine

History: Two months duration of intermittent vomiting and voluminous, mucoid to hemorrhagic diarrhea with associated tenesmus, urgency, and flatulence, weight loss, anorexia, and lethargy. Dog had access to a large, unfenced area containing ponds and drainage ditches bordering a suburban residential development. Physical examination revealed a moderately enlarged popliteal, submandibular, prescapular, and inguinal lymph nodes and melena.

Gross Pathology: Via laparotomy: Diffusely thickened, firm to sclerotic pancreas; uniform, moderate hepatomegaly; generalized mesenteric lymph node enlargement; multifocal, transmural nodules throughout large and small intestine.

Laboratory Results: CBC – mild, normocytic, normochromic, non-regenerative anemia (PCV = 30), mild increase in band neutrophils (900/μl: range 0-300/μl)

Serum Biochemistry Profile – hyperglobulinemia (7.2 g/dl: range 2.5-4.3 g/dl), increased serum alkaline phosphatase (ALP) (269 IU/l: range 20-155IU/l), increased serum aspartate aminotransferase (AST) (55 IU/l: range 1-37 IU/l), increased serum alanine aminotransferase (85 IU/l: range 3-5- IU/l), E. canis and Lyme serology – negative, Rocky Mountain Spotted Fever titer – 1:1024, Urine specific gravity – 1.030, 3+ proteinuria

Lymph Node Aspirate – popliteal and prescapular nodes: reactive lymphocyte hyperplasia

Fecal Float - negative

Contributor's Morphologic Diagnosis: Multifocal, severe, coalescing, granulomatous, transmural enteritis with villous blunting and fusion, dilated lacteals, and numerous, intralesional, schistosome ova

Contributor's Comment: The diagnosis of heterobilharziasis, caused by *Heterobilharzia americana*, considered the primary blood fluke of dogs, was confirmed by performing direct and saline sedimentation of feces (ova are difficult to recover by fecal floatation) and a miracidia hatching technique described by Goff and Ronald. An ELISA for schistosomiasis commonly used in human medicine was used in this case with positive results. The test may have application in the ante-mortem diagnosis of heterobilharziasis. The test can be performed by Dr. Bruce Hammerburg at North Carolina State University (919-513-6226).

Histologically, granulomas and numerous schistosome ova were scattered throughout the liver, pancreas, small intestine, colon, and a mesenteric lymph node. The stomach was least affected with only a focal microgranuloma in the submucosa and no ova seen. The ova had a round to ovoid, thick, ~100 um, yellowish to clear hyalinized wall, often collapsed and were either empty or contained a developing miracidium. The inflammatory cell infiltrate was similar in all involved organs and composed of single to coalescing granulomas composed of epithelioid macrophages and multinucleate foreign-body giant cells, usually surrounding parasitic ova, admixed with varying numbers of eosinophils, neutrophils, lymphocytes, and plasma cells. Ova and the granulomatous inflammation were distributed transmurally in the small intestine, portally in the liver, and randomly throughout 50% of the pancreatic specimen. Minimal inflammation was present in the colon and a mesenteric lymph node. There were numerous pigment-laden macrophages in the liver. The pigment likely represents blood pigment, a waste product of the flukes, which consume red blood cells.

The life cycle of *H. americana* is complex. The ova shed in the feces begin to hatch in approximately 30 minutes, releasing the highly motile miracidia. The miracidia then infect the intermediate host, one of two susceptible species of snail (*Lymnea cubensis* and *Pseudosuccinea columella*), where asexual reproduction occurs. The cercaria, released from the snail into the water, then penetrate the skin of a definitive host that enters the water, with the aid of a proteolytic enzyme. In the definitive host, the flukes mature and infect the portal and mesenteric veins, where they reside in copula and reproduce sexually. The morphology of the ova and distribution of the lesions in the abdominal viscera is typical of that previously reported in the dog and raccoon. Oviposition usually occurs in the intestinal venules and for those ova that don't make it to the intestinal lumen, many of them are hematogenously disseminated to distant sites. The pattern of liver disease is likely due to spread of eggs from intestines via portal circulation. In the liver, cirrhosis can result in longstanding cases of schistosomiasis.

This case, the first reported in North Carolina, is only the second successfully treated dog (30 mg/kg of praziquantel) out of 10 cases in dogs reported in the literature. The definitive hosts for *H. americana*, other than the domestic dog, include the bobcat, nutria, swamp rabbit, opossum, white-tailed deer, wild canids such as coyotes and red wolves, and the natural definitive host and most important reservoir host, the raccoon. The range of *H. americana* in the United States is limited to the southeast as far north as North Carolina, and as far west as central Texas. The trematodes are found in Kansas as well, after transplantation of raccoons from Texas by a local hunting club.

AFIP Diagnosis: Small intestine: Enteritis, granulomatous, diffuse, moderate, with villar blunting and fusion, crypt loss and regeneration, and numerous trematode eggs, Golden Retriever mix, canine.

Conference Comment: *Heterobilharzia americana*, the cause of North American schistosomiasis, is a member of the family Schistosomatidae which includes the genera Schistosoma, Heterobilharzia, and Orientobilharzia. Distinctive features of schistosomes include non-operculate eggs and the presence of two separate sexes (hermaphroditism being the general rule in trematodes). The *Heterobilharzia americana* adult male is typically stout; the female slender. The larger male can grip multiple smaller females in a gynecophoric canal.

Although not seen in this case, hypercalcemia has been reported to occur in cases of schistosomiasis. A recent report describes hypercalcemia with a concurrent elevation in parathyroid hormone-related protein (PTHrP).

Contributor: North Carolina State University, College of Veterinary Medicine, Raleigh, NC 27606

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CASE II – 7006, 3003, 8006, 7007 (AFIP 2787591)

Signalment: 10-month-old, Male, Fischer 344 rats

History: These rats were in a surgically induced model (unilateral ligation, combined with unilateral nephrectomy) of chronic renal failure

Gross Pathology: At necropsy, nephrectomized rats were thin, dehydrated and had rough haircoats. Renal lesions (infarction, mineralization and nephropathy) were consistent with the model and model-induced exacerbation of spontaneous nephropathy. Other model-related findings included parathyroid hypertrophy; metastatic mineralization characterized by firm, white streaks in the myocardium and enlarged, firm vessels (most pronounced in the aorta); and uremic gastritis. Periarteritis nodosa was also a consistent finding in uremic rats and was most evident in the mesenteric and peripancreatic vessels.

Laboratory Results: Clinical pathology findings (anemia, markedly elevated BUN and creatinine, decreased total protein and albumin, and alterations in electrolytes) were consistent with renal failure, as expected.

Contributor's Morphologic Diagnoses: 1. Bone; moderate to marked fibrous osteodystrophy (all rats)
2. Bone marrow; slight to moderate hypercellularity (not noted in Rat 8006)

Contributor's Comment: Fibrous osteodystrophy associated with hyperparathyroidism has been described previously in the F344 rat, secondary to age-related spontaneous degenerative kidney disease. Both cortical and trabecular bone may be affected. Fibrous osteodystrophy is characterized by extensive osteoclastic resorption of bone and formation of fibro-osseous tissue, caused by prolonged and extensive secretion of parathormone secondary to renal failure. Morphologic alterations include increased numbers of osteoblasts along the trabecular bone, an increased number of osteoclasts resulting in increased bone resorption, thinning and loss of trabecular bone, and proliferation of fibrous connective tissue in the marrow space. The reference listed below (Hruska and Teitelbaum) is an excellent review of the complex processes involved in the development of renal osteodystrophy (fibrous osteodystrophy, osteomalacia, adynamic renal bone disease and mixed disease).

In this study, minimal to moderate bone marrow hypercellularity was also noted in some uremic rats. Photomicrographs supplied to the AFIP contrast an age-matched control rat, with one of the submitted uremic rats, illustrating the relative decrease in marrow adipose tissue and increased hematopoietic precursors, predominantly erythroid. Three of the rats in this submission (not noted in Rat 8006) had erythroid hyperplasia, consistent with regenerative anemia. The anemia was characterized by decreased erythrocytes, macrocytosis, hypochromasia, reticulocytosis, and polychromasia. These changes were attributed to gastrointestinal blood loss and hemolysis secondary to uremia.

AFIP Diagnosis: Femur, tibia, and sternebra: Fibrous osteodystrophy, Fischer 344 rat, rodent.

Conference Comment: Mineral homeostasis and bone formation is a complex process involving the regulatory effects of parathyroid hormone (PTH), thyroid hormone, calcitonin, and calcitriol. Further complicating the process are the roles of cytokines and factors such as interleukins, colony stimulating factors, growth factors, and tumor necrosis factor alpha (TNF-a) which are involved in both osteoblast and osteoclast activation, proliferation, and differentiation.

In normal bone remodeling, PTH, IL-1, and TNF-a stimulate osteoblasts to release collagenase and tissue plasminogen activator; deposit latent transforming growth factor B (TGF-B) in bone matrix; and secret soluble factors. These soluble factors, which include RANKL, M-CSF, GM-CSF, IL-6, and IL-11, stimulate the proliferation and activation of osteoclasts. Collagenase and tissue plasminogen activator release osteopontin, bone sialoprotein, and collagen fragments. Osteopontin and bone sialoprotein contain specific amino acid sequences, recognized by osteoclast integrins, which may direct chemotaxis and allow adherence.

Active osteoclasts resorb bone resulting in increased calcium levels and the release of latent TGF-B. Sufficient levels of local calcium have an inhibitory effect on osteoclasts. TGF-B decreases osteoclast activity as well, but it also (along with other growth factors) recruit osteoblasts into the resorption site and activates them. New bone formation is initiated with the eventual deposition of lamellar bone.

With respect to fibrous osteodystrophy secondary to renal disease, abnormal calcium and phosphorous handling by the kidney as well as decreased production of calcitriol results in decreased ionized calcium and a subsequent increased and continuous production of PTH. Osteoclast activity is increased; osteoblastic activity is also increased but results in woven bone rather than lamellar bone; and fibrous connective tissue is deposited in the marrow cavity by activated fibroblast like cells. The net result is decreased bone mass in long bones.

Contributor: Lilly Research Laboratories, Greenfield, IN 46140

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CASE III – D95-151 (AFIP 2548595)

Signalment: 4-month-old, Domestic longhair, intact male, feline

History: The animal was presented to the Tuskegee University School of Veterinary Medicine's small animal clinic with a history of polyuria/polydipsia. The owner also stated that the animal was not using the litter box and was urinating all over the house and its cage. This had been going on for approximately one week. The animal had soft stools, was depressed, and lethargic. Blood and urine were submitted for laboratory analysis. All clinical signs, hematology, serum chemistry, and urinalysis were consistent with Diabetes Mellitus and the owner requested euthanasia of the animal.

Gross Pathology: Excessive amounts of fat were deposited in normal fat depots: i.e. subcutaneous, pericardial, omental, and perirenal. The liver was enlarged and bronze. The pancreas was mottled.

Laboratory Results: Hematology: elevated erythrocyte count and PCV.

Serum chemistry: glucose (411 mg/dl), ALP (70 IU/L), ALT (116 IU/L), CK (400 IU/L), T bili (5.0 mg/dl), TP (10.3 g/dl); BUN and creatinine were normal.

Urinalysis: SG (1.046), protein (3+), glucose (4+), pH (6.0), positive occult blood

Contributor's Morphologic Diagnosis: Vacuolar degeneration of Islet cells, diffuse, with focal infiltration of lymphocytes, moderate to severe, pancreas, cat.

Contributor's Comment: These sections of pancreas are characterized by enlargement of the islets. The enlarged islets are composed of swollen cells with large, discrete cytoplasmic vacuoles. The nuclei of these cells are condensed and generally eccentric. A few normal appearing islet cells persist in some islets. In some sections an infiltrate of small lymphocytes is adjacent to a swollen islet. Some sections also contain multiple microscopic foci of fat necrosis and mineralization.

In man and most animal species, Diabetes mellitus (DM) is classified into two basic types. Type I (insulin dependent) usually occurs in younger individuals with an absolute insulin deficiency. Type II (non-insulin dependent) usually occurs in older individuals with a relative insulin deficiency. In cats, DM is typically associated with characteristic degenerative lesions in the islets, while the remainder of the pancreas is within normal limits. The most common feline islet lesion is amyloid deposition. Feline insular amyloidosis is very similar to human Type II DM. The other specific islet cell lesion (vacuolar degeneration) has been reported in cats that are resistant to large doses of exogenous insulin and is believed to develop as an exhaustion phenomenon. The vacuoles in these cases contain large amounts of glycogen that has resulted in displacement of organelles toward the periphery of the cell. However, infections with certain viruses may cause selective islet cell damage that is associated with inflammatory infiltrates composed of lymphocytes and macrophages. Degenerative insular lesions in humans with Type I DM are frequently associated with heavy infiltrates of T lymphocytes with CD4+ and CD8+ markers in and about the islets. This case presented in a young cat that had a clinical course and degenerative islet cell lesions resembling those of Type I DM in man.

AFIP Diagnosis: Pancreas, islets: Degeneration, vacuolar, diffuse, moderate, Domestic Longhair, feline.

Conference Comment: Although islet cell vacuolar degeneration is characteristic of diabetes mellitus (DM), islet amyloidosis is a more common finding, with up to 80% of spontaneously diabetic cats developing the lesion. Ninety percent of humans with type-II DM and 100% of diabetic cynomolgus macaques develop islet amyloidosis.

Islet amyloidosis is derived from islet amyloid polypeptide (amylin), which is a normal secretory product of islet B cells and is co-secreted with insulin. Amyloid deposition in the islets occurs before the onset of clinical signs; the amino acid sequence between positions 20 and 29 of the amylin molecule predispose it to fibril formation. The exact pathogenesis of amylin in DM is unclear but it is thought that amylin acts both directly and indirectly on B cells and is associated with significant loss of islet B cells. Recent reports suggest the role of amylin in the induction of islet B cell apoptosis. Ultrastructural findings such as membrane blebbing,

chromatin margination, nuclear shrinking and cytoplasmic vacuolation support this theory.

Contributor: Tuskegee University, School of Veterinary Medicine, Tuskegee, AL, 36088

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CASE IV – A01-137 (AFIP 2787434)

Signalment: Captive born, 4-year-old, male rhesus monkey (*Macaca mulatta*)

History: This monkey was inoculated with SIVmac239 at 1 year of age. An outbreak of an upper respiratory infection, characterized by coughing, sneezing and a nasal discharge, occurred in two groups of monkeys. This animal was one of two SIV-infected monkeys that were found dead. *Streptococcus pneumoniae* was isolated from nasopharyngeal cultures from approximately 40% of the monkeys exhibiting signs of an upper respiratory infection of several days duration.

Gross Pathology: The monkey was thin. A scant pale yellow exudate was adhered to the omentum and mesentery, which were hyperemic. A similar exudate was adhered to the visceral tunica vaginalis of the testicles. The liver was moderately enlarged with rounding of the hepatic borders. The gall bladder wall was mildly and diffusely thickened; the bile was pale yellow and watery. The pleural surfaces of both lungs were mottled yellow-pink and red; the texture of the lungs was normal.

Laboratory Results: Adenovirus was isolated from nasopharyngeal and lung specimens obtained at necropsy. *Streptococcus pneumoniae* was isolated from cultures obtained from the peritoneal cavity, tunica vaginalis and bile. By immunohistochemistry, adenovirus-positive cells are present in the bronchial

epithelium, *Pneumocystis carinii*-positive organisms are seen in some alveolar spaces.

Contributor's Morphologic Diagnosis: 1. Lung: Bronchitis, suppurative and mucinous, acute, mild, multifocal with intralesional diplococci and basophilic intranuclear inclusion bodies, rhesus macaque (*Macaca mulatta*), nonhuman primate.
2. Lung, bronchi: Bronchial and submucosal glands, cryptosporidia, rare, multifocal.
3. Lung: Interstitial pneumonia with type 2 pneumocyte hyperplasia, mild to moderate, mulitfocal with intra-alveolar histiocytes and protozoa.
4. Lung, peribronchial arteries: Bacteremia.

(Note: The extent of the adenoviral bronchitis and alveolar pneumocystosis are variable depending on the section).

Contributor's Comment: SIV infection causes an AIDS-like disease in macaques. Commonly observed findings in macaques with SIV-induced disease include immunosuppression with depletion of peripheral blood CD4 positive T lymphocytes. Lymphadenopathy is frequently observed in the early stages of SIV infection while lymphoid depletion is characteristic of advanced disease. Wasting, peripheral blood cytopenias and opportunistic infections are common sequelae in SIV-induced disease.

The morphologic changes in the lungs are primarily due to opportunistic infections secondary to SIV-induced immunosuppression. Adenoviral infection of the lungs, pancreas and alimentary tract are not uncommon in immunosuppressed macaques. Pulmonary adenoviral infection generally results in a multifocal to coalescing, necrotizing bronchopneumonia; adenoviral infection confined to the bronchial epithelium, as demonstrated by immunohistochemistry in this case, is unusual. *Pneumocystis carinii* infection of the lungs is also a common opportunistic infection. Cryptosporidiosis is more commonly observed in the alimentary tract although involvement of the trachea, and less frequently the lungs, has been observed.

Fibrinosuppurative meningoencephalitis is the most commonly reported manifestation of *Streptococcus pneumoniae* infection in monkeys. However, *S. pneumoniae* can also cause pneumonia, polyserositis and/or septicemia in Old World monkeys and apes. *Streptococcus pneumoniae* has a propensity for serosal surfaces and colonizes the pleural lining, pericardial sac, peritoneal lining and/or meninges. A fibrinosuppurative exudate with intralesional diplococcal bacteria is the typical microscopic presentation due to infection with *S. pneumoniae*.

Streptococcus pneumoniae has been isolated from nasal and pharyngeal cultures from healthy macaques. Outbreaks of illness appear to be associated with

stress or occur as a secondary bacterial infection following viral-induced upper respiratory tract infections. Monkeys with primary septicemias may be found dead with no premonitory signs of illness. Clinical signs vary depending on the sites of bacterial colonization.

Streptococcus pneumoniae is generally not regarded as an opportunistic infection associated with SIV-induced disease. While most outbreaks of *S. pneumoniae* occur in monkeys not infected with SIV, immunosuppressed monkeys are at greater risk for developing infections.

Although this monkey had multiple opportunistic infections associated with AIDS, the cause of death was *S. pneumoniae* septicemia. Numerous Gram positive diplococci were seen within pulmonary blood vessels. Lesser numbers of extracellular diplococci were identified within the suppurate exudate of the bronchial lumina; rare diplococci were seen within the cytoplasm of intrabronchial macrophages. Although a blood culture was not obtained at necropsy, pure cultures of *S. pneumoniae* were isolated from the exudate in the abdominal cavity, tunica vaginalis and from the bile.

AFIP Diagnosis: 1. Lung: Bronchitis, proliferative, subacute, diffuse, mild, with basophilic intranuclear inclusion bodies and surface associated protozoa, protozoal etiology consistent with *Cryptosporidium parvum*, rhesus monkey (*Macaca mulatta*), nonhuman primate.
2. Lung: Alveolar histiocytosis, multifocal, mild, with intrahistiocytic and extracellular fungi, etiology consistent with *Pneumocystis carinii*.
3. Lung: Pneumonia, interstitial, acute, diffuse, mild, with intravascular diplococci and rare intra alveolar syncytia.

Conference Comment: The contributor has provided a concise discussion of this case. Phylogenetic classification of *Pneumocystis carinii* has been problematic for years. Until only recently, *P. carinii* was considered a protozoan; difficulty growing the organism in vitro, resistance to broad-spectrum antifungals, and response to treatment with anti-protozoal drugs supported this idea. Recent genetic sequencing of *P. carinii* has now placed it unequivocally in the kingdom Fungi. Although the precise taxonomic position is uncertain, it has been placed among the *Ascomycete*.

As a fungus, *P. carinii* is atypical. Typical fungal cell membranes contain ergosterol. With *P. carinii*, ergosterol is replaced by cholesterol (explaining the resistance to antifungals). Additional features of this atypical fungus include an insubstantial cell wall, little genetic material, and advanced mechanisms for varying surface antigens.

There are many different kinds of *Pneumocystis* organisms and considerable genetic divergence indicates that there are multiple species rather than different

strains. Individual species of *Pneumocystis* are believed to be host specific, as isolates from one host do not infect other host species.

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