The Armed Forces Institute of Pathology
Department of Veterinary Pathology
WEDNESDAY SLIDE CONFERENCE
2004-2005

CONFERENCE 17
16 February 2005

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Smithsonian National Zoological Park

CASE I – S03-2084 (AFIP 2936462)

Signalment: 3.5 year-old, male, Asian Elephant (*Elephas maximus*).

History: This juvenile elephant from the zoological garden, Zürich, refused to eat in the morning, showed signs of colic and depression and did not urinate. In the afternoon the tongue appeared cyanotic, the periorbital skin was swollen and the animal collapsed and died.

Gross Pathology: The main pathological changes were extensive hemorrhages particularly in the heart, the intestines and mesentery as well as in the subcutaneous tissues (Figs 1-3). The latter were also edematous in the head region. The myocardium, particularly the right atrium, was severely hemorrhagic and dark red. The liver was markedly swollen.

Contributor's Morphologic Diagnosis: Generalized severe petechial to extensive hemorrhage in nearly all organs and tissues. Intranuclear endothelial inclusion bodies (only in capillaries) in the heart, liver and tongue.

Etiology: Endotheliotropic Herpesvirus (Elephant herpesvirus-1)

Contributor's Comment: Histologically nearly every tissue was congested and most had areas of hemorrhage. There were early signs of hypoxic degeneration in the liver. Intranuclear inclusion bodies in the endothelium of the capillaries in the heart, liver and tongue were observed.

Death due to this disease typically occurs as a result of cardiac failure following herpesviral induced capillary injury and extensive myocardial hemorrhage. It is also

typical that inclusion bodies may be found in the endothelium of capillaries of the heart, liver and tongue, but never in larger vessels.

The death of an Asian elephant due to Elephant Endotheliotropic Herpesvirus was first described in a juvenile Swiss circus animal in 1990.[1] The animal presented here is the third juvenile elephant which has died as a result of a herpesvirus infection in Switzerland.

The significance of this disease for the survival of elephant populations in captivity became apparent after a retrospective study published a decade later revealed that deaths due to herpesvirus, but unrecognized at necropsy, had occurred in both Asian and African elephants as early as 1983.[2,3] So far, the virus has not been isolated. However, molecular methods have provided evidence that several newly identified herpesviruses are involved. The current theory is that otherwise healthy African elephants with hyperplastic lymphatic tissue in the genital tract and nodules in the skin and in the lung, which harbor herpesvirus, may be the source for a fatal infection in Asian elephants.[3,4,5] This obviously has great significance for the risk of disease transmission in connection with the translocation of animals.[6]

AFIP Diagnosis: Liver: Hepatocellular degeneration and necrosis, centrilobular, diffuse, mild, with congestion, hemorrhage, and endothelial cell eosinophilic intranuclear inclusion bodies, Asian elephant (*Elephas maximus*), proboscidea.

Conference Comment: The Asian and African elephant endotheliotropic herpesviruses (EEHV) are two novel, distinct, yet related viruses that are an important cause of fatalities in young Asian elephants, and less commonly cause death in African elephant calves [2]

Clinical signs include sudden onset of lethargy, anorexia, edema of the head, neck and thoracic limbs, cyanosis of the tongue, lymphopenia, and thrombocytopenia. Gross lesions include pericardial effusion with widespread petechial to ecchymotic hemorrhages primarily involving the heart, liver, intestine and tongue. Oral, laryngeal and intestinal ulcerations often occur. Histologically there are extensive microhemorrhages, edema, and mild lymphohistiocytic infiltrates throughout the heart and tongue. Congestion and hemorrhage cause hepatic sinusoidal expansion with mild hepatocellular degeneration, and endothelial cells of capillaries in the myocardium, tongue, and liver contain amphophilic to basophilic intranuclear inclusion bodies. Ultrastructurally, the inclusion bodies are 80-92 nm and morphologically consistent with other herpesviruses.[2]

Currently it is thought that the African elephant may be the reservoir of the herpesviruses that can cause disease in the two elephant species. African elephants carrying EEHV have typical herpetic lesions on the skin and vulva. Transmission is thought to be through intimate contact. However, direct proof of transmission has not been established. Nonetheless, it is currently recommended that Asian and African elephants be housed separately.[2]

The differential diagnosis for widespread necrosis and hemorrhage in elephants includes encephalomyocarditis virus, orbivirus, salmonellosis or other bacterial septicemia, and vitamin-E deficiency.[7] The Smithsonian National Zoological Park is developing PCR and ELISA tests to diagnose elephant herpesvirus infections.

Additional endotheliotropic viruses of veterinary importance include equine viral arteritis virus, equine Hendra virus, equine orbivirus (African horse sickness), cervid orbivirus (epizootic hemorrhagic disease), ovine orbivirus (bluetongue), hamster parvovirus, rat parvovirus (Kilham rat virus), canine adenovirus type 1 (infectious canine hepatitis), porcine adenovirus, bovine adenovirus, and adenovirus of deer.[2]

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CASE II – ND-2 (AFIP 2935568)

Signalment: 12-year-old American bison (*Bison bison*) cow.

History: The animal was found dead on pasture with no prior clinical signs of illness.

Gross Pathology: Pale, hemorrhagic nodules were present in multiple tissues: nearly all skeletal muscle groups (intercostals, epaxial and hypaxial groups, quadriceps, gluteals, semimembranosus, semitendinosus, deltoids, etc.), heart, liver, kidney, spleen, walls of the forestomachs, small and large intestines, lung and brain.

Laboratory Results: Specials staining of replicate sections of tumor tissue yielded the following results: Desmin +, Factor VIII -, trichrome -, vimentin +, PTAH +, myoglobin +, and muscle specific actin +. Electron microscopy of tumor samples showed cross striations consistent with myocyte origin.

Contributor's Morphologic Diagnosis: Disseminated rhabdomyosarcoma.

Contributor's Comment: Rhabdomyosarcomas are described as embryonal, botryoid, alveolar and pleomorphic. The embryonal variety is classified into two types, large round cell and myotubular. It has been reported in a variety of species, including cows and sheep. Botryoid rhabdomyosarcomas are a distinct entity most commonly found in the canine bladder. It is considered a variant of the embryonal type. The alveolar subtype has been reported in large and small animals, and shows a distinct histologic pattern of tumor cells supported by a prominent fibrovascular stroma. Least common of the subtypes in animals is the pleomorphic variant. As the name implies, there is considerable cellular pleomorphism with minimal connective tissue involvement. Morphologic characteristics of the tumor in this bison cow would seem most consistent with the more rare pleomorphic variant.

Rhabdomyosarcomas of the limbs, trunk, and neck typically present as nodules within muscle. While usually pale on cut surface, hemorrhage and necrosis associated with continued growth can change the appearance. Necrosis occurs when tumor cells outgrow their blood supply. Metastatic disease occurs primarily

in muscle tissue and less commonly in other organs. Microscopically, the cells are highly pleomorphic with unusual morphology characterized by racquet shapes, multinucleation, vacuolated cytoplasm, and strap cells. Striated myofibrils may or may not be visible by light microscopy. Immunohistochemical testing for specific proteins such as desmin, vimentin, myoglobin, and actin can help with diagnosis.

A search of the literature indicates that this is the first report of a rhabdomyosarcoma in a bison.

AFIP Diagnosis: Skeletal muscle; heart; kidney: Sarcoma, favor rhabdomyosarcoma, American bison (*Bison bison*), bovine.

Conference Comment: Based on the H&E histomorphology alone, conference attendees unanimously diagnosed a sarcoma with a differential list including rhabdosarcoma, fibrosarcoma, leiomyosarcoma, hemangiosarcoma, and neurofibrosarcoma.

Several histochemical and immunohistochemical stains are helpful in narrowing the differential diagnosis. With Masson's trichrome, moderately abundant blue-staining collagen is demonstrated separating neoplastic cells, which stain red. In the contributor's laboratory, neoplastic cells are negative for Factor VIII-related antigen, which does not support a diagnosis of hemangiosarcoma. Neoplastic cells exhibit diffuse immunoreactivity for vimentin, which supports mesenchymal origin, and exhibit multifocal immunoreactivity for desmin and muscle specific actin, suggesting it is a neoplasm of muscle origin. Neoplastic cells multifocally exhibit positive immunoreactivity for myoglobin, which is specific for striated muscle. In addition, phosphotungstic acid hematoxylin (PTAH) is used to highlight cross-striations, staining them blue. Depending on the laboratory results, immunohistochemistry can assist in determining the histogenesis of a tumor, but should never be used alone when making a diagnosis. In this case, the contributor also evaluated the tumor utilizing electron microscopy and results are consistent with myocyte origin. Conference attendees discussed the potential difficulty of evaluating an electron micrograph of a rhabdomyosarcoma within a skeletal muscle sample, especially if the sample were taken from the periphery, in areas where the neoplastic cells separate and surround pre-existing myocytes. In this case, ideally one would perform EM on the mass in the kidney.

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CASE III – 8053 (AFIP 2944793)

Signalment: Tissue from an adult female Vietnamese potbellied pig (*Sus scrofa*).

History: This animal came from a Vietnamese potbellied pig rescue establishment. The animal had been recently introduced into a herd of mixed sexes, most of which had been neutered. She had a history of hematuria for the previous 2 weeks. Ultrasound revealed pyelonephritis, along with pus in the uterus and urinary bladder. The owner attempted to give oral antibiotics without success and the animal was euthanized.

Gross Pathology: The animal had extensive subcutaneous and abdominal body fat. Thick, mucopurulent exudate was present in the vaginal lumen. The mucosa of the urethra and urinary bladder were streaked with hemorrhages, and the urine was cloudy. The ureters were distended and thickened. Purulent exudate could be extruded into the pelvis of the kidneys if the ureters were compressed. The renal pelvis was bilaterally dilated by considerable tan flocculent debris. There was also edema of the adipose tissue of the mesentery, around the pancreas, and near the adrenal glands. The adipose tissue in this general area contained multiple chalky white areas. Two ascarids were present in the lumen of the duodenum; one of these protruded from the pancreatic duct and extended 12 cm into the duct itself.

Laboratory Results: A heavy growth of *Eubacterium suis* was isolated from the kidney.

Contributor's Morphologic Diagnosis: Acute fibrinosuppurative pancreatitis and fat necrosis, with pancreatic duct obstruction by an adult ascarid.

Contributor's Comment: *Ascaris suis* is the largest enteric nematode of pigs. Adults reside in the lumen of the small intestine, and the females may be up to 40 cm in length. Pigs are most often exposed in dirt lots where earthworms and dung beetles may ingest larvated eggs and serve as reservoirs. Piglets may ingest eggs attached to mammary nipples, but prenatal infection is not thought to occur. L2 larvae leave the eggs in the intestine, penetrate the hepatic portal system and molt

to L3. These migrate via the blood from the liver to the lungs where they exit pulmonary capillaries, molt to L4, and migrate up airways. They are then swallowed and develop into enteric adults. Most pathology attributed to ascarids is a result of migration of large numbers of L3 larvae through the liver (white spot disease or milk-spotted liver) or lungs, where pneumonia results.

Ascaris suum is a close relative of *Ascaris lumbricoides*, the human ascarid, with which it shares a similar shape and size. Ascarids are sporadically responsible for blockage of the pancreatic duct in a variety of species, including human beings, especially in developing countries where 60% of the children and 30% of adults are parasitized. In people, the mechanism of pancreatitis is most often compression of the pancreatic duct by blockage of the bile duct or common duct, with obstruction of the pancreatic duct alone being less common. This pig had rather localized pancreatitis, and pigs have separate entrances for the pancreatic and biliary ducts at the duodenum. Living parasites slip in and out of ducts in people with relative ease, and occasionally bypass large obstructions such as gall stones in the ducts. Children are less commonly affected, perhaps because of the small diameter of the hepatic and pancreatic ducts, although they can develop severe pancreatitis. There is a 3:1 prevalence of women over men in adult patients. Chronic pancreatitis also has been described in horses as a result of migration of *Strongylus equinus*, occasionally with resulting diabetes, and there is a single report of an ascarid becoming lodged in the pancreatic duct of a horse. *Trichospiruria leptostoma* commonly inhabits the pancreatic ducts of wild-caught common marmosets. Occasional pancreatic fibrosis and exocrine insufficiency have been reported.

Characteristics of ascarid nematodes include coelomyarian musculature, and uninucleate intestinal epithelium with a low microvillous border. Numerous thick shelled eggs are present in the coelom, as characteristic of an adult female.

AFIP Diagnosis: Pancreas; peripancreatic fat; and duodenum: Pancreatitis, neutrophilic and eosinophilic, acute, multifocal, moderate, with vasculitis, fibrinous peritonitis, necrotizing steatitis, focal mural duodenitis, pancreatic duct ectasia, ulceration, and intraluminal adult ascarid, Vietnamese potbellied pig (*Sus scrofa*), porcine.

Conference Comment: The contributor provides a thorough overview of the lifecycle of *Ascaris suum* and the lesions associated with infestations by the nematode.

As pathologists, it can be tempting to play the "vet game" and diagnose *A. suum* infestation based on finding a large nematode in the pancreas of a pig. However, with a basic understanding of common histomorphological features of the various groups of metazoans, one can easily identify the parasite of this case as a nematode, and further classify it as an ascarid.

There are six groups of commonly seen metazoan parasites: nematodes, acanthocephalans, trematodes, cestodes, arthropods, and pentastomes. When evaluating a parasite histologically, it is important to note the following features: type of body covering and body wall, presence or absence of a body cavity, location and type of musculature, presence and type of digestive tract, presence and type of reproductive organs. Below is a simple table to identify the parasite to its group:[8]

GROUP	GENERAL SHAPE	BODY CAVITY	DIGESTIVE TRACT	STRIATED MUSCLE	SPECIAL DIAGNOSTIC FEATURES
Cestode	Flattened dorso-ventrally	--	--	--	1. calcareous corpuscles 2. scolex 3. tegument
Trematode	Flattened dorso-ventrally	--	+	--	1. suckers 2. tegument 3. blind ceca 4. yolk gland 5. hermaphroditic
Acanthocephalan	Spherical	+	--	--	1. hypodermis 2. lemniscus 3. two muscle layers 4. proboscis
Nematode	Spherical	+	+	--	1. cuticle 2. musculature
Arthropod	Tend to be spherical	+	+	+	1. chitinized exoskeleton 2. jointed appendages 3. tracheal tubes
Pentastomes	Spherical	+	+	+	1. chitinized exoskeleton 2. digestive glands 3. sclerotized openings

Once the organism has been identified as a nematode, it must be further classified into one of the following groups: Aphasmids or Phasmids. Aphasmids lack a tiny pair of sensory papillae (the phasmids) on the caudal end; however, these are not readily identifiable on histologic section. The morphological features that distinguish them from phasmid nematodes are hypodermal bands with associated nuclei, and prominent esophageal glands that form a stichosome. The Phasmids consist of the Rhabditoids, Oxyurids, Ascarids, Strongyles, Spirurids, and Filarids. Both the Rhabditoids and Oxyurids have a rhabditoid esophagus composed of a corpus, isthmus and bulb. The Strongyles have a cuticle, which occasionally is ridged, and all have an intestine composed of few multinucleated cells and a

prominent brush border. Spirurids can be very diverse, but all adult females in this group produce embryonated eggs. Filarial nematodes are small and produce distinctive larvae called microfilariae.[2]

Ascarids are large worms that are found, as adults, in the intestines of their host. Larval ascarids may be found in other tissues in both the definitive and intermediate hosts. Adult ascarids have a cuticle, a pseudocoelom, coelomyarian musculature, large lateral chords, and less prominent dorsal and ventral chords, a simple esophagus, a large intestine lined by uninucleate cuboidal cells and a low brush border. Adult female ascarids produce eggs which contain a uninucleate zygote covered by a thick shell. Larval ascarids of mammals commonly have lateral alae.[2]

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CASE IV – 8-81-04 (AFIP 2956553)

Signalment: Male, yearling Mule Deer (*Odocoileus hemionus)*.

History: A male, yearling mule deer was observed lying down near a rural residence in southwest Montana. The animal was unable to stand, had tachypnea, and moist airway sounds were audible from a distance.

Gross Pathology: Numerous tan foci occurred throughout the lungs, kidney and liver. Tuberculosis was suspected and the carcass was buried. Two visiting veterinarians who performed a field necropsy submitted specimens.

Laboratory Results: *Yersinia pestis* was cultured from the lung and kidney.

Contributor's Morphologic Diagnoses: 1. Lung: Pneumonia, necrotizing, suppurative, multifocal to coalescing, severe, with intralesional bacterial rods. 2. Kidney: Nephritis, necrotizing, suppurative, acute, multifocal to coalescing, severe.

Contributor's Comment: Plague is endemic in many areas of the western United States. *Yersinia pestis* is maintained in the environment by a variety of rodent species and their associated fleas. Reports of plague are usually seasonal with the greatest incidence between March and October. Susceptibility differs both in domestic and wild species. In domestic animals, the cat and dog are most frequently infected. Besides rodents and rabbits, plague has been reported in mule deer, pronghorn antelope, mountain lions and bobcats in the western United States. There are three clinical manifestations: bubonic, septicemic, and pneumonic. Lymphadenitis of bubonic plague occurs after inoculation of the organism through the skin or mucous membrane by flea bites or direct penetration. Septicemia can occur subsequent to dissemination of the infection from the infected lymph nodes or by direct introduction into the blood vasculature. Primary pneumonic plague occurs after inhalation of infected material. This particular case represents the septicemic form. *Yersinia pestis* is occasionally isolated from domestic cats in this region in Montana.

AFIP Diagnoses: 1. Lung: Pneumonia, necrotizing, suppurative, subacute, multifocal and coalescing, severe, with vasculitis, and large colonies of bacilli, mule deer (*Odocoileus hemionus*), cervid.
2. Kidney: Nephritis, necrotizing, suppurative, subacute, multifocal and coalescing, severe, with vasculitis, and large colonies of bacilli.
3. Kidney: Nephritis, interstitial, lymphoplasmacytic, chronic, multifocal, with mineralization.

Conference Comment: Of the eleven species of *Yersinia*, family Enterobacteriaceae, only four are considered to be primary pathogens: *Y. pestis*

(plague in mammals), *Y. enterocolitica* (yersiniosis in mammals and birds), *Y. pseudotuberculosis* (yersiniosis in mammals and birds), and *Y. ruckeri* (red mouth in fish).[3]
Y. pestis remains endemic in certain rodent populations on five continents due to an elaborate interaction of the agent, fleas, vertebrate hosts, and the environment. Enzootic hosts, such as voles, mice, and rock squirrels, serve as reservoirs and do not suffer 100% mortality. These animals may experience a transient bacteremia and thereby transmit the infection to fleas. The fleas may then transmit bacteria to other enzootic hosts, thereby maintaining the disease, or they may transmit it to epizootic rodent hosts. These hosts, such as prairie dogs, have a low resistance to plague morbidity and mortality. Subsequently there is a high death rate, which favors spread of plague, amplifies the intensity of the epizootic, and increases the risk of human infection as infected fleas disseminate from dead hosts.[3]

The primary mode of transmission of *Y. pestis* in mammals is via flea bite. Other modes of transmission include ingestion of, or exposure to, another mammal infected with *Y. pestis*. Another rare but effective mode of transmission is inhalation of aerosolized bacteria by a mammal in close proximity to an animal with pneumonic plague.[3]

As mentioned by the contributor, bubonic, pneumonic, and septemic plague are three clinical manifestations of *Y. pestis* infection. In many cases they represent a continuum as illness, if left untreated, will often progress from one form to the next. The bubonic form results from flea bite inoculation of bacteria and subsequent acute local inflammation of the lymph node draining the inoculation site. The Latin root "bubo", meaning "swelling", is descriptive of the lymphadenomegaly that occurs. Primary septemic plague is defined as bacteremia without the presence of palpable buboes. Secondary septemic plague occurs when bacteria from buboes enter the bloodstream. Primary pneumonic plague may result from inhalation of aerosolized droplets of *Y. pestis* from an animal with pneumonic plague.[3] This form has a very rapid incubation period (1-6 days) and is usually fatal if not treated within the first 24 hours of illness.[4] Secondary pneumonic plague may result from hematogenous spread of bacteria to the lungs in the septemic form. Clinically, the septicemic and pneumonic forms are much more severe and almost always result in death.[3]

Y. pestis is a potential bioterrorism agent, and if aerosolized, pneumonic plague could induce many human casualties. Accordingly, the Centers for Disease Control (CDC), lists it as a Category A agent. Category A agents are those agents that have the greatest potential for inflicting large numbers of human casualties, can be manufactured and disseminated on a large scale, require significant efforts in public health preparedness, and are most familiar to the public.[4]

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*Sponsored by the American Veterinary Medical Association, the American College of Veterinary Pathologists and the C. L. Davis Foundation.