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VIRGINIA ANIMAL DIAGNOSTIC NEWSLETTER

A joint publication between Virginia Department of Agriculture and Consumer Services and the Virginia Tech Animal Laboratory Services

EDITORIAL

Chronic Wasting Disease

Overview
Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (TSE) that affects North American cervids, including white-tailed deer, moose, elk, and mule deer. First discovered in 1967 in a herd of Colorado mule deer, it has since been detected in 27 North American states and two Canadian provinces, Norway, Sweden, Finland, and South Korea. Localized infection rates as high as 30% in wild populations have been reported.

What is CWD, and how does it spread?
Chronic wasting disease belongs to a family of conditions called transmissible spongiform encephalopathies (TSEs) that are rapidly progressive and invariably fatal. These diseases are caused by prions, or naturally-occurring misfolded proteins, that accumulate in and damage lymphatic and neural tissue. Other TSEs include bovine spongiform encephalopathy (BSE or "mad cow") in cattle, scrapie in sheep and goats, and Creutzfeldt-Jacob disease (CJD) in humans. The CWD prion protein gene is transmitted horizontally and is highly contagious. Direct horizontal transmission occurs when naive animals are exposed to infectious saliva, urine, or feces of CWD-affected animals. The prion is thought to cross the gastrointestinal epithelium after ingestion and crosses to the associated lymphoid tissues, including retropharyngeal and tonsillar lymph nodes. From the lymphoid centers, the prion likely travels the sympathetic fibers and progresses to the central nervous system. Indirect horizontal transmission can also occur through exposure to contaminated fomites. Vertical transmission is rare but has contributed to infection in wild elk populations.

What are the clinical signs?
CWD is characterized by a long incubation period (18-24 months), in which animals may look and act normal. Clinical signs are often nonspecific and include progressive weight loss, decreased social interaction, loss of awareness, loss of fear of humans, polyuria, polydipsia, polyphagia in the face

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of weight loss and cachexia, flaccid and hypotonic facial muscles, excessive salivation, and ataxia. It is hypothesized that affected animals are more susceptible to vehicular and predatory trauma, leading to an underreporting of neurologic signs.

What is the prevalence and impact?
The prevalence of CWD is variable and depends on biotic factors (e.g., sex and age) and geographic abiotic factors (e.g., soil characteristics). However, prevalence has been reported to be as high as 30% in wild cervid populations and as high as 90% in captive populations, with more than 175 captive cervid facilities being affected in the United States. Since its initial discovery in the late 1960s, the host range of CWD has grown to include moose (*Alces alces*), North American elk (*Cervus canadensis* and *Cervus elaphus elaphus*), white-tailed deer (*Odocoileus Virginianus*), red deer (*Cervus elaphus*), sika deer (*Cervus nipon*), reindeer (*Rangifer tarandus*), European moose (*E. alces alces*, Eurasian Elk), mule deer (*Odocoileus hemionus*) and black-tailed deer (*O. hemionus columbianus*).

The high disease prevalence in cervid populations has a devastating economic impact on affected communities. Management and surveillance of CWD in Wisconsin from 2002-2006 cost an estimated $32.3 million. Some captive cervid facilities in the area required total depopulation and government compensation for the losses. According to the 2016 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation, an estimated $156.9 billion was spent by Americans participating in fishing, hunting, or other wildlife-associated recreational activity. Revenue from these activities is used to support conservation efforts, and the impact of CWD can affect communities that depend on financial support generated by wildlife-related activities.

There is currently no evidence that chronic wasting disease affects humans; however, the outbreak of bovine spongiform encephalopathy (BSE) in the United Kingdom in the late 1980s has drawn concerns about other animal TSEs crossing the species barrier. Some experimental studies using humanized mice and cynomolgus monkeys did not result in CWD transmission. Still, other studies have shown that CWD prions can be transmitted to cattle, cats, sheep, goats, squirrel monkeys, and macaques via oral or intracerebral inoculation. The CDC recommends that hunters in endemic areas strongly consider having all animals tested before consuming the meat.

How can we control CWD?
Control of CWD is mainly dependent on the participation of hunters and landowners in surveillance and monitoring programs. Many states have programs in which hunters can help reduce the strain on local agencies by learning how to extract the retropharyngeal lymph nodes from harvested animals and submit them for testing. The CDC also has published guidelines for hunters on how to reduce their exposure, including not consuming meat from a sick or already-dead animal, wearing gloves when handling cervid meat, minimizing contact with the brain and spinal cord, avoiding "baiting" deer with piles of feed where individuals may congregate, and only having meat processed at facilities where animals are processed individually. Studies evaluating control measures found that continued and frequent culling is a successful strategy to reduce CWD prevalence. Culling can be accomplished by non-selective population harvest, targeted removal of infected animals, or seasonal harvest by hunters. A transmucosal vaccine has been developed, but the efficacy of control was not apparent.

For farmed cervids, the USDA-APHIS has created a herd certification program in which herds may be classified as CWD-positive (an animal tested positive), CWD-exposed (there has been a CWD-positive animal in another herd within the last five years), or "epidemiologically linked herd" (herds that were in contact with animals that previously resided with a CWD-positive animal). CWD-positive or CWD-exposed herds should be quarantined for five years or depopulated.

Chloe Carr DVM
Virginia Maryland College of Veterinary Medicine.

HORSES

Neurologic Anaplasma in a Horse

An adult horse was evaluated for tachycardia, ataxia, and decreased response to stimuli. A CBC revelated mild anemia, increased band neutrophils, and thrombocytopenia. Rare morulae were observed in neutrophils, suggestive of Anaplasma phagocytophilum. Anaplasmosis was confirmed by PCR on an EDTA whole blood sample. Neurologic impairment and other clinical signs resolved with tetracycline-class antimicrobial treatment. The most common clinical signs of anaplasmosis in horses are fever, depression, decreased appetite, lower limb edema, icterus, and reluctance to move, but neurologic abnormalities, including ataxia and altered mentation, have been described. The organism is transmitted by ticks and disease is seasonal. ViTALS started offering an Anaplasma phagocytophilum PCR test for horses earlier this year. While morulae in neutrophils can be visualized on a blood smear during acute infection in some cases, molecular detection of the pathogen in anticoagulated whole blood is more sensitive for diagnosis.

Tessa LeCuyer DVM, PhD, DACVM, Virginia Tech

PIGS

Gastric carcinoma in a pig

A four-year-old female Vietnamese potbellied pig was submitted for necropsy with a history of chronic gastric ulceration (one-month duration) which progressed to complete GI stasis and marked gastric distension. On postmortem examination there were multiple firmly adherent fibrous adhesions throughout the peritoneum and pleura, the stomach was markedly distended and filled with viscous to watery digesta, and the pylorus was focally and circumferentially constricted and firm, obstructing gastric outflow. Histology of the affected pylorus revealed a gastric carcinoma with transmural infiltration, desmoplasia, and lymphatic invasion throughout the omentum. Gastric carcinomas in pigs are in pigs are uncommon compared to dogs and cats. Gastric carcinoma in a Vietnamese potbellied pig has been described in a case series of alimentary-associated carcinomas in pigs (JAVMA 2009 Dec 1;235(11):1336-41).

Thomas Cecere DVM, PhD, DACVP, Virginia Tech

AVIAN

Nutritional deficiency in turkey poults

Six 3-week-old turkey poults were received for postmortem evaluation. The farmer reported losing about 30 birds a day over the last week, and at the time of submission had lost 45% of the flock. They reported that poults would become weak and die, and that treatment with various herbal products had little impact. The poults were small for their given age, and had no feed in the GI tracts. All had soft bones, with tarsometatarsi that bent rather than breaking. Their beaks were easily bent and rubbery, and some growth plates appeared widened. Four had unilateral conjunctivitis, and two had caseous material blocking the choana and multifocal to coalescing caseous plaques in the esophagus and crop. Cytology of the crop revealed abundant inflammatory cells, bacteria, and squamous epithelial cells. Histopathology revealed abnormal ratios of the zones of pre-hypertrophy and hypertrophy to the zone of proliferation in growth plates, and squamous metaplasia and epithelial hyperplasia in the esophagus and crop. The lesions were consistent with rickets and squamous metaplasia due to vitamin and mineral deficiencies. Nutritional issues can cause devastating losses, and can mimic infectious disease. In this case once the feeders were cleaned and new feed provided the mortality began to decline. The exact cause of the nutritional issues was not identified.

Hailey Quercia DVM, MS, RAHL Harrisonburg

Fungal infection in a parrot

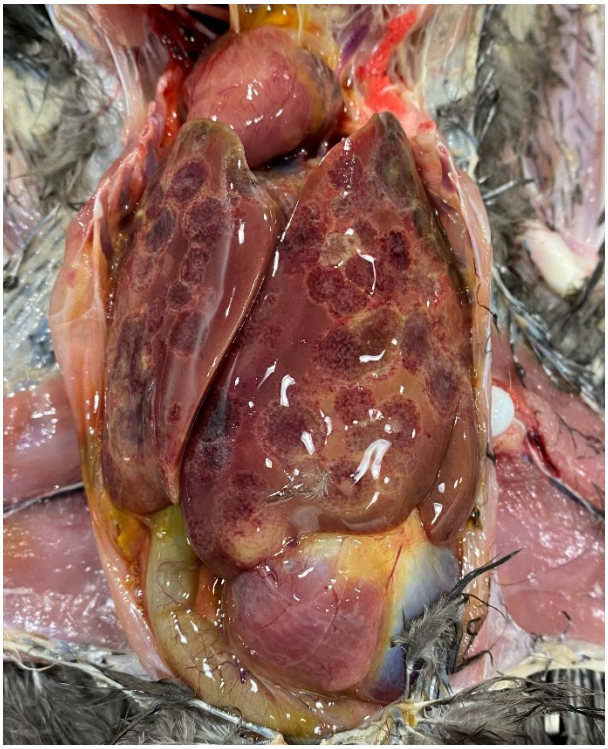
A nine-month-old Ring Neck Parrot was presented for necropsy, having been found dead without any earlier signs of illness. The posterior half of the right lung was dark red with white foci. The air sacs were clear. The liver was mottled with pale areas throughout. Sections of affected lung had nearly confluent mats of fungal hyphae with infiltration of lymphocytes, macrophages, and heterophils. The liver had severe and extensive coagulative necrosis of the centrilobular regions, consistent with hypoxic damage.

Phillip Sponenberg DVM, PhD, Virginia Tech.

Histomoniasis in a turkey poult

A ten-week-old turkey poult was submitted to necropsy after dying on its own. He was in a group of ten poults and only this one had died so far. The liver had multifocal to coalescing areas of necrotizing to granulomatous hepatitis (see image) with numerous intralesional round protozoal structures that measure 15-25 um diameter. The cecum had severe transmural necrotizing to granulomatous typhlitis with a thick, pseudomembrane covering the mucosa and numerous intra-histiocytic protozoal structures. Protozoal structures were highly compatible with Histomonas Meleagridis, which conferred a final diagnosis of blackhead diseases. Histomonas meleagridis is a parasitic protozoan that infects birds including turkeys, chickens, peafowl, quail, and pheasants. It causes infectious enterohepatitis and can be deadly in turkeys. It is transmitted by direct cloacal contact with infected birds or contact with fresh droppings of infected birds. No medications are currently approved as treatments for Histomoniasis. Good biosecurity, good litter management and separating affected birds may help decrease the spread of the parasite within a flock.

Tabitha Moore DVM, RAHL Warrenton/Harrisonburg.



Polyoma virus infection in a parrot

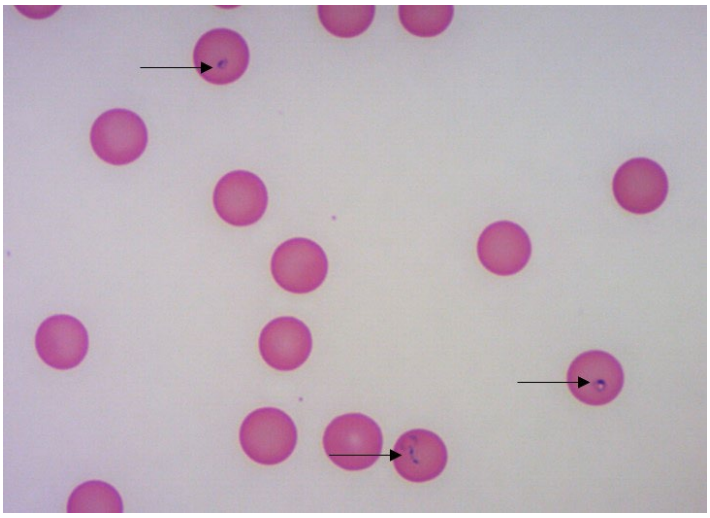
A fledgling small green parrot (no species given) was presented for necropsy, having been found dead without earlier signs of illness. The liver was very mildly swollen and subtly mottled. Sections of liver revealed extensive random areas of necrosis, with minimal inflammation although extramedullary hematopoiesis was present in several viable areas. Rare hepatocytes had large pale nuclei with marginated chromatin. Similar nuclear changes were present in many cells in the spleen. These nuclei are consistent with inclusion bodies from polyoma virus, a common cause of early mortality in many parrot species.

Phillip Sponenberg DVM, PhD, Virginia Tech.

COMPANION AND EXOTIC ANIMALS

Babesia gibsoni in a puppy

An 8-week-old, intact female, mixed breed dog was presented to the Virginia-Maryland College of Veterinary Medicine Veterinary Teaching Hospital emergency department for acute lethargy and anorexia. On presentation, the puppy was 6-8% dehydrated with pale, tacky mucous membranes, a prolonged capillary refill time, and a tense abdomen which appeared uncomfortable on palpation. A CBC revealed a marked non-regenerative anemia (hematocrit 12.2%, RI 37.3-61.7; reticulocytes 99.9 k/µL, RI 10.0-110.0) and thrombocytopenia (platelets 40 k/µL, RI 148-484). There were a few small platelet clumps at the feathered edge, but the platelet density was subjectively moderately to markedly decreased. Numerous small (approximately 1-2 µm diameter) ring-shaped piroplasms, consistent with Babesia gibsoni, were noted in the erythrocytes (see photo).



Peripheral blood smear, Modified Wright stain, 1000x magnification. Peripheral blood from an 8-week-old mixed breed dog. The dog was markedly anemic with numerous intra-erythrocytic protozoan parasites (arrows) consistent with Babesia gibsoni.

Babesia spp. are tick-borne intra-erythrocytic protozoan parasites which infect many species including dogs, cats, horses, cattle, and humans. Clinically important species in dogs include Babesia canis and Babesia gibsoni. Though transmission is tick-borne in most Babesia spp., B. gibsoni can be spread via dog fighting bite wounds, blood transfusions, and vertically (presumed transplacentally). Infection can range from subclinical to a lethal hemolytic crisis or multiple-organ dysfunction syndrome (MODS). Clinical signs, when present, typically are non-specific and include anorexia, lethargy, weakness, pyrexia, lymphadenomegaly, and splenomegaly. Clinicopathologic abnormalities include anemia and thrombocytopenia as was seen in this case.

The age of this patient highlights possible vertical transmission in this case as well as the importance of parasite prevention in pregnant dogs for both the dam and puppies.

Natalia Strandberg, DVM, MS, DACVP, Virginia Tech

Copper-associated hepatopathy in a dog

A 2-year-old, spayed female, Dalmatian dog presented to VMRCVM Teaching Hospital due to a week-long history of lethargy and vomiting. Her physical exam was unremarkable. A complete blood count was performed and only revealed a mild leukocytosis characterized by mature neutrophilia. However, the chemistry panel indicated severe hepatocellular injury and cholestasis. A fine-needle aspirate of the patient's liver showed mild hepatocellular hyperplasia and vacuolar degeneration. In addition, a light turquoise, refractile particulate pigment was observed within hepatocytes (arrow, left), confirmed with Rhodamine stain to be copper (red pigment, right). Copper-associated hepatopathy can be primary or secondary. Primary copper accumulation can be genetic or metabolic and this essential mineral tends to accumulate in the centrilobular areas of the liver. Secondary accumulation can occur as a result of chronic hepatitis and copper can be diffusely distributed in the liver parenchyma or located in the periportal areas. Dietary composition of food might play a role in the pathogenesis of both primary and

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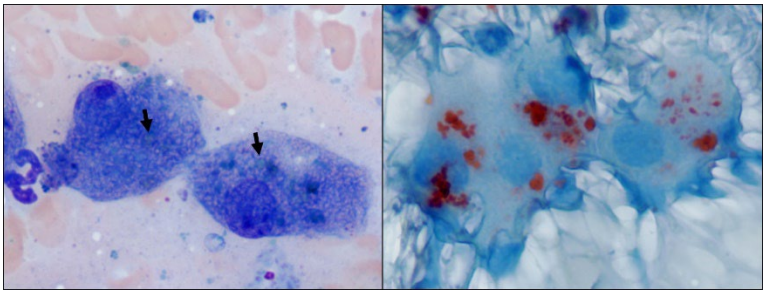
and secondary copper-associated hepatopathy. Hereditary copper accumulation has been described in Bedlington terriers, Labrador retrievers, Dobermann pinchers, West Highland terriers, and Dalmatians. Concentrations above 1,000 µg/g of liver dry weight is speculated to be able to cause hepatic injury.

Priscila B. S. Serpa, DVM, MSc, DSc, DACVP, Virginia Tech



Presumptive Crotalid Envenomation in a Cat

A 6-year-old, male, neutered Domestic Shorthair cat was noted by the owners to be lethargic and somewhat limp when picked up. The cat had come in from outdoors the day before, and the owners noticed the fur was slightly matted on the cat's back, but assumed the cat had been under the car and gotten into some oil or grease. The cat was taken to a veterinary clinic and seemed painful along its back. Clipping the fur revealed 2, 1-2 mm puncture wounds, which were about 2.5 cm apart, just caudal to the last rib and lateral to the vertebral column. Other findings on physical examination included a fever of 106.6 F and ventroflexion of the neck. Bloodwork showed nonregenerative or pre-regenerative anemia with toxic neutrophils. Chemistry values were within normal limits. The wounds were cleaned and the cat was treated with fluids, antibiotics, buprenorphine, and dexamethasone. Over the next 5 days of hospitalization, the hematocrit continued to drop and the cat became icteric. The cat continued to be lethargic and painful and stopped eating and drinking. The owners elected euthanasia and the body was submitted for necropsy. In addition to the puncture wounds and icterus, gross examination revealed a slightly enlarged, yellow liver with rounded edges and tan discoloration of the skeletal muscle near the puncture wounds. Histologic examination showed hepatic lipidosis with single hepatocyte necrosis, pigment casts in the renal tubules (likely hemoglobin), and necrosis of the epaxial muscle with minimal inflammation.
The history, clinical findings, hematology results and gross and histologic changes in this case are most consistent with envenomation by a crotalid snake (rattlesnake or copperhead). In particular, the muscle necrosis in the absence of significant inflammation near the puncture wounds, along with the evidence of hemolytic anemia, strongly support envenomation. Based on the spacing of the presumptive fang marks, this was a large snake (at least 1 meter in length). Crotalid venom includes a variety of toxins that cause local necrosis, hemolysis, and, in some cases, neurotoxicity. Hepatic lipidosis was most likely secondary to anorexia, but may have also contributed to the cat's neurologic signs.

Teresa Southard DVM, PhD, DACVP, Virginia Tech

Intranuclear coccidiosis in a tortoise

An adult female hingeback tortoise with a history of anorexia and progressive lethargy died spontaneous and was submitted for necropsy. Gross examination revealed an enlarged mottled gray/tan liver, catarrhal exudate within the oral cavity and esophagus, and multiple mucoid plaques on the mucosal surface of the tongue. On histologic examination there was hyperplastic to erosive stomatitis/glossitis and esophagitis and rare mucosal epithelial cells contained intranuclear protozoal organisms. Chronic lymphohistiocytic and heterophilic inflammatory foci were in the liver, kidneys, pancreas, and lung. Samples of lung and kidney submitted to the University of Florida were positive for intranuclear coccidia by qPCR, confirming the diagnosis of Intranuclear coccidiosis of testudines (TINC). First recognized in the 1990s, this emerging disease has been described in a growing number of chelonian species including multiple tortoise species, Easter box turtles, and Arakan forest turtles. The causative agent is thought to have a fecal-oral transmission route. Infection results in a systemic disease with inflammatory lesions in multiple organs including those of the alimentary, respiratory, cardiovascular, urogenital, and integumentary systems.

Thomas Cecere DVM, PhD, DACVP, Virginia Tech

LABORATORY NEWS

ViTALS

Dr. Natalia Strandberg has accepted our offer of a Clinical Track position in Clinical Pathology! We are thrilled that she has decided to stay. She has an interest in exotic and wild animal medicine and is looking to grow the exotic caseload.

We had our AAVLD accreditation site visit June 12-13th, and are eagerly awaiting the committee report. The accreditation process is focus on quality and continued improvement, and the committee input is important to keep us focused on excellence in the laboratory.

Tanya LeRoith DVM, PhD, DACVP, ViTALS Director.

VDACS

The VDACS Laboratory System has been working to expand test offerings, including a Diagnostic Bovine Respiratory PCR Panel, a Ruminant Abortion PCR Panel, a new platform for Salmonella PCR, and Fibrinogen testing on blood submissions.

A new serologist, Elizabeth Hinson, started working at the Warrenton RAHL in June. A new bacteriologist, Amanda Hamilton, will be starting at the Lynchburg RAHL in August. We are very excited to have new staff on board and to continue to evaluate new tests for our clients.

Jessica Walters DVM, PhD, DACPV, Program Manager, Office of Laboratory Services

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