May 2025: VOLUME 6, ISSUE 2

VIRGINIA ANIMAL DIAGNOSTIC NEWSLETTER

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

EDITORIAL

Infectious Laryngotracheitis (ILT) in the Shenandoah Valley

Recently in the Shenandoah Valley, Infectious laryngotracheitis was detected in multiple commercial chicken flocks. Referred to as "ILT" this is a disease caused by a herpesvirus that only infects chickens, some pheasants, rarely peafowl, and very rarely turkeys. It causes a severe respiratory disease, often killing 10% or more of the flock. However, in the past this was not a major concern in mainland Virginia. In the past ten years, only two cases were diagnosed by VDACS in commercial flocks. Similarly in small flocks over the same period, VDACS diagnosed an average of <2 cases per year in the entire commonwealth.

During active ILT infection signs range from mild tracheitis and conjunctivitis to severe hemorrhagic tracheitis. In the latter case, growers and owner may see streaks of blood on the walls of enclosures, from birds shaking blood and dead tissue out of their throats in order to breathe. Infected birds may extend their necks out to breathe, gasp, or make strange whistling or honking noises. [Figure 1] This is due to dead cells forming tracheal "casts" which partially or fully block the trachea. [Figure 2] Eventually these can suffocate the bird. Those that survive infection may take two weeks to fully recover but will carry the virus with them for life. Like other herpes viruses, ILT virus will go dormant within the bird, only to reactivate and spread during periods of stress.

Currently, the outbreak in the valley is caused by viruses in the genotype six group, a cluster of virulent ILT viruses. They are closely related to other genotype six

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viruses causing outbreaks in other southeast states. This virus can be spread in numerous ways. It can enter a flock on shoes, clothing, shared equipment, or even wild birds. Adding birds to an established flock is a risk since recovered but infected chickens can appear healthy while still shedding ILT virus and infecting others. Flocks are also at risk of infection if close to a road with passing trucks of infected birds, or if untreated litter from an infected flock is spread nearby.

Once on a farm the virus spread slowly through the flock from birds contacting respiratory or ocular exudate from infected flock mates. The virus also survives well in waterers and drinkers spreading to birds when they drink. It spreads slowly enough that birds on one side of the house may appear healthy while those on the other end are sick.

Suspicion of ILT may occur following necropsy of sick and dead birds. Suspicious lesions, especially tracheitis and tracheal casts, should prompt follow up testing with histology or PCR testing. PCR testing can also be performed on live birds. Histology was the traditional gold standard test, because the ILT virus forms inclusions within cells that can be seen under the microscope. [Figure 3] Before the PCR was available, pathologists and histology technicians would rush through collection and preparation of tissues so that slides could be read on the same day and give a quick diagnosis. With the advent of PCR testing, a diagnosis can be gained even faster with less effort.

There is no cure for the virus. In commercial settings sanitizing waterlines and vaccinating remaining birds can limit the impact. In small flocks, isolation of sick birds and cleaning of the coop and equipment with common household disinfectants can similarly limit spread. All infected farms should be quarantined to prevent spread while the virus is active, usually four weeks from the first sign. Once the houses are empty, waterlines and other equipment should be disinfected, and the building closed and heated to 100 F for three days to destroy the virus. It is especially important to heat the litter and destroy the virus before spreading it on fields.

Vaccines are available for commercial flocks. Recombinant (vectored) vaccines are usually given in the hatchery to chicks. The other vaccines available are typically only used in areas with an active ILT challenge as they must be carefully managed. In small flocks, the best means of prevention is through good biosecurity and not adding juvenile or adult birds to the flock, who may carry the virus. Building the flock through hatching eggs or chicks carries the least amount of risk.

Currently teams from poultry companies are working together with the Virginia Poultry Federation to monitor and track infected farms, manage vaccine use to prevent the virus from spreading, plan safe movement of birds, and ensure disinfection of infected farms. This combined effort is the best chance to stamp out the virus here in the valley. Until then maintaining tight biosecurity and testing of suspect flocks will aid in that goal.

Hailey Quercia DVM, MS, DACPV
Laboratory Director/Poultry Diagnostician
Harrisonburg Regional Laboratory

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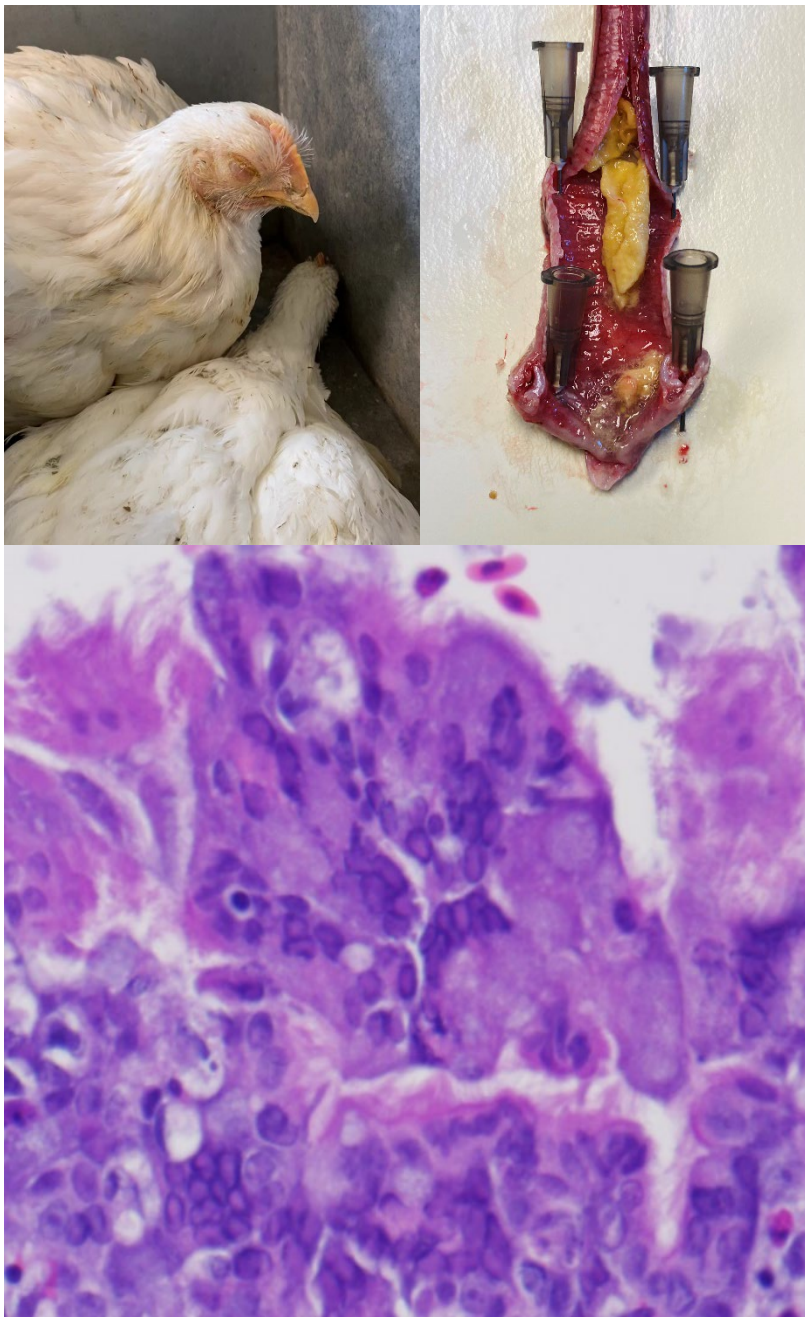

Figure 1 (Upper left): Broiler chickens infected with ILT. Upper bird showing conjunctivitis and nasal discharge, lower bird has neck fully extended to breathe. Figure 2 (Upper right): Trachea from ILT infected bird. Opened to show tracheal casts blocking the airway. Figure 3 (Lower figure): Tracheal cells from ILT infected bird. ILT inclusions are intranuclear, appearing as an eosinophilic central inclusion with a basophilic "halo" of marginated chromatin. Also common are multinucleated syncytial cells

Bovine

Vitamin D deficiency in feeder steers

A ~275 lb. Holstein steer was submitted for necropsy after a prolonged inability to rise. The steer was living outdoors, but the diet was largely comprised of commodity feeds. The right distal femur in the region of the physis had a complete transverse fracture with surrounding hemorrhage. Microscopically, there were very thin bony trabeculae and a hypocellular bone marrow that contained abundant amount of fibrin occasionally mixed with hemorrhage. Several other down animals were reevaluated after necropsy and were also found to have long bone fractures. The diet was evaluated by a nutritionist and found to be completely lacking in supplemental vitamin D. Rickets was diagnosed based on the history, diet, and necropsy findings. Vitamin D or phosphorus deficiency, as well as calcium and phosphorus imbalance, can lead to rickets, a disorder of the growth plates of young animal bones that can lead to pathologic fractures as seen here. Although the animal was housed outdoors, where sunlight and grazing can usually meet their needs, heavy commodity feeding in this case likely reduced fresh forage intake.

Taylor Young DVM, Diagnostician and Laboratory Director, Lynchburg RAHL.

Lead toxicity in a cattle herd

A 3-month-old Angus heifer was submitted for necropsy after showing signs of chewing and staggering while walking, rapidly followed by death. Several other calves had died less than 48 hours prior, but no adults had been affected. Routine diagnostics found only minor signs of rumenitis. A mineral panel was completed on liver and found lead present at 42.36 ug/g (normal < 3 ug/g), which is diagnostic for lead toxicity. Lead toxicity most commonly affects young animals due to their natural curiosity and propensity to explore their environment by licking. Neurologic signs are most common. Lead batteries and automotive fluids are common sources of lead, and in this case, heavy machinery was being stored in the same pasture as the cattle.

Taylor Young DVM, Diagnostician and Laboratory Director, Lynchburg RAHL.

Mycotic rumenitis and lungworms in a bull

A 2-y-o Hereford bull was submitted for necropsy in poor condition after passing. There is bilateral conjunctivitis and body condition is 2/9 and scant orange adipose present within the peritoneal space. The rumen contains white fungus on the dorsal aspects, the contents of the rumen are very dry, and the abomasum contains multiple trichobezoars. There is mild hepatomegaly with a moderate to significantly enlarged gallbladder. The gastrointestinal tracts are empty with minimal mucoid coated feces present. There is significant emphysema within the mediastinum. The trachea, when cut on cross section has several small white nematodes presents which continue within the bronchi and then into the distal bronchioles. Toward the tips of the lungs the bronchioles appear completely occluded with these white nematodes. All cranial lymph nodes appear enlarged and reactive on cross section. Histology was submitted and confirmed to be bacterial pneumonia, pulmonary nematodiasis (lungworm infection) and mycotic rumenitis. Samples were submitted for fungal culture which indicated fungus was isolated but could not be identified due to overgrowth on the plate.

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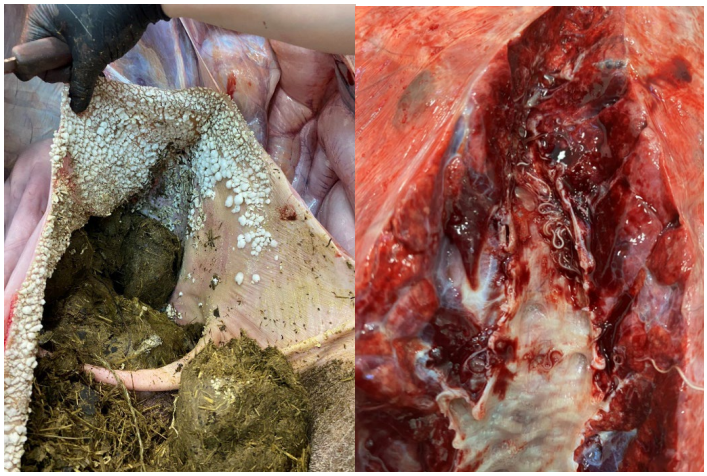


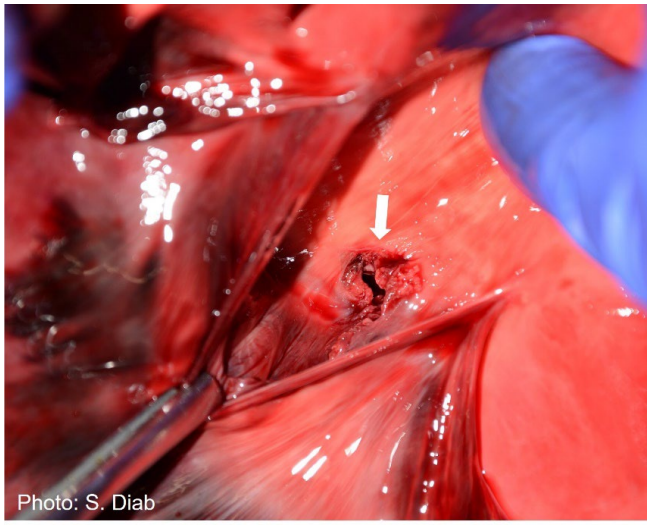
Left: Mycotic ruminitis, with multifocal white plaque-like areas of thickening of the mucosa. Right: Numerous white nematodes (lung worms) present in the lumen of the bronchus

Jamie Horstmann-Blinn DVM - Harrisonburg RAHL

Horses

Sudden death due to aortic rupture in a horse

A 14-year-old gelding collapsed shortly after exercise. The animal exhibited bracing posture, dyspnea, and arrhythmic heart sounds before passing naturally. Gross examination revealed severe hemorrhage along the beginning of the aorta and the aortic arch and approximately 750 mL of blood in the pericardial sac (hemopericardium). A closer examination of the aorta just above the aortic valves, revealed a 1 cm long aortic rupture. The cause of death was diagnosed as aortic rupture leading to cardiac tamponade, a life-threatening condition where excess fluid or blood builds up in the pericardial sac, resulting in a decreased cardiac output, low blood pressure and shock. Although uncommon, this is a well-recognized cause of sudden death in equine athletes.



Focal aortic rupture in a horse, with a transmural perforation at the base of the aorta.

Santiago S. Diab DVM, DACVP - Virginia Tech

Equine degenerative myeloencephalopathy

An 8-year-old Dutch Warmblood gelding was evaluated for neurologic disease following a history of behavioral changes, difficulty collecting, and tripping. Spinal ataxia with hypometria and weakness were diagnosed based on clinical findings. No evidence of dynamic spinal cord compression was present on myelography. Due to worsening clinical signs, the horse was euthanized and submitted for postmortem examination. Microscopic lesions compatible with equine degenerative myeloencephalopathy were detected in the brainstem and spinal cord (caudal cervical and cranial thoracic segments). Although the etiopathogenesis of this condition is incompletely understood, evidence suggests that oxidative stress related to vitamin E deficiency contributes to neurodegeneration, and genetic factors likely play a role as well (Journal of Veterinary Internal Medicine. 2024;38:431-439).

Thomas Cecere DVM, PhD, DACVP - Virginia Tech

Temporohyoid osteoarthropathy in a horse

A 20-year-old, female, Rocky Mountain Horse presented to the VMCVM for acute colic signs and was diagnosed with a pelvic flexure impaction. Right sided facial nerve paralysis was diagnosed during the workup. A tight fitting halter was the suspected cause of the neurologic deficit. The impaction resolved with medical treatment, but the horse was having difficulty eating and drinking. She was able to swallow normally, but could not prehend food or move a bolus of food to the pharynx. Upper airway endoscopy showed enlargement of the right stylohyoid bone. While hospitalized, the animal became febrile and was treated with TMS, enrofloxacin, and metronidazole. Testing for EHV-1 was negative. The horse was euthanized and submitted for necropsy. Gross examination of the right temporohyoid joint showed bony proliferation and fusion of the joint. This finding was interpreted as termorohyoid osteoarthropathy. Examination of the lungs revealed bilateral, firm, tan foci in the cranial lung lobes. On histologic examination, these areas corresponded to clusters of alveoli filled with degenerate neutrophils, macrophages, and erythrocytes, mixed with small amounts of plant material and bacterial colonies. These findings were interpreted as aspiration pneumonia. Temporohyoid osteoarthropathy is a condition in horses characterized by thickening and eventually fusion of the joint between the stylohyoid bone and the petrous part of the temporal bone of the skull. Secondary fractures can also occur. The enlarged joint can compress the neighboring facial and vestibulocochlear nerves, leading to paralysis of the muscles of facial expression, vestibular signs, and hearing loss. The cause of temporohyoid osteoarthropathy is not completely understood, but risk factors include trauma to or inflammation of the temporohyoid joint.

Teresa Southard DVM, PhD, DACVP - Virginia Tech

Avian

Myelocytomatosis in a one-and-a-half-year-old hen

A 1.5-year-old Ameraucana hen presented for necropsy, following an eight-day period of progressive reluctance to ambulate and anorexia. Despite antibiotic and NSAID treatment, her condition continued to worsen, and she was euthanized.

On intake, the hen was noted to be in excellent body condition. At the thoracic inlet, attached to the medial surface of the sternum was a firm white lamellated irregularly shaped mass. This was disseminated throughout the coelomic cavity, covering the intercostal nerves, the lungs, and the kidneys. Histologically the tumors were determined to be myelocytomatosis, a granulocyte cell cancer.

Myelocytomatosis is a rare cancer in chickens, caused by infection with Avian leukosis virus subgroup J, member of the avian retrovirus group. Usually found in meat type birds, it spreads vertically from hen to chick, horizontally between birds, or on contaminated needles during blood draw or vaccination. It is rarely reported in small flocks, and along with other retroviruses was eliminated from United States commercial breeding stock.

Ancillary testing found the hen had never been exposed to mycoplasma or common viruses such as reovirus, infectious bursal disease, and infectious bronchitis, indicating good biosecurity for the flock. This raises some concern for the virus being introduced as a vertical transmission event.

This case indicates that Avian leukosis virus subgroup J is present in the region and a differential to consider.

Hailey Quercia DVM, MS, dACPV, Poultry Diagnostician and Laboratory Director, Harrisonburg RAHL

Parasitic encephalitis in a one-year-old rooster

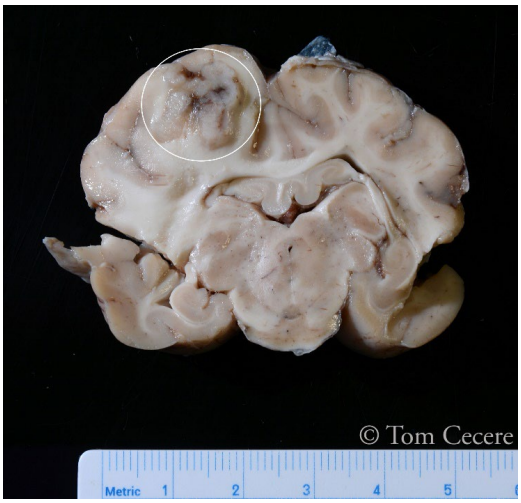
A one-year-old silver laced Wyandotte rooster presented for euthanasia and necropsy. Ten days prior he began avoiding the flock and walking in circles. This developed into him no longer moving, eating, or drinking, and the owner suspected he was blind. On physical exam he circled to the right, and lacked both menace and pupillary light reflex (PLR) in the right eye, and PLR in the left eye. On necropsy, the main gross lesion was a pale-yellow swelling at the optic chiasm. In the eyes, the pathologist identified severe lymphoplasmacytic and granulomatous inflammation of ocular structures including the optic nerves, along with detachment of the retina. Present in areas of inflammation were protozoal parasites. Inflammation and parasites were also identified in meninges, brain, spinal cord, and peripheral nerves. There are many different types of protozoal parasites that can cause encephalitis in birds, but a likely suspect in this case is Toxoplasma gondii. The parasite is infamous for being able to infect most mammals, including humans, causing health issues in pregnant or immuno-compromised individuals, and for being spread in the feces of cats. It is known that free range or pastured chickens are commonly infected with T. gondii, with parasites found in muscle. Consumption of undercooked poultry is therefore another risk factor for T. gondii infection. More recent research found that the heart and brain of infected birds had the highest levels of parasites. However, while chickens are often infected with T. gondii, they very rarely show any signs of illness. There have been few reported cases of clinical disease, but in some neurological signs and encephalitis were reported, similar to this case. The reasons why only some birds develop disease are still not known.

Hailey Quercia DVM, MS, dACPV, Poultry Diagnostician and Laboratory Director, Harrisonburg RAHL

Companion Animals and Wildlife

Brain metastasis tumor in a dog with neurological signs

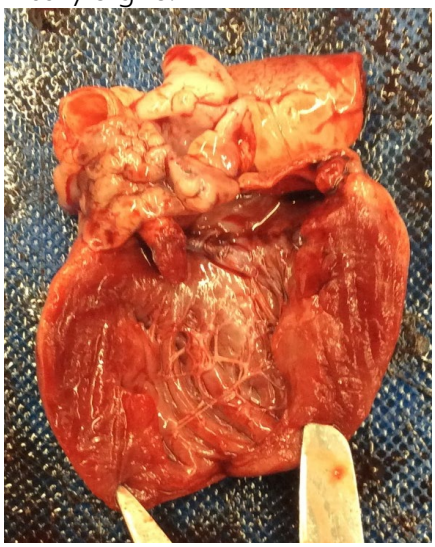
A 13-year-old, spayed female Belgian Malinois presented with a history of seizures that had recently worsened, leading to ataxia (loss of coordination), sudden blindness, and continuous seizure activity requiring emergency treatment. Despite intervention, humane euthanasia was elected. Examination of the brain revealed a firm, well-demarcated mass measuring approximately 1.5 cm in diameter in the frontal lobe, causing compression of the surrounding brain tissue (white circle in the photo). In the lungs, two firm, well-demarcated masses were identified in the right cranial lung lobe, measuring approximately 2.5 × 2 × 1 cm and 3 × 2.5 × 2 cm. Microscopic examination showed that both the brain and lung masses were composed of the same type of abnormal epithelial cells. Special immunohistochemical testing confirmed the diagnosis of a metastatic squamous cell carcinoma, suggesting the cancer originated in the lung and spread to the brain. This case highlights the critical role of thorough diagnostic evaluation in older pets with sudden neurological signs, as cancer in distant organs can metastasize to the brain.



Santiago S. Diab DVM, DACVP - Virginia Tech

Anomalous chorda tendinea in a cat

A 3-year-old female Domestic Short Hair cat was submitted for necropsy, after being found dead. The cat was seen last time the night before, and nothing suspicious was noted. This animal had good body condition. The most significant findings were restricted to the thoracic cavity, with the presence of pulmonary congestion and edema. The heart weight was 16 g. The left ventricle displayed anomalous chordae tendinae, characterized by numerous string like projections across the ventricle. Anomalous chorda tendinea is a rare congenital defect characterized by the presence of numerous fibrous cords connecting the left ventricular wall in different directions. Usually, animals with this malformation die suddenly without any premonitory signs.

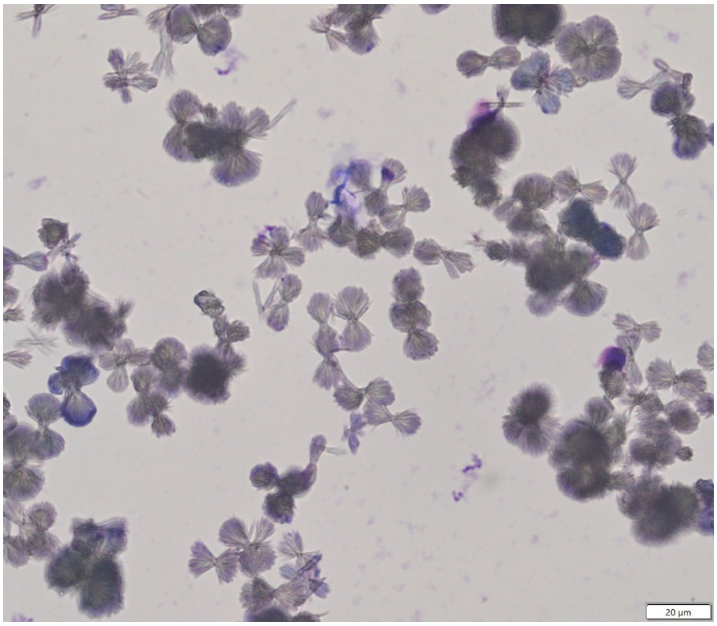


Left ventricle of a cat. Numerous string-like projections in different directions.

Francisco R Carvallo DVM, DSc, DACVP, Virginia Tech

Ammonium bi-urate crystalluria in a puppy

A 3 month-old Shih Tzu-mix puppy was presented to the Virginia Maryland College of Veterinary Medicine Veterinary Teaching Hospital for a 2-week history of bloody diarrhea which was mildly responsive to treatment with metronidazole and deworming and which progressed to weight loss, lethargy, and anorexia. The puppy was ataxic and dull on presentation with generalized muscle wasting a distended. Routine CBC and serum chemistry revealed a mild microcytic, hypochromic anemia, consistent with iron deficiency and a mild leukocytosis due to neutrophilia and monocytosis, consistent with chronic inflammation. The serum chemistry results showed moderate elevation in ALT activity (254 U/L; RI: 8 - 75 U/L), but was otherwise unremarkable. The dipstick portion of the urinalysis revealed 2+ proteinuria, but was otherwise within normal limits. Evaluation of urine sediment showed many pale gold to brown needle-like crystals which were in “bundles of wheat” and spherical arrangements (Figure 1). The primary differential for the “bundle of wheat” morphology includes sulfonamides and ammonium biurate. Given the lack of history of sulfonamide administration, the clinical picture, and the dark brown color of some of the crystals, ammonium biurate was considered the likely identity of the crystals. Further diagnostic workup was declined at this time and the puppy was euthanized two days later due to decline of the patient’s condition, so presence of a portosystemic shunt was not confirmed.



Canine urine sediment, modified Wright stain, 50x oil objective. There are numerous pale gold to brown needle-like crystals arranged in “bundles of wheat” structures with fewer spherical arrangements. The morphology is consistent with either sulfonamide or ammonium biurate crystals with the brown color being more suggestive of the latter.

Ammonium biurate crystalluria can be seen in some healthy dogs of certain breeds (English bulldogs, Dalmatians), but is an indication of liver failure of portosystemic shunt in all other cases. Ammonium biurate crystals tend to be dark gray-brown and have a “thornapple” morphology, but

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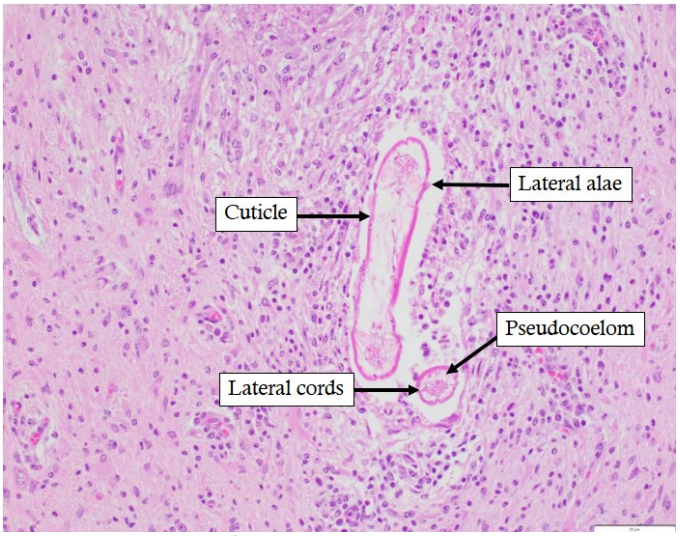
"bundle of wheat" and spherical arrangements of needle-like crystals can also be seen.

Natalia Strandberg DVM, MSc, DACVP (Clinical Pathology)

Cerebral nematodiasis in a juvenile Bennett's wallaby

A 1-year-old Bennett's wallaby joey was euthanized after a one-month history of progressive neurologic dysfunction with circling, ataxia, and head tremors. Ancillary diagnostics including radiographs, fecal floatation, and bloodwork were within normal limits, and test results for toxoplasmosis, lead toxicity, and Highly Pathogenic Avian Influenza were negative. No gross abnormalities were identified in the brain or spinal cord, and all other findings were considered incidental. On histologic evaluation, in all analyzed sections of brain, there are multifocal areas of gliosis and perivascular inflammatory infiltrates composed of lymphocytes, plasma cells, macrophages, and occasional neutrophils. Within the thalamus, there is a focal granuloma characterized by a core of necrotic cellular debris surrounded by numerous foamy macrophages, lymphocytes, plasma cells, and rare eosinophils. In the center of the granuloma, there are transverse and longitudinal profiles of nematode larvae that are approximately 50 microns in diameter and have a thin cuticle with lateral alae. The pseudocoelom is internally lined with polymyarian-coelomyarian musculature and there are two lateral cords along the digestive system. Although formalin-fixed paraffin embedded tissue scrolls of the cerebrum were submitted for DNA retrieval and PCR, definitive parasite identification could not be achieved. The wallaby's neurologic disease in this case was attributed to cerebral nematodiasis (neural larva migrans), which was supported by the compatible clinical history and histopathologic features in the cerebrum with intralesional nematode larvae. Based on the histomorphology of the nematode larvae and known raccoon exposures at the zoological institution, the most likely etiologic agent in this case is Baylisascaris procyonis. In the veterinary literature, there are only two reports of cerebral nematodiasis in wallabies, including: 1) Baylisascaris-associated neural larva migrans in a group of juvenile Bennett's wallabies in a zoological population in Canada, and 2) a case report of naturally-occurring neural larva migrans in a 3-year-old female Bennett's wallaby in Australia that was attributed to the rat lungworm, Angiostrongylus cantonensis, based on its histomorphology. Ultimately, our case and those reported in the veterinary literature demonstrate the susceptibility of wallabies to cerebral nematodiasis and highlight potential considerations for their health under managed care.



Histopathology of the brain. Transverse/sagittal section of a nematode, with all its histologic features, surrounded with a moderate granulomatous inflammation.

Alexandra Reddy DVM - Virginia Tech
Francisco R Carvallo MV, DSc, DACVP - Virginia Tech

Laboratory News

ViTALS

We are thrilled to announce that three of our pathology residents, Abraham Adeyemo, Marlie Nightingale, and Laura Quishpe Contreras, passed Phase I of their pathology boards on their first attempt! We are extremely proud of them and their mentors, and look forward to their continued success.

Anna Hassebroek, pathologist and NBAF regional scientist hosted her first meeting as the liaison between NAHLN, NBAF, and diagnostic labs in the Southeast. The meeting was a great opportunity for USDA to learn about the challenges that diagnostic labs have currently and foresee, providing valuable input for future funding efforts.

NAHLN agreements for 2025 have been processed, and ViTALS is prepared to support VDACS and VDWR in testing for African Swine Fever, Antimicrobial Resistance, Avian Influenza, Classical Swine Fever, Foot and Mouth Disease, Infectious Salmon Anemia, Newcastle Disease, Swine Influenza, Viral Hemorrhagic Septicemia, and Chronic Wasting Disease.

The College of Veterinary Medicine held their faculty awards ceremony in April, and ViTALS faculty Roger Ramirez-Barrios, Jessica Gilberte, and Natalia Strandberg were recognized for their excellence in teaching and research.

The 4th year veterinary students ended their clinical rotations, and a new class of students started in the clinics on May 5th. We are excited to see where the class of 2025 goes, and to begin to train the next generation of veterinarians and diagnosticians. We are looking forward to seeing what their future holds!

Tanya LeRoith DVM, PhD, DACVP. ViTALS Director

VDACS

CoreOne for Labs went live on February 3rd with the billing portal opening on March 3rd. While our staff are still learning the ins and outs of the new system, the client portal and ability to pay via credit card has been well received. We are continuing to make tweaks in reporting and have purchased a data analytics add-on to be able to better utilize data inputs state-wide.

The expanded histopathology partnership between VDACS and ViTALS has been extremely successful. Diagnosticians from VDACS and pathologists from ViTALS have enjoyed collaborating to review and solve cases, as well as benefitting from the increased caseload and variety that comes with partnering the two institutions. We look forward to continuing to work together more formally!

Bulk tank surveillance testing for Highly Pathogenic Avian Influenza Virus in milk continues at the Harrisonburg Regional Animal Health Laboratory, now including samples from both the Harrisonburg and Wytheville regions. Over 550 bulk tank samples have been tested so far with over 1100 samples tested to date (all testing negative). The laboratory is expecting to continue to enhance surveillance numbers with changing federal orders. Additionally in Harrisonburg, the introduction of a wild type Infectious Laryngotracheitis into commercial chicken farms has significantly increased testing needs. In Warrenton, analysts are continuing to trouble shoot in bringing in CL SHI testing capabilities and have been working to bring in Avian Influenza and Newcastle Disease testing. They have also received the probes for Equine Herpes Virus- 4 and will be working to bring that into the EHV panel. The Wytheville Laboratory is deep in the yearly fish screening testing for the Department of Wildlife Resources. By the end of April, fish testing had resulted in a total of 695 bacterial cultures, 675 IFAs, 840 virology tests on three different cell lines, and 180 parasitology tests for Whirling Disease. The Lynchburg Laboratory now has the capability to accept Trich testing in saline rather than traditional incubation pouches, and is working to expand other testing.

We are always open to feedback and ideas for testing to further assist our agricultural community. It is our goal to make this lab system the best it can be to further serve our clients. Our partnership with the Virginia Tech Animal

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Laboratory Services Lab (ViTALS) continues to evaluate ways to collaborate and expand testing services. Whether by utilizing data for reports or enhancing testing capabilities, we look forward to continuing to work together and with all of you.

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

LABORATORY LOCATIONS

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