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VIRGINIA ANIMAL DIAGNOSTIC NEWSLETTER

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EDITORIAL

Mycoplasmosis - a proverbial thorn in your side

What is it?

Mycoplasmosis is a disease characterized primarily by respiratory signs in common types of poultry. While chickens and turkeys are the most frequently affected and most susceptible, all bird species, including song birds, can be carriers and show clinical signs. While multiple species of *Mycoplasma* exist, the most common species associated with illness in backyard poultry are *Mycoplasma gallisepticum* and *Mycoplasma synoviae*. These two species of *Mycoplasma* are specific to birds and are not shared with other livestock that may cohabitate with them.

Mycoplasma gallisepticum (MG) is generally associated with chronic respiratory disease in chickens and turkeys. Transmission (by aerosol or by egg) occurs when carrier animals are commingled with susceptible birds. Additional stress, such as cold weather, overcrowding, vaccination, or concurrent viral infection, may contribute to the spread of the disease. In its mildest form, uncomplicated MG may manifest simply as respiratory distress, coughing, sneezing, and ocular discharge. Frequently, however, severe acute disease and increased mortality can occur when MG interacts with secondary viral or bacterial pathogens (e.g., *E. coli*, Newcastle Disease, Avian Influenza, Fowl Coryza, Fowl Cholera, Infectious Bronchitis Virus, Infectious Bursal Disease Virus). With such mixed infections, birds can develop significant systemic disease and have lesions of tracheitis, sinusitis, rhinitis, pneumonia, arthritis encephalitis, and ophthalmitis.

Mycoplasma synoviae (MS) is the second most frequently seen *Mycoplasma* infections in poultry. While not as severe as *Mycoplasma gallisepticum*, it can cause mild respiratory signs, lameness due to infectious synovitis, and immunosuppression. Commonly, co-infection with MS can be associated with other bacterial and viral pathogens, similarly to MG. It is spread through direct contact, fomites (i.e., shoes, equipment), and by egg transmission (hen to chick).

Are there treatment options?

Keeping *Mycoplasma*-free flocks is the best way of prevention. Treating infected birds with antibiotic medication may alleviate signs keeping a closed flock and practicing biosecurity and disease and

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Virginia-Maryland
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VIRGINIA DEPARTMENT
OF AGRICULTURE AND
CONSUMER SERVICES

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lesions, but does not always eliminate the infection and can produce carrier birds. Preventing introduction onto the farm by prevention management is important in controlling the disease. Bringing birds from poultry shows, auctions, or untested sources is a common source of disease. Separating new birds away from the existing flock for 2-4 weeks always helps to see if they have any disease. Visitors and contaminated equipment from other flocks should be avoided to control the disease.

Most strains of *Mycoplasma* are susceptible to tetracycline, erythromycin, spectinomycin, tylosin, and enrofloxacin. Antibiotics are usually given for 5-7 days in feed or water. Although antibiotic therapy may help alleviate clinical symptoms and lesions, it will not eliminate *Mycoplasma* from the flock, as most flocks require identifying and breeding seronegative stocks.

Why is this a big deal?

Signs of Mycoplasmosis frequently appear with the introduction of new birds that are seemingly “healthy” from an unknown flock into new premises. Unfortunately, because of the carrier state of the disease, birds that appear healthy, once stressed, become shedders of the pathogen. In this way, *Mycoplasma* acts like a virus (although it is not). Once the bacterial agent is shed, any naïve populations can be significantly affected and have increased morbidity and mortality (depending on co-infections). Ultimately, with *Mycoplasma*, the key is “like to like”. Carrier birds can enter *Mycoplasma*-positive premises with no problems, but problems arise when naïve *Mycoplasma*-negative birds or flocks are exposed to carriers who become shedders.

This proverbial “thorn” becomes especially painful during the spring and summer when “swap” meets (sales of birds from unknown health status source flocks at local farm stores and auctions) pick up in frequency. The solution? Education and enhanced biosecurity are key, because once this thorn gets in, it won’t come out!

Jessica Walters DVM, PhD, DACPV

Program Manager, Office of Laboratory Services.
VDACS - Harrisonburg RAHL

HORSES



Caterpillar ingestion

A 21-year-old American Quarter Horse mare with a 3-day history of inappetence and lethargy was evaluated by a veterinarian and found to have multiple cavitory effusions on ultrasound. Neoplasia was suspected, and the horse was euthanized. Postmortem examination revealed tricavitory effusion characterized by severe, chronic fibrinous pericarditis with mixed bacteria. These findings mirrored features described in horses with fibrinous pericarditis associated with the ingestion of tent caterpillars. Fibrinous pericarditis due to ingestion of eastern tent caterpillars has been reported to occur in

conjunction with cases of mare reproductive loss syndrome, and another publication described similar cases of fibrinous pericarditis in horses in Saskatchewan associated with an outbreak of forest tent caterpillars. The exact etiopathogenesis of this condition, including the role of the tent caterpillars in inducing disease, is not completely understood.

Thomas Cecere DVM, PhD, DACVP, Virginia Tech

Fatal head and neck trauma in a horse

A 15-year-old Standardbred mare was being led out to a pasture when she reared up and fell onto a concrete floor, hitting the right side of her head and neck. She bled profusely from the nose and died within 15 minutes of the fall. Necropsy revealed hemorrhage in the right cervical skeletal muscle, around the wing of the atlas, a retropharyngeal hematoma, chip fractures of the nuchal crest, and diffuse subdural hemorrhage. The subdural hemorrhage and increase in intracranial pressure was the likely cause of death, is one of the most common causes of head injury in horses and is typically seen in young horses or excitable horses that are being led or restrained. When the horse

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rears up, the height of the head is often more than 10 feet, and the force of the collision with the ground or floor generates a shock wave that can fracture smaller bones away from the point of impact. Additionally, the acceleration/deceleration forces on the brain can cause diffuse axonal injury and can rupture the veins that bridge between the dura mater and the brain, leading to a subdural hematoma.

Teresa Southard, DVM, PhD, DACVP, Virginia Tech

Avian



Fowl cholera in breeder chickens

Ten male broiler breeder chickens were submitted, with a history of being lame. The flock had been treated with penicillin with no improvement. They were 14 weeks old and had recently been given pre-move vaccinations. When the birds were examined, most were unwilling to stand with swollen hock joints. Over half had large scabbed ulcerations in one wing web or evidence of incorrect vaccine placement. Nine had excessive viscous, opaque yellow fluid in one or both hock joints. Pure *Pasteurella multocida* was isolated on culture.

Pre-move vaccinations are given to protect the adults and ensure adequate antibodies are passed on to their chicks. Both live and killed vaccines are given in sites such as the breast muscle and the wing web. In wing web vaccines, a dye can be included, and some vaccines cause swelling or “take”, all of which is used to monitor for proper vaccination technique. Mild or slightly stronger Fowl cholera vaccines (live attenuated strains of *Pasteurella multocida*) are often given in the wing web. These vaccines must be kept temperature-controlled and properly given to prevent adverse reactions. Roosters are very sensitive and should be given the mild strain.

In this case, it is believed that the vaccine was either the stronger strain or was overheated, leading to active infection and disease. This case illustrates the importance of proper vaccine handling and administration and the potential of assessing vaccination while performing necropsies.

Hailey Quercia DVM, MS, Harrisonburg RAHL

COMPANION AND LABORATORY ANIMALS



Splenic hemangiomatosis in a dog

A 7-year-old female German shepherd dog was taken to an emergency clinic, with a history of fever and anorexia. The clinical evaluation revealed pyometra, so abdominal surgery was performed in order to remove the uterus and ovaries. During this procedure, the surgeon noted that the spleen had numerous nodules dispersed throughout the organ. He removed the spleen and sent it for histologic evaluation to the Harrisonburg RAHL. The histologic examination of the splenic masses revealed the presence of splenic hemangiomatosis, a condition seldom reported in humans and dogs. Splenic hemangiomatosis is characterized by numerous, variable-sized vascular channels lined with a monolayer of endothelial cells dissecting through the splenic parenchyma. Due to the lack of reports in the literature, it is not clear if hemangiomatosis is a vascular malformation or a neoplastic process. If this finding is restricted to the spleen, the prognosis is good.

Francisco R Carvalho DVM, DSc, DACVP, section of veterinary pathology, VDACS

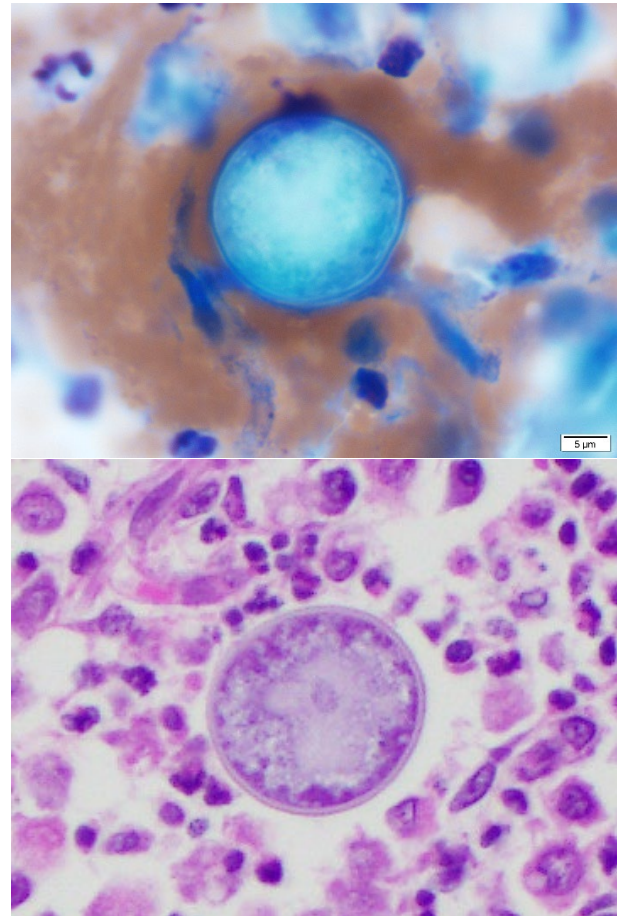
Coccidioidomycosis in a dog

A 6-year-old, spayed female Yorkshire Terrier mix was referred to the VMRCVM Teaching Hospital emergency service due to bicavitory effusion, chronic weight loss, lethargy, and labored breathing for 2 months duration. The owners moved from California/Arizona to Virginia, and the dog had been diagnosed with coccidioidomycosis (aka Valley Fever) two years before. Abnormalities observed on physical examination included tachypnea with abdominal effort, increased bronchovesicular sounds on all quadrants, tense and distended abdomen, enlarged right superficial cervical lymph node, and moderate periodontal disease. More importantly, imaging confirmed bicavitory effusion and revealed splenomegaly, abdominal and thoracic lymphadenopathy, and pulmonary atelectasis. Laboratory data showed a nonregenerative normocytic normochromic anemia, inflammatory leukogram, hypoglycemia, hypoalbuminemia, hyperglobulinemia, and evidence of cholestasis. Cytologic evaluation of pleural fluid was consistent with a neutrophilic exudate, while superficial cervical and abdominal lymph nodes were characterized by neutrophilic and macrophagic lymphadenitis. The spleen aspirate revealed marked lymphoid hyperplasia and extramedullary hematopoiesis. No infectious agents were identified in any sample, and given the history, serum fungal antigen testing was recommended. Results returned positive for coccidioidomycosis. Due to the rapid patient decline, the family elected humane euthanasia.

Gross examination revealed abdominal and thoracic effusions with multiple adhesions between the organs. Lymph nodes, including the prescapular, popliteal, mesenteric, and tracheobronchial, were enlarged and mottled on the cut surface. Dozens of small tan nodules multifocally expanded the pleura, lung parenchyma, and peritoneum. Histologic examination showed that the nodules were clusters of macrophages, neutrophils, lymphocytes, and plasma cells, often centered on round structures, up to 100 um in diameter, with refractile walls surrounding granular basophilic material, consistent with *Coccidioides* sp. spherules.

Coccidioidomycosis is a systemic fungal infection caused by *Coccidioides immitis* and *C. posadasii*. The fungus is dimorphic, forming hyphae in the environment and yeast-like structures within a host animal. Only the hyphal form is infectious. *Coccidioides* spp. are endemic in warm, dry areas of the southwest United States, Mexico, Central and South America. Dogs typically become infected by inhaling the arthrospores, leading to inflammation in the lungs and draining lymph nodes. The infection can also spread to distant sites, causing

disseminated disease, as in this case. Coccidioidomycosis also infects humans and a variety of other mammals.



Left: Postmortem impression smear of lung tissue showing a 20-um spherule with double-contour wall and blue-green granular protoplasm. Diff-Quik, 100x objective. Right: Histologic image of a *Coccidioides* sp. spherule surrounded by neutrophils and macrophages in a mesenteric nodule. Hematoxylin and eosin staining.

Priscila B. S. Serpa, DVM, MSc, DSc, DACVP (Clinical), Virginia Tech
Teresa Southard, DVM, PhD, DACVP, Virginia Tech

Protozoal enterocolitis in a snake

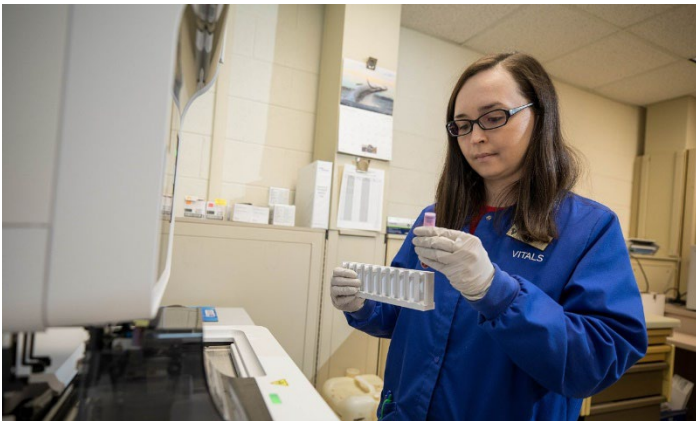
A juvenile elegant bronzeback arboreal snake (*Dendrelaphis formosus*) was imported from Asia to a closed colony. Its health history prior to import was unknown, and the snake was behaving and eating normally immediately following import. The snake was found dead one and a half weeks following import. At postmortem examination, the gastrointestinal tract was nearly completely devoid of digesta, and no other significant gross

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lesions were present. Histologic examination revealed segmental necrotizing and ulcerative enterocolitis with numerous nematodes in the intestinal lumen and myriad protozoal trophozoites within affected segments of mucosa. The morphologic features of the protozoal trophozoites were suggestive of amoeba. A report describing similar lesions and protozoa in snakes found that in the majority of cases these organisms were *Entamoeba invadens* (confirmed by immunohistochemistry), but in other cases protozoan parasites with similar morphology were identified in association with fibrinous or diphtheritic inflammation and were identified as *Monocercomonas* spp. Protozoal infection is thought to be potentiated by other risk factors including stress and concurrent infection/parasitism - both of which were present in this snake.

Thomas Cecere DVM, PhD, DACVP, Virginia Tech

LABORATORY NEWS



ViTALS

This summer has brought some changes to our faculty and staff. We are excited to announce that Santiago Diab will be joining us in August as an Anatomic Pathologist. Dr. Diab trained at the California Animal Health and Food Safety Laboratory (CAHFS) in San Bernardino, spent another 9 years there as a faculty member, and brings years of large animal necropsy experience. Welcome to ViTALS and the New River Valley!

Our amazing Quality Manager, Jennifer Rudd, has left ViTALS to focus on her family business full-time. We are so grateful for her years with ViTALS, and for all her work in getting the lab accredited by AAVLD and NAHLN, and ensuring that we maintain the accreditation standards that our clients have come to expect. We will miss her terribly but are thankful that she has agreed to continue to help us through the

transition. Moreover, if you're ever in Floyd, VA, be sure to stop by Cocoa Mia and say hello. We are extremely fortunate that Lauren Lytle has moved seamlessly into her role as QM, and we are excited to see where her skills and expertise take ViTALS in the future!

We also want to welcome Jessica Haley, who has joined us in microbiology. Jessie is a 2020 VT graduate and completed her MLS at RU/Jefferson. Rachel Edwards has also joined our staff in clinical pathology. Rachel is a VT and RU graduate and comes to us from the UVA Core Lab. Welcome, Jessie and Rachel!

Tanya LeRoith DVM, PhD, DACVP, ViTALS Director

VDACS

The VDACS Laboratory System has been working diligently to improve outreach communications and promotions, and to expand and improve testing modalities. Our staff have hosted booths at two fairs so far this year, both in Fauquier and Rockingham Counties, and will have representatives at two more this fall- Washington and Amherst Counties. We are excited to be able to show off our system and what we do! Lab staff in both Lynchburg and Wytheville have been working to bring on NAHLN testing associated with their new Level 1 NAHLN Branch Lab designations.

We are continuing to move through the process of our Capital Expansion Projects affecting three of our facilities- Warrenton, Harrisonburg, and Lynchburg. These projects will result in these facilities' expansion and minor remodeling, and we are excited to see the outcome!

Over the past few months, multiple staff have been attending training focusing on necropsy, quality management, and laboratory diagnostics. We are thrilled to have the opportunity to send staff to these trainings and for our system to utilize the benefits of these opportunities to grow!

We are excited to have a new Diagnostician/Laboratory Director starting in September at the Lynchburg Laboratory- Dr. Taylor Young, who comes to us from a mixed animal practice in central Virginia. Necropsy services will begin again there on November 1st, and we look forward to serving client needs in central Virginia again!

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

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