Laboratory Services Division
Animal Health Laboratory

# AHL Newsletter

**AHL Newsletter, Volume 29, Number 3** **September 2025**

***In this issue:***



**AHL Newsletter**
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*Its mission is to inform AHL clients and partners about AHL current activities, and laboratory-based animal disease events and disease trends.*  *Ideas and opinions expressed herein do not necessarily reflect the opinions of the University or the editor.*

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# Update from the Director

*The view from the Director's office*

September is the time of year to restart planning initiatives following the summer break. Here at the AHL, we are already examining investments in equipment purchases and replacements. The latter is an ongoing process that seems to be accelerating in recent years - has anyone else been informed that the company will no longer "support the software", requiring the purchase of new equipment although the previous version still works perfectly fine? One of our recent purchases is the BD Kiestra InoqulA sample processor which automates the processing of samples and streaking of bacteriology plates. This equipment is used in larger medical and hospital laboratories and is one of the few options available for this purpose. Our previously used plate streakers are no longer available for purchase or repair, and our only other option was a return to hand streaking of each bacteriology plate. AHL inoculates around 350 bacteriology plates every day, and hand streaking would require additional staff and increase the risk of repetitive strain injuries. InoqulA provides consistency and uniformity in sample processing and streaking and also prepares slides from the inoculum for Gram staining. Overall, this purchase appears to be a necessary and innovative investment for AHL's microbiology laboratory.
Please refer to the article "New AHL Bacteriology transport media – ESwabs" in this newsletter for additional information on appropriate samples for bacteriology testing.

I hope you all had a wonderful summer and are prepared for the renewal that every fall seems to generate.

*Maria Spinato, Director*

*Animal Health Laboratory, University of Guelph, Guelph, ON.*

# Update from Specimen Reception and Client Services

*Tim Pasma, Jennifer Zoethout*

*Animal Health Laboratory, University of Guelph, Guelph, ON.*

AHL Newsletter 2024;28(3):3.

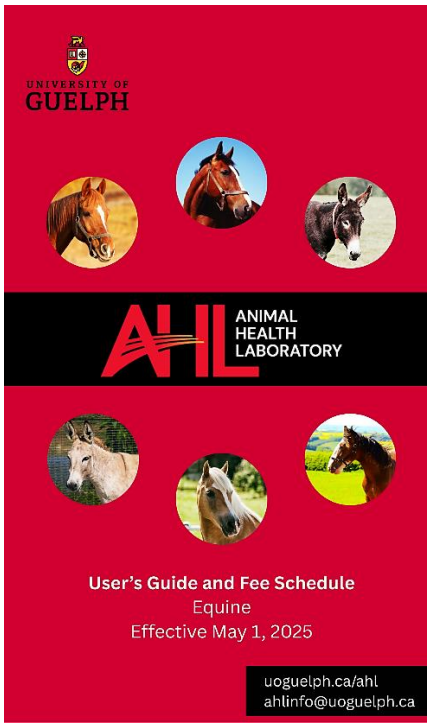
## Proper use of waybills

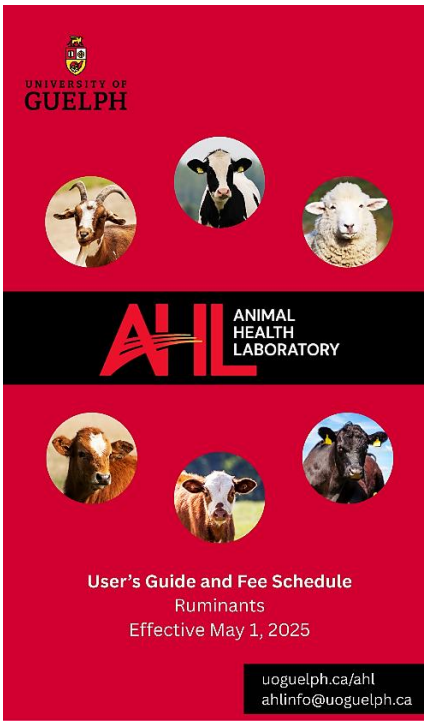
The AHL offers free shipping for laboratory submissions within Ontario by providing waybills for shipping via Purolator. **These waybills are to be used once only and are valid for 1 year from the issue date.**

Purolator has brought to our attention that some clients are using the same waybill multiple times. In these cases, Purolator will return the shipment and charge a fee associated with the return to the client. Please ensure that you use shipping waybills properly to avoid delays in sample submissions and unexpected costs.

## Coming soon – Equine and ruminant pocket guides

We will be sending a pocket-sized version of our User's Guide and Fee Schedule to clinicians practicing with equine and ruminant species. These pocket guides will be small enough to take with you in the glove compartment of your vehicle or in your pocket. They will contain the same information found in our regular fee guide, but will only include equine or ruminant species-specific information.



# Update on *Salmonella* spp. serotyping available at AHL

*Durda Slavic*

*Animal Health Laboratory, University of Guelph, Guelph, ON.*

AHL Newsletter 2025;29(3):4.

At AHL *Salmonella* spp. isolates are routinely forwarded to the Public Health Agency of Canada (PHAC) for serotyping. This testing frequently takes 3-4 weeks or longer if environmental *Salmonella* spp. are submitted. To provide our clients with alternative and faster turn-around-times, the Bacteriology section at AHL recently validated the Check&Trace *Salmonella* serotyping method (CTS). This is a PCR-based method that can be used to differentiate more than 100 different *Salmonella* serotypes that are most frequently isolated from poultry environmental samples, as well as from clinical samples in all animal species. Most relevant clinical *Salmonella* serotypes that can be differentiated by CTS are shown in (**Table 1**).

If faster turn-around-time (TAT) for serotyping is needed to include/exclude any of the serotypes from (**Table 1**), this test can be requested. The test can only be performed on pure bacterial cultures of isolates already identified as *Salmonella* spp. The current price per isolate is $81, and the test code is 'bssero'. If culture and/or bacterial identification are needed, extra charges will apply.

For additional information, including the expanded list of *Salmonella* spp. serotypes, please contact ahlbact@uoguelph.ca.

**Table 1.** Some of the *Salmonella* serotypes differentiated by Check&Trace test.

| | | |
|---|---|---|
| *S.* Abaetetuba | *S.* Heidelberg | *S.* Panama |
| *S.* Agona | *S.* Idikan | *S.* Paratyphi B (Java) |
| *S.* Alachua | *S.* Indiana | *S.* Poona |
| *S.* Albany | *S.* Infantis | *S.* Putten |
| *S.* Anatum | *S.* Javiana | *S.* Reading |
| *S.* Berta | *S.* Kentucky | *S.* Rissen |
| *S.* Bovismorbificans | *S.* Kedougou | *S.* Saintpaul |
| *S.* Braenderup | *S.* Kiambu | *S.* Sandiego |
| *S.* Brandenburg | *S.* Kingston | *S.* Schwarzengrund |
| *S.* Bredeney | *S.* Liverpool | *S.* Senftenberg |
| *S.* Cerro | *S.* Livingstone | *S.* Stanley |
| *S.* Choleraesuis | *S.* London | *S.* Tennessee |
| *S.* Corvallis | *S.* Mbandaka | *S.* Thompson |
| *S.* Cubana | *S.* Minnesota | *S.* Typhimurium |
| *S.* Derby | *S.* Molade | *S.* Typhimurium monophasic (1,4,[5],12:i:-) |
| *S.* Dublin | *S.* Montevideo | *S.* Uganda |
| *S.* Enteritidis | *S.* Muenchen | *S.* Urbana |
| *S.* Gallinarum | *S.* Muenster | *S.* Virchow |
| *S.* Pullorum | *S.* Newport | *S.* Worthington |
| *S.* Give | *S.* Ohio | *S.* Yoruba |
| *S.* Goldcoast | *S.* Oranienburg | *Salmonella* 4,[5],12:d:- |
| *S.* Hadar | *S.* Orion | |
| *S.* Havana | *S.* Ouakam | |

# New AHL Bacteriology transport media - ESwabs

*Durda Slavic and Sarah Lippert*

*Animal Health Laboratory, University of Guelph, Guelph, ON*

AHL Newsletter 2025;29(3):5.

AHL is replacing Amies gel-based swabs with ESwabs, a new transport system for microbiology testing. ESwab is a multi-purpose collection and transport system consisting of a flocked swab and 1 mL of liquid Amies transport medium in a screw cap tube. Upon collection, the clinical specimen is eluted from the flocked swab into the liquid medium and maintains viability of a variety of microorganisms for up to 48 h at both room and refrigerated temperatures. Uniform suspension of the specimen results in more consistent sample application on different culture plates and can be used for multiple tests. This system can be used for aerobic, anaerobic, *Mycoplasma*, *Ureaplasma* and fungal cultures, as well as PCR testing. The liquid medium in ESwabs is also suitable for the transport of small tissue samples for culture submission.

Please note that these tubes are not suitable for virus isolation – VTM swabs are still the best option for this test.


We highly recommend the use of ESwabs for all your microbiology testing needs. The price is comparable to current gel-based swabs. For pricing and ordering please contact ahlinfo@uoguelph.ca.

# OAHN Update – September 2025

*Mike Deane, Tanya Rossi*

*Animal Health Laboratory, University of Guelph, Guelph, ON.*

Throughout the summer, the OAHN networks have completed and published multiple research projects, veterinary reports, and species-specific resources. We anticipate a busy fall, with new reports from the companion animal, equine, poultry, and swine networks.

## Published OAHN Research Projects

The OAHN networks conduct species-specific research, focused on diseases that affect Ontario animals. You can see a full list of these research projects (both in-progress and complete) here: OAHN Projects.

The latest completed projects are below:

- OAHN Swine Project: Prevalence of PHEV in growing pig submissions to the AHL
- OAHN Wildlife Network Project: Muskrat Health and Disease Surveillance
- OAHN Wildlife Project: Botulism Test Development

## New Resources

The Companion Animal, Equine, and Small Ruminant networks have created valuable resources for vets and owners. Below, find links to the resources published this summer.

Companion Animal:

- Antimicrobials for Use in Dogs (table)
- Influenza A diagnostic testing

Equine:

- Infographic: How Gestation Length Affects Limb Deformities in Thoroughbred Foals
- Infographic: How Farm Size & Biosecurity Shape Strongylus vulgaris Risk in Horses
- Infographic: Understanding Equine Gammaherpesvirus EHV-2 & EHV-5

Small Ruminants:

- Fact Sheet: Cysticercus ovis: Protecting Your Flock and Income

## New Reports

These are the most recent reports published by OAHN. We publish regularly, so be sure to check back in between newsletters to see what's new. To view any of the veterinary reports below, please click on the link for each report, or go to OAHN.ca and navigate to the species in which you are interested.

**Swine Network – Q1 2025 Vet Report**

- 3 New OAHN Swine Projects
- Porcine Epidemic Diarrhea (PEDV)/ Porcine Deltacoronavirus (PDCoV)
- OAHN Veterinary Clinical Impression Survey Veterinary Comments
- Animal Health Laboratory Immediately Notifiable Disease Review
- Laboratory Diagnostic Reports
- Ontario Slaughter Statistics
- CanSpotASF Surveillance Update
- OAHN Project Update- Porcine Hemagglutinating and Encephalomyelitis Virus (PHEV)
- International Disease Topics of Interest Summary

**Poultry Network - Q2 2025 Vet Report**

- Leveraging Artificial Intelligence-Based Decision Support Tools for the Control of Avian Influenza Outbreaks
- Newcastle disease
- Reovirus – Summary of Genotypes by Year in Ontario
- Poultry Veterinary Survey Highlights – Q4 2024
- Events and News

**Small Ruminant Network - Dec 2024 – Apr 2025 Vet Report**

- Animal Health Laboratory Q4 2024 & Q1 2025 Case Summary Data
- Mastitis Pathogens from Milk Cultures Submitted to the Animal Health Laboratory in 2023 and 2024
- Provincial Abattoir Slaughter and Condemnation Data
- International Disease Topics of Concern

**Bovine Network - Feb – Apr 2025 Vet Report**

- Q1 Bovine Data from AHL
- Bovine IBR Abortion Summary
- *Salmonella* Agona
- Global Surveillance Update
- Condemnations data from Ontario Abattoirs
- Malignant Catarrhal Fever 101

**Equine Network - Q1 2025 Vet Report**

- Strangles and EHV-1 Resources
- Bits N Snips: Gammaherpesvirus, *S. vulgaris*
- Network Member Reports
- Syndromic and lab Surveillance Dashboard
- Equine Research
- ResearchONequine
- Ontario Equine Disease Surveillance Summary

**Companion Animal Network - Jan-Apr 2025 Veterinary Need-2-Know Update**

- OAHN Spring Survey and Lab Data: Key Results
- Rabies Update: Foxes in the North
- Feline Disease Cluster, Middlesex
- Ontario Lepto Trends, 2023-2024
- CAPCvet Graphs
- Echinococcus Update
- Don't-miss Resources!
- US Canine Imports

# Ovine white liver disease in a yearling ewe

*Dominique Comeau*

*Animal Health Laboratory, University of Guelph, Guelph, ON*

AHL Newsletter 2025;29(3):9.

A yearling ewe was submitted for postmortem examination at the Animal Health Laboratory following a history of anorexia, depression, and weight loss lasting several weeks, eventually leading to euthanasia. Discharge around the eyes and mouth was also described as the clinical condition progressed. Several animals had died sporadically in the past year with a similar presentation, and variable underlying causes including parasitism and mineral imbalances were identified. On gross examination of this ewe, the blood was thin and watery, supporting anemia, and the mucus membranes were off-white with no evidence of jaundice. There was severe effusion in the abdomen, thorax, and pericardium with at least three liters of serous fluid in the abdomen. The liver was diffusely pale and very firm. Histologically, this correlated with severe bridging fibrosis effacing up to 50% of the liver parenchyma. Abundant lipid vacuolation was noted in the remaining hepatocytes, along with ceroid pigment and scattered neutrophils (**Fig. 1**). In addition, there was hypertrophy of astrocytes (neuronal support cells) in the brain, suggestive of early hepatic encephalopathy (**Fig. 2**).

Ancillary testing was performed to investigate possible parasitism or metabolic disease. A small burden of nematodes was found, but was not considered sufficient to be a major cause of the pathologic findings. A mineral panel performed on liver found that copper levels were within normal limits, ruling out copper toxicity. This was an important differential diagnosis; however, it was considered less likely due to the lack of jaundice, yellow discoloration of the liver, or dark discoloration of the kidneys. This ewe had cobalt levels too low to be detected. The normal reference interval for this mineral in sheep is 0.025-0.085 ug/g, and the lower limit of detection for our laboratory method is 0.006 ug/g. This result, together with the histologic findings, was considered diagnostic for ovine white liver disease.

Cobalt is an essential trace element in sheep and other ruminants, and it is required for the synthesis of vitamin B12 by the ruminal flora. Low levels are typically associated with sandy or high pH soil, or intensive cropping leading to leaching of the soil. Studies have demonstrated reduced activity of several vitamin B12-dependent enzymes within hepatocytes of lambs with experimentally-induced white liver disease, which hinders the oxidation of fatty acids and is the probable cause of lipid accumulation and secondary hepatocellular injury in this disease. Lesions of hepatic encephalopathy, as noted in this ewe, are occasionally reported in cases of white liver disease. Liver damage leads to increased levels of circulating ammonia, causing injury to the brain.

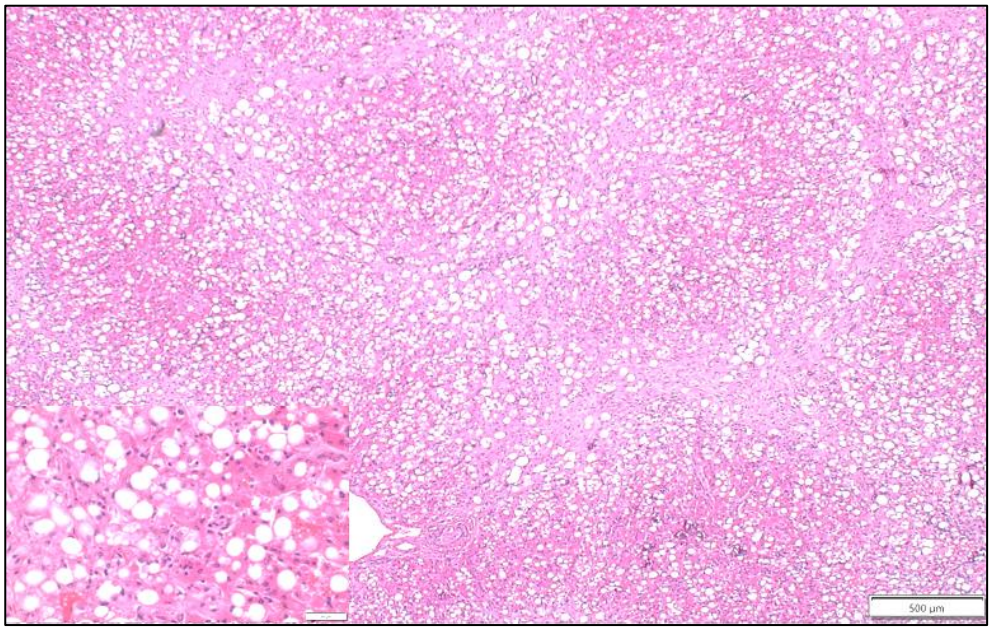



**Figure 1.** Bridging fibrosis throughout the liver (pale areas) with marked vacuolar change throughout (clear spaces). Inset shows higher magnification of vacuoles with intermingled inflammatory cells. H&E stain.

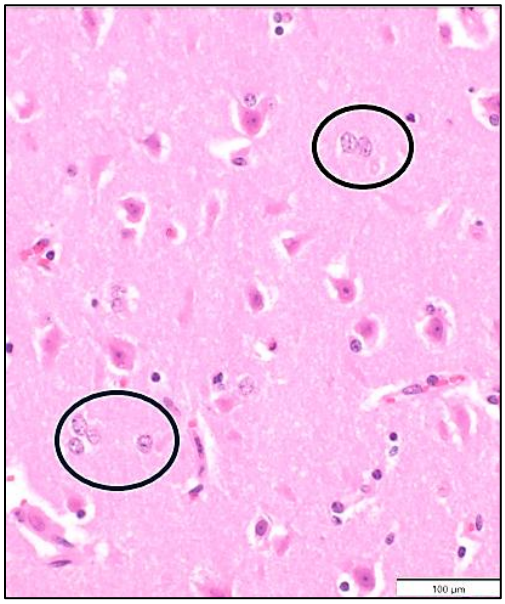



**Figure 2**. Section of the cerebrum showing swollen and clustered astrocytes (circled). H&E stain.

**References**

1. Kennedy S, et al. Histopathologic and ultrastructural alterations of white liver disease in sheep experimentally depleted of cobalt. Vet Pathol 1997;34(6):575–584.
2. Ulvund MJ. Ovine white-liver disease (OWLD) pathology. Acta Veterinaria Scandinavica 1990;31(3):309–324.

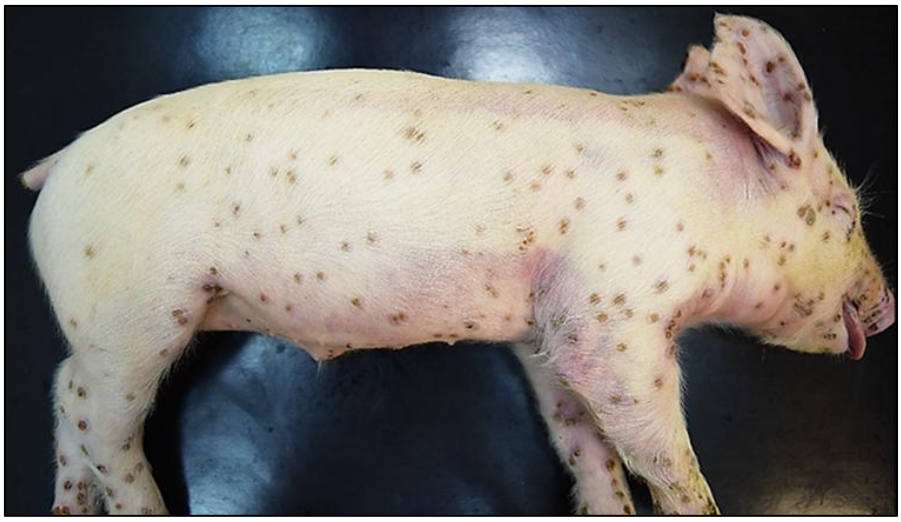
# SWINE

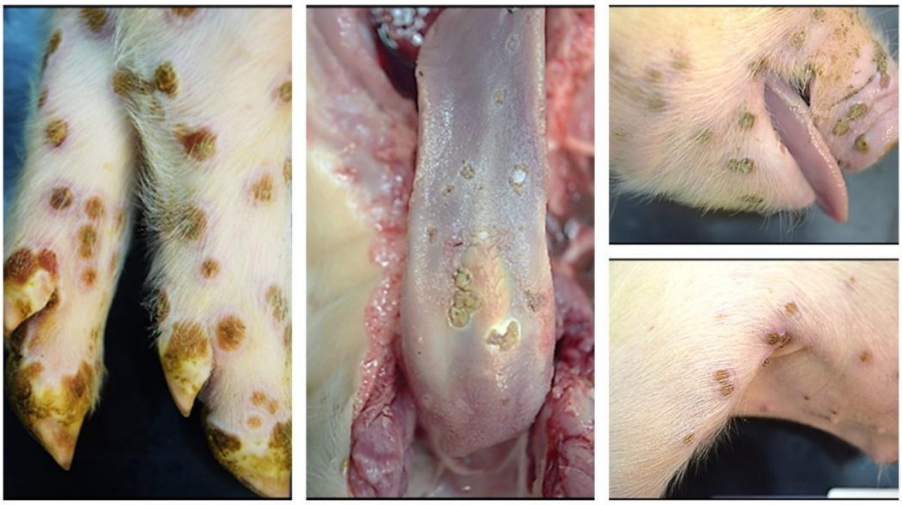
## What is your diagnosis?

*Josepha DeLay, Hannah Jansen*

*Animal Health Laboratory, University of Guelph, Guelph ON (DeLay), South West Vets, Stratford, ON (Jansen)*

AHL Newsletter 2025;29(3):11.





Multiple discrete ulcerative and proliferative, crusted skin lesions were identified in 2 neonatal piglets in a single litter. The sow was unaffected. Lesions involved haired skin, lip margins, tongue, and coronary band.

What are your differential diagnoses? Stay tuned for the answer in the December 2025 AHL Newsletter.

# Respiratory infection in a Harris's hawk (*Parabuteo unicinctus*) by *Cyathostoma* spp.

*Felipe Reggeti, Jacob Avula, Ginger Louws*

*Animal Health Laboratory, University of Guelph, Guelph, ON (Reggeti, Avula); Campus Estates Animal Hospital, Guelph, ON (Louws)*

AHL Newsletter 2025;29(3):12.

A 10-year-old female Harris's hawk was presented to the referring veterinarian with a history of coughing and sneezing. The patient had been suspected of having aspergillosis the previous year. On clinical examination, a bloody mucoid discharge was observed. A complete blood count revealed a mild leukocytosis of 25.6 × $10^9$/L (reference interval [RI]: 5–15×$10^9$/L) and a mild eosinophilia of 6.4×$10^9$/L (RI: 0–4×$10^9$/L). The heterophils were well-granulated, with no evidence of a left shift or toxic changes (**Fig. 1**). These hematological findings were non-specific, although eosinophilia can be associated with fungal infections, parasitism, hypersensitivity reactions, mixed inflammation, and, less commonly, paraneoplastic syndromes.

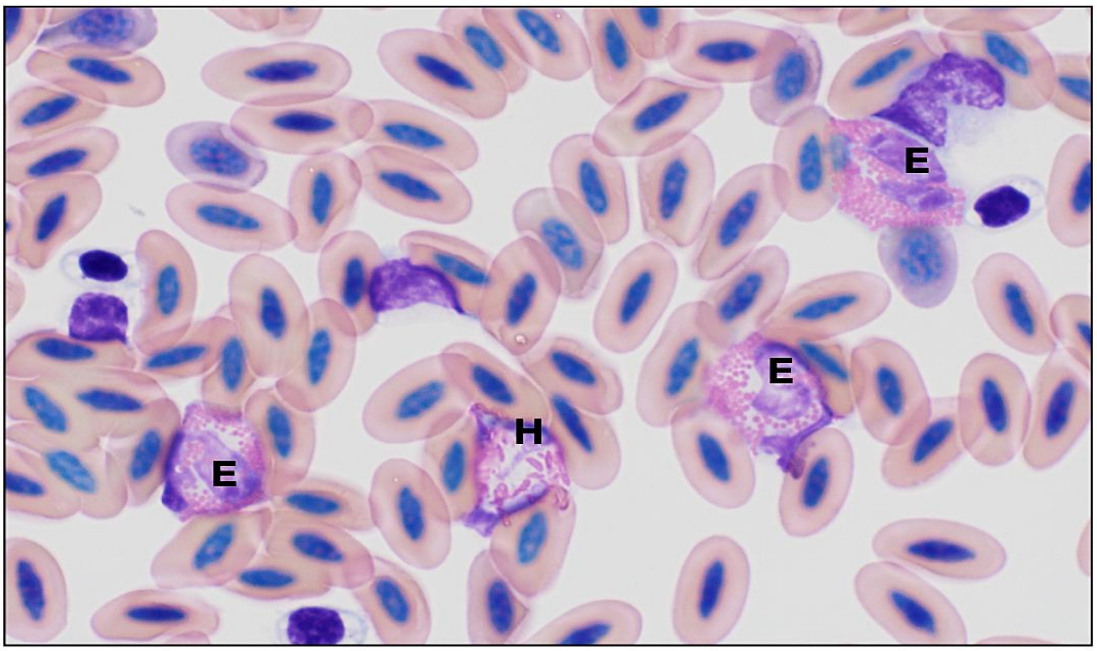



**Figure 1**. Blood smear examination showing eosinophilia. Eosinophils **(E)** can be distinguished from heterophils **(H)** by their smaller round eosinophilic-staining granules. Wright's stain, 100x.

Fluid collected from the endotracheal tube (ETT) was submitted to the Animal Health Laboratory,

University of Guelph, for cytological examination. The sample consisted of mixed inflammatory cells within a mucoid background, with low numbers of red blood cells, unremarkable epithelial cells, a mixed population of free bacteria, and a few contaminants (plant material). Numerous large, blue-green staining oval structures, suggestive of parasitic ova, were scattered throughout the sample. A few "vesicular" structures with granular contents were also noted, possibly environmental contaminants (**Fig. 2**).

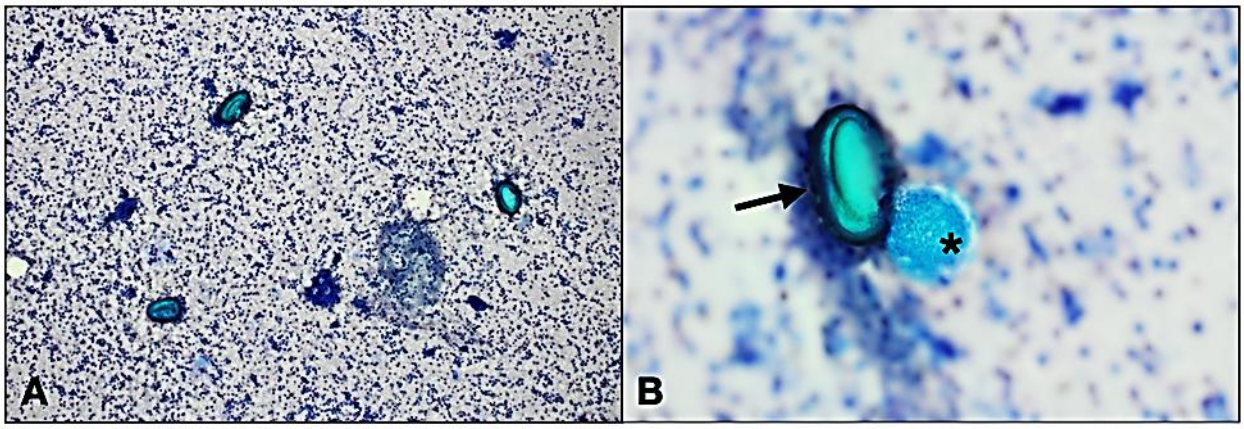
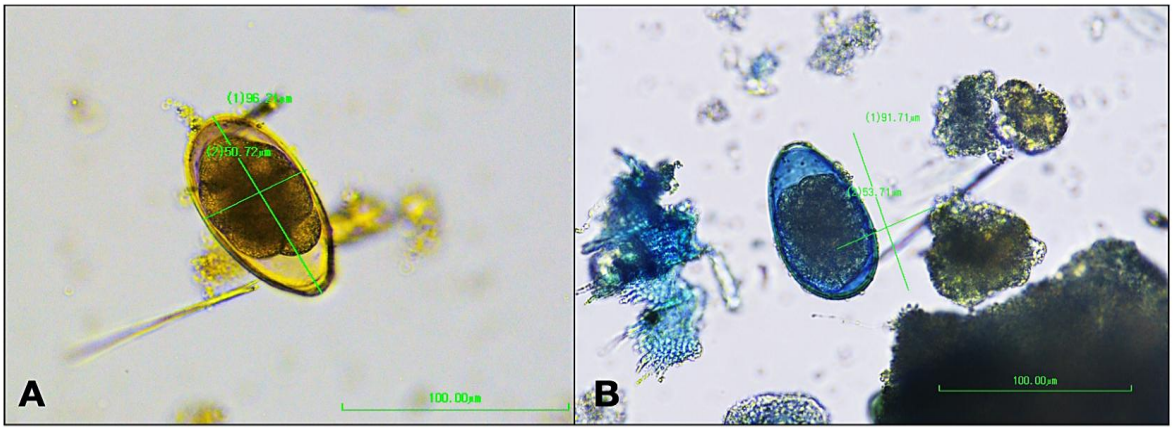



**Figure 2**. Cytology smears of endotracheal tube fluid. **2A**. Several oval structures are observed within a proteinaceous background with mixed inflammatory cells. Wright's stain, 10x**. 2B**. Oval structures compatible with parasite ova (arrow) and vesicular structure of unknown significance with granular content (asterisk). Wright's stain, 40x.

Additional diagnostic testing included centrifugal fecal flotation for parasite ova identification, and fungal culture, considering the patient's history of suspected aspergillosis and the presence of unidentified structures resembling fungal spores in the cytology of the ETT fluid. Fecal flotation revealed unioperculated parasite ova with morphometric range of 84-96 µmx48-53 µm which were identified as *Cyathostoma* spp. (**Fig. 3**). The fungal culture was negative, suggesting that the spore-like structures




observed were probably environmental contaminants.

**Figure 3**. Fecal flotation. Unioperculated parasite ova with morphological features consistent with *Cyathostoma* spp. **3A**. Ova recovered on fecal float. **3B**. Ova stained with methylene blue showing the single polar operculum.

The patient was treated with ivermectin at 0.3 mg/kg orally, administered twice at a three-week interval. Clinical symptoms resolved, and follow-up radiographs showed resolution of the parabronchial pattern and improved air sac definition. A repeat fecal test was negative for parasite ova.

Tracheal worms of birds are parasitic nematodes belonging to the family *Syngamidae* which includes two genera: *Syngamus* and *Cyathostoma*. *Cyathostoma* spp. has been reported in predatory birds in Canada, including hawks and owls. Adult parasites reside in the trachea and produce eggs that are coughed up, swallowed, and subsequently passed in the feces. In the environment, infective larvae develop within the eggs, which may be ingested by paratenic (carrier) invertebrates such as earthworms. These invertebrates can transmit the infection when consumed by other animals. Interestingly, raptors typically do not feed directly on such invertebrates, but are likely infected through predation on rodents with infected intermediate hosts in their gastrointestinal tracts. Wild birds infected with *Cyathostoma* spp. are often asymptomatic but may present with emaciation and respiratory signs such as sneezing, dyspnea, and abnormal respiratory sounds. These signs are associated with air sacculitis and the presence of parasites in the trachea. Antemortem diagnosis can be achieved via fecal flotation by identifying morulated eggs measuring approximately 75–90 µm×45–60 µm with a single polar operculum.

**Reference**

1.Fernando MA, Barta JR. Tracheal worms. In: Parasitic Diseases of Wild Birds. Atkinson CT, Thomas NJ, Hunter DB, eds. John Wiley & Sons, 2009:343–354.

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# Remember that ‘other’ poultry foreign animal disease! Newcastle disease/avian paramyxovirus 1

*Emily Martin, Davor Ojkic*

*Animal Health Laboratory, University of Guelph, Guelph, ON.*

AHL Newsletter 2025; 29(3):14.

Considering the ongoing threat of avian influenza circulating in wild birds, it can be easy to develop tunnel vision when it comes to other disease threats to poultry flocks. Recently, in June of 2025, avian paramyxovirus 1 was identified in a commercial pigeon operation in British Columbia. This virus was confirmed to be a pigeon-adapted strain of Newcastle disease (pigeon paramyxovirus 1 (PPMV-1)). The classification and naming of Newcastle disease virus (NDV) has evolved over time. While it is still commonly referred to as NDV, the current term is avian paramyxovirus 1 (APMV-1). The World Organization for Animal Health (WOAH) continues to use both NDV and APMV-1 in its communications.

Because multiple strains of APMV-1 exist, the progression of the disease depends on several factors including the viral strain, the species infected, the age of bird, concurrent infections, stress levels and immune status. As a result, clinical signs are not necessarily pathognomonic. The general organ systems affected include the respiratory tract, digestive tract and nervous system. A few examples of potential signs include high fever, dyspnea, diarrhea, depression, nervous signs, mucosal/serosal bleeding, partial or complete loss of egg production and abnormal egg formation (e.g., abnormal colour, shape, watery

albumen). In vaccinated birds, lesions are often absent, or minimal. Transmission occurs rapidly after exposure to infected feces, respiratory secretions or infected fomites (food, water, equipment). The average incubation period is 5-6 days, but it can range from 2-15 days. Mortality rates vary depending on the strain and host, but can be up to 100%. The virus can survive for weeks in the environment. Classification of pathotypes based on clinical signs in chickens is outlined in **Table 1**.

**Table 1.** Pathotypes of Newcastle Disease (WOAH Terrestrial Manual 2021)

| Pathotypes (increasing severity) | General clinical signs |
|---|---|
| Subclinical | Enteric infection usually |
| Lentogenic | Respiratory infections – mild or subclinical |
| Mesogenic | Low mortality (<50%)<br>Respiratory signs, occasional nervous signs |
| Neurotropic velogenic (nvNDV) | High mortality<br>Neurologic signs and respiratory signs |
| Viscerotropic velogenic (vvNDV) | Hemorrhagic lesions in GI tract, possible neurologic signs |

Wild birds are not all equally susceptible to the virus, but many species can be infected. They can act as reservoirs for the virus, resulting in endemic infection and sporadic outbreaks. According to the Canadian Wildlife Health Centre (CWHC), APMV-1 infection was last detected in Ontario in 2024 in cormorants. The presence of this virus in wildlife represents a potential source of infection for domestic birds. While Newcastle disease is endemic in commercial poultry in many countries around the world, commercial poultry in Canada have remained free of this disease for decades, resulting in its classification as a foreign animal disease (FAD).

APMV-1 is an enveloped RNA virus that utilizes specific viral attachment proteins to bind and enter host cells. The hemagglutinin-neuraminidase (HN) protein facilitates viral attachment to the host cell surface. Along with HN, the fusion (F) protein enables fusion of the viral envelope with the host cell membrane, allowing viral entry. In infected cells, the F protein is initially produced as an inactive precursor, F0, which lacks fusion activity. Activation occurs when host proteases cleave F0 into two subunits, F1 and F2, which are essential for membrane fusion and viral entry. The amino acid sequence at the F protein cleavage site determines whether this cleavage can occur efficiently. The F protein is considered the main virulence determinant of APMV-1. Virulent APMV-1 strains possess multiple basic amino acids at the cleavage site, enabling cleavage by ubiquitous host proteases found in a wide range of tissues. This allows for systemic viral spread and contributes to enhanced pathogenicity.

Tissues, oropharyngeal swabs, and cloacal swabs are used to detect acute APMV-1 infection by PCR. The matrix (M) gene is a standard target for PCR due to its highly conserved nature across all strains, making it suitable for initial screening regardless of APMV-1 pathotype. In Canada, Newcastle disease is reportable due to its significant impact on poultry and trade. A positive APMV-1 M gene PCR result will trigger a mandatory notification to the CFIA, and samples will be sent to the National Centre for Foreign Animal Disease (NCFAD) in Winnipeg for confirmatory testing and further characterization to determine the strain's virulence and to guide control measures.

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https://doi.org/10.3389/fvets.2022.936251.

# Pulmonary artery rupture in a stillborn Standardbred foal

*Emily Brouwer*

*Animal Health Laboratory, University of Guelph, Guelph, ON.*

AHL Newsletter 2025; 29(3):17.

A stillborn foal was submitted to the Animal Health Laboratory for diagnostic workup for suspected infectious placentitis. There had been concerns on the farm for the previous three breeding seasons, with the submitting veterinarian reporting increased incidence of placentitis cases estimated at 1-2 per season out of 15-25 mares. Some of the foals born to mares with diagnosed placentitis have been poor doers, and one was suspected to have in utero sepsis.

The dam of this foal was reported to have a one week history of mucohemorrhagic vulvar discharge, and was diagnosed with placentitis based on elevated combined thickness of the uterus and placenta via transrectal ultrasound (14.3 mm). The mare was treated with trimethoprim sulfa, flunixin meglumine, and altrenogest (Regu-Mate). The mare subsequently had a red bag delivery of a stillborn foal at 329 days gestation.

On gross examination, the placenta was noted to be diffusely edematous, up to 1 cm thick in the non-gravid horn, and there were tortuous, dilated lymphatic vessels on the allantoic surface. The chorionic surface was opaque, diffusely tan-brown and thickened with scattered petechial hemorrhages. The umbilical cord was 54 cm long with no twists. The amnion was unremarkable.

On internal examination, the pericardial sac was distended and taut, and contained approximately 200 mL of clotted blood. There was a focal, elliptical rent in the pulmonary artery measuring 22 mm x 5 mm on the serosal surface that was surrounded by intramural hemorrhage (**Fig 1.)**. On cut section, the defect in the intimal surface measured approximately 5 cm x 1 cm, and had ragged edges (**Fig. 2)**.

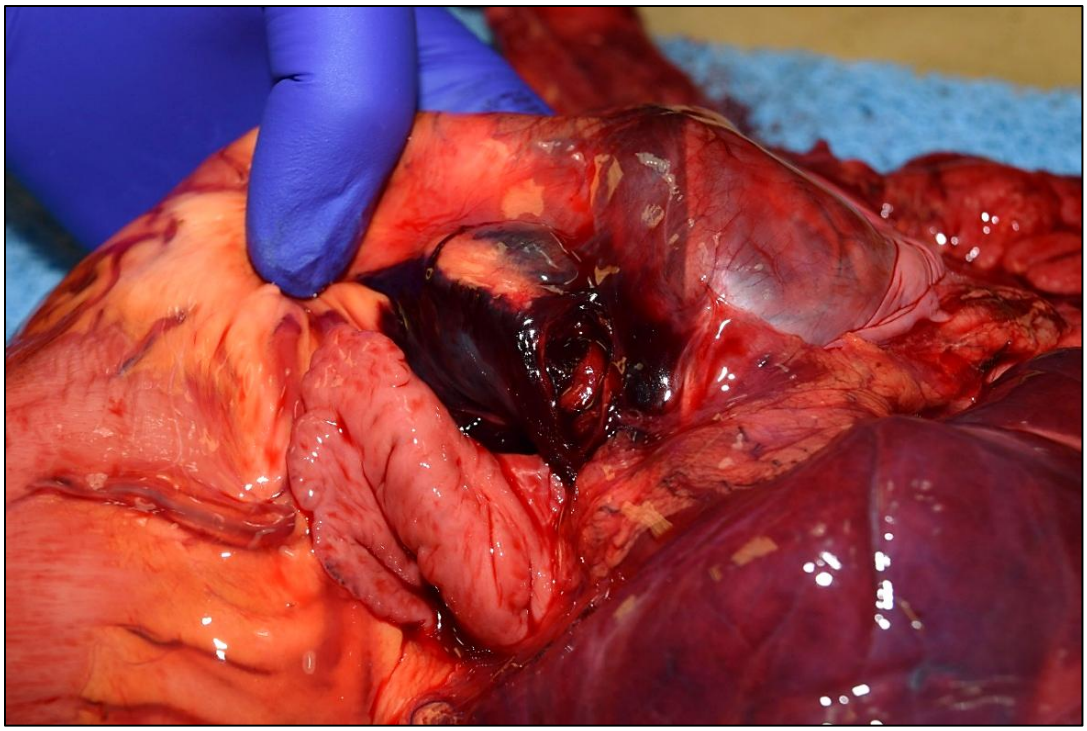

**Figure 1.** Pulmonary artery rupture, serosal surface.

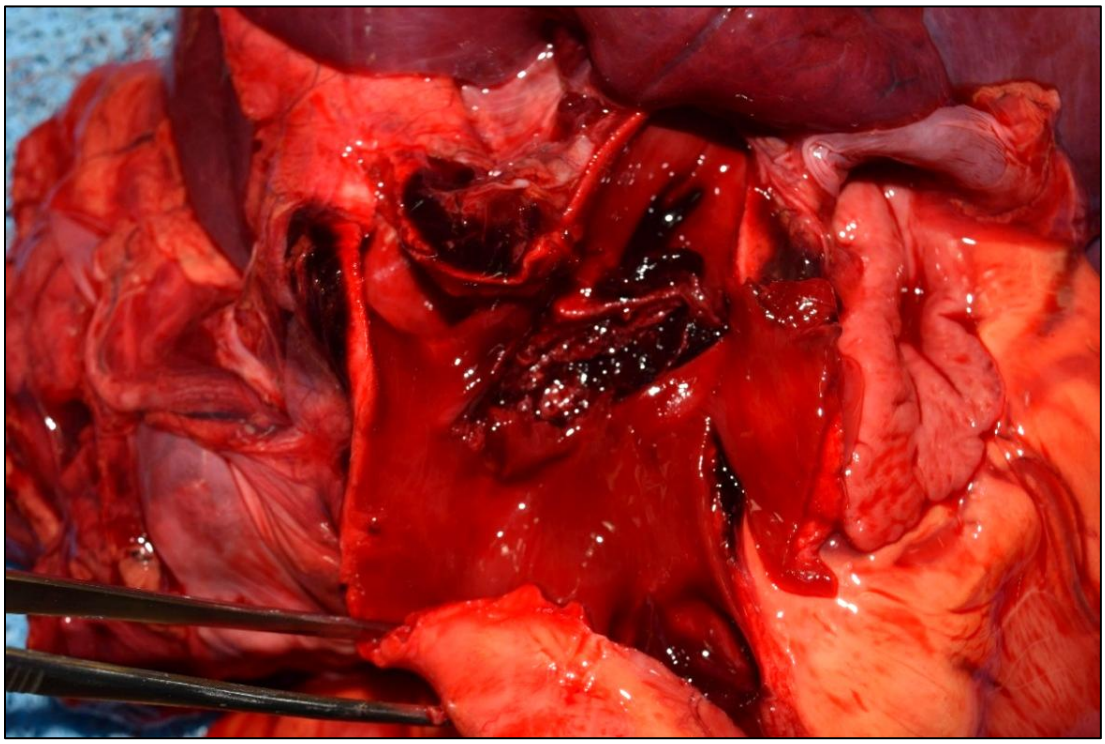

**Figure 2.** Pulmonary artery rupture, intimal surface

On gross examination, the foal was diagnosed with pulmonary artery rupture and severe hemopericardium, as well as placental edema.

Histologically, the placenta was severely, diffusely edematous and the vessels were markedly dilated. There was no histologic evidence of an inflammatory or infectious process in the sections of placenta or fetal tissues. Bacterial culture of the placenta isolated low numbers of *E. coli* in mixed culture (interpreted as contamination), and there were no bacterial pathogens isolated from either the fetal lung or stomach content. *Leptospira* spp., equine arteritis virus, and equid herpesvirus 1 (both neuropathogenic and non-neuropathogenic variants) were not detected by PCR.

Based on these findings, spontaneous vascular rupture was suspected to be the result of vascular fragility- either related to an inherited collagen defect (such as Marfan syndrome) or copper deficiency. Fetal copper levels were elevated (89 µg/g, reference 4.0-7.5 µg/g), which was considered normal as fetuses will store copper at the expense of the dam. This finding ruled out copper deficiency as a cause of vascular fragility. Fragile foal syndrome type 1, an inherited disease that results in fragile skin, hematomas, and accumulation of fluid (among other lesions) was considered, but ultimately was not tested for due to the signalment. The mutation responsible for fragile foal syndrome type 1 has been reported predominantly in warmbloods, and much less frequently in other breeds such as Thoroughbreds, American Sport Ponies, Knabstruppers, and Haflingers.

In the absence of an inflammatory component associated with the placental edema, the placental lesions were suspected to be the result of impaired fetal circulation related to cardiac tamponade. The underlying cause of vascular rupture was not identified.

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# Mallory-Weiss syndrome in a cat with an intestinal foreign body

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An 18-month-old male Bengal cat was presented for postmortem examination after being found dead in a large pool of blood.

He was neutered three days prior, and by all accounts was clinically normal immediately following his surgery. He was given a 0.1 mg/kg dose of meloxicam subcutaneously, and discharged with oral meloxicam for four days. On the second day following his surgery when he was brought in for a recheck examination, the owners noted that he had vomited twice overnight, had stopped eating, and was 7% dehydrated. The veterinarian advised the client to discontinue the meloxicam, and the cat was brought home following some supportive care including subcutaneous fluids, maropitant injection, and mirtazipine. The following morning, the owners heard vomiting and a splashing sound, and when they investigated, they found him deceased.

On gross postmortem examination, the cat was found to have markedly pale oral mucus membranes and conjunctiva. There was hemorrhagic fluid in the oral cavity and dried blood at the nares, around the mouth, on the ventral chest, and on the forelimbs.

On internal examination, the stomach contained scant dark brown, granular fluid, consistent with digested blood. There was a linear tear in the gastric mucosa, approximately 3 cm long and 1 cm wide, extending from the cardia of the stomach up into the distal esophagus (**Fig. 1**). The trachea and esophagus both contained granular dark brown fluid, similar to that noted in the stomach. The small intestine was markedly dilated, thin walled, and contained abundant dark brown fluid. Tightly lodged in the distal jejunum/proximal ileum was a 3.6 cm x 1.5 cm diameter piece of dense, green foam (**Fig. 2**). Apart from visceral pallor, no other significant internal abnormalities were noted.

The cause of death was determined to be the combined effects of acute blood loss and obstructive asphyxia due to the fluid in the trachea.

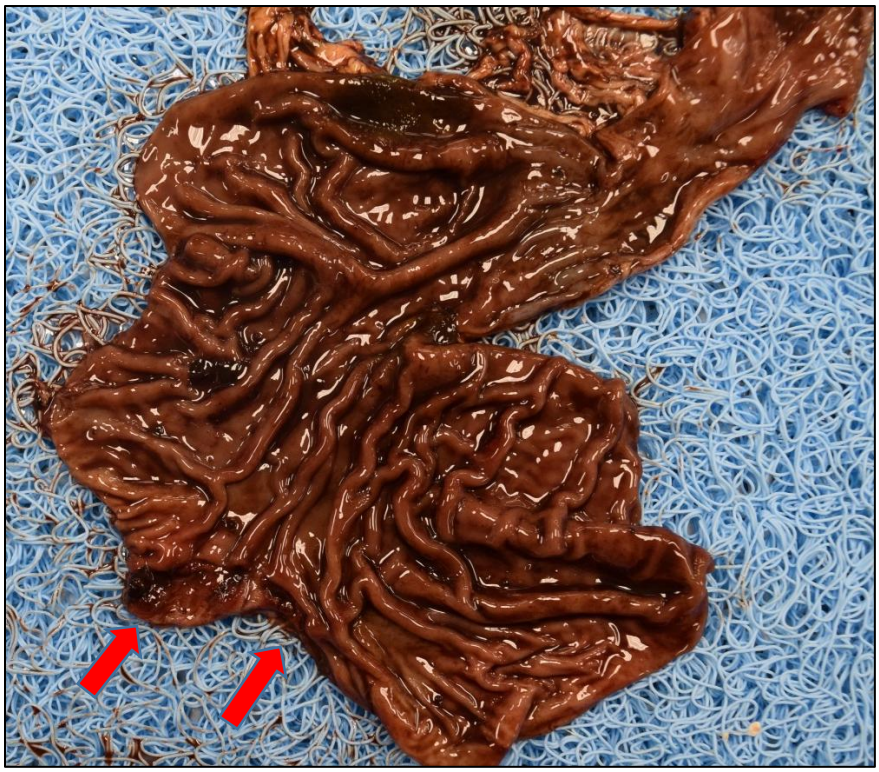

**Figure 1.** Linear tear in the gastric mucosa (arrows).

**Figure 2.** Obstructive foreign body (green foam) in the lumen of the distal small intestine.

The cause of sudden, intractable vomiting was interpreted to be the obstructive foreign body identified in the distal small intestine. The gastroesophageal tear was a spontaneous, non-penetrating mucosal laceration interpreted to be the result of forceful vomiting. The location of the lesion was not grossly compatible with (or in the proper location for) NSAID-related ulceration, which was the main differential diagnosis based on the history of post-surgical meloxicam treatment.

Spontaneous linear gastric tears in humans, known as Mallory-Weiss syndrome, result from increased intragastric pressure. This increased pressure can be endogenous, such as with forceful retching or vomiting, or exogenous (e.g., endoscopic insufflation, transesophageal echocardiography). These mucosal tears are characteristically longitudinal, and tend to occur at the gastroesophageal junction. The precise mechanism is not well understood, but the prevailing theory is that excess pressure in the abdomen causes gastric contents to rush into the esophagus, resulting in increased intragastric pressure, and mucosal tears that reach the submucosal vasculature. Hematemesis is the typical presenting sign, but other clinical signs can include melena, dizziness, syncope, and epigastric pain. In cats, the literature is sparse, but this syndrome has been reported in a cat with chronic vomiting related to atrophic gastritis and *Helicobacter pylori* infection.

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# Atypical poorly granulated mast cell tumor in a cat

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A fourteen-year-old female spayed DSH cat presented for a firm freely movable cutaneous swelling on the face that had been growing gradually over several months. Incisional punch biopsies were taken and microscopic examination with routine hematoxylin and eosin staining revealed a neoplasm of uncertain cell type. Based on the tumor morphology, the primary differential diagnosis was atypical poorly granulated mast cell tumor; other differentials included histiocytic sarcoma and progressive histiocytosis (**Fig. 1**). Toluidine blue, a special/histochemical stain was done on the biopsy sections and highlighted variable numbers of cytoplasmic granules in a significant proportion of neoplastic cells confirming the diagnosis of atypical poorly granulated mast cell tumor (**Fig. 2**). The low mitotic rate of 2 mitotic figures in 2.37 $mm^2$ was predictive of benign behavior and good response to treatment with surgical excision.

Approximately 20% of cutaneous neoplasms in cats are cutaneous mast cell tumors. The head, neck and trunk are common sites. Three histologic subtypes are recognized: well-differentiated, pleomorphic and atypical poorly granulated. Unlike canine cutaneous mast cell tumors, a well-tested and widely accepted grading scheme does not exist for prognostication. Most feline cutaneous mast cell tumors of all histologic subtypes are benign and can be cured by complete surgical excision; however, a small proportion can spread to local lymph nodes or viscera, or may be associated with disseminated cutaneous

disease. Mitotic rate is the best histologic criteria for differentiation between benign and potentially aggressive feline cutaneous mast cell tumors. Atypical poorly granulated mast cell tumors are the least common subtype, the most difficult to diagnose histologically due to the sparse cytoplasmic granularity of the neoplastic cells, and have been reported to regress spontaneously in some cases.

Definitive diagnosis of neoplasms can sometimes be difficult with hematoxylin and eosin staining alone, even with provision of relevant clinical histories and good gross descriptions. Toluidine blue is a dye with an affinity for the cytoplasmic granules in mast cells and is frequently used in cases of suspected mast cell neoplasia in all species to confirm or rule out the diagnosis. If toluidine blue staining results are inconclusive, immunohistochemical staining using an antibody to cKIT (CD117), a protein expressed by mast cells, can be helpful for diagnosis. In this case, toluidine blue staining results were diagnostic.

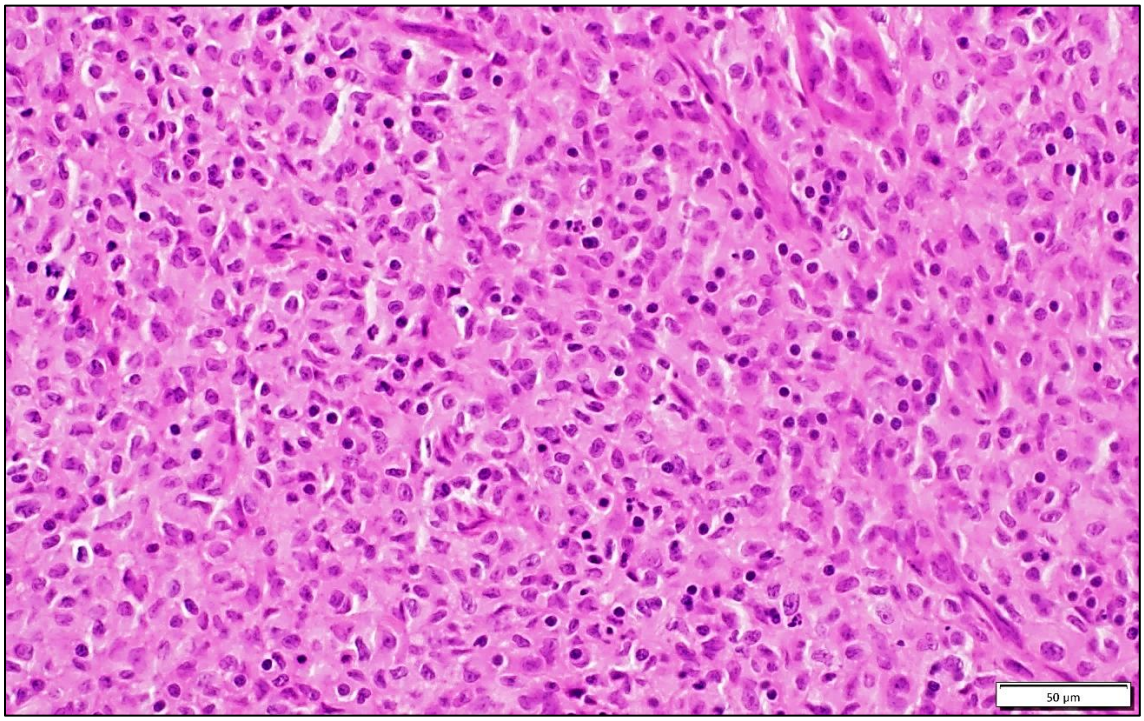


**Figure 1.** This cutaneous neoplasm is composed of sheets of round to spindle-shaped cells. Cytoplasmic granules are not visible in the neoplastic cells with hematoxylin and eosin staining. H&E stain.

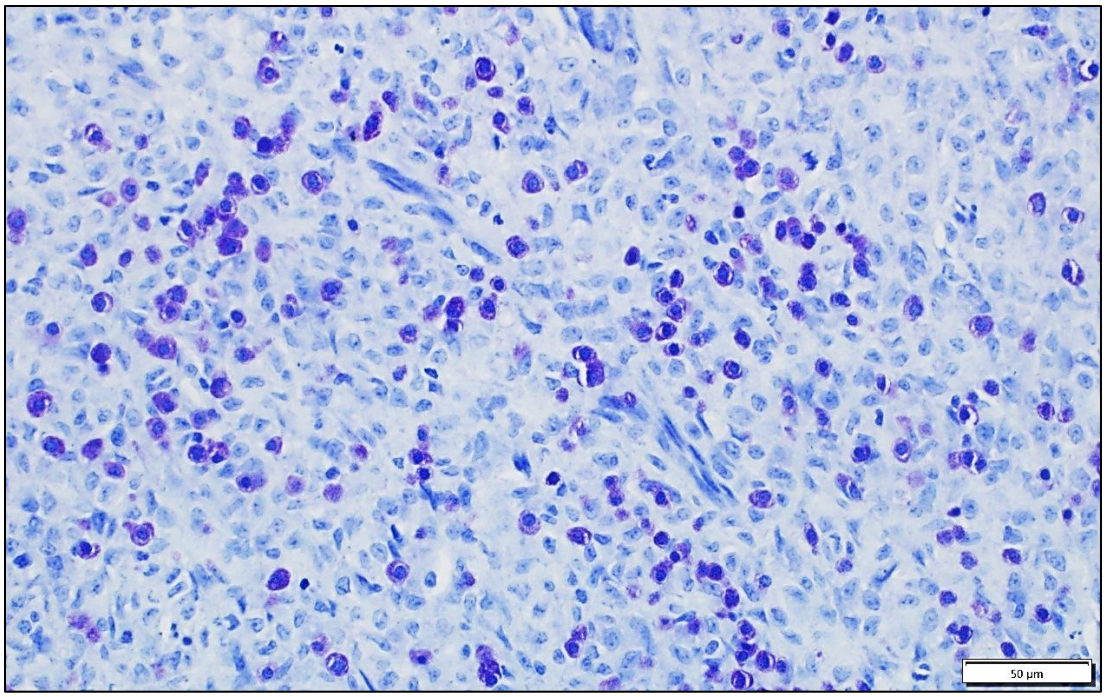


**Figure 2.** Few to many red-purple cytoplasmic granules are appreciated in most neoplastic cells with toluidine blue staining. TB stain.

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2. Herrera C, Henry C. Mast cell tumors in cats: Clinical update and possible new treatment avenues. Journal of Feline Med and Surg 2013;15:41-47.

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# Systemic canine adenoviral infection in a puppy

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A six-week-old puppy was submitted to the Animal Health Laboratory for postmortem examination. The puppy presented to the primary care veterinarian for lethargy, anorexia, and wheezing. On presentation, he had prominent swelling of the face and enlarged submandibular lymph nodes. Treatment was initiated for presumed puppy strangles, however, the puppy passed shortly after.

On examination at the AHL, there was moderate subcutaneous edema along the mandibles and ventral neck, along with enlargement of the submandibular lymph nodes. The only other significant change was subjective enlargement of the liver and the mesenteric lymph nodes. Histologically, the most striking

finding was multifocal random necrotizing hepatitis with large basophilic intranuclear inclusion bodies in affected hepatocytes (**Fig. 1**). Vascular endothelium was reactive in all levels of the brain, and the vessels were often surrounded by thin rims of macrophages and lymphocytes (**Fig. 2**). There was a mild interstitial pneumonia. The lesions in the skin of the jaw were not consistent with puppy strangles (a pyogranulomatous dermatitis would be expected), but instead showed a fibrinoid vasculitis and abundant edema.

Hepatitis with large intranuclear viral inclusions like those observed in this young puppy is typical of infectious canine hepatitis. Given the presence of cerebral and pulmonary inflammation, canine distemper virus was a key differential diagnosis in this case, and the possibility of multiple viral infections was also considered. This animal tested negative for canine distemper virus, but both the liver and the brain tested positive for canine adenovirus 1 (causative virus of infectious canine hepatitis). The Ct vales were between 17 and 23, indicating a moderate to high viral load. The presence of canine adenovirus was further confirmed in the liver using immunohistochemistry (**Fig. 3**).

Canine adenovirus 1 most often targets the liver leading to hepatitis and variably severe clinical disease. Vascular endothelial cells are also a common target for this virus, and injury to these cells can lead to rare systemic presentations of this disease, such as encephalopathy. In this case, cerebral vasculitis leading to perivascular inflammation and edema was diagnosed alongside the typical hepatic lesions associated with this virus. While neurologic disease due to vascular injury is rare in domestic dogs, it is the most common presentation of infection with canine adenovirus 1 in wild canids such as foxes.

The vascular changes in the submandibular tissues were different than those noted in the brain. Therefore, it is suspected, but not confirmed, that this vascular injury was virally induced. It is not certain if canine adenovirus 1 was the cause of the facial swelling in this animal.

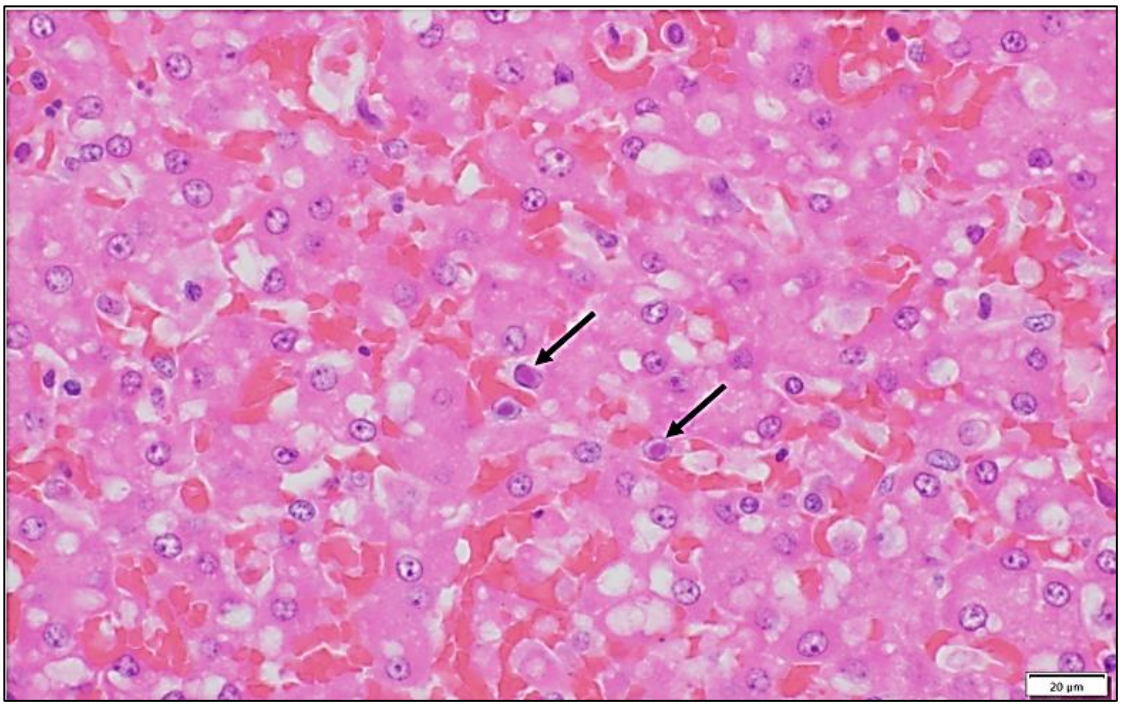


**Figure 1.** Section of liver showing congestion, vacuolation, and single cell death indicating hepatic injury, as well as multiple large basophilic intranuclear viral inclusions (arrows). H&E stain.

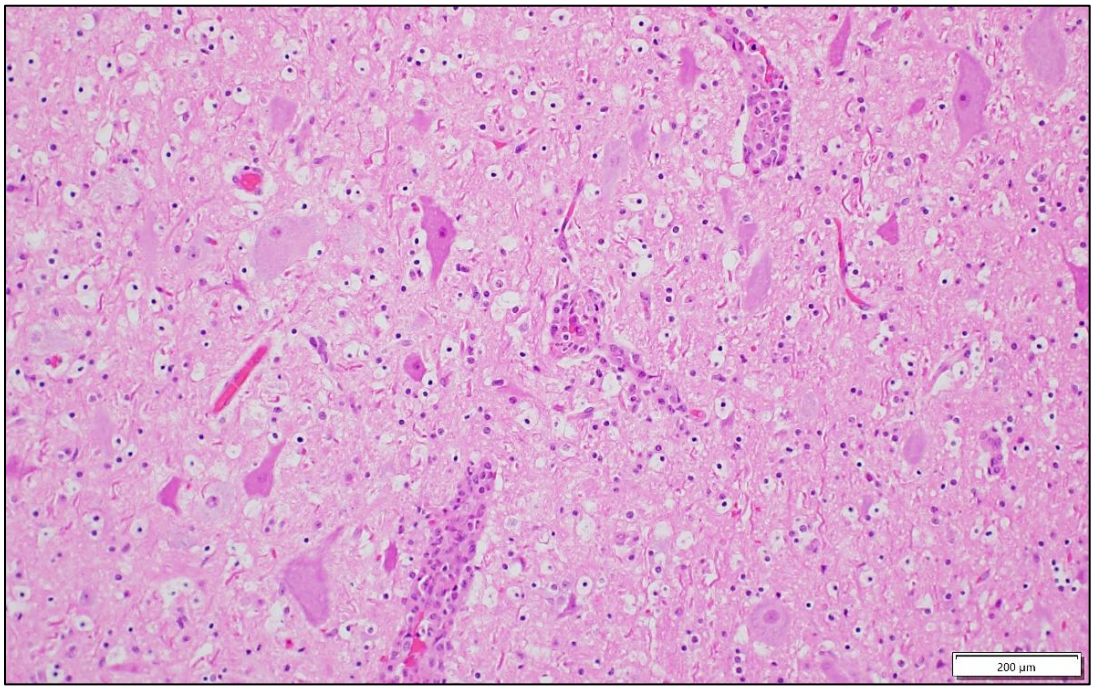


**Figure 2.** Multiple vessels in the brainstem with large cuffs of lymphocytes, macrophages, and fewer neutrophils. H&E stain.

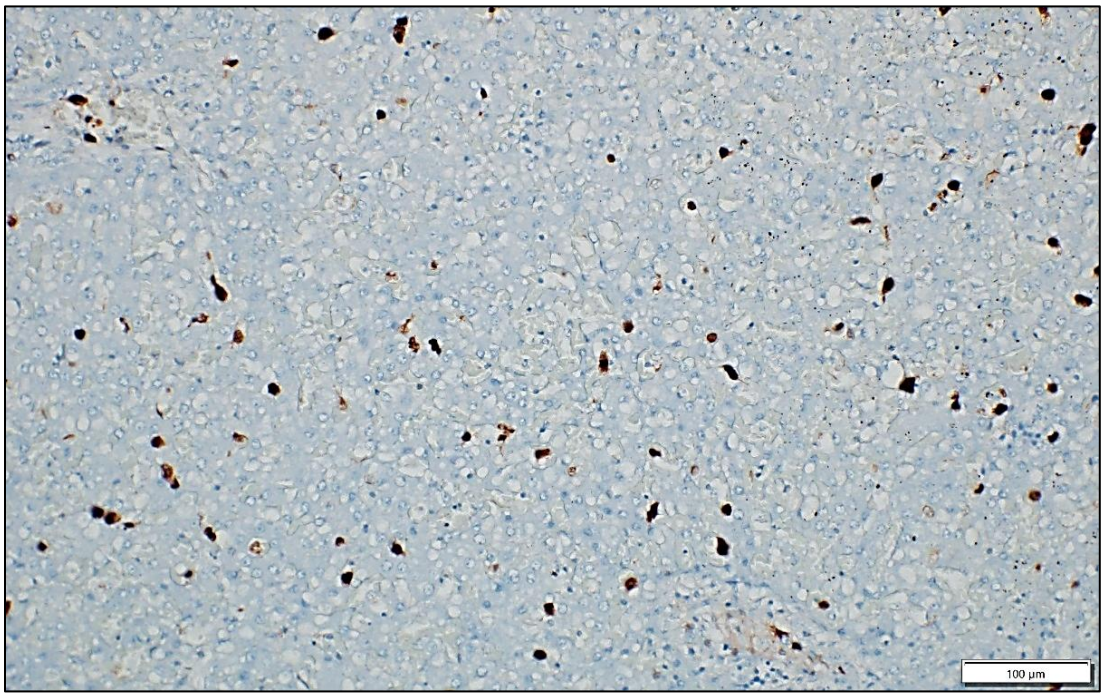


**Figure 3:** Section of liver showing numerous hepatocytes with strong positive immunoreactivity for adenovirus (brown pigment). DAB chromogen.

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# Visceral larval migrans in an imported dog

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A 3-year-old mixed breed dog imported as a stray from Africa presented with acute seizures and vomiting. Vomiting resulted in aspiration pneumonia and accelerated the patient's decline. Bloodwork identified renal injury, and there were multifocal white spots on the kidneys at postmortem. Histopathology of the kidneys revealed multiple granulomas with infiltrating eosinophils and necrotic centers. Rare nematode larvae were found in the center of these granulomas, prompting the diagnosis of verminous nephritis secondary to visceral larval migrans (VLM) (**Fig. 1**). Migrating larvae were not seen histologically in any other organ.

VLM is an atypical cause of kidney injury in a dog, given the prevalence of deworming treatment in Ontario. Parasites implicated in VLM include nematodes such as *Toxocara canis*. Puppies can present with patent intestinal infections, but when eggs are ingested by older dogs or humans, larvae can develop and penetrate the intestinal mucosa and migrate through the blood, arresting in various organs. Cases of VLM are often asymptomatic, but this depends both on the organ affected, and the parasite burden. When the burden is high and/or the organ affected is vital, like the kidney, or as in cases of ocular or neural larval migrans, symptoms can be severe. In this case, it was undetermined whether seizures were secondary to kidney injury, or due to VLM that was not captured in histologic sections. The consequences for dogs and the zoonotic potential of some nematode infections underscore the importance of preventative deworming treatment.

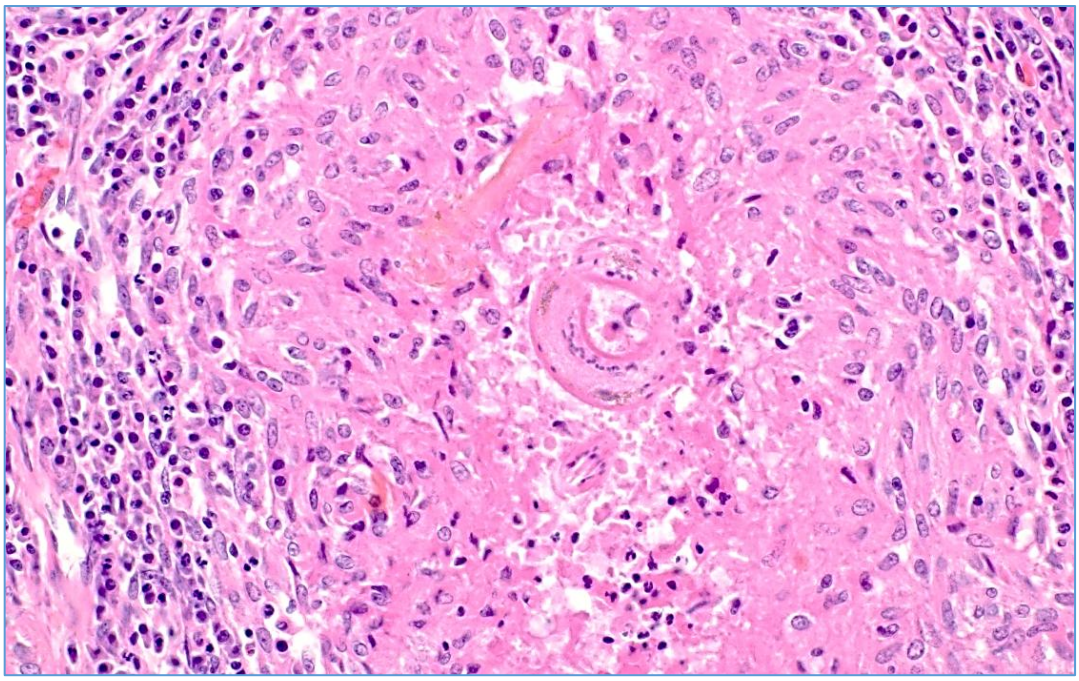

**Figure 1.** Canine, kidney. A coiled larval nematode embedded in a granuloma. H&E stain.

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