Laboratory Services Division
Animal Health Laboratory

AHL Newsletter

AHL Newsletter, Volume 25, Number 4 **December 2021**

In this issue:

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The *AHL Newsletter* is published quarterly (March, June, September, December) by the Animal Health Laboratory, Laboratory Services Division, University of Guelph.

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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AHL holiday hours 2021/2022

Except for Sat. Dec. 25 (closed – no service), AHL-Guelph is open every day from Fri. Dec 24th until Mon Jan 3, 2022 with limited services. The University of Guelph is officially closed during this period.

Thurs. Dec. 23	Guelph and Kemptville - All laboratory sections open with full service
Fri. Dec 24	All laboratory sections open with limited services (Guelph - 9:00 am to 5:00 pm; Kemptville 8:30-4:30 pm)
Sat. Dec. 25	Guelph and Kemptville laboratories closed
Sun. Dec 26	Guelph: specimen receiving, emergency mammalian postmortems; Kemptville closed
Mon. Dec. 27	Guelph: specimen receiving, emergency mammalian postmortems; Kemptville closed
Tues. Dec. 28	Guelph: specimen receiving, emergency mammalian postmortems; Kemptville closed
Wed. Dec. 29	All laboratory sections open with limited services (Guelph - 9:00 am to 5:00 pm; Kemptville 8:30-4:30 pm)
Thurs. Dec. 30	All laboratory sections open with limited services (Guelph - 9:00 am to 5:00 pm; Kemptville 8:30-4:30 pm)
Fri. Dec. 31	All laboratory sections open with limited services (Guelph - 9:00 am to 5:00 pm; Kemptville 8:30-4:30 pm)
Sat. Jan. 1	Guelph: specimen receiving, emergency mammalian postmortems; Kemptville closed
Sun. Jan. 2	Guelph: specimen reception, emergency mammalian postmortems; Kemptville closed
Mon. Jan. 3	Guelph: specimen receiving, emergency mammalian postmortems; Kemptville closed
Tues. Jan. 4	Guelph and Kemptville - All laboratory sections open with full service

Guelph **drop box and fridges available 7AM to 10PM** and Kemptville **drop box and/or fridges are available 365/24/7** for specimen drop off.

For full details, please see our website – www.ahl.uoguelph.ca

Note: Last day for system generated invoicing is Monday December 20th, 2021.

Season's Greetings from the staff of the

Animal Health Laboratory

Update from the Director

The view from the Director's office

This fall has been a very busy period at the AHL. It has been very satisfying to view the return of students to the University of Guelph campus. Scientific meetings and conferences have proceeded, albeit still virtually for now. While on-line meetings enable more people to access the most recent scientific information safely and inexpensively, most of us agree that they are a poor substitute for in-person meetings. The hallway conversations and information discussions during refreshment breaks that occur when we gather together produce an expanding network of personal connections that strengthen and support our animal health sector.

Together with our colleagues at OMAFRA, CFIA and the Canadian Animal Health Surveillance Network (CAHSN), we observe with concern the spread of African swine fever and highly-pathogenic avian influenza around the world. We continue to hold discussions with our partners to ensure a high level of emergency preparedness should one of these high consequence diseases reach Canada. Preparedness includes working to ensure a stable source of materials and reagents during the world-wide supply shortages that we are all experiencing. A dedicated group of AHL staff are also reviewing and updating protocols for emergency testing. We are ready!

This fall, we added two new professionals to the AHL team. Dr. Tanya Rossi is the OAHN and data manager, and Ms. Erika Chadwick is the clinical pathology technical supervisor. You can view their biographies in the OAHN update and Staff highlights section. Welcome Tanya and Erika!

Despite the concern posed by yet another variant of SARS-CoV-2 virus (hello omicron), we are still approaching the end of 2021 with some degree of optimism and hope. From all of us at AHL, we extend our very best wishes to you and your families for the holiday season, and for a safe and healthy 2022.

Maria Spinato, Director

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

Specimen Reception update

Jim Fairles

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

AHL Newsletter 2021;25(4):4.

Cold weather shipping reminder

It's that time of year again when we need to start thinking about preventing samples from freezing. Specimens such as EDTA blood are rendered useless when frozen. Formalin will also freeze, creating artifacts in fixed tissue. It can be difficult to protect samples shipped during the winter from severe cold. Even 10% neutral-buffered formalin will freeze in harsh winter weather conditions. To prevent formalin freezing, add 1 mL of ethanol per 10 mL of formalin.

Samples that should not be frozen should be shipped inside insulated containers with minimal cold packs. Use of room temperature cold packs will help prevent temperatures from dipping too low. If you have any concerns about the best way to ship critical samples, please contact the AHL. ahlinfo@uoguelph.ca

Humane transport of live animals for postmortem

The AHL encourages submission of live animals for immediate euthanasia and postmortem, to ensure a high rate of diagnostic accuracy in poultry cases, and in outbreaks of diarrhea in young pigs and ruminants only. **We remind submitters that live animals must be transported humanely.** The Provincial Animal Welfare (PAW) Act requires that veterinarians report all suspected cases of animal abuse to the PAWS (Provincial Animal Welfare Services). AHL veterinarians will confer with PAWS inspectors to determine if an investigation is warranted in suspected cases of inhumane transport.

Examples of inappropriate restraint and transportation methods include:

- hog-tying calves, adult sheep or goats;
- baby pigs or chickens placed in sealed plastic tubs or styrofoam containers;
- pigs or chickens submitted in tied feed sacks;
- compromised animals in open air (back of a truck) in any type of weather.

Examples of acceptable transport containers include:

- dog or cat kennels for small pigs;
- cardboard boxes of appropriate size with ventilation holes for baby pigs or chickens;
- poultry crates;
- large dog kennels or enclosed bedded truck cabs for larger pigs, calves, small ruminants.

All containers should be of sufficient size to avoid crowding or smothering, and animals should be protected from extreme temperatures. Animals that cannot be transported humanely should be euthanized on-farm, and the practitioner is encouraged to collect appropriate samples for diagnostic testing. **Please contact the AHL and request a consultation with a pathologist if assistance is required for sample selection when performing on-farm postmortems**. *AHL*

OAHN Update – December 2021

Mike Deane, Tanya Rossi

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

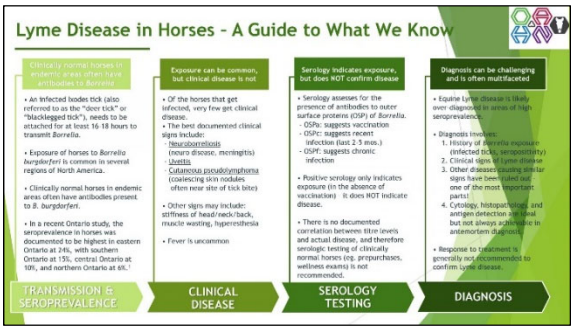
OAHN is pleased to announce that Dr. Tanya Rossi has joined OAHN as the Animal Health Network and Data Manager. Tanya graduated from OVC with a DVM in 2009, and practiced mixed large animal practice for two years before returning to the University of Guelph to earn a PhD in Epidemiology. Her research has focused on chronic and infectious diseases in large animals (particularly equines), and she has worked on vaccine safety and COVID-19 surveillance at PHAC. She also has a strong interest in diagnostic test validation, and surveillance systems. She is excited to work with the OAHN expert networks and hopes to contribute her own expertise to this important initiative.

New Resources, Reports, and Podcasts

Throughout the summer and fall, OAHN has been very busy creating new animal health resources, and finishing up many research projects. In addition, we held our quarterly species-specific meetings and created the resultant veterinary and owner/producer reports which reflect what is being seen in veterinary practice, labs, and abattoirs throughout Ontario. To view the reports, go to OAHN.ca and navigate to the species you are interested in.

New resources include:

- a Bovine Anaplasmosis fact sheet
- a Lyme Disease in Horses infographic
- poultry fact sheets on Fowl Adenovirus, Inclusion Body Hepatitis, and Avian Reovirus
- the Companion Animal Expert Network Public Health Update 2021
- two new bovine podcasts: Bovine Anaplasmosis with Dr. Kathryn Reif and Disease testing in newly introduced cattle project summary and quarterly lab data



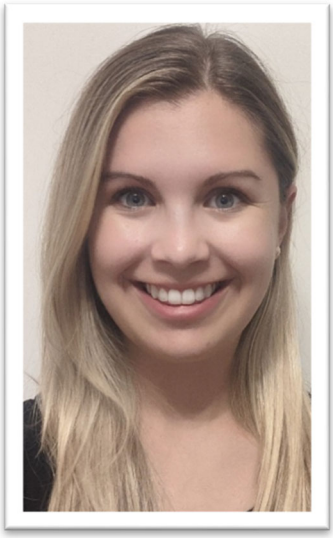
Completed projects include:

- Mid-season treatments for Varroa mites, residue testing in Honey (Bee Network)
- Investigation of ovine herpesvirus-2 as the cause of an idiopathic fatal vasculitis syndrome in Ontario sheep (Small Ruminants Network)
- Prevalence of seroconversion to SARS-CoV-2 among dogs, cats and ferrets in close contact with humans with confirmed or probable COVID-19 (Companion Animal Network)
- Investigation of an elevated number of condemnations for septicemia in veal (Bovine Network - login needed to view)

We are looking forward to a busy and productive winter which will hopefully see many new publications from our ongoing research projects, and more resources to help veterinarians, pet owners, and industry members. *AHL*

Staff highlights

Erika Chadwick joins Clinical Pathology as Technical Supervisor



Erika Chadwick has joined the AHL as Technical Supervisor of the Clinical Pathology section of the Animal Health Lab. Erika has a Bachelor of Science (Honours Chemistry), a Master of Clinical Science (M. Cl. Sc.), and is currently working on her MBA.

Tanya Rossi is the new Animal Health Network (OAHN) and Data Manager

Dr. Tanya Rossi has accepted the position of Animal Health Network and Data Manager at the AHL. Dr. Rossi has a DVM and PhD in epidemiology, and has worked as a veterinarian in mixed animal practice. Her post-doctoral research involved investigating respiratory disease outbreaks at racetracks, and she has worked with PHAC as a modelling epidemiologist. Dr. Rossi has also worked on the near real-time dashboards that AHL is developing to enhance our disease surveillance capabilities.

Antimicrobial susceptibility testing: Part 1 Test methods

Đurđa Slavić

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AHL Newsletter 2021;25(4):7.

When performing **a**ntimicrobial **s**usceptibility **t**esting (AST), all diagnostic labs in North America are expected to follow the standards and guidelines established by Clinical Laboratory Standard Institute (CLSI). The goal of the institute is to promote accurate and reproducible AST results, as well as appropriate reporting and interpretation by standardizing all aspects of AST. A sub-committee on veterinary antimicrobial susceptibility testing (VAST) establishes guidelines for veterinary laboratories that include: testing methods, bacterial density, media and drug types, drug dilutions, incubation times, quality control (QC) requirements, and - most importantly - interpretative criteria. Some of these recommendations will be discussed in an antimicrobial susceptibility testing series in AHL newsletters, starting with this article on test methods. The full document is available in Labnote 64: https://www.uoguelph.ca/ahl/ahl-labnote-64-antimicrobial-susceptibility-testing-ast

Test methods

There are two different methods frequently used by veterinary diagnostic laboratories to determine in vitro susceptibility of bacteria to antimicrobial agents: disk diffusion and microbroth dilution.

The disk diffusion test, also known as Kirby Bauer (KB), is a test where a standardized bacterial suspension is plated on different plate types and disks impregnated with specific antimicrobials are placed on top of it. The drug concentrations on the disks are based on plasma steady-state drug concentrations in humans after IV dosing, although many have now been assessed in veterinary species (see Part 5). However, other than as indicated for bovine mastitis, the pharmacology may not reflect the relevant site of infection. After a specific incubation time, the inhibition of bacterial growth is measured and expressed in millimeters (**Fig. 1**).

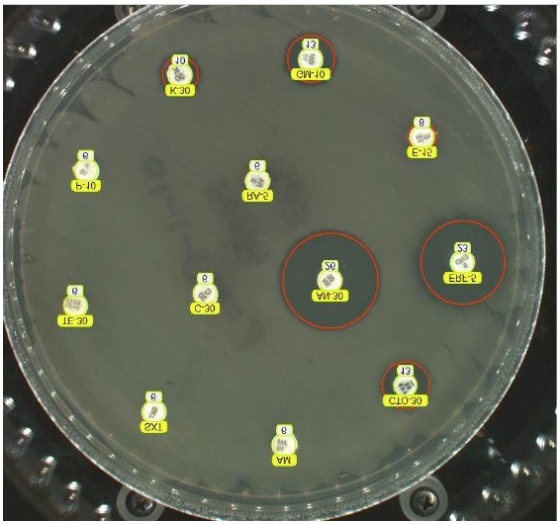

Figure 1. Disk diffusion plate demonstrating disks impregnated with antibiotics placed on an agar plate with the bacterial species of interest. Note the red circles highlighting the zones of inhibited bacterial growth.

Microbroth dilution, also known as **m**inimal **i**nhibitory **c**oncentration (MIC), is a test where a standardized bacterial suspension is dispensed on a 96 well commercially available plate with each well containing different dilutions of antimicrobial agents. After a specific incubation time, the first well with no growth (**Fig. 2**) in a series of wells with different antimicrobial concentrations of the same agent denotes the minimal inhibitory concentration and it is expressed in µg/ml. MIC data need to be interpreted with pharmacokinetic data from the literature or the antimicrobial product data to estimate the drug concentration that is achievable at the site of infection.

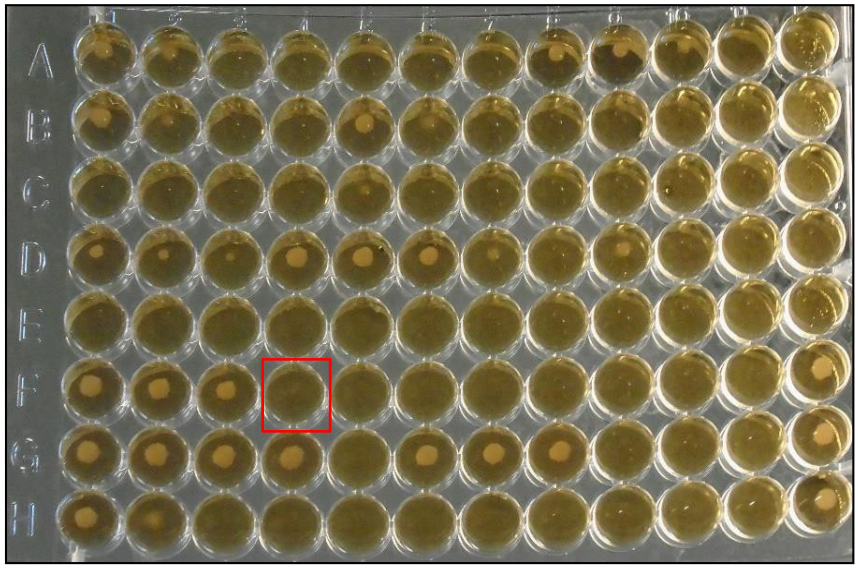

Figure 2. Microbroth dilution plate demonstrating a series of wells containing "buttons" of bacteria growing in broth. The first well in each row containing no growth (button) indicates the minimal inhibitory concentration for that particular antibiotic. For example, red square in row F.

RUMINANTS

Salmonella Dublin in a goat kid

Amanda Mansz

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2021;25(4):9.

Following the sudden death of a one-week-old dairy goat kid, a range of formalin-fixed and frozen tissues were submitted to the AHL for histopathology and bacterial culture. Histologic lesions were comprised of a **neutrophilic enteritis**, **suppurative peritonitis**, **suppurative lymphadenitis**, **embolic hepatic necrosis** and **multi-organ thrombosis**. Bacterial culture of the intestinal content, kidney and liver each isolated a culture of Group D *Salmonella* spp., later serotyped as *Salmonella* Dublin. Additional history revealed a group of veal calves had recently been introduced onto the farm, and that calves had been in close contact with this goat kid.

Salmonella Dublin was first detected in cattle in Ontario in 2012, and continues to be an important pathogen for the Ontario bovine dairy industry. The transmission of *S.* Dublin typically occurs via the fecal-oral route. The presence of an infected non-clinical carrier in the herd provides a source of long-term environmental contamination. Carrier animals will intermittently shed the bacterium in manure, milk, saliva, and urine leading to clinical infection of susceptible animals. Primary colonization of the bacteria in the gastrointestinal tract is followed by bacteremia and systemic spread to organs including lung, liver, spleen and kidneys. In calves, *S*. Dublin infection generally presents clinically as septicemia and pneumonia, often accompanied by fever, anorexia, and depression. Bloody diarrhea, arthritis and/or meningitis may also occur. Calves that survive the infection may become carriers for life. Identifying carrier animals is challenging, and improving biosecurity, sanitation, and colostrum quality are the mainstays for reducing the risk of disease transmission.

S. Dublin has been documented in the literature as causing disease in goats, however, it is considered uncommon and case reports are sparse. **To date, this is only the second case (the first in 2018) where *S.* Dublin has been diagnosed as the cause of septicemia and death in a goat kid at the AHL**. *AHL*

Reference

Rosenbaum Nielsen L. Review of pathogenesis and diagnostic methods of immediate relevance for epidemiology and control of *Salmonella* Dublin in cattle. Vet Microbiol 2013;162(1):1-9.

What bugs are causing BRD in young dairy calves?

David Renaud

Department of Population Medicine, University of Guelph, Guelph, ON

AHL Newsletter 2021;25(4):10.

It seems that an age-old question is: what bugs/pathogens are responsible for causing bovine respiratory disease (BRD) in dairy calves, especially in the fall? To provide an answer about the pathogen profile causing BRD on farms that are having a respiratory disease outbreak or a challenge with chronically high levels of respiratory disease in their calves, deep nasopharyngeal swabs can be a practical on-farm tool. These swabs will sample the respiratory and associated epithelium of the nasopharynx. The collection of a meaningful sample can be done in a few minutes. Contamination can be an issue when using these swabs which can compromise the clinical interpretation; however, using a guarded swab, such as a guarded mare uterine swab, will help minimize nasal contamination.

To evaluate the pathogen profile responsible for causing BRD on several Ontario dairy farms, a descriptive study was undertaken on farms that were having challenges with respiratory disease.

During 2021, several different clinics and farms participated in this descriptive study. In total, 68 animals from 14 different farms were sampled using deep nasopharyngeal swabs. These farms mostly housed calves indoors in groups, and were either experiencing a respiratory disease outbreak at the time of sampling, or had a chronic-active situation. Calves that were sampled were either sick or poor-doing, and had not been previously treated with antimicrobials or vaccinated within the preceding 2 weeks. The age of the calves varied from 2 weeks to 6 months of age; however, the majority were under 6 weeks of age.

The samples were submitted to the Animal Health Laboratory where they were cultured or underwent PCR testing for BHV-1, PI3, BRSV, bovine coronavirus BVDV and *Mycoplasma bovis*.

The bacterial results highlight the high levels of *Pasteurella multocida* isolated and positive *Mycoplasma bovis* PCR tests from the calves (**Fig. 1**).

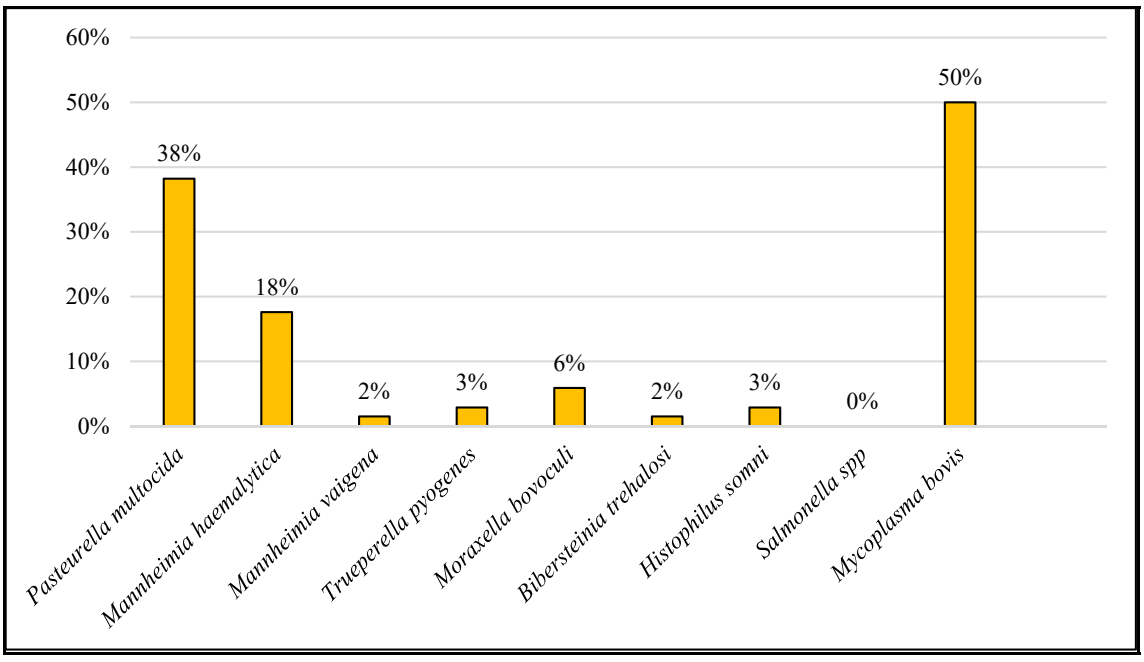

Figure 1. Results of bacterial panel from 68 dairy calves on 14 dairy farms in Ontario that were experiencing a respiratory disease challenge.

The viral results demonstrate that overall, a small number of calves had a viral infection, and of these, a moderate number were infected with bovine coronavirus (**Fig 2**.).

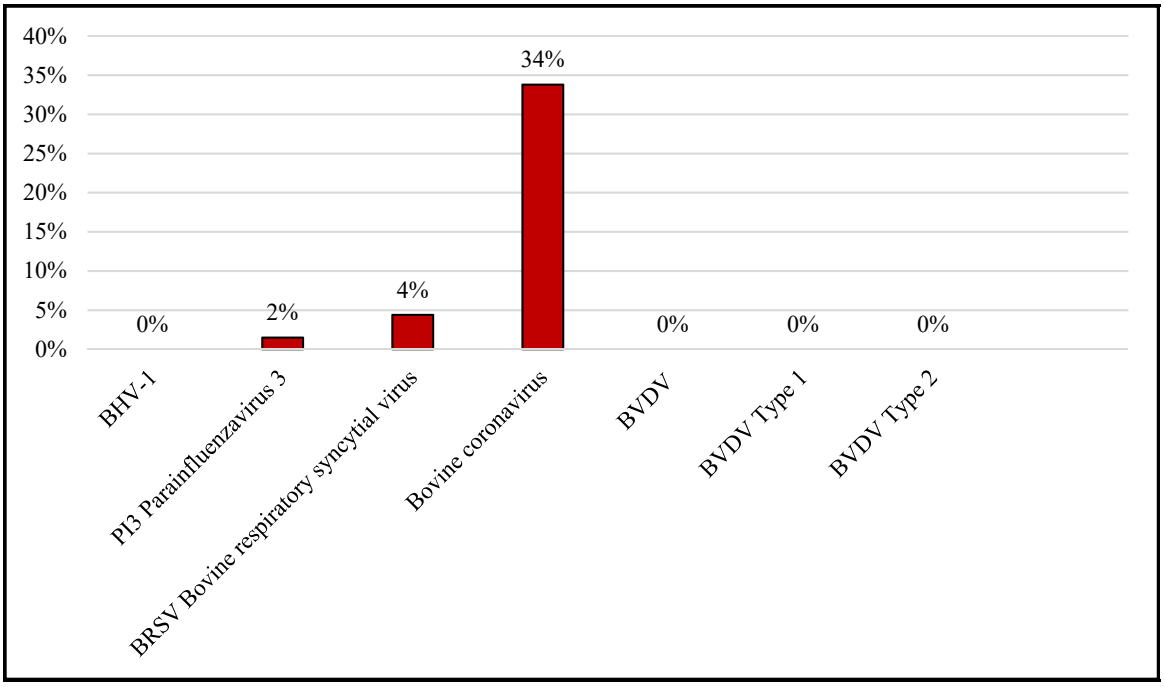

Figure 2. Results of viral PCR panel from 68 dairy calves on 14 dairy farms in Ontario that were experiencing a respiratory disease challenge.

On a herd level basis, most farms had a mixed population of pathogens isolated, including: *Pasteurella multocida* (10/14), bovine coronavirus (10/14), *Mannheimia hemolytica* (8/14), and *Mycoplasma bovis* (8/14). With respect to the traditional viral pathogens, very few farms were positive for BRSV (1/14), PI3 (1/14), BHV-1 (0/14), or BVDV (0/14).

There are some interesting findings from the samples collected, but also some lessons learned. We found that deep nasopharyngeal swabs can be useful to determine the pathogens present in a herd, especially when an appropriate calf necropsy is not available. In addition, we found that the swabs are easy to use and only minor bleeding in the nostril was observed in some of the calves.

To request a comprehensive bovine respiratory disease panel at AHL, submit 2 deep nasopharyngeal swabs (normally guarded uterine swab) for each animal and order test code brsppnl.

Note: separate samples are needed for bacteriology (gel media) and PCR (VTM or dry swab). The swabs cannot be transferred into alternate media upon arrival at the lab. More information on the AHL comprehensive bovine respiratory disease panel is available in the September 2021 AHL Newsletter: https://www.uoguelph.ca/ahl/comprehensive-bovine-respiratory-disease-panel.

Funding for this descriptive study was provided by Merck Animal Health.

SWINE

Porcine Circovirus 2-associated vasculitis in a young farmed wild boar

Emily Rätsep, Stéphanie Bourgon

Animal Health Laboratory, University of Guelph, Kemptville, ON (Rätsep), Vankleek Hill Veterinary Services, Vankleek Hill, ON (Bourgon)

AHL Newsletter 2021;25(4):12.

On a wild boar farm with a history of respiratory signs, 4 animals had complete hind limb paralysis and 5 more demonstrated some degree of weakness. Approximately 20 animals had been treated by injectable Ivomec 10 days prior for the perceived respiratory issue. On physical exam, rectal temperatures were normal, heart rate and respiratory rates were increased for all affected pigs, and increased lung sounds were noted. There were no associated central nervous signs (cranial nerve reflexes remained), and forelimb movement was intact in all animals. Pigs showing weakness or paralysis were treated with 5 mg of Dexamethasone 5 by intramuscular injection. One animal was found dead, two animals were euthanized by the owner, and one 6-month-old sow was euthanized by intracardiac injection with T-61 and was submitted for postmortem examination at the AHL in Kemptville.

Postmortem findings for the submitted sow included voluminous, dark red lungs that failed to collapse on opening of the thorax, petechial hemorrhages on the bladder and a focally extensive area of hemorrhage observed within a lumbar spinal cord segment (L3-4). No obvious cause of the spinal cord hemorrhage was observed. A viral cause of pneumonia was suspected based on the gross findings in the lungs.

Histologically, a marked lymphohistiocytic vasculitis was observed in multiple tissues, including the heart, bladder, colon, esophagus, tonsils, and kidney. Vasculitis was also apparent within sections of spinal cord associated with areas of hemorrhage observed grossly, and within the associated pia matter. Focally, the area of hemorrhage within the spinal cord resulted in significant disruption of the parenchyma and injury to the grey and white matter in this location (**Fig. 1**). Other than moderate pulmonary edema, no histological lesions were observed in the lungs.

The presence of vasculitis in multiple organs raised suspicion of infection with porcine circovirus 2. Lung submitted for viral PCR confirmed the presence of porcine circovirus 2 (PCV-2, positive with Ct 11.56) and viral infection was further confirmed by immunostaining for PCV-2 antigen within mononuclear cells in the vascular lesions in multiple tissues, notably spinal cord and lung (**Fig. 2**). All viral testing performed on this pig (as part of the OAHN swine small herd postmortem project) was otherwise negative.

There are several syndromes associated with infection by PCV-2 in domestic pigs, including postweaning multisystemic wasting syndrome or PMWS. These syndromes are now collectively known as porcine circovirus associated disease (PCVAD), and have been reported in both domestic pigs and farmed wild boar (1,2). Of these syndromes, vasculitis is considered the hallmark lesion of the more severe forms of PCVAD (3). In these reports, an acute lymphohistiocytic vasculitis was often accompanied by pulmonary edema (as was observed in this case). While lesions in the brain or spinal cord are only rarely observed (4), the presence of systemic vasculitis combined with a suspected direct cytopathic effect on these vessels by the PCV-2 virus (5) would explain hemorrhage in multiple organs, including spinal cord.

PCV-2 infection with vasculitis and subsequent spinal cord hemorrhage is the presumed underlying cause of the spinal cord lesion, thereby explaining the history of weakness progressing to paralysis in this pig. **Though rare, it is worth considering the possibility of PCV-2 infection as a potential cause of neurological signs and paralysis in pigs, especially in those with a history of respiratory signs and wasting**. In this case, a vaccination program was implemented on farm, with improvement reported in the breeding sows and piglets. *AHL*

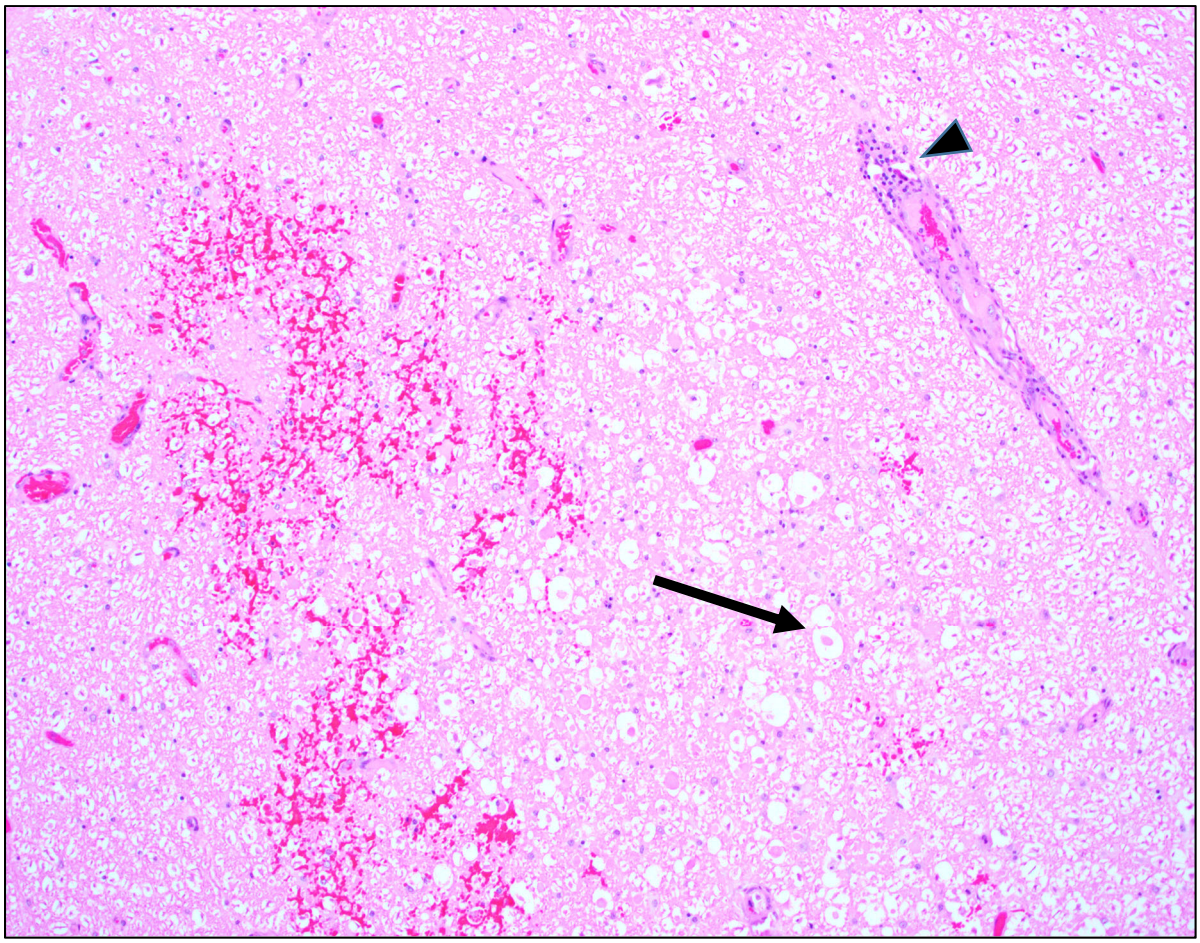

Figure 1. Hemorrhage within the spinal cord white matter with disruption of the parenchyma and damage to axons (arrow). Lymphocytes and macrophages are present within the adventitia of an adjacent vessel in this area (arrowhead). H&E stain.

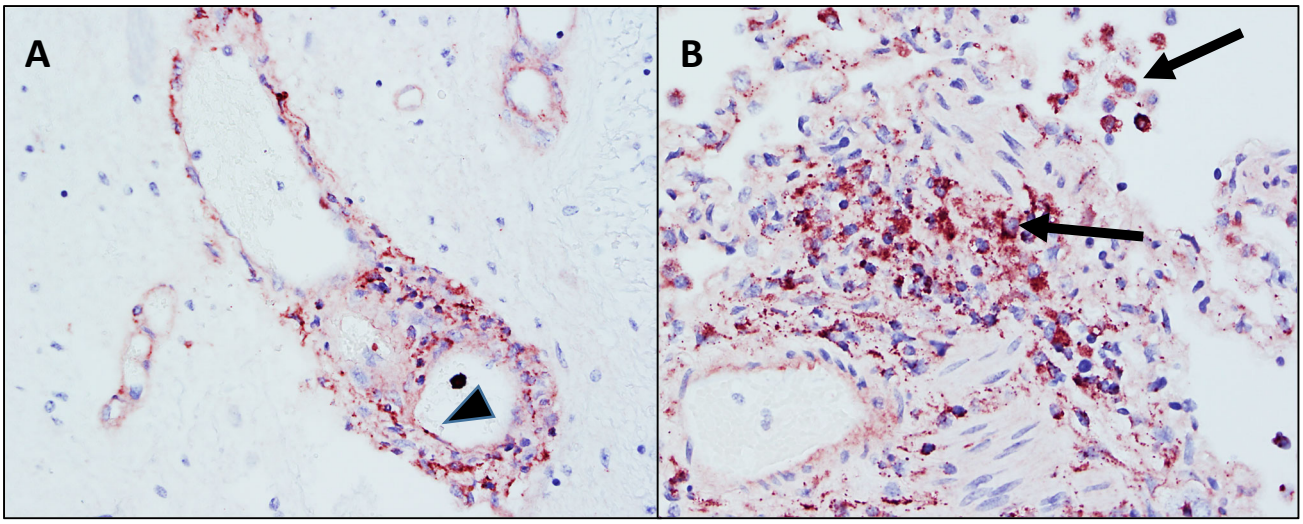

Figure 2. Porcine circovirus 2 infection in a pig. Spinal cord (2A) and lung (2B) with immunohistochemistry for PCV2. There is positive immunostaining of scattered endothelial cells (arrowhead) in the spinal cord, and within alveolar macrophages and perivascular/peribronchiolar mononuclear cells in the lung (arrows). Immunohistochemical staining for PCV-2 antigen, Nova Red chromagen.

References

1. Gillespie J, et al. Porcine circovirus type 2 and porcine circovirus-associated disease. J Vet Intern Med 2009;23:1151-1163.
2. Vicente, J, et al. Epidemiological study on porcine circovirus type 2 (PCV2) infection in the European wild boar (*Sus scrofa*). Vet Res 2004;35(2):243-253.
3. Langohr IM, et al. Vascular lesions in pigs experimentally infected with porcine circovirus type 2 serotype B. Vet Pathol 2010;17(1):140-147.
4. Seeliger FA, et al. Porcine circovirus type 2-associated cerebellar vasculitis in postweaning multisystemic wasting syndrome (PMWS)-affected pigs. Vet Pathol 2007;44:621-634.
5. Resendes AR and Segalés J. Characterization of vascular lesions in pigs affected by porcine circovirus type 2-systemic disease. Vet Pathol 2015;52(2):497-504.

Sudden death in sows: AHL pathology results, 2010-2021

Josepha DeLay

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2021;25(4):14.

Diagnostic investigation of sudden (unexpected) death cases in sows and gilts can be challenging, mainly due to the logistics of performing a necropsy on these large animals. Transportation to a diagnostic laboratory can be difficult to accommodate, and on-farm necropsies are awkward due to sow size. Tissue autolysis and decomposition are often rapid due to the combined effects of the relatively high environmental (indoor) temperature, and the animal's size and fat content, maintaining a high carcass temperature following death.

To summarize and better understand the causes of sudden death in Ontario sows and bred gilts, sow mortality cases submitted to the Animal Health Laboratory were reviewed. The AHL laboratory information management system (LIMS) database was searched for relevant cases involving sows or gilts that were received between January 2010 and November 2021. Only those cases with a pathology component (necropsy and / or histopathology) and for which the clinical history was compatible with sudden (unexpected) death were included in the review.

A total of 43 sow or gilt sudden death cases were submitted to the AHL over this 12-year period (range 0-10 cases / year, average 4 cases / year). The submissions included 2 necropsies carried out at the AHL and 41 on-farm necropsies, with histopathology and various other diagnostic tests subsequently conducted at the AHL.

The causes of death in all submitted sows and gilts, based on histopathology findings and results of ancillary tests, are listed in (**Table 1**). No definitive cause of death was identified in 10/43 animals (23%). Among the remaining cases, the cause of death was attributed to systemic disease or to respiratory, gastrointestinal, cardiovascular, or musculoskeletal disease. Systemic disease was diagnosed most frequently [18/43 (42%) cases] and included septicemia, polyserositis, and suspected heat stroke. Definitive etiologies for septicemia were identified in 6 cases, including *Streptococcus suis* (5 cases) and *Streptococcus dysgalactiae* ssp. *equisimilis* (1 case). For other cases, a diagnosis of systemic disease was based on histopathology findings.

Table 1. Causes of sudden death in sows and gilts submitted to the AHL, 2010-2021.

Cause of death / body system affected	Number of cases	Comments
Unknown cause of death	10	
Systemic	18	Septicemia: 13 cases Polyserositis: 3 cases Suspected heat stroke: 2 cases
Respiratory	9	Bacterial pneumonia +/- influenza: 8 cases Parasitic pneumonia: 1 case
Gastrointestinal	4	Porcine proliferative enteritis / *Lawsonia*: 4 cases
Cardiovascular	1	Acute myocardial degeneration, r/o ionophore toxicity, vitamin E / selenium deficiency, sepsis

The level of detail about herd mortality rates and duration that was provided in clinical histories was highly variable. From cases with on-farm necropsies, a description of gross findings was included for only 15/43 (35%) of cases. This information is valuable to pathologists when interpreting histologic findings in light of the clinical context. Not including necropsy findings with a diagnostic submission is a missed opportunity for communication with diagnosticians about important features of the disease condition in the herd.

For most cases necropsied on-farm, organs sampled for histopathology included lung and a good range of filtering organs such as liver, spleen and kidney. Despite the clinical history of sudden death, vital organs such as heart, brain, and skeletal muscle were collected and submitted from relatively few cases: brain - 2/43 (5%) cases; skeletal muscle - 6/43(14%) cases; heart - 19/43 (44%) cases.

This case series describes the cause of death in sows dying unexpectedly, and for which relatively subtle or no gross lesions were evident during postmortem examination. Those sows with an obvious cause of death, such as splenic torsion or gastric ulceration and hemorrhage, would not typically have further

diagnostic testing performed and would not be included in the database searched for this review. As a result, this data is not representative of the overall causes of sudden death in sows and gilts. Among the animals examined, a cause of death was not identified in a disappointing, but expectedly high number of cases (23%). Thorough gross examination and collection of a wide range of tissues for histopathology could improve the diagnostic success rate for sudden death cases involving all ages of swine, including sows and gilts. Brain is especially difficult to sample on-farm in mature swine, however a lateral approach for brain removal is helpful in these animals (see AHL Labnote 33: https://www.uoguelph.ca/ahl/ahl-labnote-33-brain-removal-field-postmortems). *AHL*

Reminder: OAHN swine small herd postmortem project

The AHL will continue to accept submissions for the small herd postmortem project **only until March 31, 2022.** To enroll a case in the project, please contact Dr. Josepha DeLay, AHL at 519-824-4120 ext. 54576 or jdelay@uoguelph.ca. More information about the project is located on the AHL website: https://www.uoguelph.ca/ahl/oahn-swine-small-scale-herd-postmortem-project-may-2020.

Severe myositis associated with avian encephalomyelitis in chickens

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Two-week-old chickens with signs of neuromuscular disease were submitted to the Animal Health Laboratory (AHL) and the Department of Pathobiology, Ontario Veterinary College for postmortem examination. The clinical problems included reduced feed intake, runting, lethargy, and lameness. Approximately 10-15% of the flock was reluctant or unable to stand while others were alert and eating, but seemed less active than normal. The chickens were housed in a well-managed facility with regulated environmental temperatures and adequate access to feed and water. Hypoglycemia-spiking mortality syndrome or avian encephalomyelitis (AE) were suspected by the referring veterinarian.

The postmortem findings were nonspecific. Some birds were thin, and there was a wide variation in body weight. The crops were empty, and a small amount of feed and bedding material was present in the proventriculus and gizzard. The small and large intestines contained variable amounts of dark green liquid. Other findings included partially resorbed yolk sacs, mild splenic enlargement, and pulmonary congestion.

Histologically, there were prominent lesions in the skeletal muscle and heart. The muscle tissue was infiltrated with numerous lymphocytes and plasma cells. Myofibre necrosis and atrophy were especially severe in the skeletal muscle (**Fig. 1**). Skeletal muscle in the limbs, vertebral column, and esophagus was affected. In the gastrointestinal tract, there were infiltrates of lymphocytes and plasma cells in the muscular wall of the proventriculus, ventriculus, small intestine, and cecum. Such lesions in the proventriculus are considered pathognomonic for AE (**Fig. 2**). In contrast, the lesions in the nervous system were very mild. The brain contained occasional perivascular cuffs of lymphocytes and plasma cells, scattered foci of gliosis, and rare neuronal chromatolysis. Mild gliosis in the spinal cord and axonal degeneration in the peripheral nerves were also observed.

The diagnosis of AE was supported by the characteristic lesions in the viscera and CNS, and the detection of avian encephalomyelitis virus in the brain by PCR. Vaccination is an effective means of controlling AE, but the vaccination status of the source breeder flock for these chickens is not known. Hypoglycemia-spiking mortality syndrome was the other clinical differential diagnosis, but serum glucose concentrations were normal and compatible lesions were not observed.

Although myositis is known to occur with AE, the severity of myopathy in this case was unexpected, and it probably explains most of the clinical signs. The possibility of concurrent nutritional deficiency or ionophore toxicosis was considered, but the likelihood seemed low and further testing was not pursued. Skeletal muscle is not routinely sampled for histopathology when investigating AE. Over the past decade, 8 out of 18 AE cases in the AHL database included skeletal muscle in the histopathologic description; in all 8 cases there was inflammation, degeneration, and/or necrosis noted in the skeletal muscle, with varying degrees of severity. **Although diagnostic lesions are typically found in the brain,**

spinal cord, and viscera, it is recommended to also include skeletal muscle for histopathology when investigating potential cases of AE in chickens. *AHL*

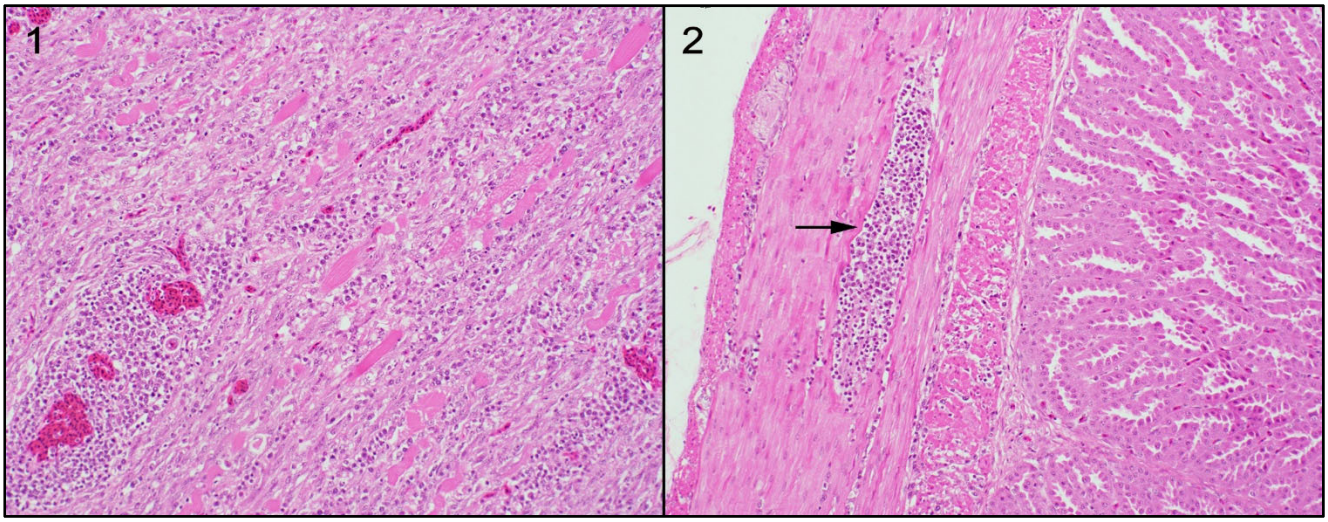

Figure 1. Skeletal muscle with lymphoplasmacytic inflammation, myofibre necrosis and atrophy. H&E.
Figure 2. Lymphoplasmacytic infiltration of the muscular wall of the proventriculus (arrow). This lesion is pathognomonic for avian encephalomyelitis. H&E.

Reference

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Knemidocoptes mutans (scaly leg mite) in backyard flocks

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Scaly leg mites (*Knemidocoptes mutans*) infect both wild birds and domestic poultry. Transmission is by direct contact and all life stages occur on the bird. These mites live in areas of unfeathered skin. They burrow into the skin of the shanks and feet causing irritation, tissue swelling, enlarged scales and hyperkeratosis, resulting in the lesions having white, powdery, exfoliating crusts (**Fig. 1, 2**). The shanks and feet appear to have thickened skin and the scales can be seen to protrude (**Fig. 3, 4**). If left untreated, deformity of the legs and claws can result, and birds can become crippled. Eventually, there is slow spread of this parasite through the flock.

Treatment of individual birds is required. Daily application of an oil-based product (e.g. petroleum jelly, paraffin, mineral oil) will soften the crusts. Then, the affected areas can be washed with mild soap to loosen and remove crusts. Treatment should continue for at least 2 weeks or until the legs return to normal appearance. Insecticide treatment is an option, but must be administered on the advice of a veterinarian. *AHL*

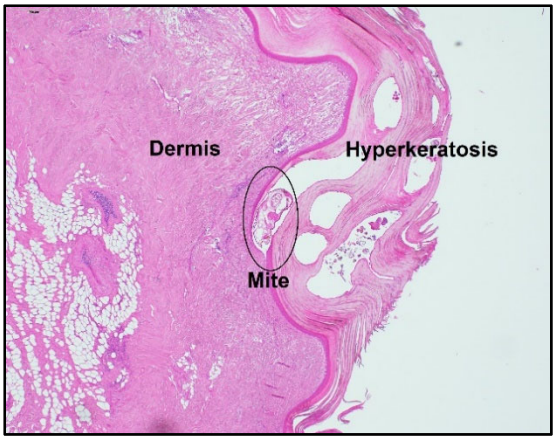

Figure 1. Histology of skin showing hyperkeratosis and burrowed mite (H&E, 4x).

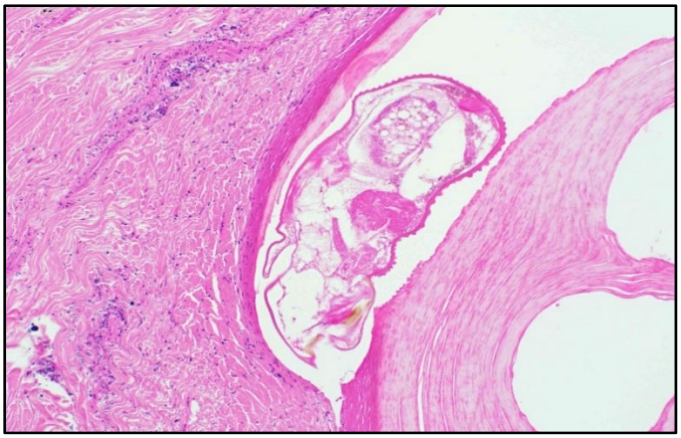

Figure 2. Higher magnification of mite (H&E, 20x).

Figure 3. Thickened skin and raised scales.

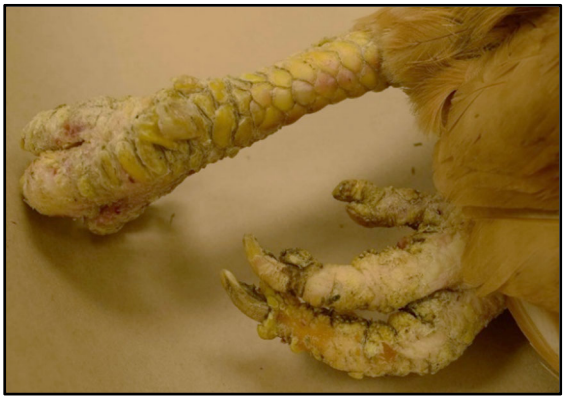

Figure 4. Swollen feet and exfoliating crusts.

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New Test: Serpentovirus (reptile nidovirus) RT-PCR

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Respiratory disease in captive snakes is an all too common and significant concern for pet owners and the exotic animal veterinarians. It is a complex and multifactorial problem resulting from complex interactions between environmental husbandry factors (e.g. temperature, humidity), host anatomical factors (elongate trachea and poorly developed mucociliary escalator apparatus, large sac-like lung, lack of diaphragm), and infection with a variety of different primary and opportunistic pathogens. Reptiles are also cryptic creatures, and disease is often unapparent to the owner until the problem is quite advanced and open-mouth breathing is observed. This can make treatment particularly frustrating, especially when multiple coinfections are present. A variety of bacterial (often opportunistic gram-negative upper respiratory tract commensals), parasitic (lung mites, lung worm), fungal, and primary viral (e.g. ophidian paramyxovirus, inclusion body disease, nidoviruses) pathogens are recognized. In recent years, reptile nidoviruses (also known as serpentoviruses) have emerged as particularly significant respiratory pathogens in reptiles.

With this in mind, the Animal Health Laboratory has developed a real-time PCR assay for the detection of nidoviruses with the goal of helping veterinarians to better differentiate nidovirus as a possible cause for respiratory disease, and to assist them in their management of reptile collections. The RT-qPCR assay is based on published methods (3,6). Upon request, PCR sequencing can be performed for further identification. Lung and distal trachea collected at the time of postmortem is the preferred sample for testing deceased snakes, while deep tracheal swabs or trans-tracheal lung lavage samples are preferred for testing clinical cases.

The majority of nidovirus infections to date have been documented in snakes within the Pythonidae (pythons) family, but infections have also been documented in Boidae (boas), Colubridae (the largest family of snakes), and Homalopsidae (water/mud snakes), as well as in a few individual lizard species (e.g. Australian shingleback lizards and veiled chameleons) and some testudines (e.g. Bellinger and Murray River turtles). Nidovirus infection typically results in a prominent proliferative interstitial pneumonia, and is now commonly referred to as nidovirus-associated proliferative disease (NPD). Although the lung is the primary site of infection and lesions, the virus also has tissue tropism for the oral cavity and upper alimentary tract, and this can be helpful for differentiation from other viral infections (**Fig. 1**). Therefore, submission of either the whole head or the excised epiglottis, surrounding oral tissue and upper trachea and esophagus is essential when submitting tissues for histological confirmation of NPD. *AHL*

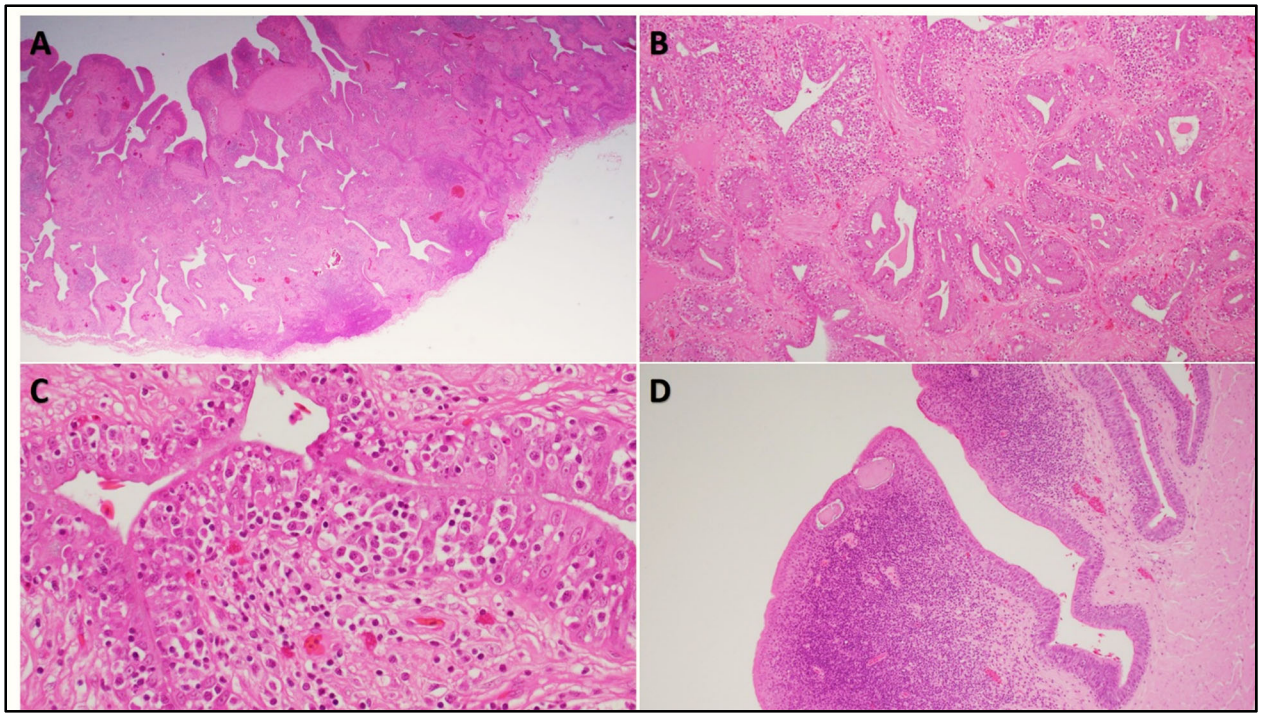

Figure 1. Lung and oropharynx in an adult Burmese python (*Python bivittatus*) with nidovirus-associated proliferative disease (NPD). **A. Lung:** Faveolar and sub-faveolar septal walls are thickened with increased amounts in intervening smooth muscle and fibrous connective tissue that markedly narrow and constrict the air spaces. Hyperplastic pseudo-follicular lymphoid aggregates are scattered throughout the lung. **B. & C. Lung:** The lining respiratory epithelium consists of tall columnar ciliated epithelium arranged in a variably thickened pseudostratified to stratified layers with scattered areas of epithelial swelling and degeneration. Constricted air spaces occasionally contain entrapped homogenous eosinophilic proteinaceous fluid and there is widespread loss of apical cilia. **D. Oropharynx:** The oropharyngeal mucosa is similarly thickened and hyperplastic with large submucosal lymphocyte aggregates. H&E stain.

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B-cell chronic lymphocytic leukemia with monoclonal gammopathy in a horse

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An 18-year-old Clydesdale female horse was examined for vague signs of colic. The mare was agitated and painful during urination. A complete blood count (CBC) revealed a mild neutropenia [1.78 x10e9/L (*RI* 2.8-7.7 x10e9/L)], mild regenerative left shift [bands: 0.59 x10e9/L (RI 0.0-0.2 x10e9/L)], moderate lymphocytosis [14.65 x10e9/L (RI 1.3-4.7 x10e9/L)] and mild to moderate monocytosis [2.57 x10e9/L (RI 0.1-0.8 x10e9/L)]. Upon examination of the blood smear, the lymphocytes were heterogeneous suggesting a reactive cell population, although the lymphocyte count was considered unusually elevated and monitoring was warranted. The biochemistry profile revealed a mild to moderate hyperproteinemia due to hyperglobulinemia [53 g/L (RI 26-41 g/L)]. Urinalysis on a free flow sample showed 2-5 leukocytes/400x and 3+ bacteria; *Escherichia coli, Enterococcus casseliflavus* and *Enterobacter cloacae* were isolated on bacterial culture. These findings, along with elevated serum amyloid A [765 mg/L (RI 0-20 mg/L)], indicated inflammation, antigenic stimulation and possible urinary tract infection (UTI).

A follow-up CBC showed a marked leukocytosis [60.5 x10e9/L (RI 5.1-11.0 x10e9/L)] due to marked lymphocytosis (41.75 x10e9/L) and marked monocytosis (13.92 x10e9/L). The lymphocytes were variably sized, with basophilic cytoplasm and round to indented/lobulated to convoluted nuclei with coarsely clumped chromatic pattern and no visible nucleoli (**Fig.1**). Some of the larger lymphoid cells were probably misidentified as monocytes. These findings are consistent with leukemia, probably of lymphocytic origin (chronic lymphocytic leukemia vs. leukemic phase of lymphoma).

Since laboratory findings supported both inflammation and neoplasia, serum protein electrophoresis (SPE) was performed to characterize the hyperproteinemia. A relative hypoalbuminemia [26 g/L (RI 30-37 g/L)] was noted. The hyperglobulinemia was characterized by increased beta-2 globulins [29 g/L (RI 2-9 g/L)], outlined by a narrow-based peak, consistent with a monoclonal gammopathy (**Fig.2**). These findings were consistent with an immunoglobulin-producing B-cell or plasma cell neoplasm. To further investigate B-cell origin, immunophenotyping by flow cytometry (FC) was performed on EDTA blood. This method identified a population of variably-sized cells that were moderately positive for CD18 and CD21, and uniformly positive for MHC II. These findings are most consistent with a B-cell chronic lymphocytic leukemia (B-CLL).

An interesting feature noted in this case was the deep nuclear indentation and convolution of the neoplastic cells, which is more commonly associated with T-cell neoplasms; however, SPE and FC were confirmatory for B-cell origin. Also, the paraproteins resulting in a monoclonal peak migrated in the beta-2 region, suggesting an IgM or IgA gammopathy, although immunoglobulin isotype characterization was not attempted.

Clinical signs improved after treatment with TMS and enrofloxacin for the presumptive UTI, and owners considered chemotherapy for the leukemia. However, the mare lost a significant amount of weight,

clinical signs recurred, and the owners elected humane euthanasia. Chronic lymphocytic leukemias have been infrequently reported in horses (1,2), and information on clinical outcome is sparse. Chemotherapy with prednisolone-chlorambucil was attempted in one horse with B-CLL; however, due to inadequate response to treatment and poor prognosis, the animal was euthanized 6 weeks after initial presentation (2). Differentiation between lymphocytic leukemia and stage-V-lymphoma can be challenging or impossible. In the case reported here, clinical presentation (lack of lymphadenopathy) and cytological and immunophenotypic features of the neoplastic lymphocytes strongly suggested B-CLL; however, primary lymphoma (e.g. GI tract) with secondary bone marrow/blood involvement could not be entirely excluded.

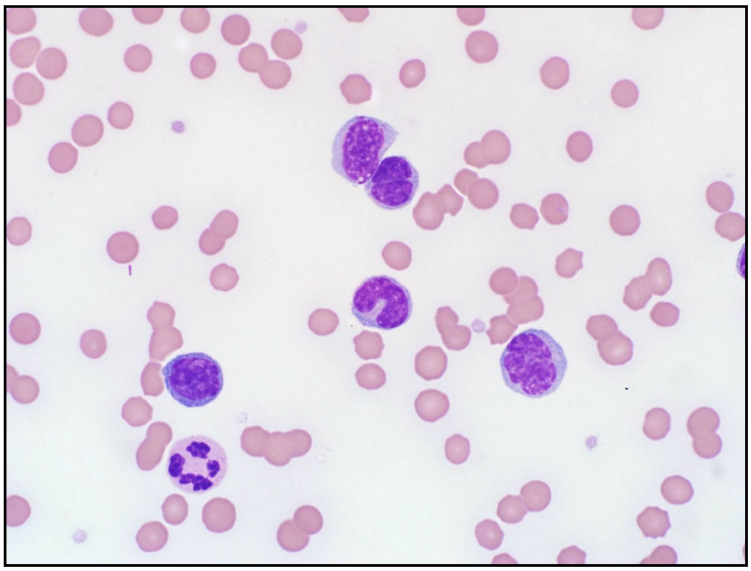
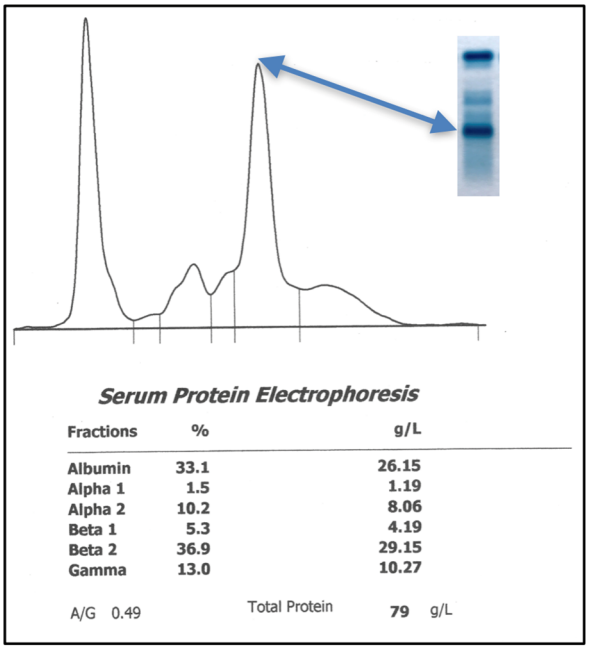


Figure 1: Blood smear examination. There is a population of atypical lymphocytes with frequent nuclear indentation. Wright's stain (400X).



Serum Protein Electrophoresis

Fractions	%	g/L
Albumin	33.1	26.15
Alpha 1	1.5	1.19
Alpha 2	10.2	8.06
Beta 1	5.3	4.19
Beta 2	36.9	29.15
Gamma	13.0	10.27

A/G 0.49 Total Protein **79** g/L

Figure 2: Serum protein electrophoresis. The discrete band noted on the cellulose acetate strip (insert) is represented by a narrow-based peak in the beta-2 globulin region of the densitogram, consistent with a monoclonal gammopathy.

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Streptococcus equi ssp *zooepidemicus* and canine respiratory disease

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The bodies of 2 young adult female mixed breed dogs were submitted to the Animal Health Laboratory for postmortem examination. Both dogs originated from multi-dog housing where outbreaks of respiratory disease had recently occurred. Dog A deteriorated rapidly and died while being treated. Dog B developed pneumothorax and was euthanized.

Lung from dog A had diffuse dark red discoloration and mild rubbery consistency. Lung lesions in dog B included diffuse atelectasis, as well as unilateral (right) red-tan discoloration, firm texture (consolidation), and pleural adhesions (**Fig. 1**). Histologically in lung from both dogs, bronchiolar lumens and alveoli were filled with aggregates of neutrophils variably mixed with macrophages and fibrin. Fibrin thrombi were present in either alveolar capillaries or larger pulmonary blood vessels. In dog B, additional lung lesions included patchy alveolar hemorrhage and necrosis, and pleural and alveolar septal fibrosis. Numerous coccobacilli were present among alveolar inflammatory cells in dog A.

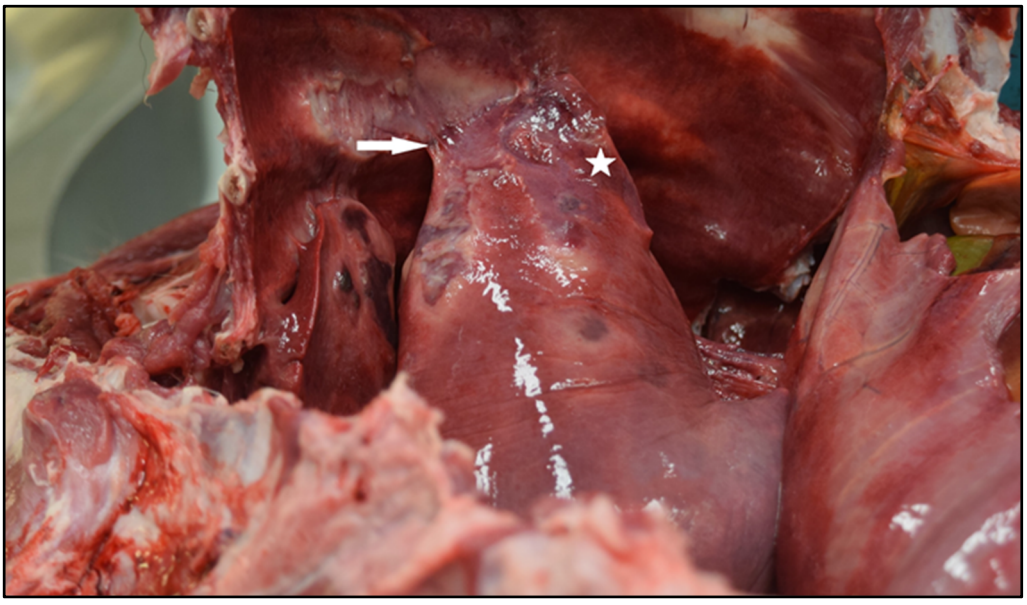

Figure 1. Lung is firmly and focally adherent to parietal pleura (arrow), and there is multifocal consolidation (asterisk) in lung parenchyma.

The inflammatory lesions in lung from each dog were typical of a bacterial etiology, and *Streptococcus equi* ssp. *zooepidemicus* (*S. zooepidemicus*) was isolated in moderate to large number from lung of both animals. PCR tests for various viral pathogens (canine parainfluenza virus, canine adenovirus 2, canid herpesvirus-1, canine distemper virus, influenza A virus) identified concurrent, low level infection with canine parainfluenza virus in dog 1, but did not detect any of these viruses in lung from dog 2.

***S. zooepidemicus* is recognized as a contributor to the canine infectious respiratory disease (CIRD) complex ('kennel cough'), and may be the main pathogen identified in respiratory disease outbreaks in kennels and shelters.** Rapid progression of pneumonic clinical signs and a poor prognosis have been described in dogs with respiratory disease attributed to *S. zooepidemicus*. Typical lesions are similar to those present in the dogs in this report, including fibrinosuppurative and necrotizing or hemorrhagic bronchopneumonia, sometimes with pleuritis.

Although more frequently encountered as an opportunistic pathogen in horses, *S. zooepidemicus* has also been isolated from other species in addition to canids. Recent outbreaks of *S. zooepidemicus* in swine have been associated with a highly pathogenic strain containing specific SzM gene mutations which were not detected in either canine isolate evaluated in this case report. *AHL*

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Feline gastrointestinal eosinophilic sclerosing fibroplasia in two cats

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Duodenal lesions were identified in two domestic short-haired cats consisting of incidental mural thickening and ulceration in a 6-month-old cat presenting for workup of post-anesthetic mortality, and an intramural mass in a 3-year-old cat exhibiting gastrointestinal symptoms. Examination of the duodenum in both cats revealed a similar histologic lesion. Expanding the intestinal wall were anastomosing trabeculae of eosinophilic matrix (collagen) interspersed with streams of plump spindle cells (reactive fibroblasts) and eosinophils, compatible with feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) (**Figs. 1, 2**).

There have been approximately five cases of FGESF submitted to the AHL from 2015-2021. Cats tend to present clinically with gastrointestinal symptoms (vomiting, weight loss) and may have a palpable abdominal mass. Cats of various ages are reportedly affected, ranging from 14 weeks-16 years, although middle-aged cats are overrepresented. Relevant and relatively consistent clinical findings include: peripheral eosinophilia; a notably 'gritty' resistance to fine needle aspiration due to the collagen content of the mass; and aspirate cytology dominated by eosinophils. Gross lesions are described as focal raised firm masses that can expand to obstruct the lumen, most often located near the pylorus or ileocecocolic junction, but appearing anywhere along the gastrointestinal tract. Histologic lesions can also appear in adjacent lymph nodes.

The underlying cause of FGESF is currently unknown. Prior to emerging as a distinct entity, differential diagnoses for FGESF included fibrosarcoma, extra-skeletal osteosarcoma or a sclerosing variant of

gastrointestinal mast cell tumor. Various bacteria have been detected within FGESF masses by culture or fluorescence in situ hybridisation (FISH). Zygomycetes and nematodes have been less frequently associated with FGESF. However, no infectious agent has been consistently implicated, and ulcerated FGESF masses are often considered secondarily infected by normal intestinal flora. A mechanical component to the pathogenesis has been suggested, with trauma from foreign bodies or ingested hair at sites of gastrointestinal narrowing (pylorus, ileocecocolic junction), allowing for bacterial invasion and subsequent inflammation. **FGESF lesions may represent an immune-mediated, self-perpetuating eosinophilic inflammatory response following exposure to unknown antigens, with a genetic predisposition for a dysregulated immune response being the primary contributor**. The significance of any breed predisposition or hereditary component is currently undescribed. It has also been suggested that FGESF may represent another aspect of the feline eosinophilic granuloma complex known to produce oral and cutaneous lesions.

Given the few reported cases of FGESF, the prognosis is not well known, but is considered guarded. With complete excision, immunosuppression and antibiotic therapy, many cats see resolution of the clinical signs without evidence of recurrence, but there are reported exceptions. *AHL*

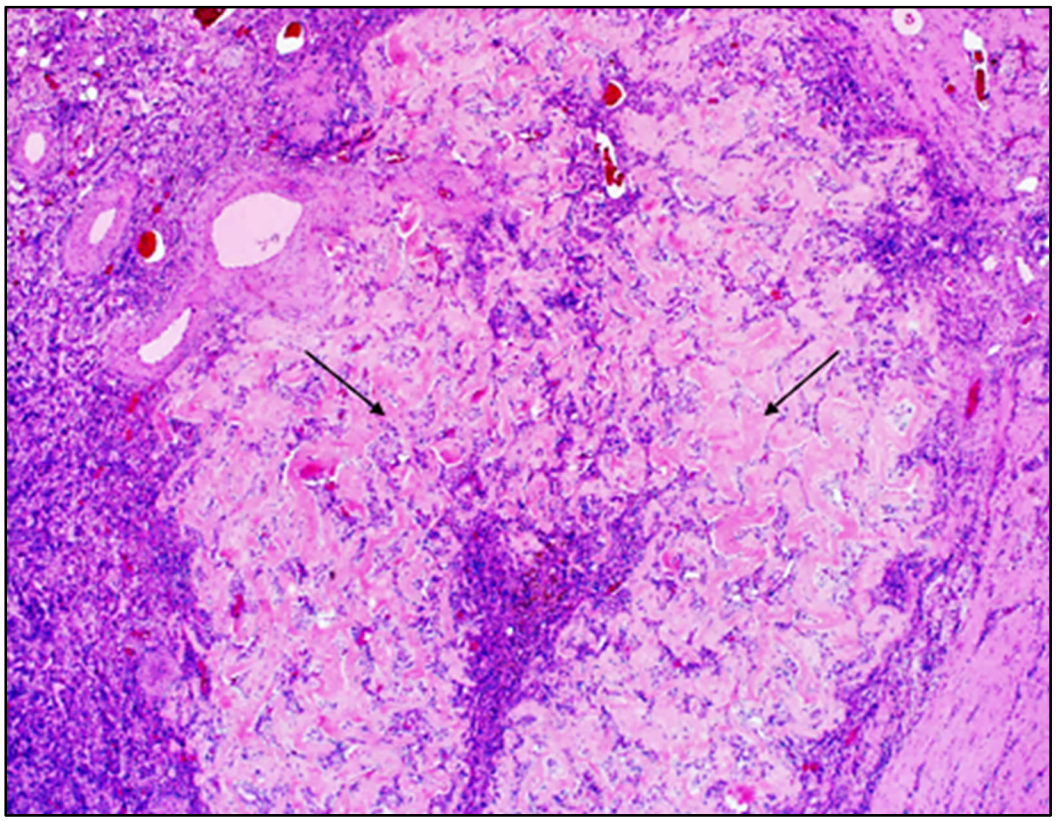

Figure 1. Expanding the duodenal wall are anastomosing trabeculae of deeply eosinophilic collagen (arrows). H&E.

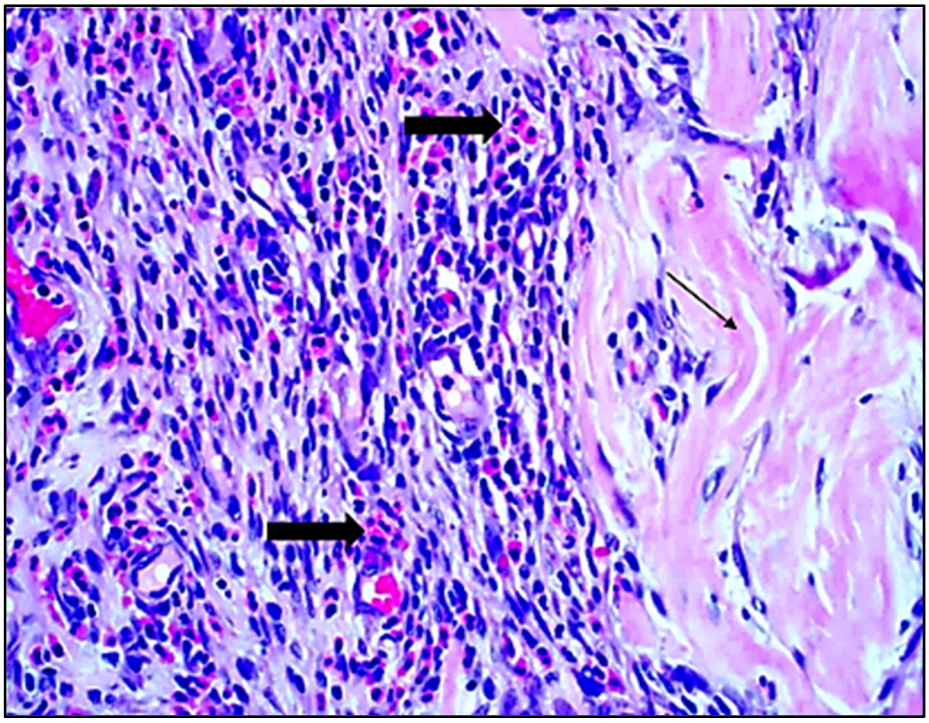

Figure 2. Reactive fibroblasts and eosinophils (thick arrows) surround the collagen trabeculae (thin arrow). H&E.

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