Joint Pathology Center
Veterinary Pathology Services

WEDNESDAY SLIDE CONFERENCE 2026-2027

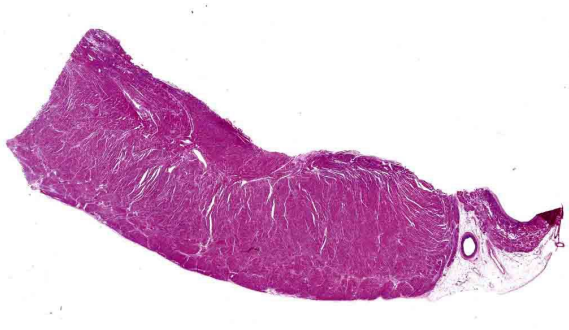
Conference #6 — 16 September 2026

CASE I:

Signalment:
18-month-old intact female Toy Manchester Terrier, canine

History:
This dog was found dead in her kennel with pink-tinged discharge from the mouth. She had no reported clinical signs.

Gross Pathology:
The carcass weighed 4.2kg and was in good nutritional and postmortem condition. The heart weighed 42.7g (1% of body weight; adult normal: 0.43-0.99%). The left ventricular free wall, interventricular septum, and right ventricular free wall were 10mm, 7mm, and 4mm thick, respectively. The left atrial appendage was larger than the right. The stomach contained a small amount of dog food and green beans.



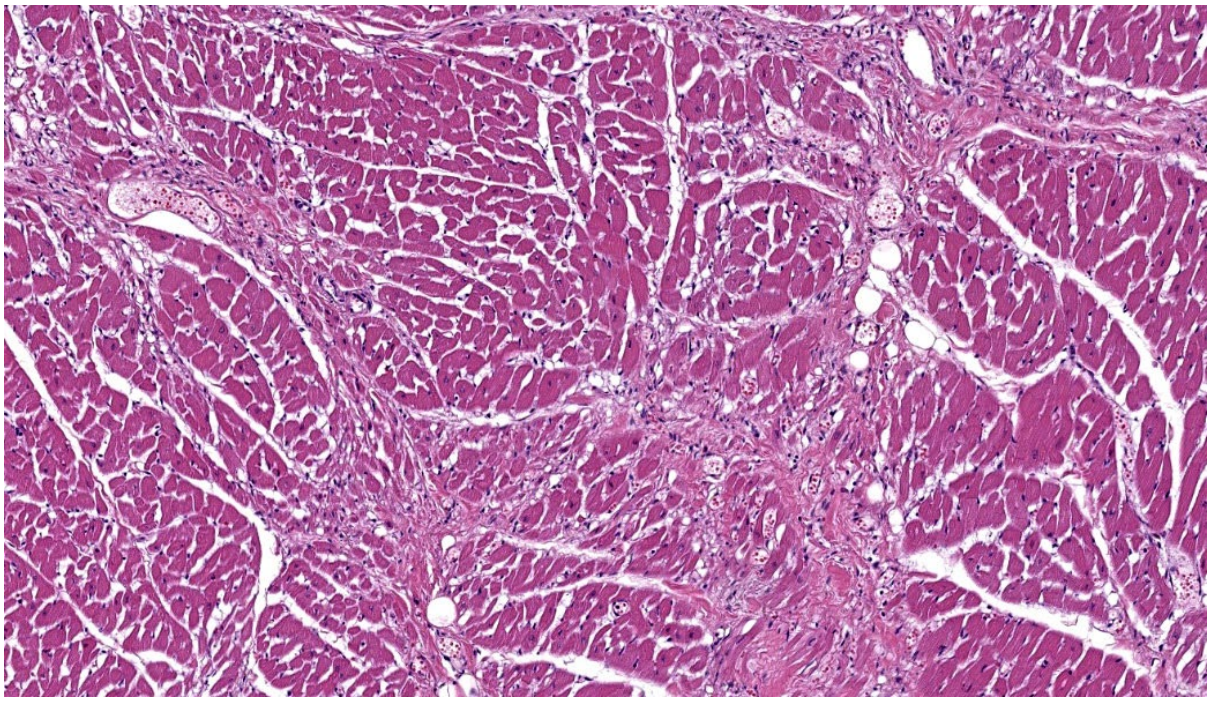
1-1. Heart, dog: A section of right ventricular free wall is submitted for examination. (HE, 9x)

Laboratory Results:
None available

Microscopic Description:
Slides include sections of the interventricular septum and right ventricular free wall or left ventricular free wall. Replacing approximately 20% of the myocardium are multifocal to coalescing areas of fibrosis associated with low numbers of lymphocytes and plasma cells. Some areas of fibrosis contain adipocytes. There is degeneration, necrosis, and rarely mineralization (not present in all sections) of entrapped myofibers. Small groups of myocytes distant from areas of fibrosis are also necrotic. Trichrome staining of the affected myocardium is a beautiful thing.

Contributor's Morphologic Diagnoses:
Marked multifocal myocardial necrosis and fibrosis

Contributor's Comment:
The signalment and postmortem findings are consistent with juvenile cardiomyopathy of Toy Manchester Terrier dogs.[1] In the published series of 14 cases, the oldest was 13.5 months and the other 13 cases were less than 10 months old. This dog was 18 months old, so slightly older than expected. Sudden death without clinical signs is a common history for these puppies.[1] The puppy in this report had food in her stomach indicating a good appetite



1-2. Heart, dog: A network of interstitial and perivascular fibrosis criss-crosses the myocardium. (HE, 209x)

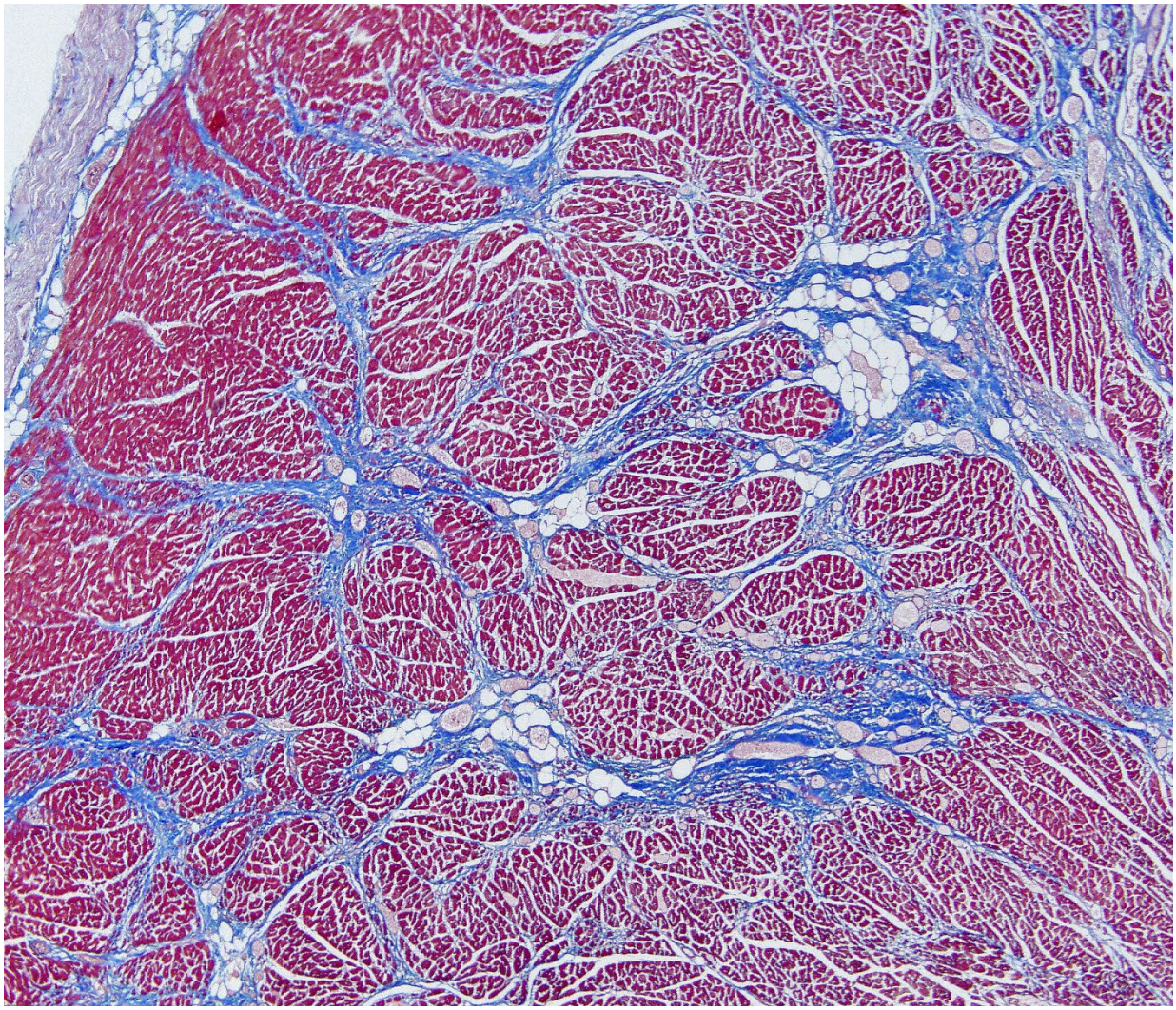
prior to death. There were no gross or microscopic lesions suggesting pre-existing congestive heart failure. The heart in this dog was not visibly dilated, but gross evidence of dilation is milder in the Toy Manchester Terriers than in Portuguese Water Dogs with juvenile dilated cardiomyopathy.[1] The left atrial appendage was larger than the right in our case, a finding that was also described in some of the published cases, most likely due to mitral valve insufficiency resulting from dilation.

Contributing Institution:
University of Tennessee College of Veterinary Medicine
Department of Biomedical and Diagnostic Sciences
Biomedical and Diagnostic Sciences

JPC Diagnoses:
Heart, ventricular free wall: Fibrosis, chronic, multifocal to coalescing, marked with cardiomyocyte degeneration, necrosis, and loss.

JPC Comment:
The term "cardiomyopathy" is used for diseases of the myocardium for which a pre-existing cause, such as a congenital heart defect, valvular disease, hypertension, or other abnormalities, is not previously diagnosed. In humans, the most common five types of cardiomyopathy based on gross and histologic changes of the affected heart include dilated, hypertrophic, arrhythmogenic, restrictive, and non-compaction[2]; all except for non-compaction have been diagnosed in one or more domestic species.

In the dog, dilatative cardiomyopathy is the second most common cardiac disorder following degenerative valvular disease.[3] Two main forms of DCM exist in the dog – those with a dietary basis and those with a genetic basis. Dietary forms of DCM in the dog are associated with the feeding of grain-free, legume-rich diets with abundant potatoes and a lack of



1-3. Heart, dog: A Masson's trichome better demonstrates the myocardial fibrosis, or to quote the contributor, "Trichrome staining of the affected myocardium is a beautiful thing." (Masson's trichrome, 200x) (*Photo courtesy of:* University of Tennessee College of Veterinary Medicine, Department of Biomedical and Diagnostic Sciences).

more traditional feed grains including rice and corn, although a direct causal line has not yet been elucidated. Other diet-driven causes of DCM include diets lacking in taurine and/or carnitine.[3]

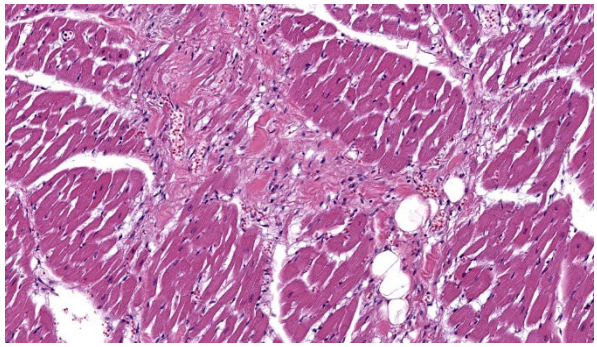
Genetic causes of canine DCM often display a combination of breed predispositions and often cluster in areas where related animals live. Commonly affected breeds include Doberman Pinschers (it is estimated that up to 58% of Dobermans may develop DCM), Airedales, Cocker Spaniels, Boxers, Bulldogs, Great Danes, Golden retrievers, and a number of others.[3] It tends to occur more commonly in middle-aged and older dogs, however, in some breeds, such as the Toy Manchester Terrier (the subject of this case) and the Portuguese Water Dogs, it is frequently diagnosed before a year of age.[1,3] One or more genetic mutations has been identified in many, but not all breeds, but is largely beyond the generalized scope of this discussion, with the exception of *ABCC9*, whose missense variant which, when homozygous, results in sudden death in Toy

Manchester terriers before the age of two years. The ABCC9 protein is a regulatory subunit of cardiac K_{ATP} channels; cardiomyocytes lacking functional ABCC9 have reduced fatty acid oxidation and oxygen consumption and are unable to respond to cell stress, leaving affected animals to die suddenly under stressful conditions.

DCM is characterized by systolic dysfunction (heart failure), dilation of both atria and both ventricles (although the diagnosis of DCM is typically predicated on dilation of the left side of the heart), and attenuation and eccentric mural hypertrophy (as opposed to concentric hypertrophy, which is seen in cases of hypertrophic cardiomyopathy, a far less common issue in the dog).[3]

The histologic hallmarks of cardiomyopathy in all species are relatively straightforward: myofiber loss and fibrosis (both of which contribute to the lack of contractility of the failing myocardium. The fibrous connective tissue component of the degenerating heart may be associated with fatty infiltration as well (which is discussed in more detail in the next case).

The fibrosis identified in cardiomyopathy most commonly takes one of three forms: replacement fibrosis, reactive/interstitial fibrosis, and perivascular fibrosis.[2] In these cases, the delicate balance between synthesis and degradation of the extracellular matrix is significantly altered toward the former by the activation and differentiation of cardiac fibroblasts into myofibroblasts, and the secretion of higher amounts of extracellular matrix under the influence of transforming growth factor-beta.



1-4. Heart, dog: More dense compacted fibrosis ("replacement fibrosis") multifocally replaces bundles of myofibers Mature adipocytes often infiltrate within areas of myofiber loss. (HE, 381x)

The histologic appearance of fibrosis can be classified into three different subtypes, all of which are present in this particular case.

- Replacement fibrosis occurs following the death of cardiomyocytes, and the histologic appearance is that of compacted bands of densely packed fibrous connective tissue which truly replaces cardiomyocytes.
- Interstitial fibrosis is characterized by the deposition of collagen as well as increased extracellular matrix, which imparts a more loosely packed appearance to the fibrous connective tissue. It is present in areas in which there is no loss of cardiomyocytes, and often requires histochemical stains, including Masson's trichrome or Movat pentachrome to identify it. It may be associated with aging, inflammation, or the initial stages of pressure overload.
- Finally, perivascular fibrosis, as the name implies, is seen primarily in the areas of the coronary arterioles, and is typically seen with hypertension.

References:

1. Legge CH, López A, Hanna P, et al Histological characterization of dilated cardiomyopathy in the juvenile toy Manchester terrier 2013;**50**(6):1043-1052.
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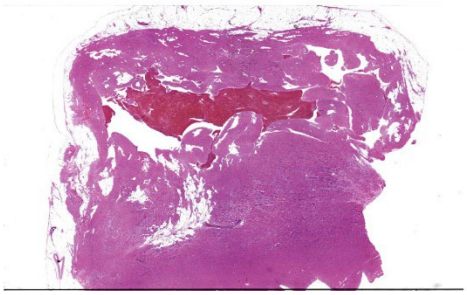
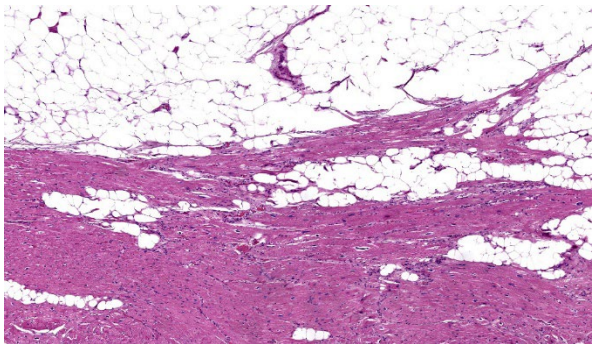
3. Donovan TA and Kelly, KM. Cardivascular System. *In*: Jubb, Kennedy and Palmer's Pathology of Domestic Animals, , 7th ed, Vol. 3, Elsevier Publishing, 2026, pp. 59-65
4. Furrow E, Tate N, Minor K, Martinson S, Larrabee S, Anttila M, Sleeper M, Henthorn P. An *ABCC9* Missense Variant Is Associated with Sudden Cardiac Death and Dilated Cardiomyopathy in Juvenile Dogs. Genes (Basel). 2023 Apr 27;14(5):988. doi: 10.3390/genes14050988. PMID: 37239348; PMCID: PMC10218235.

CASE II:

Signalment:
9.3-year-old male Indian origin rhesus macaque *(Macaca mulatta)*



2-1. Heart, rhesus macaque: The subepicardial myocardium and scattered áreas within the interventricular septum are infiltrated by sheets of mature adipocytes. (HE, 7x)



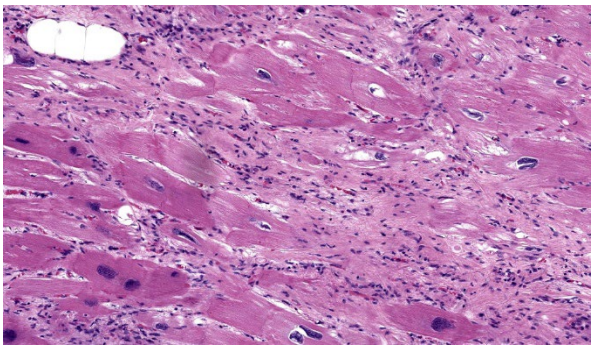
2-2. Heart, rhesus macaque: Higher magnification of infiltration of the subepicardial myocardium by mature adipocytes. (HE, 71x)

History:
This animal was used in an infectious disease study and had been experimentally infected nine days prior to euthanasia. The animal was otherwise clinically healthy.

Gross Pathology:
The heart was moderately enlarged and flabby (heart and ventricular weights could not be collected). Abundant adipose diffusely expanded the epicardium, obscuring the myocardium. Both ventricles were dilated, and there was multifocal interstitial fibrosis as well as fatty replacement within the myocardium, particularly within the interventricular septum. Other gross lesions were consistent with experimental manipulations.

Laboratory Results:
None available

Microscopic Description:
Slide contains a cross section of interventricular septum and right ventricular free wall. There is copious epicardial fat which frequently infiltrates and separates, surrounds, and dissects the cardiomyocytes of the outer myocardium, with occasional individual or small islands of cardiomyocytes encircled by abundant well-differentiated unilocular (white) adipocytes and/or mature fibrosis. There are scattered small foci of individual



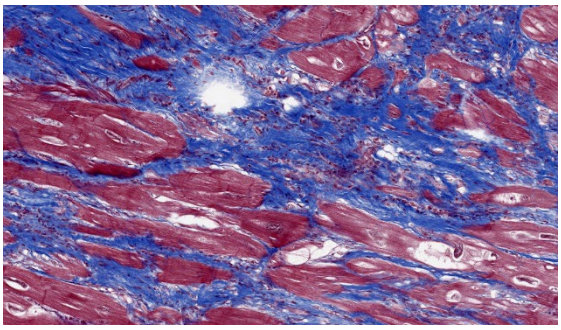
2-3. Heart, rhesus macaque: In other areas, there is degeneration of myofibers with replacment fibrosis. Nuclei within affected cardiomyocytes are hypertrophic and mildly pleomorphic. There is hyperplasia of cardiac interstitial cells. (HE, 200x)

cardiomyocyte degeneration necrosis with bland histiocytic infiltrates. There is frequent cardiomyocyte hypertrophy with numerous karyomegalic "boxcar" nuclei. There is occasional cardiomyocyte disarray with basketweave formation. There are also hypercontracted cardiomyocytes, particularly at-the cut edges of the section (artifact). There are frequent interstitial perivascular white adipocytes in the myocardium (normal).

Contributor's Morphologic Diagnoses:
Heart, RVFW and IVS: Cardiomyocyte degeneration, necrosis, and loss with fibrofatty replacement, multifocal to coalescing, chronic, moderate to severe, with cardiomyocyte hypertrophy

Contributor's Comment:
The gross and histologic lesions in the heart are consistent with arrhythmogenic cardiomyopathy, formerly arrhythmogenic right ventricular dysplasia.[2,4]

Although commonly involving the right ventricle, left and biventricular disease may occur. The characteristic features of this entity are progressive epicardial to endocardial loss of cardiomyocytes with fibrofatty replacement, with or without aneurysms of the RVFW, particularly at the RV outflow tract. ACM is a genetic disease, with autosomal dominant with incomplete penetrance and autosomal recessive forms. The vast majority of human cases in which a cause is identified result from mutations in desmosomal genes, most often plakophilin-2 *(PKP2)*. Other mutated desmosomal genes include desmoplakin *(DSP),* desmoglein-2 *(DSG2),* plakoglobin *(JUP),* and desmocollin-2 *(DSC2)*. Implicated non-desmosomal genes include alpha-T-catenin *(CTNNA3),* N-cadherin *(CDH2),* transmembrane protein 43 (*TMEM43),* lamin A/C *(LMNA),* desmin *(DES),* sodium voltage-gated channel alpha subunit 5 *(SCN5A),* phospholamban *(PLN),* and ryanodine receptor 2 *(RYR2)*. However, in up to 50% of human cases, no mutation has been identified. Antemortem diagnosis can be difficult due to an absence of specific unique diagnostic criteria, variable expressivity, and incomplete penetrance in relatives. EKG, cardiac imaging, endomyocardial biopsy, and genetic testing are the mainstays of diagnosis. Diagnosis may occur post-mortem, particularly in otherwise healthy young athletes with sudden death. In veterinary medicine, ACM has been reported in cats, dogs (boxers, English bulldogs), horses, a rhesus macaque, two chimpanzees, and a bonobo.[1,3,5,7] In boxers, deletions in the intercalated disc gene striatin *(STRN)* cause AD ACM, with worse disease in homozygotes.



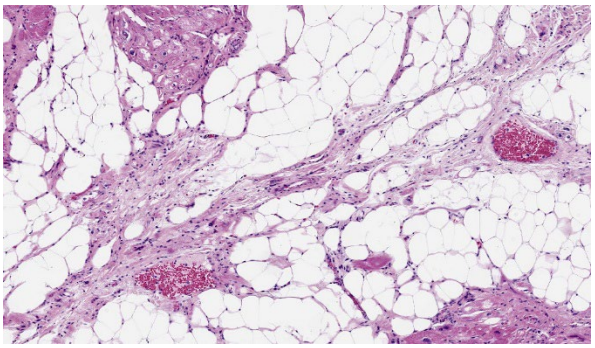
2-4. Heart, rhesus macaque: A Masson's trichome demonstrates the amount of fibrosis in the microscopic field seen on the HE section in Figure 2-3. (Masson's trichrome, 200x)

ACM should be differentiated from adipositas cordis, in which there is diffuse fatty infiltration of the ventricular myocardium or interventricular septum without fibrosis or cardiomyocyte abnormalities or destruction.

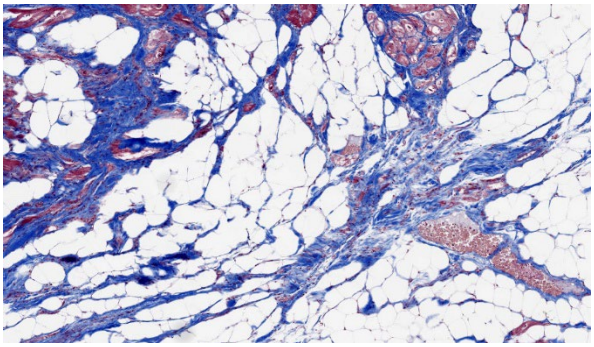
Contributing Institution:
Pathology Department
NIH/NIAID
Integrated Research Facility
https://www.niaid.nih.gov/about/integrated-research-facility

JPC Diagnoses:
Heart: Fibrofatty infiltration, chronic, multifocal to coalescing, severe, with myofiber karyomegaly, hypertrophy, degeneration, and loss.

JPC Comment:
This case was selected to provide a contrast between the histologic changes seen in dilated cardiomyopathy (Case 1 in this conference) versus arrhythmogenic right cardiomyopathy this case. It also demonstrates a nice interspecies contrast between the relatively bland degenerating cardiomyocytes in the dog, and the visually striking nuclear changes seen in late-stage cardiomyopathy in primates.



2-5. Heart, rhesus macaque: Some areas within the interventricular septum are infiltrated by both mature adipocytes and replacement fibrosis. (HE, 136x)



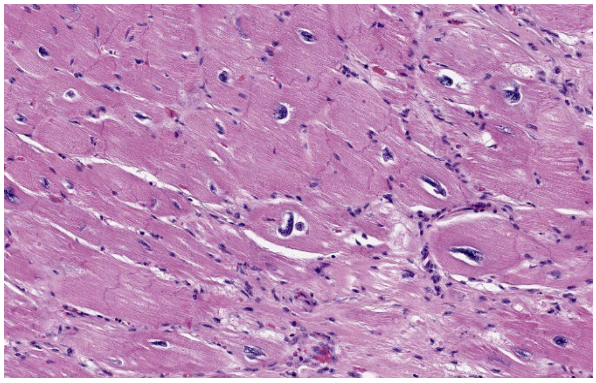
2-6. Heart, rhesus macaque: Masson's trichrome demonstrates the amount of fibrosis in the microscopic field seen on the HE section in Figure 2-5. (Masson's trichrome, 150x)

This is not the first time that this condition has been seen in the WSC – it has been previously seen in three different species – WSC 2009 Conference 5, Case 3 (dog), WSC 2018 Conference 13, Case 3 (horse), and WSC 2020 Conference 18, Case 2 (rhesus macaque).

ARVC is a genetic disease (and familial in 50% of cases) linked to defects in over 13 different genes that are vital components of the intercalated disc, particularly the adherens junction. The degeneration of intercalated disks with mutated structural proteins results in weaking of desmosomes, ultimately resulting in the loss of cardiomyocyte adhesion, cardiomyocyte apoptosis and an opportunity for adipocytes to infiltrate between separated and degenerating cardiomyocytes.[8] Other mutations in this syndrome more directly affect cardiomyocytes, to include connexin 43, whose mutation results in abnormal gap junctions between cardiomyocytes which are required for the proper movement of ions, nucleotides and protein and sugar molecules needed for prompt repolarization of myocytes following contraction. This particular mutation ultimately impairs the influx of sodium into car-

diomyocytes resulting in asynchronous contraction of myocytes and ultimately, the arrhythmias from which this disease gets its name.[9] (A more detailed description of the genetic mutations and their fascinating effects on the myocardium is present in WSC 2020, Conference 18 Case 2.). The right ventricular wall is thought to be predisposed to development of this lesion as a result of being thinner and less able to withstand the rigors of exercise due to defects in cardiomyocyte adhesion.[10] Sudden death is seen in athletes (both animal and human), while progressive heart failure is exhibited by more sedentary individuals (pass the pork rinds and put on the ballgame, please!)

One of the more intriguing changes in the section (and of the other rhesus macaque with ARVC in the WSC, but not the dog or horse case) is the nuclear pleomorphism that is seen within cardiomyocytes adjacent to areas of fibrosis. Studies of this change in human cardiomyopathy have described it a common change and in affected cases, is seen in proximity to areas of fibrosis.[11]



2-7. Heart, rhesus macaque: Cardiomyocyte degeneration with loss of cross striations, vacuolation of the sarcoplasm, karyomegaly, and nuclear pleomorphism. There are thin bands of fibrous connective tissue separating cardiomyocytes. (HE, 202X)

While early studies promoted polyploidy as the cause of the nuclear alterations in various forms of cardiomyopathy in humans, recent investigations have focused on more complex interaction between the cardiomyocyte cytoplasm and it nucleus in related process known as mechanosensing and mechanotransduction.[12,13] A vast oversimplification of these complex interplay is as follows: actin and desmin filaments as well as microtubules form connections between the sarcomere and the nuclear envelope which result in changes in nuclear morphology and gene expression.[12] Mechanical forces are transmitted from the sarcomere via desmin filaments in the Z-disks. Nuclear envelope proteins such as laminin A/C then interact with chromatin at the nuclear periphery, resulting in an increase or decrease in chromatin organization and corresponding changes in gene expression. A much more detailed accounting of these processes and the structures and proteins involved can be found in the references below.[12,13]

References:

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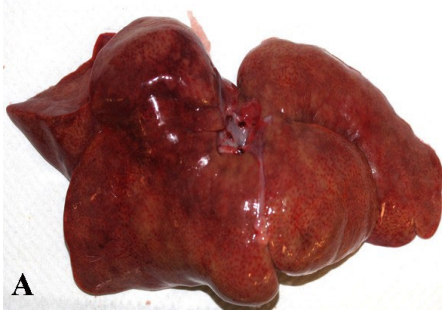
CASE III:

Signalment:
9-year-old, spayed, female, domestic shorhair cat (Felis catus)



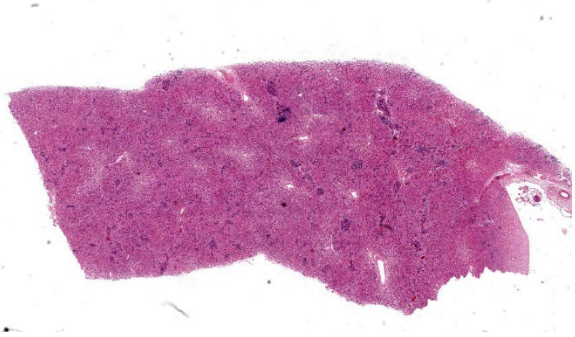
3-1. Liver, cat: The liver is grossly enlarged and pale. (*Photo courtesy of:* The University of Georgia, College of Veterinary Medicine, Department of Pathology, Tifton Veterinary Diagnostic and Investigational Laboratory, http://www.vet.uga.edu/dlab/tifton/index.php)

History:
The cat had a history of vomiting, decreased appetite, and pale mucus membranes. Hemoparasitism was suspected. Blood parasites were not observed in smears from peripheral blood. There was moderate lymphocytosis with high differential count for lymphocytes, and monocytes and a low differential count for neutrophils (neutropenia). Low RBC, HGB, and HCT values (anemia) were recorded. RBC morphology was normocytic normochromic. Due to poor response to palliative / symptomatic treatment, and poor prognosis the cat was euthanized.

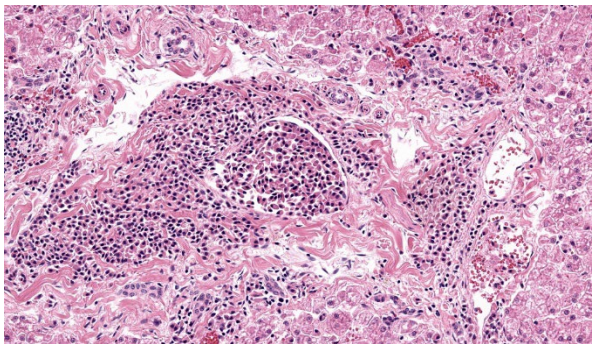
Gross Pathology:
On gross examination, the cat was found in very good nutritional condition with abundant subcutaneous and body fat. The spleen was markedly enlarged and weighed 210 grams. The liver was moderately enlarged, weighed 184 grams and pale yellowish in color. Various body lymph nodes were also moderately enlarged.

Laboratory Results:
Fluorescent antibody test for Feline leukemia virus and Feline parvovirus was negative.

Histopathologic Description:
Large numbers of round cells diffusely infiltrated the hepatic sinusoids, periportal areas and formed varisized aggregates. Similar round cells were present in scattered hepatic blood vessels. The round cells have distinct cell borders, ample to abundant finely granular eosinophilic cytoplasm, and round paracentric to eccentric nuclei. Nucleoli were indistinct. Mitotic cells were very rare. The round to polygonal cells stained positive for mast cell granules by Toluidine blue stain. Similar round cells diffusely infiltrated and effaced the splenic parenchyma and filled the medullary as well as sub-capsular sinuses of the sections of various body lymph nodes including the mesenteric, pre-scapular, and submandibular lymph nodes (sections not included in the slide). Toluidine blue stained impression smears made from cut splenic surface yielded round to polygonal cells with abundant metachromatic cytoplasmic granules consistent with mast cells (not included in the slide).



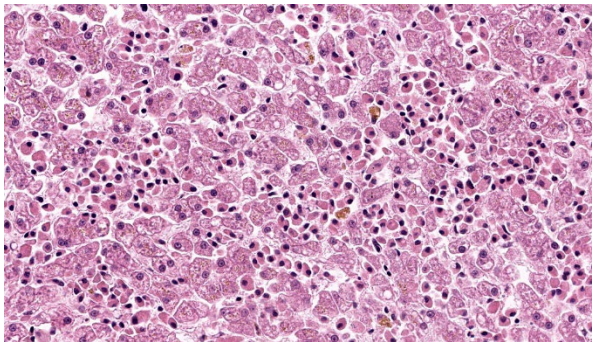
3-2. Liver, cat: Subgross magnification of the submitted section of liver demonstrates several scattered cellular aggregates, often filling vessels. (HE, 9x)



3-3. Liver, cat: In this sublobular area, the vein is filled with neoplastic mast cells that extend into the adjacent tissue. (HE, 381x)

Contributor's Morphologic Diagnoses:
mastocytosis (mast cell leukemia), liver

Contributor's Comment:
Diffuse infiltrative mast cells in spleen, liver, and various lymph node with vascular invasion is consistent with mast cell leukemia (mastocytosis / mastocytaemia). Given the severe splenomegaly observed grossly, and diffuse infiltration of splenic parenchyma by mast cells, the mast cell tumor apparently originated from the spleen and invaded other



3-4. Liver, cat: Neoplastic mast cells are present within sinusoids. Hepatocytes are swollen with cytoplasmic glycogen, lipid and lipofuscin granules. (HE, 589x)

tissues including liver. The initiating factors that induce mast cell proliferation and mast cell leukemia are largely unknown, but in humans and dogs a derangement of the c-kit receptor and /or its ligand likely plays a primary role in the disease process. Marked splenomegaly is the characteristic gross lesion of mastocytosis in the cat.[4]

Cats with mastocytosis exhibit signs of systemic illness such as depression, anorexia, lethargy, weight loss and vomiting[1,3] as was observed in this case. Mastocytemia in cats, unlike in dogs, is distinctively observed in cats with mast cell tumors and is frequently associated with the splenic mast cell tumor. Mastocytaemic cats with mast cell tumor have a lower hematocrit (HCT) value (anemia) than those without mastocytaemia.[5] Anaemia of chronic disease, blood loss due to gastrointestinal ulceration, splenic erythrocyte sequestration, removal of antibody-coated erythrocytes by splenic macrophages and erythrophagocytosis by neoplastic mast cells are suggested to underlie mast cell tumor associated anemia.[1,2]

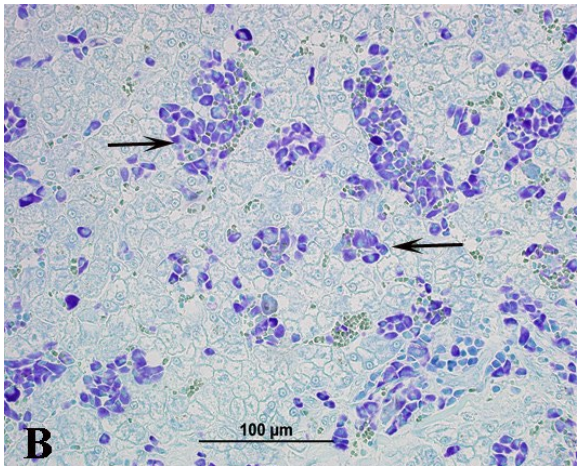
Mastocytaemia may be detected by examination of peripheral blood smear, bone marrow aspirate, or a buffy-coat (BC) layer.[1] Buffy coat examination is the preferred method and should be mandatory in cases of suspected mast cell tumor or cats with splenomegaly or vomiting of undetermined cause.[5] Cytology on fine needle aspirates of affected organs is usually diagnostic and histopathology of biopsy from affected tissue is confirmatory[3]. Splenectomy renders long survival time in cats with splenic mast cell tumor and mastocytosis.[1]

Contributing Institution:

The University of Georgia, College of Veterinary Medicine, Department of Pathology,
Tifton Veterinary Diagnostic and Investigational Laboratory
Tifton, GA 31793,
http://www.vet.uga.edu/dlab/tifton/index.php

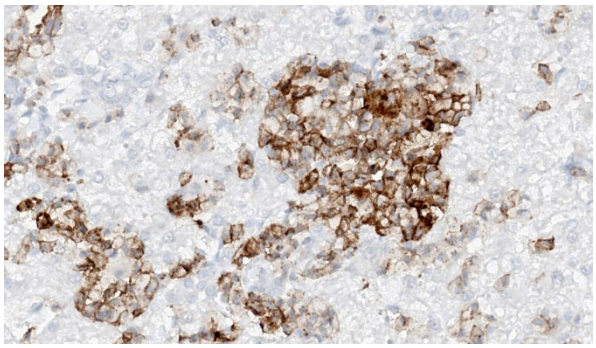
JPC Diagnoses: Liver: Mast cell tumor.

JPC Comment:
While we essentially agree with the contributor's diagnosis of mastoleukemia in this case (due to the presence of neoplastic cells within blood vessels and the hepatic sinusoids), we



3-5. Liver, cat: A Toluidine blue stain demonstrates metachromatic cytoplasmic granules. (Toluidine blue, 1334x) (*Photo courtesy of:* The University of Georgia, College of Veterinary Medicine, Department of Pathology, Tifton Veterinary Diagnostic and Investigational Laboratory, http://www.vet.uga.edu/dlab/tifton/index.php)

prefer the diagnosis of hepatic mast cell tumor in this case. Although this section shows numerous mast cells within blood vessels, and the clinical history includes anemia (suggesting the possibility of bone marrow infiltration), the lack of peripheral blood or bone marrow samples for evaluation makes an evidence-based diagnosis of "mastoleukemia" somewhat problematic. That small quibble aside, this section is an excellent example of a visceral mast cell tumor in a cat.



3-6. Liver, cat: Neoplastic cells demonstrate strong cytoplasmic immunoreactivity for CD117 (c-kit). (anti-CD117, 780x)

Visceral mast cell tumors are less common than their cutaneous counterparts, comprising up to 50% of all feline MCTs)[6,7] and are most commonly seen in one or more of the following: spleen, liver, gastrointestinal tract, lymph nodes, and bone marrow.[6] These tumors are reported to account for 15-25% of all feline splenic tumors, and 4% of all intestinal neoplasms.[7] They may be seen in association with cutaneous neoplasms, but there is no definitive evidence that they are the result of visceral metastasis from a cutaneous primary.[7] Mastocytemia may be seen with both cutaneous and visceral mast cell tumors; evaluation of peripheral blood smears, bone marrow aspirates, or buffy coat smears is required for the definitive diagnosis of mastocytemia.

The gross appearance of visceral mast cell tumors typically includes organomegaly and pallor of the affected organ, particularly the spleen. In the intestine, they are typically noncircumferential and result in a loss of mural layering on ultrasound evaluation.[7]

Unlike cutaneous mast cell tumors in cats, which may demonstrate any of three well-documented morphologies (mastocytic, histiocytic and pleomorphic), visceral mast cell tumors in cats tend to be populated with cells resembling those of the cutaneous mastocytic type – round cells with few granules and minimal cellular atypia.[7] The lack of granules on HE section is typical of feline mast cell tumors, and are reminiscent of formalin-sensitive mucosal mast cells in many species; the use of alcohol fixatives such as Carnoy's fixative or alcohol based cytologic stains such as Wright-Giemsa will often demonstrate these granules which are nearly invisible in formalin-fixed tissue.[7] Visceral mast cell tumors, like their cutaneous counterparts, typically have few infiltrating eosinophils, but may have aggregates of lymphocytes scattered throughout the neoplasm.

CD117 immunohistochemistry appears to be of more limited utility in the cat than the dog, with one study reporting that 35% of splenic MCTs and 33% of enteric MCTs were positive for this immunomarker.[9] Fortunately, most visceral MCTs are diagnosable on HE section, with a relative few requiring immunomarkers to exclude lymphoma for the differential diagnosis. In a study of feline gastrointestinal mast cell tumors, CD117 immunostaining did not appear to correlate with survival outcomes.[11]

Finally, all visceral mast cell tumors should be considered high-grade aggressive tumors, unlike their cutaneous counterparts. Splenectomy of cats with splenic MCTs has been associated with median survival times of 12–19 months,[6,10] and is almost three times that of non-splenectomized cats.[10]

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CASE IV:

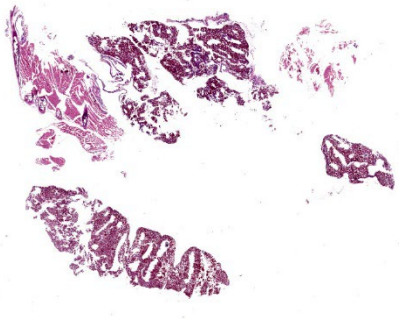
Signalment:
1-year-old male Gould finch (*Erythrura gouldiae*)

History:
The patient suffered for a long time from chronic respiratory disease, characterized by coughing, respiratory distress mostly evident during physical activity and environmental stress.

The patient was found suddenly dead in the cage by the owner.

Gross Pathology:
The finch was severely cachectic. The air sacs were severely thickened, opaque and whitish. The lungs appeared severely congested and multifocally fleshy and firm.

Laboratory Results:
None Available



4-1. Lung, rainbow finch: Multiple sections of congested lung are submitted for examination. (HE, 15x)

Microscopic Description:
Section of lung and air sac.

Lung is diffusely characterized by hyperemia of alveolar capillaries and bronchial vessels. Multifocally in the lumina of bronchi, alveoli and on the surface of air sacs several mites are evident. Mites are characterized by chitinous exoskeleton, joint appendages, thick muscular layer, intestine with lumen occasionally containing red blood cells and reproductive tract with numerous eggs (arthropods consistent with *Sternostoma tracheacolum*).

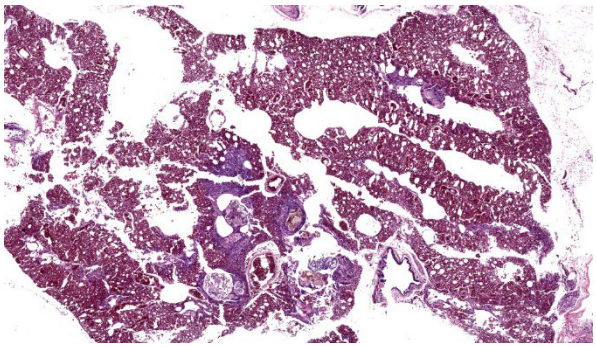
Mites are often surrounded by rows of granular macrophages admixed with foreign-body giant multinucleated cells and lesser number of lymphocyte and plasma cells.

Contributor's Morphologic Diagnoses:
Lung MD: severe, multifocal, chronic, granulomatous bronchopneumonia and aerosacculitis with intralesional arthropods consistent with *Sternostoma tracheacolum*.

ND: Respiratory acariasis

Contributor's Comment:
Mites of the family *Rhinonyssidae Trouessart*, 1895, popularly known as nasal mites, are obligatory endoparasites of birds that specifically target the respiratory system[1]. This family is organized in eight genera, including about 500 species, and has been described around the world. *Sternostoma* represents one of the genera belonging to the family Rhinonyssidae. *Sternostoma tracheacolum* Lawrence, 1948 is spread worldwide and has been recorded in both captive and wild birds. *Sternostoma tracheacolum* has been described for the first time in captive canaries (*S. canarius*) by R. F. Lawrence in 1947 in South Africa. This species resides throughout the respiratory system, ranging from the trachea to the air sacs, and is known to cause complications. In particular, *Sternostoma tracheacolum* is the only species of mite from the family Rhinonyssidae that is capable of infecting not only the nasal passages of birds but also the tracheae, lungs and air sacs causing serious respiratory problems[2]. The most common clinical signs are dyspnea, coughing, sneezing, and in severe infestations the host may die from asphyxiation or due to severe pneumonia and inflammation of air system[3].

Sternostoma tracheacolum is a generalist parasite infecting many species of hosts. In a large-scale survey involving 103 species and 502 individual birds, the author found the parasite in 40 species and in 87 birds with a prevalence of 17%[3].



4-2. Lung, rainbow finch: Within one section of lung, within the air bronchi, there are cross sections of multiple 2.75mm diameter lung mites. (HE, 75X)

High prevalence of *Sternostoma tracheacolum* was found also in house sparrow (Passer domesticus)[2]. The broad geographic distribution of *Passer domesticus* and its adaptability to different ecosystems around the world including urban and rural areas, make *Passer domesticus* an important reservoir for a number of pathogens of interest and concern for public and animal health, including *Sternostoma tracheacolum*[2].

Tidemann et al. (1992)[4] documented the occurrence of *Sternostoma tracheacolum* in wild passerines in Australia where the prevalence of this mite infection was 62% in the Gouldian finch, *Erythrura gouldiae* (Gould, 1844). The authors reported that *Erythrura gouldiae* had 34.1 mites/bird (1 – 102 mites); mites were found in different anatomical locations. The authors suggested that *Erythrura gouldiae* has not been able to regain its former population status due to *Sternostoma tracheacolum* infection. According to the IUCN Red List of Threatened Species, the Gouldian finch has been endangered since the 1980s[2].

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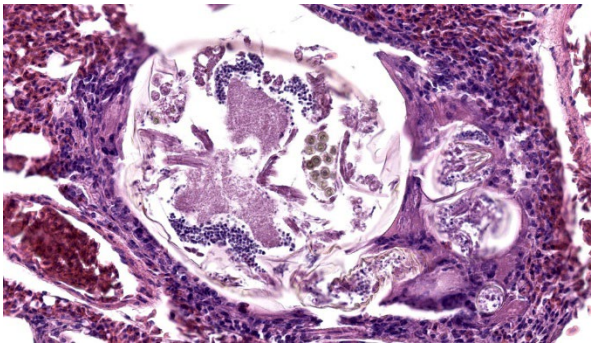
JPC Diagnoses:

1. Lung: Bronchopneumonia, granulomatous, chronic, multifocal to coalescing, moderate with adult mites and mild granulomatous and proliferative airsacculitis.
2. Bone marrow: Fat atrophy, chronic, diffuse, severe.
3. Skeletal muscle: Myocyte atrophy, chronic, diffuse, severe.



4-3. Lung, rainbow finch: Within one section of lung, within the air bronchi, there are cross sections of multiple 2.75mm diameter lung mites. Mites are characterized by a chitinized brown exoskeleton, jointed appendages containing skeletal muscle, a blue granular nervous system, and an reproductive tract with numerous eggs. The mite is surrounded by multiple layers of macrophage and few foreign body-type macrophage containing brown-black mite pigment. (HE, 750X)

JPC Comment:
First things first... How did this brightly colored bird get its name? The bird, a colorful passerine within the ranks of Australian fauna, was named by British ornithologist Sir John Gould from several specimens he received from British naturalist Benjamin Bynoe. Although this species had been described years prior by French naturalists Jacque Bernard Hombron and Honore Jacquinot. Ignoring that pesky fact, Sir John Gould named the finch *Amadina gouldiae* after his dead wife Elizabeth. The genus *Amadina* is a related genus of parrotfinches, and the species has also spent time in the genera *Chloebia* and *Erythrura.* It also goes by a range of given names, with the rainbow finch describing its colorful plumage, the Gouldian finch, and the Lady Gouldian finch.

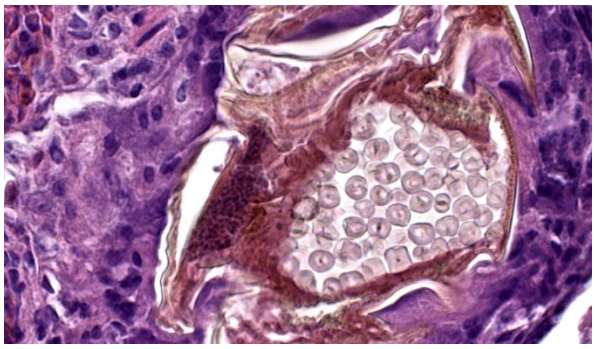
The contributor has provided an excellent review of the rhynonyssid or nasal mites of finches and related species, to include Ster-

natstoma tracheocolum, but perhaps a brief review of other nasal and respiratory mites are in order.

Staying with avian species, the air sac mite is a common parasite of a wide range of poultry, including chickens, turkeys, gamebirds, canaries and pigeons. These mites result in air sac disease, and light infections are well-tolerated, but heavier infections result in granulomatous inflammation of the airways, lungs, and pneumatic bones.

Respiratory mites are not unheard of in mammalian species as well. *Orthohalarachne* (which infects sea lions and walruses) and *Halarachne* sp. (which infect seals and sea otters), which have piercing mouthparts can cause significant irritation, and may transmit bacterial pathogens such as *Streptococcus phocae* as well. How this occurs is not yet clear. It may simply pierce the respiratory mucosa with their mouthparts, may diminish the immune system in affected animals, or it could represent a true host/vector/pathogen relationship.

A few more well-known mites parasitize dogs and non-human primates. *Pneumonyssoides caninum,* a global parasite that appears concentrated in Scandinavia. Presence of this mite results in sneezing (both conventional and reverse, nasal pruritus, serous discharge, anosmia, and epistaxis, and aberrant migration may result in lacrimation, orbital cellulitis, and nervous signs. The mite has even once been suggested to be a cause of gastric dilation-volvulus due to aerophagia associated with reverse sneezing.[8]



4-4. Lung, rainbow finch: High magnification of the reproductive tract with eggs. (HE, 1522x)

Finally, one of the most well-known respiratory mites known to pathologists and laboratory animal medical practitioners is *Pneuonyssus simicola*, the air sac mite of non-human primates. These mites are seen with a frequency of almost 100% in wild and wild-caught Old World primates. Histologic findings include granulomatous to eosinophilic bronchitis and bronchiolitis due to the presence of mites and their excreta. Over time, the presence of the mites, their golden-brown iron-laden excreta, and the inflammation that all of this creates results in bronchiectasis, and the formation of what is known as colloquially referred to "mite houses" in the lungs of affected macaques.

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