Joint Pathology Center
Veterinary Pathology Services

WEDNESDAY SLIDE CONFERENCE 2025-2026

Conference #17 — 28 January 2026

CASE I:

Signalment:
Female Indian ringneck parrot/parakeet (*Psittacula krameria*)

History:
Trouble breathing, wings out.

Gross Pathology:
There was moderate gelatinous hydrothorax and ascites. The lungs were congested and edematous.

Laboratory Results:
Transmission electron microscopy - lung: Intranuclear and intracytoplasmic herpesviral replication complexes

- Psittacine herpesvirus 1 PCR - lung: negative
- Herpesvirus PCR and sequencing: positive - Psittacid herpesvirus 5

Microscopic Description:
The bronchi and parabronchi contain numerous large syncytia with large amphophilic-to-eosinophilic intranuclear inclusions that marginalize the chromatin. Bronchi and parabronchi also often contain foamy macrophages, pale basophilic mucus, homogeneously eosinophilic edema fluid, fibrin, cellular debris, and sloughed necrotic epithelial cells.

Contributor's Morphologic Diagnoses:
Lung: Pneumonia, histiocytic, with syncytia and intranuclear inclusion bodies

Contributor's Comment:
Pulmonary lesions in this parrot were consistent with a herpetic pneumonia (cause: Psittacid alphaherpesvirus 5). *Herpesviridae* is a large family of double-stranded DNA viruses that can infect a wide variety of animal species, including fish, mollusks, reptiles, birds and mammals.[1,3] A hallmark of this family of viruses is persistent infection (or latency) with intermittent or continuous shedding. Herpesviruses can be sub-classified into alpha-, beta-, or gamma-herpesviruses. The subfamily *Alphaherpesvirinae* is further subdivided into the following genera: *Iltovirus*, *Scutavirus*, *Simplexvirus*, *Varicellovirus,* and *Mardivirus*.

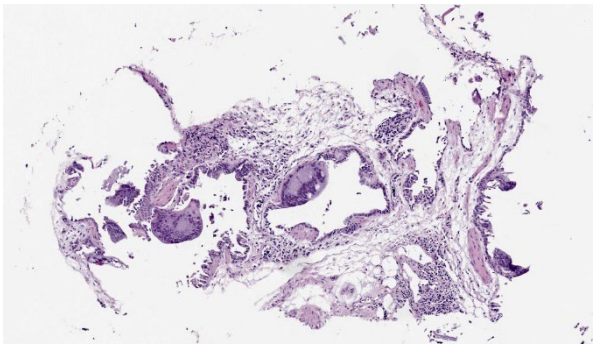

Figure 1-1. Bronchus and parabronchus, Indian ringneck parakeet: One section of mildly autolytic bronchus and parabronchus is submitted for examination. There are numerous cytomegalic multinucleated viral syncytia within the airway epithelium which project into the lumen of the airways. nucleated viral syncytia within the airway epithelium which project into the lumen of the airways. (HE, 188X)

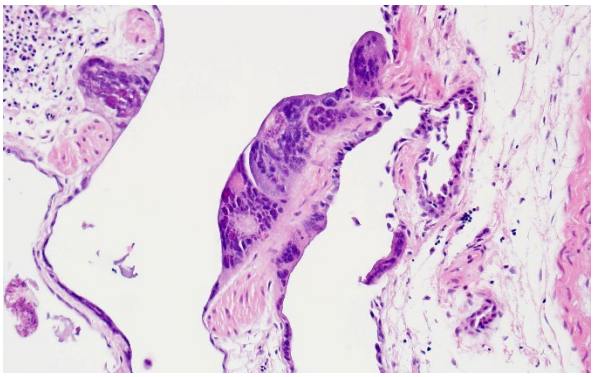

Figure 1-2. Bronchus and parabronchus, Indian ringneck parakeet: Higher magnification of viral syncytia. (HE, 188X) (Photo courtesy of: California Animal Health and Food Safety Laboratory, Sa Bernardino Laboratory. https://cahfs.vetmed.ucdavis.edu/)

The subfamily *Betaherpesvirinae* is further subdivided into the following genera: *Cytomegalovirus*, *Muromegalovirus*, *Proboscivirus*, *Quwivirus*, and *Roseolovirus*. Similarly, the subfamily, *Gammaherpesvirinae* is also subdivided into several genera – *Lymphocryptovirus*, *Bossavirus*, *Macavirus*, *Manticavirus*, *Patagivirus*, *Percavirus*, and *Rhadinovirus*.[1]

Herpesviruses of veterinary significance and their classification:[3]

- Subfamily *Alphaherpesvirinae*: Bovine herpesvirus 1 (Infectious bovine rhinotracheitis virus, Infectious pustular vulvovaginitis virus), Bovine herpesvirus 2 (Mammillitis, Pseudo-lumpy skin disease virus), Bovine herpesvirus 5 (Bovine encephalitis virus), Canid herpesvirus 1, Caprine herpesvirus 1, Cercopithecine herpesvirus 1 (B virus disease of macaques), Cercopithecine herpesvirus 9 (Simian varicella virus), Equid herpesvirus 1, Equid herpesvirus 3 (Equine coital exanthema virus), Equid herpesvirus 4 (Equine rhinopneumonitis virus), Felid herpesvirus 1 (Feline viral rhinotracheitis virus), Gallid herpesvirus 1 (Avian infectious laryngotracheitis virus), Gallid herpesvirus 2 (Marek's disease virus), Human herpesvirus 1, Human herpesvirus 2, Human herpesvirus 3 (Varicella-zoster virus), Saimiriine herpesvirus 1 (Herpesvirus tamarinus), Chelonid herpesvirus 5
- Subfamily *Betaherpesviriane*: Murid herpesvirus 1 and *2*, Elephantid herpesvirus (Endotheliotropic elephant herpesvirus), Suid herpesvirus (Porcine cytomegalovirus), Macacine herpesvirus 3 (Rhesus cytomegalovirus)

- Subfamily *Gammaherpesvirinae*: Malignant catarrhal fever (cause: Alcelaphine herpesvirus 1, Ovine herpesvirus 2), Bovine herpesvirus 4, Equid herpesvirus 2, Equid herpesvirus 5, Equid herpesvirus 7 (Asinine herpesvirus 2), Human herpesvirus 4 (Epstein-Barr virus), Saimiriine herpesvirus 2 (Herpesvirus saimiri), Otarine herpesvirus 1

There are a number of herpesviruses of significance in birds including *Gallid alphaherpesvirus 1* (GaAHV1; causative agent of infec

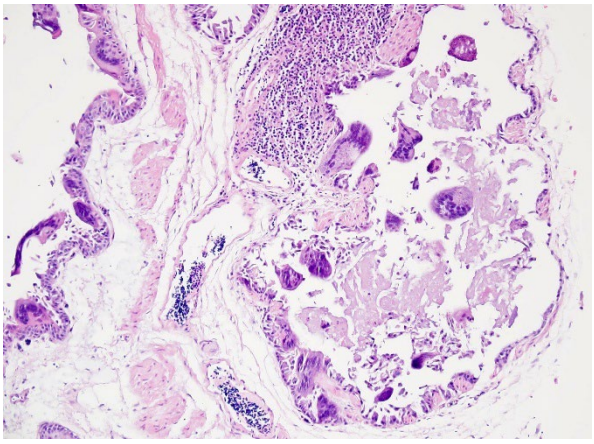

Figure 1-3. Bronchus and parabronchus, Indian ringneck parakeet: The lumen of the airway contains sloughed epithelium and viral syncytia, edema fluid, few histiocytes, and small amounts of cellular debris. (HE, 400X) (Photo courtesy of: California Animal Health and Food Safety Laboratory, Sa Bernardino Laboratory. https://cahfs.vetmed.ucdavis.edu/)

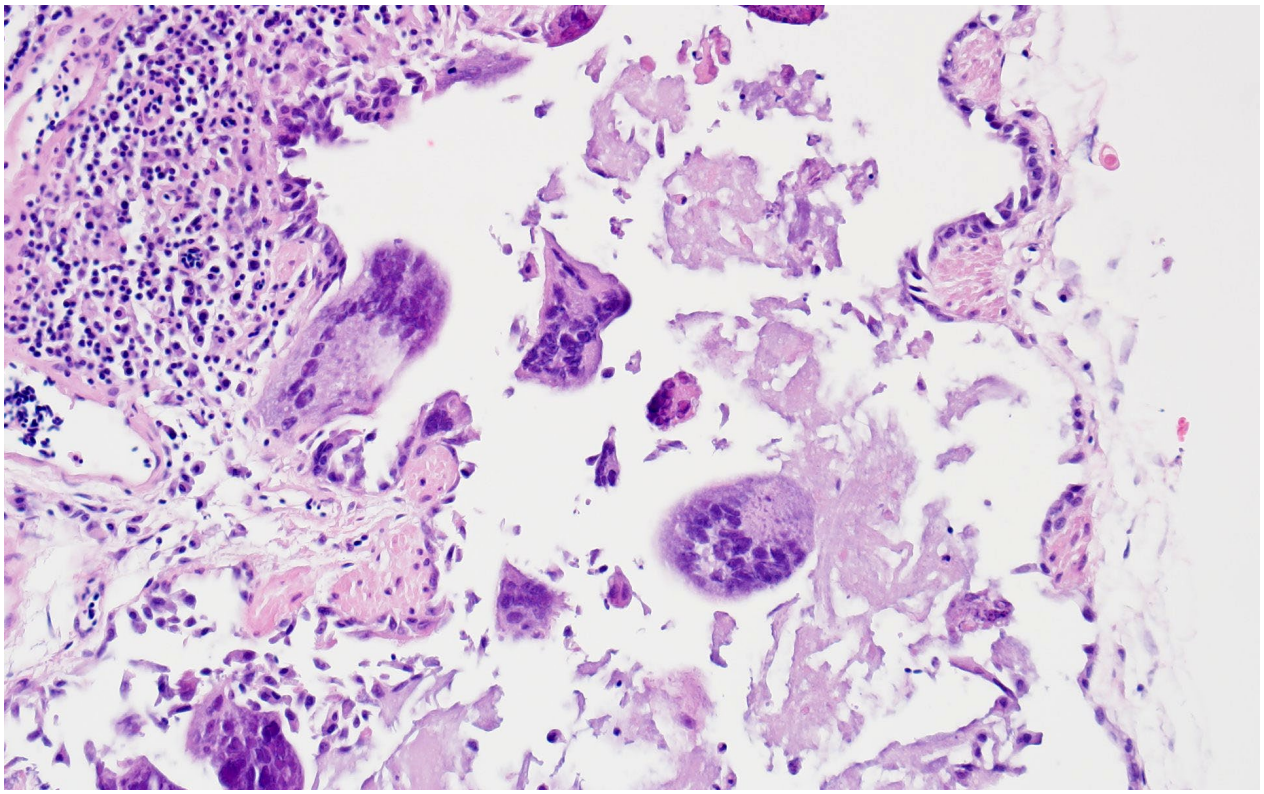

Figure 1-4. Bronchus and parabronchus, Indian ringneck parakeet: Higher magnification of luminal contents and viral syncytia. (HE, 400X) (Photo courtesy of: California Animal Health and Food Safety Laboratory, Sa Bernardino Laboratory. https://cahfs.vetmed.ucdavis.edu/)

tious laryngotracheitis), *Gallid alphaherpesvirus 2* (GaAHV2; causative agent of Marek's disease), *Anatid alphaherpesvirus 1* (AnAHV1; causative agent of duck viral enteritis), *Psittacid alphaherpesvirus 1* (PsAHV1; causative agent of Pacheco's disease and papillomatosis), and *Columbid alphaherpesvirus 1* (CoAHV1; pigeon herpesvirus; fatal disease in hawks, owls, and falcons). In psittacines, at least 5 herpesviruses have been named; all of the aforementioned viruses cluster in the genus *Iltovirus*.[2]

- *Psittacid alphaherpesvirus 1* (PsAHV1)
- Provisionally named Psittacid alphaherpesvirus 2 (PsAHV2)
- Provisionally named Cacatuid alphaherpesvirus 1
- Provisionally named Cacatuid alphaherpesvirus 2
- Provisionally named Psittacid alphaherpesvirus 3 (PsAHV3)
- Provisionally named Psittacid alphaherpesvirus 5 (PsAHV5)

Respiratory herpesviruses have been described in Indian ringneck parrots as early as the 1990s.[4,8] Similar gross and microscopic lesions have been reported in psittacines infected with PsAHV3 and PsAHV5, including the formation of large syncytia and intranuclear inclusions throughout the respiratory tract.[2,6,7] Thus far, PsAHV5 has been detected in Indian ringneck parakeets, Alexandrine parakeets, Bourke's parrots, and possibly eclectus parrots. Concurrent infections, such as Aspergillosis, are common.[2]

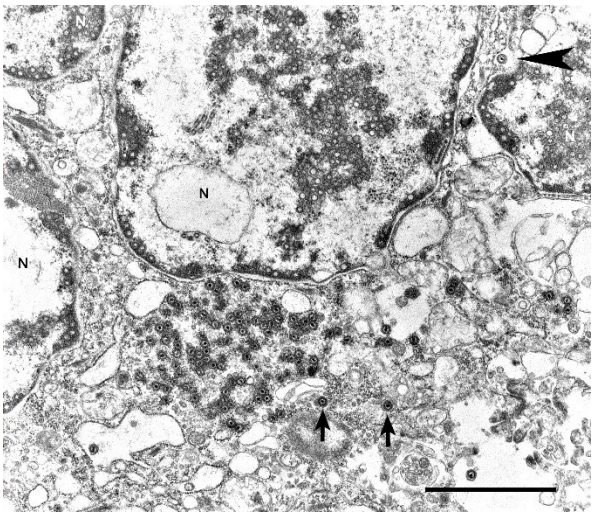

Figure 1-5. Bronchus and parabronchus, Indian ringneck parakeet: Syncytial cell with nuclear replication and assembly and cytoplasmic replication complexes. Note numerous nucleocapsids positioned at the nuclear membrane. A maturing enveloped virion is trafficking through the nuclear membrane (black arrowhead). Virions near cisterns (arrows) are gaining the final envelope and tegmental proteins. N = nuclei. Bar = 2µm.

Contributing Institution:
California Animal Health and Food Safety Laboratory (San Bernardino Branch)
https://cahfs.vetmed.ucdavis.edu/

JPC Diagnoses:
1. Tubular reproductive tract, air sac, lung: Epithelial necrosis, acute, multifocal, moderate, with intranuclear viral inclusions and numerous syncytia.
2. Liver: Hepatitis, necrotizing, acute, multifocal, random, moderate with microbes consistent with microsporidia.

JPC Comment:
The JPC's own Dr. Elise LaDouceur moderated this year's avian-focused seventeenth conference. This first case provided participants with a panoply/salmagundi/"dog's breakfast" of tissues to sort through to achieve a diagnosis. The characteristic intranuclear viral inclusion bodies of herpesvirus were best seen in the air sac respiratory epithelium, which also rewarded conference-goers with some exceptional viral syncytia. Of the potential herpesviral culprits, psittacine alphaherpesviruses (PsAHV) 1, 3, and 5 have been reported in psittacine species, including Indian ringneck parakeets, and PsAHV-5 was isolated by the contributor in this case. The contributor provided some beautiful electron microscopy (EM) photos from this bird that demonstrated intranuclear herpesviral virions, with some virions budding from the nuclear envelope to become enveloped themselves, and others hanging around the rough endoplasmic reticulum (RER) to acquire additional proteins. The JPC is grateful to the contributor for providing these excellent educational images!

In addition to the respiratory epithelium, a tubular structure adjacent to the ovary was also affected by PsAHV-5 (i.e., had syncytia and epithelial necrosis). The smooth muscle wall and cellularity of this structure is consistent with either oviduct or remnant Wolffian duct. This suggests that PsAHV-5 may have a broader epitheliotropism beyond respiratory epithelium.

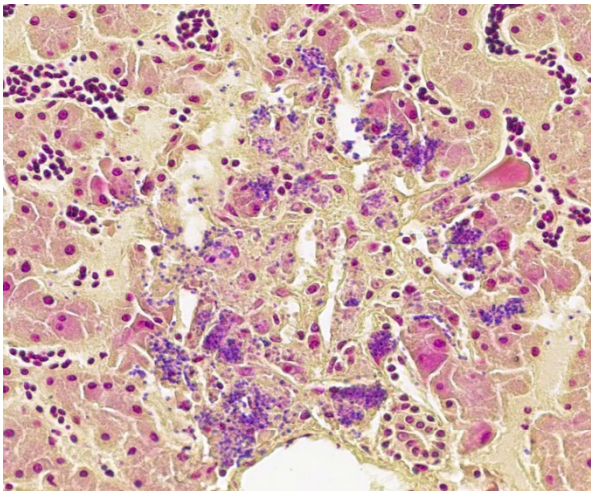

Figure 1-6. Liver, Indian ringneck parakeet: There are gram positive, round to oval, intracellular organism in regions of hepatocellular necrosis. Organisms have an eccentric clearing, most consistent with microsporidial spores. (Brown and Brenn (B&B) Gram stain, 400X)

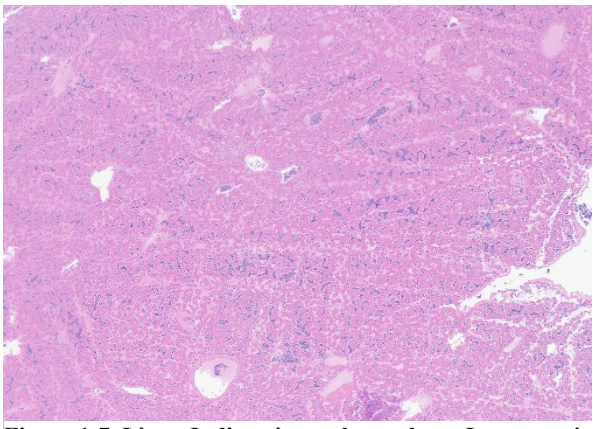

Figure 1-7. Liver, Indian ringneck parakeet: Low magnification of the liver demonstrates numerous, linear clefts filled with pale eosinophilic fluid typical of "freeze thaw" artifact. (HE, 188X)

The liver had multifocal, random hepatocellular necrosis centered on microbes that were intracellular (within hepatocytes), round to ovoid, and 1.5-2.5µm with an eccentric clearing. These organisms were gram-positive, argyrophilic on GMS, and positively staining on Giemsa; PAS and acid-fast stains were negative. Conference participants discussed at length the potential identity of these organisms. The primary differential is *Encephalitozoon hellum*, which is a microsporidian parasite in psittacines. *E. hellum* is an intracellular parasite that is ovoid, 1.5-2.5 µm, and has a predilection for the liver.[5] It is most common in immunosuppressed animals, which was likely in this case given the herpesvirus infection. Other differentials raised by participants included chlamydiosis, which can cause intracellular bacterial inclusions in the liver, but the individualized organisms were too large for chlamydiosis. Additionally, *Chlamydia spp.* would be expected to be gram-negative (unlike the gram-positive organisms in this case).[5] Candidiasis was also considered; however, yeasts should be positive for PAS (unlike the PAS negative organisms in this case) and is typically larger than 1.5µm.[5] Ultimately, confirmatory diagnosis of *E. hellum* would require molecular investigation or ultrastructural analysis (transmission electron microscopy) revealing polar filaments.[5]

Participants also noted the characteristic "freeze-thaw" artifact of the liver that is best appreciated at low magnification and causes acicular clefts filled with pale eosinophilic fluid. This is a classic artifact to be aware of!

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CASE II:

Signalment:
2-wk-old domestic duck (*Anas platyrhynchos* var *domestic*a)

History:
The duck was intranasally inoculated with Asian H5N1 subtype highly pathogenic avian influenza (HPAI) virus isolated in 2008. Cloudy eyes appeared on the third day after inoculation. The duck died on day 6 post inoculation.

Gross Pathology:
Pancreatic focal necrosis and myocarditis were observed.

Laboratory Results:
N/A.

Microscopic Description:
The corneal stroma exhibited edema, scattered cellular debris, and inflammatory cell infiltrates that were predominantly located in the middle to deep layers of the stroma. Inflammatory cells were heterophils and foamy macrophages. Many corneal stromal spindle cells (keratocytes) became swollen. Corneal endothelium was diffusely lost, and the Descemet's membrane was exposed to the anterior chamber. Lymphoplasmacytic inflammation and focal loss of the muscular cells were observed in the iris. Inflammatory infiltrations were trapped in the pectinate ligament of the iridocorneal angle. Mild edema and inflammatory cells were observed in the conjunctiva.

Figure 2-1. Eye, anterior segment, duckling: Multiple sections of the anterior segment, including the cornea, iris, ciliary body and lens are submitted for examination. (HE, 11X)

Contributor's Morphologic Diagnoses:
1. Keratitis, heterophilic, with edema and diffuse corneal endothelial loss.
2. Iritis, lymphoplasmacytic.

Contributor's Comment:
The H5Nx subtype HPAI virus is a global threat to the poultry industry and wild birds. The popular clinical signs of domestic ducks infected with the HPAI virus are depression, anorexia, decreased egg production, neurologic signs, and mortality. [9,10,17] In addition, corneal opacity (sometimes termed cloudy eye or blindness) is one of the characteristic and frequent findings in infected ducks. [1,6,9,13,18] Corneal ulceration and exophthalmos are rare findings in experimental infection.[17] Ocular change by HPAI has been recorded in some waterfowl species, including bar-headed geese, cracking geese, herring gulls, laughing gulls, mallard ducks (including domesticated breeds), swan geese, and wood ducks.[1-3,6,8,14,16]

The corneal opacity is probably attributed to keratitis caused by the H5N1 subtype HPAI virus. The diffuse to focal loss of corneal endothelial cells was consistently observed in ducks inoculated with the virus.[18] The corneal

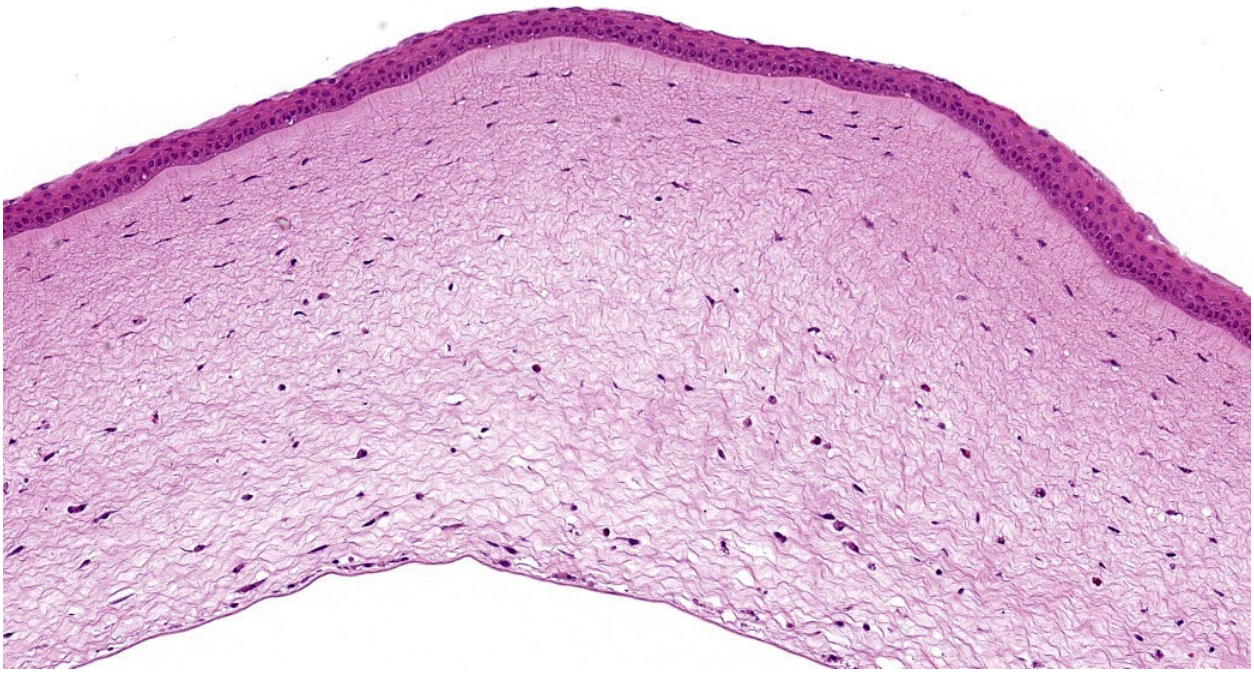

Figure 2-2. Cornea, duckling: There is marked edema of the corneal stroma, which is most profound on the inner 66% of the corneal stroma. There is scattered loss of corneal keratocyte nuclei. The endothelium is diffusely lost. (HE, 333X).

endothelium is a functional cell monolayer that maintains corneal transparency.[15] The corneal endothelium transports water from the stroma to the anterior chamber and functions as a physical barrier to water movement.[14] Although controversial, corneal endothelial cells are thought to have a limited capacity to regenerate.[5,12,15] Therefore, an extensive loss of these cells can lead to corneal stromal edema and inflammation resulting in corneal opacity. Recovery from the opacity has been reported in geese and ducks experimentally infected with the H5N1 HPAI virus.[8,18]

The H5N1 HPAI virus can replicate in various types of ocular cells in domestic ducks. The type A influenza viral antigen can be detected by immunohistochemistry in the corneal endothelial cells for short period before its loss.[18] The virus antigens can also be detected in the epithelial and stromal cells of the cornea, and cells of the iris, pectinate ligament, ciliary process, and pecten.[18]

Contributing Institution:
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JPC Diagnoses:
Eye (globe): Corneal endothelial and stromal necrosis, acute, diffuse, severe, with granulocytic and lymphocytic keratitis and anterior uveitis.

JPC Comment:
This case was an excellent example of how subtle lesions can clue a pathologist in to a specific etiology and how the evaluation of what is missing in a tissue is just as important as what is present. The lesions in this case were largely referrable to the necrosis and loss of corneal endothelium due to the HPAI infection. The corneal endothelium is responsible for removal of fluid from, and overall hydration of, the cornea. Its absence

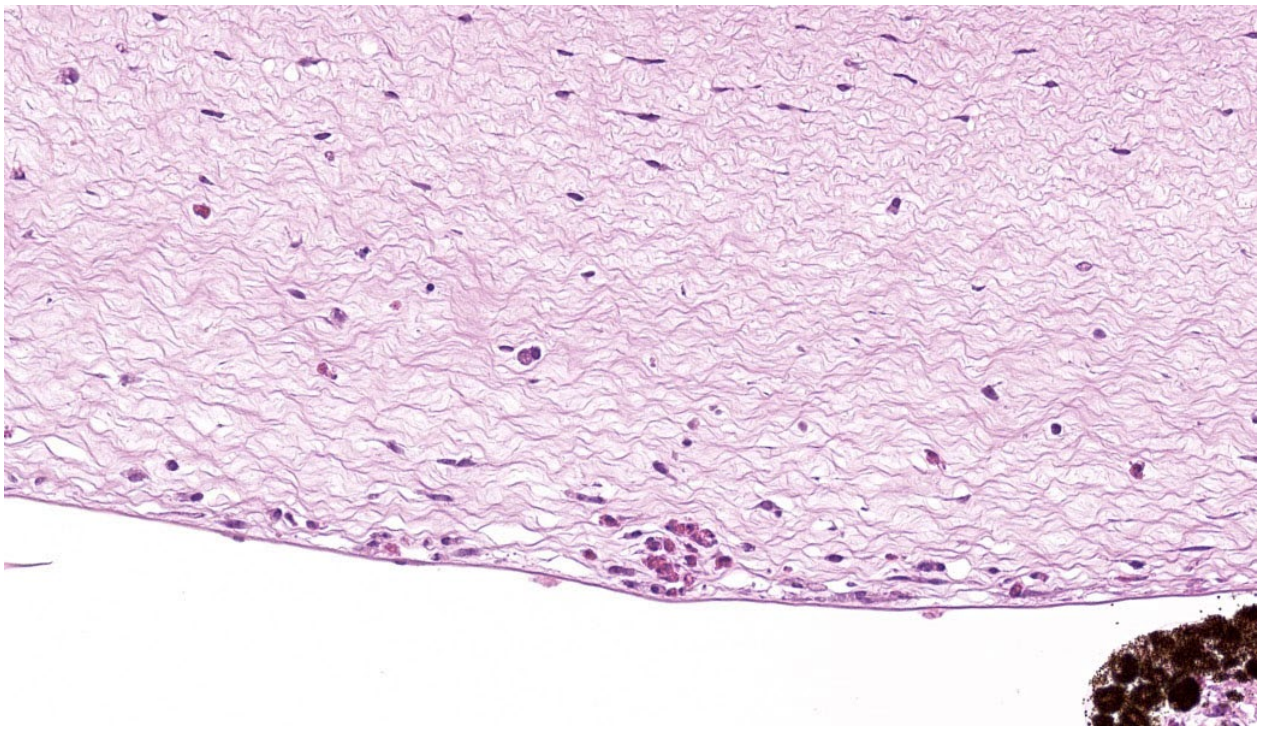

Figure 2-3. Cornea, duckling: There is diffuse loss of the corneal endothelium, marked stromal edema and infiltration of the inner layers of the cornea stroma by individualized and small groups of granulocytes. (HE, 681X).

contributed to marked corneal edema and subsequent stromal necrosis in this case. Stromal necrosis in the cornea can be a tricky diagnosis to make but is characterized by a lack of nuclei and slight tinctorial change to the stromal fibers of the cornea. These findings were most pronounced in the inner portions of the cornea (i.e., in the region closest to the corneal endothelium). While there are multiple causes of keratitis in Anseriformes (e.g., trauma, bacteria, fungi, WNV, immune-mediate disease), the specific loss of corneal endothelium appears to be unique to HPAI.[11]

HPAI viruses primarily derive their virulence from the hemagglutinin (HA) protein, which effectively enables entry into host cells. HPAIs are usually of the H5 or H7 subtypes. Influenza A viruses also make use of, amongst many other virulence factors, non-structural protein 1 (NS1), which suppresses innate immune responses by blocking type 1 interferon production and signaling. NS1 does this by binding viral RNA and sequestering it away from cytosolic pattern recognition receptors, such as RIG1 and MDA5, effectively preventing the downstream signaling pathways that would alert the cell to the presence of viral genetic material and subsequent interferon transcription.[7] This process is crucial to enabling the virus to replicate in large numbers early in an infection and cause a high viremia. These viruses demonstrate marked tropism for endothelial cells and cause multifocal hemorrhage, edema, and necrosis. In birds, meningoencephalitis and pancreatic necrosis are the two most common lesions. Less commonly, the spleen, heart, and lungs are also affected. These lesions arise due to the vascular damage, thrombosis, and dysregulated immune responses caused by the virus within the host, leading to tissue ischemia, necrosis, and, in severe cases, multiorgan failure.[1,2,3]

Conference participants also discussed the anatomy of the avian eye and how the lesions in this case were related to loss of corneal endothelial cells. Avian eyes have several unique

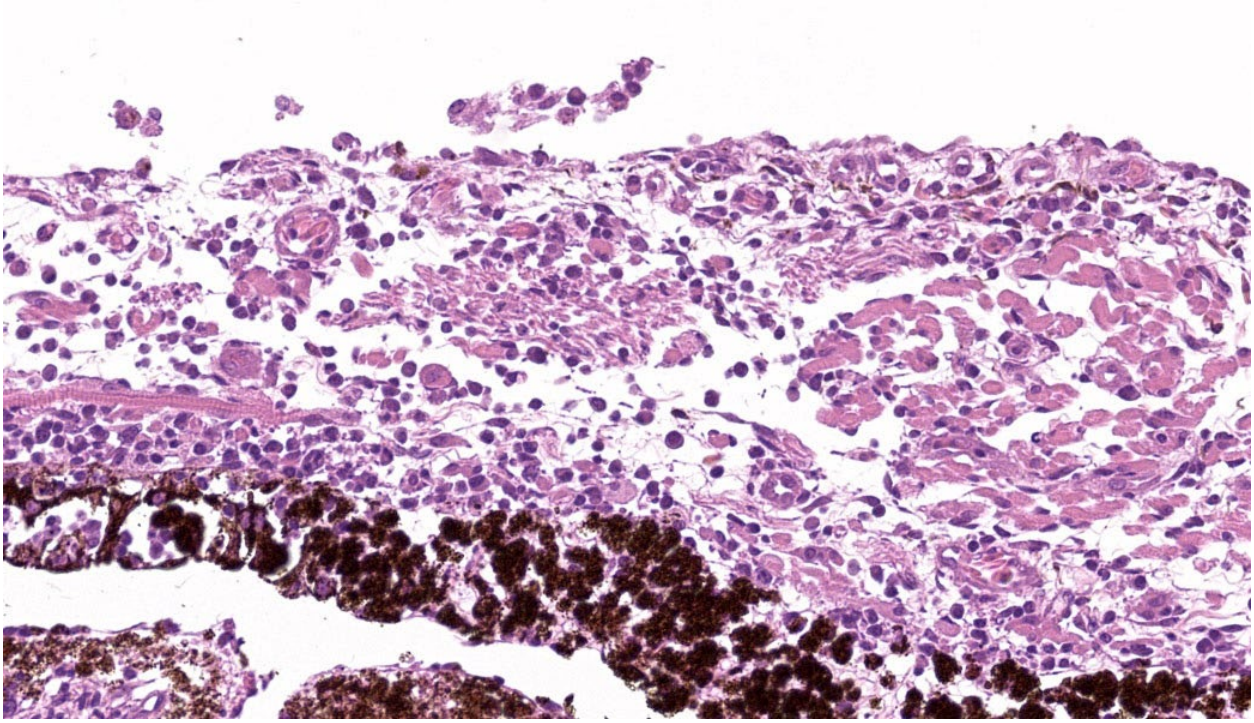

Figure 2-4. Iris, duckling: The iris is thickened and expanded by infiltration of moderate numbers of lymphocyte and plasma cells and edema which separates iridal myocytes. (HE, 811X).

anatomic features that are not present in their mammalian counterparts. The avian retina is avascular for enhanced visual acuity. Nutrition to the vitreous and retina is supplied by the pecten, which is a vascular plexus composed of small caliber blood vessels and pigmented cells. Recent research demonstrates that the inner retina receives glucose from the pecten, which also removes lactic acid.[4] Additionally, because of the pecten, trimming of an avian eye for histologic evaluation is slightly different than a mammalian eye. Rather than trimming the eye with a dorsoventral cut perpendicular to the scleral arteries at the back of the eye as is standard for mammals, avian eyes require the cut to be made parallel with the arteries to ensure that the pecten is in section. The avian sclera often entombs cartilage and/or bone, which is particularly prominent in birds of prey. In these species, the scleral ossicles are so thick that the eyes require decalcification prior to histologic processing. The iris also has various modifications depending on the species, perhaps the most striking of which is in Strigiformes (owls). Owls have xanthophores that occupy the majority of the iridial stroma and impart the dramatic yellow iridial coloration characteristic of many members of their species.[11]

References:

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CASE III:

Signalment:
Adult carrion crow (*Corvus corone*) of unknown gender.

History:
An adult carrion crow was found dead in a humane trap. Only liver was submitted for examination.

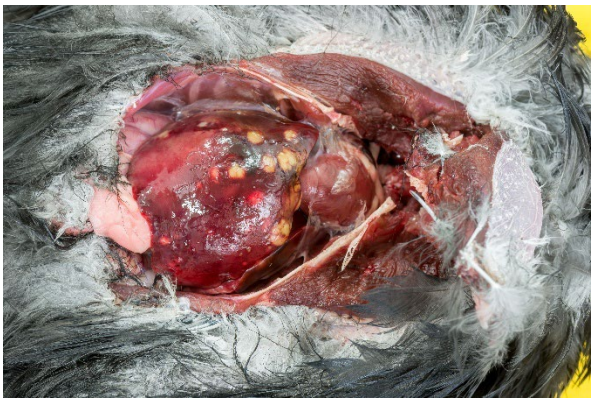

Figure 3-1. Liver, crow: There are yellow, multifocal to coalescing granulomas within the liver. (Photo courtesy of: Division of Pathology, Public Health and Disease Investigation, College of Medical, Veterinary & Life Sciences, University of Glasgow, http://www.gla.ac.uk/schools/vet/).

Gross Pathology:
In a multifocal distribution, the liver contains numerous, cream/pale yellow, moderately firm, partially confluent nodules ranging from 1x1x1mm to 6x9x7mm.

Laboratory Results:
N/A

Microscopic Description:
Approximately 70% of normal hepatic parenchyma is replaced by heterogeneously sized, multifocal to coalescing, randomly distributed foci (up to 5 mm in diameter) characterized by large, central, variably eosinophilic regions admixed with nuclear debris (necrosis), which are surrounded by inflammatory infiltrates (granulomas).

The inflammatory infiltrates consist of an inner, in most cases single layer of very large cells (up to 90 µm in diameter) with numerous (4-20) nuclei in a random arrangement (foreign body giant cells) abutting the area of necrosis, followed by a wider zone mostly comprising large (epithelioid) macrophages with occasional foreign body giant cells. Further peripherally moderate numbers of lymphocytes and plasma cells, and rare heterophils are present, multifocally situated within concentrically arranged collagen with attendant spindle cells.

The areas of necrosis consist of variably intense eosinophilic amorphous central regions, which towards their periphery multifocally exhibit more distinct cellular outlines of homogenously eosinophilic (necrotic) cells and often small to moderately sized (10 – 30 µm) round clear spaces. Small numbers of granulomas contain short basophilic rods (length 3-5 µm) within the less densely arranged necrotic material. Additionally rare individual heterophils also are seen.

The intima of a large portal blood vessel which only contains a thin to moderately thick layer of smooth muscle tissue (interpreted as large vein) in a multifocal to coalescing distribution is moderately expanded by small numbers of macrophages admixed with larger numbers of lymphocytes and plasma cells, and rare heterophils, whilst the lumen of this vessel contains moderate to large numbers of monocytes, erythrocytes and heterophils and small numbers of lymphocytes and plasma cells. Focally, the mural lymphoplasmacytic infiltrates are associated with a small, well-defined accumulation of macrophages.

Additionally, the portal area described above contains small numbers of cavernous structures, which in small areas

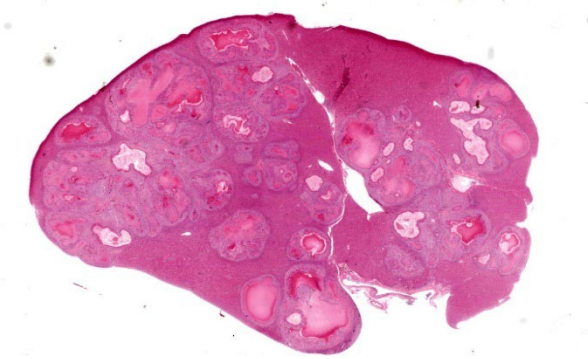

Figure 3-2. Liver, crow: Approximately 50% of the section of liver is effaced by coalescing granulomas. (HE, 9X)

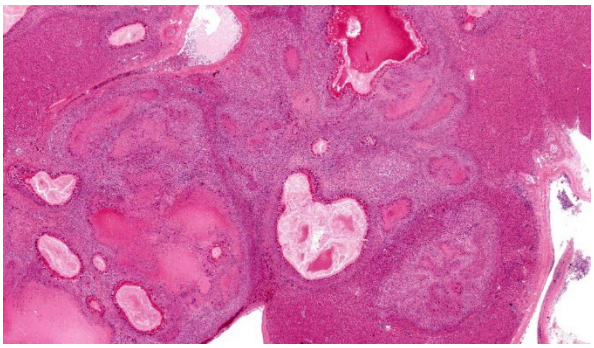

Figure 3-3. Liver, crow: Higher magnification of coalescing granulomas, with a central core of eosiiophilic cellular debris . (HE, 20X)

appears to be lined by cuboidal epithelium (interpreted as bile ducts) and contain small numbers of parasite profiles admixed with moderate numbers of desquamated epithelial cells and small numbers of macrophages and heterophils. The parasitic profiles are characterised by a thin (5-8µm) eosinophilic cuticle which lacks spines but occasionally appears ciliated, no apparent coelomic cavity, uteri containing numerous eggs and testes containing linear basophilic spermatids within the same profile (hermaphroditic organism), vitellaria containing cells with homogeneous intensely eosinophilic cytoplasm, and a ventral sucker. The eggs, measuring approximately 20 x 45 µm, are oval although often unilaterally flattened (operculated), with yellow to brown 3-5 µm thick walls and contain a miracidium (trematode, consistent with *Lyperosomum longicauda*). Small numbers of lymphocytes, plasma cells and heterophils are multifocally present in the wall of the bile ducts.

Contributor's Morphologic Diagnoses:
Liver: Hepatitis, granulomatous, multifocal to coalescing, subacute to chronic, marked to severe; with:

a) Intralesional bacilli consistent with *Mycobacterium* spp.; and
b) Phlebitis granulomatous, subacute to chronic, focal, moderate; and
c) Cholangitis pyogranulomatous, multifocal, chronic, minimal; with intralesional flukes consistent with *Lyperosomum longicauda*.

Contributor's Comment:
Histological examination of the crow liver submitted revealed extensive effacement of the liver parenchyma by numerous, variably sized granulomas, many of which contain large numbers of acid-fast short rods. Additionally, focally a large vascular profile interpreted as a large vein exhibits a predominantly lymphoplasmacytic, focally also granulomatous intramural inflammatory infiltrate, whilst small and moderately sized cavities associated with the same portal area, devoid of erythrocytes and partially lined by cuboidal epithelium (interpreted as bile ducts) contain small numbers of trematode sections.

Considering the paucity of pathological changes associated with the flukes, these very likely need to be considered incidental findings in this case. The cause of the phlebitis is not entirely clear, however, taking the nature of the inflammatory infiltrate into account, this process may need to be considered to be associated with the mycobacterial infection either via local extension or possibly also via extension from the lymphatics present within the vascular wall.

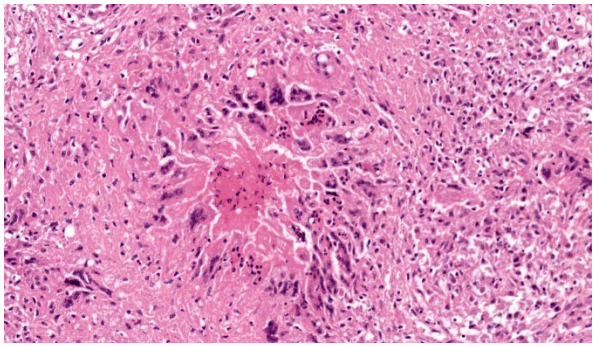

Figure 3-4. Liver, crow: High magnification of a granulomas, with a central core of eosiiophilic cellular debris, bounded by numerous epithelioid and foreign body-type macrophages. (HE, 537X)

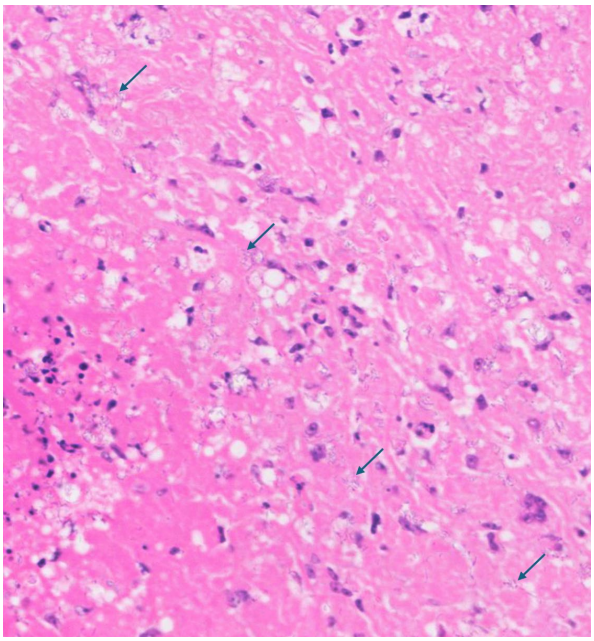

Figure 3-5. Liver, crow: There are faintly staining bacteria (arrows) within a granuloma, characteristic of mycobacteriosis. (HE, 400X)

Avian hepatic granulomas may be of viral, bacterial, fungal, protozoal or helminth aetiology, with such agents exhibiting a preference for the liver or infecting the liver in an opportunistic fashion.[13] Special stains revealed many of the hepatic granulomas in this bird to contain large numbers of short, acid-fast rods present both in macrophages and multinucleated giant cells and also extracellularly, which from their morphology and staining profile are most likely consistent with *Mycobacterium* spp.

Avian mycobacteriosis is primarily caused by *M. avium* (serotypes 1, 2, 3, and 6) and *M. genavense*, and is seen in found in many different birds including poultry, game, cage, wild and zoological species, predominantly of the North Temperate Zone. Less commonly *M. intracellulare, M. scrofulaceum*, *M. fortuitum*, *M. tuberculosis* and *M. bovis* are involved. The *M. avium* complex includes *M. avium* subsp. *avium*, *M. avium* subsp. *paratuberculosis*, *M. avium* subsp. *silvaticum* and *M. intracellulare.*[5]

Mycobacterium avium subsp. *paratuberculosis*, which causes Johne's disease in ruminants, has both been isolated from tissue and feces of Scottish carrion crows (*Corvus corone*). Additionally, histological lesions consistent with *M. avium* subsp. *paratuberculosis* were observed in these animals, albeit described to be much milder when compared to those present in this crow.[2] The infection of crows is surmised to be via the oral route, possibly from ingestion of infected rabbit or ruminant tissue. Experimentally, the contamination of livestock food by wildlife feces in turn has been determined as a possibility for the prevalence of paratuberculosis in the east of Scotland, and wild rabbits previously have been implicated as a possible wildlife reservoir for paratuberculosis.[4,12] Johne's disease is relatively commonly diagnosed in cattle of the southwest of Scotland and an increase of this disease in small ruminants in Scotland also has been reported.[10,11]

Considering crows (in experimental conditions) have also been infected with *M. bovis*, however, here only the intraperitoneal inoculation, not the oral infection, resulted in mycobacterial infection evident grossly, histologically or in pooled tissue cultures, *M. bovis* as a causative agent is considered less likely in this bird.[3] Ultimately, PCR would need to conducted to be able to confidently identify the mycobacterial (sub-) species involved.

Generally, stress, age, immune status and possible pre-existing disease are considered predisposing factors for the infection of birds with *Mycobacterium* spp; transmission may

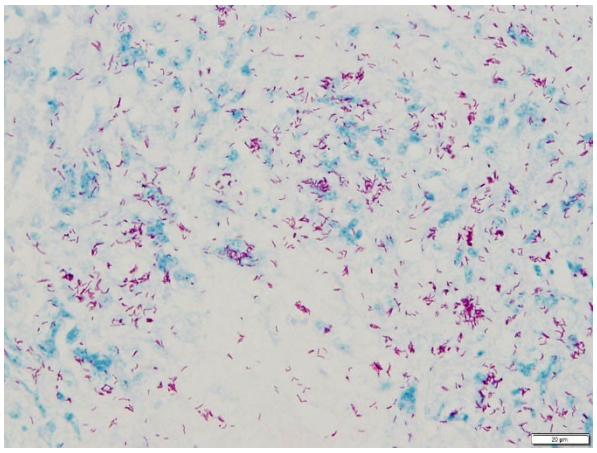

Figure 3-6. Liver, crow: Within the center of the granulomas, there are numerous acid-fast bacilli both within histiocytes and extracellularly. (Ziehl-Nielsen, 600X)(Photo courtesy of: Division of Pathology, Public Health and Disease Investigation, College of Medical, Veterinary & Life Sciences, University of Glasgow, http://www.gla.ac.uk/schools/vet/)

be oral or via aerosols. Usually, granulomatous disease is present in multiple organs, including liver, spleen, pleura, intestinal serosa, bone marrow and lung, however, previous examination of common crows with mycobacterial granulomas revealed a strong predisposition of the liver to lesions, with only rarely the spleen, peritoneum and lung affected.[9,13] To what extent other organs were involved in the crow submitted unfortunately remains unknown. However, depending on the extent and distribution of other organs involved, the mycobacterial infection may have contributed to or even caused the death of this bird.

Dicrocoeliidae, which are small, lancet-like flukes occurring in the biliary and pancreatic ducts of vertebrates, represent a large family of trematodes with over 400 species. Their life cycle usually include two (snails and arthropods) or three (snails, arthropods and amphibians/reptiles) intermediate hosts. The genera and species present with this family are distinguished from one another by the location and size of testes, ovary, vitellarium, suckers, caecum, genital pore, excretory vesicle and body form.

Lyperosomum longicauda of the genus *Lyperosomum* is the common fluke of carrion crows (*Corvus corone*) in Europe. They exhibit a relatively elongate body with a thin cuticle lacking spines. The pharynx is large, whilst the oesophagus is short. Two caeca are present. The testes are close to round, orientated slightly oblique to one another and are found close the ventral sucker, which is larger than the oral sucker. The ovary is present caudal to the testes, whilst the genital pore is located just caudal to the pharynx. The vitellaria are relatively short and the uterus contains dark brown, thick-shelled eggs approx. 32 x 21 µm in size.[7] Twenty species are present in the *Lyperosomum* genus, which may be found in intestines and livers of birds and mammals; the known life cycles include two hosts, namely terrestrial snails and insects.

Whilst most commonly trematodes in birds are present in the intestines, *Lyperosomum longicauda* is only found in the liver. This parasite also has been described in other birds including the song thrush (*Turdus philomelos*)

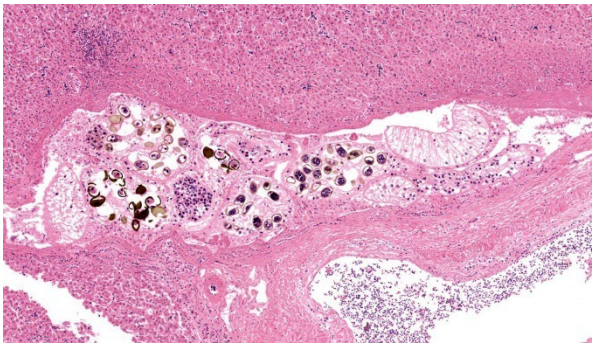

Figure 3-7. Liver, crow: A bile duct contains a tangential section of an elongate adult trematode with multiple oral suckers, a thin tegument, a parenchymatous body cavity and numerous cross sections of a uteus contain operculated eggs with miracidia. (HE,135X)

and the northern bald ibis (*Geronticus eremita*).[7] Canadian common crows (*Corvus brachyrhynchos*) exhibit *Conspicuum macroorchis* of the genus *Conspicuum*, and *Brachylecithum stunkardi* of the genus *Brachylecithum*/ *Lyperosomum* within gall bladders and bile ducts.[1]

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JPC Diagnoses:
1. Liver: Hepatitis, granulomatous, chronic, multifocal and random, severe, with faintly staining bacilli.
2. Liver: Adult trematodes.
3. Liver, large-caliber veins: Phlebitis, lymphoplasmacytic, chronic, multifocal, moderate.
4. Liver: Hepatitis, lymphoplasmacytic, chronic, multifocal and portal, mild.

JPC Comment:
This case was an excellent demonstration of mycobacteriosis in a bird. Many thanks to this contributor for providing this classic case. With careful scrutiny, the more recent granulomas contained numerous, faintly staining bacilli on H&E stain, which is typical of *Mycobacterium* spp. In many cases of paucibacillary mycobacteriosis, the bacilli can be seen at the center of "younger" granulomas, but they are characteristically poorly staining. This appearance can allow the pathologist to make a presumptive diagnosis of mycobacteriosis prior to confirmation with acid fast stains.

Paucibacillary mycobacteriosis is what is expected in most cases of *M. avium ssp avium, M. tuberculosis*, etc. due to these bacteria inciting a Th1 response with resultant strong T-cell immunity.[6] The stimulated Th1 cells produce cytokines such as IL-1 and interferon to classically activate macrophages (M1 response). This enables the formation of granulomas to wall off the invaders and creates "tuberculoid" inflammation.[6] Because the bacilli are predominantly seen only within the center of young granulomas rather than diffusely spread about, this form of mycobacteriosis is referred to as "paucibacillary." On the other than, multibacillary mycobacteriosis is characterized by diffuse, disorganized inflammation with numerous bacteria that are poorly contained.[6] This version of mycobacteriosis results from Th2 activation and subsequent production of IL-4 and IL-13. These interleukins result in alternative (M2) macrophage activation, resulting in "lepromatous" inflammation. *M. avium ssp pseudotuberculosis* (Johne's disease) and *M. leprae* (leprosy) are the classic examples of this type of mycobacteriosis, in which there are usually numerous acid-fast bacilli present throughout the inflamed tissue.[6]

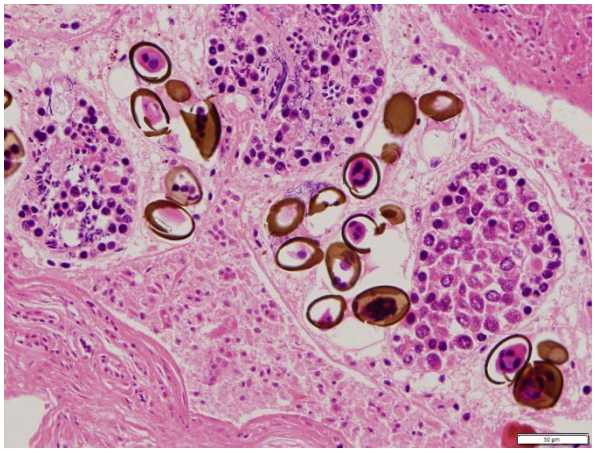

Figure 3-8. Liver, crow: High magnification of trematode eggs containing miracidia. (HE, 400X)(Photo courtesy of: Division of Pathology, Public Health and Disease Investigation, College of Medical, Veterinary & Life Sciences, University of Glasgow, http://www.gla.ac.uk/schools/vet/)

Conference participants discussed at length whether the trematodes were present within a bile duct or within a portal vein. Support for portal vein includes: the shape of the luminal structure was similar to portal veins in the surrounding liver, the lumen contained erythrocytes (along with trematodes), the animal had substantial phlebitis, and definitive epithelial lining was not identified. Phlebitis was particularly pronounced in the large portal vein profile next to the trematode. Phlebitis is an uncommon lesion of mycobacteriosis but can be seen in several other conditions in birds, including parasitism, aspergillosis, viral infection (e.g., WNV, HPAI, Paramyxovirus), sepsis, and (less commonly) immune mediated disease. Lead toxicity may also cause vasculitis, but it is typically fibrinoid vasculitis (unlikely the lymphocytic vasculitis seen in this case).[12]

Ultimately, participants were unable to definitively determine whether the trematodes were intravascular or within a bile duct. Immunohistochemistry for pancytokeratin (AE1/AE3) was attempted to look for an epithelial lining to rule in/out a bile duct as the luminal structure. Unfortunately, IHC was non-contributory (internal controls were negative), likely due to autolysis in the specimen. Dr. Chris Gardiner was consulted on this case regarding the identity of the trematodes. Dr. Gardiner confirmed that this is trematode, specifically a hermaphroditic trematode with operculated eggs. The fact that this trematode is hermaphroditic at least ruled out schistosomes, as that particular genus of trematodes are gonochoric and do not have operculated eggs. Per Dr. Gardiner, further determination of the trematode genus and species would require removal of whole specimens at necropsy and submission to a parasitologist.

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CASE IV:

Signalment:
Four-month-old, male, Oriental magpie robin (*Copsychus saularis)*

History:
The carcass was submitted from a regional zoo to perform a necropsy of the animal. Carers and veterinarians reported no history of clinical disease. It was found dead inside the house.

Gross Pathology:
The bird was in poor body condition, as assessed by a moderate to severe reduction of the bulk of muscles throughout the body, including both deep and superficial pectoral muscles (muscle atrophy). There were numerous pinpoint white foci in the liver and spleen (moderate, multifocal, acute necrotizing hepatitis and splenitis). Transversal cuts of the left humerus revealed multifocal to coalescing, irregularly shaped, well demarcated, depressed areas filled with a green soft material (severe heterophilic necrotizing osteomyelitis).

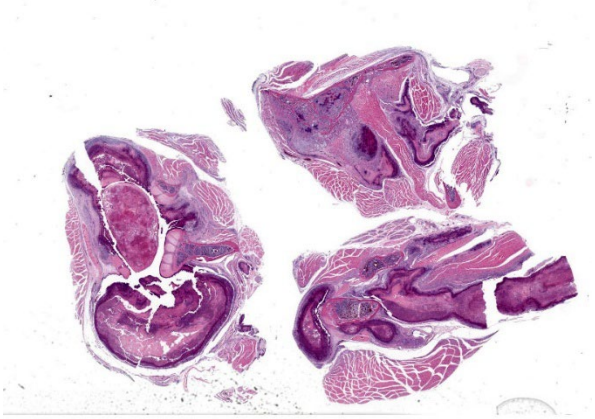

Figure 3-1. Humeroradial joint, Oriental magpie robin: Three sections of the humeroradial jointand associated soft tissues are submitted for examination. There is profound necrosis and inflammation affecting all tissues within and surrounding the joint. (HE, 12X)

Laboratory Results:
The bacteriological results revealed light growths of *Yersinia pseudotuberculosis* in the lungs and liver by using matrix-assisted laser desorption ionization time-of-flight (MALDI-TOF) mass spectrometry

Microscopic Description:
Bones and adjacent soft tissue. Multifocally throughout bone, synovial joint and muscle tissue and affecting approximately 60% of the tissue examined, there are severe, multifocal to coalescing inflammatory infiltrates. The inflammation is chiefly composed of degenerate heterophils, with fewer lymphocytes and plasma cells, embedded in eosinophilic material showing no cellular outlines (lytic necrosis) with nuclear fragmentation and loss (karyorrhexis and karyolysis); embedded within the necro-inflammatory process are common , up 100 micrometers in diameter, cocco-bacillary bacterial colonies; in some foci, the necro-inflammatory foci are bordered by macrophages forming multinucleated giant cells containing a 15-50 nuclei with a horse-shoe arrangement with occasional foamy cytoplasm. The inflamed bones show reduction of medullary bony trabeculae (osteolysis), with

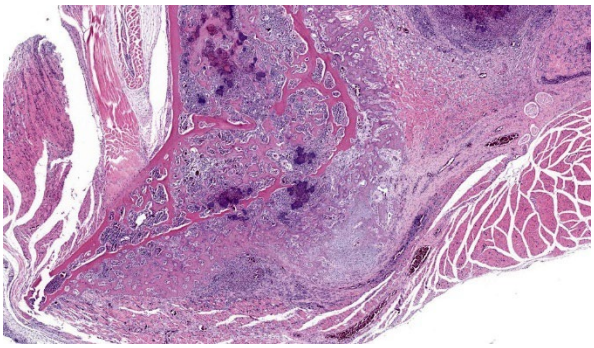

Figure 4-2. Humeroradial joint, Oriental magpie robin: There are multiple foci of necrosis within the medullary cavity of one of the bones, multifocal lysis of lamellar bone, and profound periosteal new bone formation. (HE, 55X)

multifocal scalloping and thinning of the remaining ones with an increased basophilic hue while other show empty lacunae and diffuse eosinophilia (osteonecrosis); the periosteum instead shows a common exophytic proliferation of the cortical bone with cartilage and woven bone formation (periosteal reaction).

The inflammatory infiltrate is also observed infiltrating and filling multifocal synovial joint lumina and capsules (arthro-synovitis), with a layer of fibrin embedding heterophils and covering multifocally the articular cartilage, which shows increase eosinophilia (cartilage degeneration).

The adjacent rhabdomyocytes exhibit multifocal reduction in size and diameter, with angular edges (muscular atrophy) with the closest one to the inflammation exhibiting an amphophilic sarcoplasm and linear arranged central nuclei (myotubes) and rare areas of endomysial fibrosis.

Contributor's Morphologic Diagnoses:

1. Bone: multifocal to coalescing, severe, subacute, heterophilic and necrotizing osteomyelitis, with osteolysis, periosteal reaction, osteonecrosis and with intralesional bacterial colonies.
2. Synovial joints: diffuse, severe, subacute, fibrinous and heterophilic arthro-synovitis with intralesional bacterial colonies.
3. Skeletal muscle: Severe, multifocal, subacute, heterophilic and necrotizing myositis with intralesional bacterial colonies.

Contributor's Comment:

Yersiniosis has been frequently reported in more than a hundred different species worldwide, including avian, mammals and reptiles, and it may act as a zoonotic pathogen.[3] Yersiniosis can be caused by *Yersinia pseudotuberculosis* and *Y.enterocolitica*, although the latter is more frequently involved in humans and primates cases. *Y. pseudotuberculosis* can be commonly isolated from wild animals and birds, but there are very few reports of cases of avian osteomyelitis sustained by *Y. pseudotuberculosis*.[4] A recent outbreak in a slaughter turkey flock was reported in Finland, while other older outbreaks were documented in the UK and California, still in turkeys.[1,6,8] In mammals, a Y. pseudotuberculosis osteomyelitis case was reported in a ring-tailed lemur in UK.[7]

We decided to submit this case due to the paucity of *Y. pseudotuberculosis* induced osteomyelitis in birds reported in literature. Such

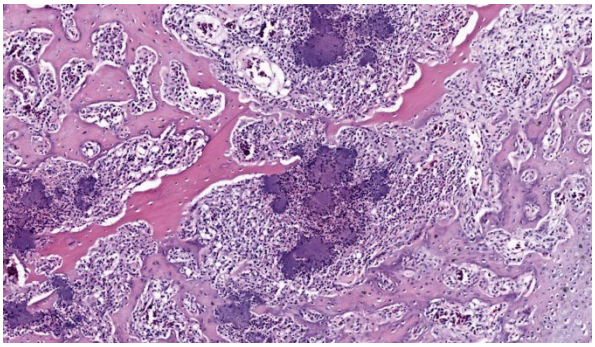

Figure 4-3. Long bone, humeroradial joint, Oriental magpie robin: Areas of bone necrosis contain large numbers of necrotic heterophils, abundant cellular debris, and large colonies of 1-2um coccobacilli. (HE, 254X)

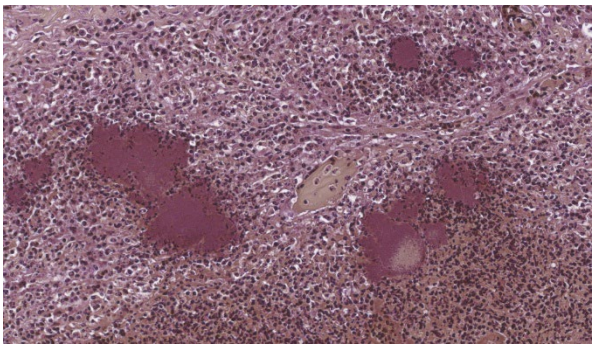

Figure 4-4. Long bone, humeroradial joint, Oriental magpie robin: Coccobacilli are gram-negative. (Brown-Hopps, 400X)

case is, to the best of our knowledge, the first pathological description of a *Y. pseudotuberculosis* induced osteomyelitis in an Oriental Magpie Robin. The present case was characterized by extensive inflammation with large bacterial colonies also spotted in other organs (spleen, liver, lung and large intestine), and therefore suggesting a systemic infection.

Yersinia pseudotuberculosis is a ubiquitous Gram-negative anaerobic coccobacillus bacterium from the Enterobacter family. The most common route of infection is oro-fecal thought contaminated food or water, although the epidemiology of the disease is considered multifactorial and highly complex. Therefore, the severity of the clinical signs and lesions would be highly variable and dependent on numerous environmental (e.g. management, temperature, etc.) and host (e.g. age, stress, parasitism and / or concomitant infections, etc.), conditions. No predisposing conditions to such infection were detected here.

Although the bacteriological culture isolated Yersinia pseudotuberculosis, other differential diagnosis for large colonies of bacteria would be: *Actinomyces sp., Actinobacillus sp. Arcanobacter sp., Corynebacterium sp., Staphylococcus sp. or Streptococcus sp.* (YACS acronym).

Contributing Institution:
University of Liverpool, Institute of Infection, Veterinary and Ecological sciences, Department of Veterinary anatomy, Physiology and Pathology.

JPC Diagnoses:
1. Wing: Osteomyelitis, tenosynovitis, myositis, and cellulitis, necrotizing and heterophilic, chronic, multifocal, severe, with pannus and large colonies of bacteria.
2. Skeletal muscle, adipose tissue: Atrophy, chronic, diffuse, severe.

JPC Comment:
The avian skeletal system has several unique functional adaptations. Birds have three types of bone: pneumatic, hematopoietic, and medullary.[5] Pneumatic bones have air sacs in the endosteum in place of bone marrow. The number and distribution of pneumatic bones varies by species, but most birds have pneumatic humeri, femurs, sternums, and skulls.[5] The remainder of the bones are hematopoietic and have bone marrow. Medullary bone is present in female birds prior to laying eggs when they deposit calcium on the endosteum, with deposits filling the marrow cavity of long bones.[5] Additionally, many bones are fused

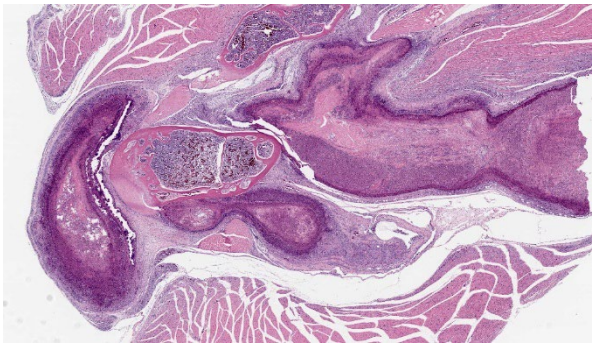

Figure 4-5. Joint space, humeroradial joint, Oriental magpie robin: The joint space is expanded and filled an inflammatory exudate, cellular debris, and bacterial colonies , which extends into the joint capsule. Bacterial colonies are present within the joint capsule (top). (HE, 34X).

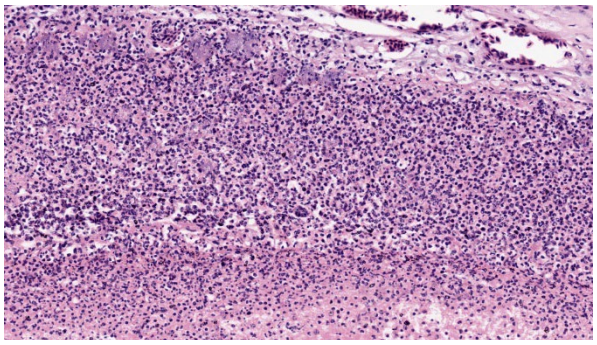

Figure 4-6. Skeletal muscle, Oriental magpie robin: Granulation tissue with inflammation and bacterial colonies extends into the adjacent, moderately atrophic skeletal muscle (HE, 380X)

(e.g., tibiotarsus, synsacrum, etc.). The slide in this case had a pneumatic bone that articulated with two hematopoietic long bones surrounded by abundant skeletal muscle; this consistent with wing where the humerus (pneumatic bone) articulates with the radius and ulna (hematopoietic bones). The leg (at the articular of the femur and tibiotarsus) was also considered, but the amount of skeletal muscle was deemed to be excessive for this location. Foci of inflammation and necrosis were present in both pneumatic and hematopoietic bone, suggestive of systemic bacterial sepsis.

Many classic features of bone and joint inflammation and healing were demonstrated in this case, including osteonecrosis and periosteal new bone formation (characterized by lattices of woven bone oriented perpendicularly to the bone's long axis). In addition to the excellent description provided by the contributor, conference participants also noted decreased basophilia of the articular cartilage. This is a common finding of arthritis and correlates to loss of proteoglycans in the chondroid matrix.[2] Additionally, one of the joints was overlain by a thin layer of fibrovascular tissue, consistent with pannus. Pannus is caused by proliferation of stromal cells in chronic arthritis and was nicely highlighted with a Masson's trichrome stain.[2]

Conference participants also discussed the cause of the severe skeletal muscle atrophy in this case. Skeletal muscle atrophy can be caused by disuse atrophy, which was presumably present in this animal. However, negative energy balance can also lead to primary skeletal muscle atrophy due to birds' high metabolic demands. Examination of body condition score in birds often involves palpation of the breast muscle, as muscling has direct cor-

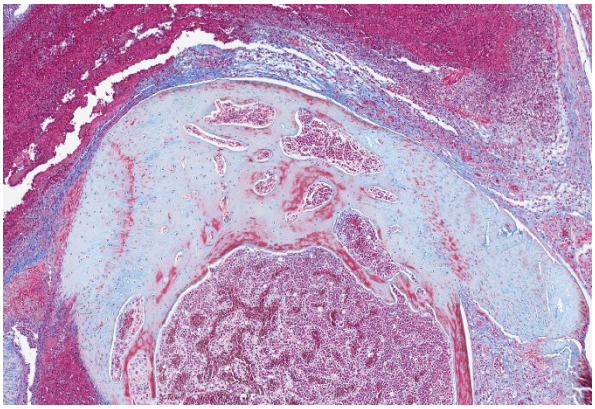

Figure 4-7. Joint space, humeroradial joint, Oriental magpie robin: The articular cartilage is covered by a thin band of fibrovascular tissue, consistent with pannus. (Masson's trichrome stain, 64X)

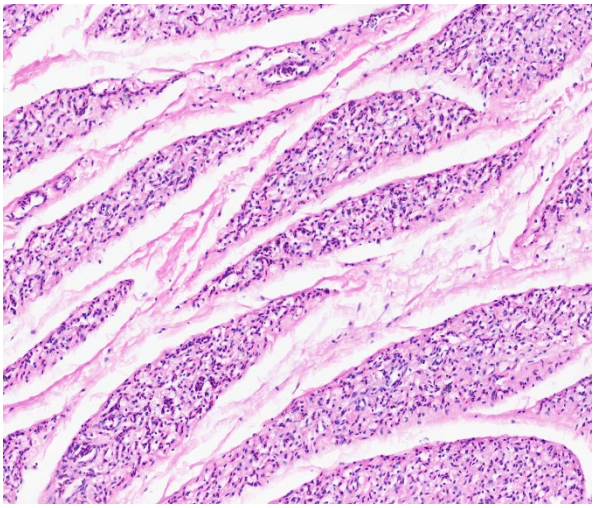

Figure 4-8. Periarticular adipose tissue, humeroradial joint, Oriental magpie robin: Markedly atrophic adipose tissue. (HE, 380X)

relation to overall nutritional status. This animal had severe adipose atrophy as well, indicative of negative energy balance, which likely also contributed to muscular atrophy.

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