



WEDNESDAY SLIDE CONFERENCE 2011-2012

Conference 21

11 April 2012

CASE I: 12874-04 (JPC 3133955).

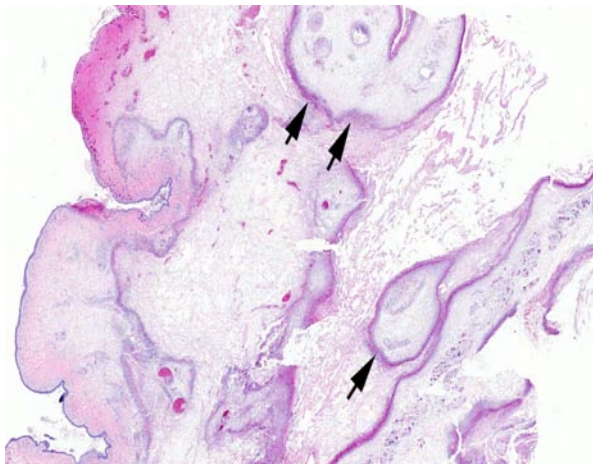
Signalment: 13-week-old male white domestic turkeys (*Melagris gallopavo*).

History: Several animals were submitted after lesions of the skin on the head were observed. 100 birds were dead and 300 displayed signs out of 13,000 birds.

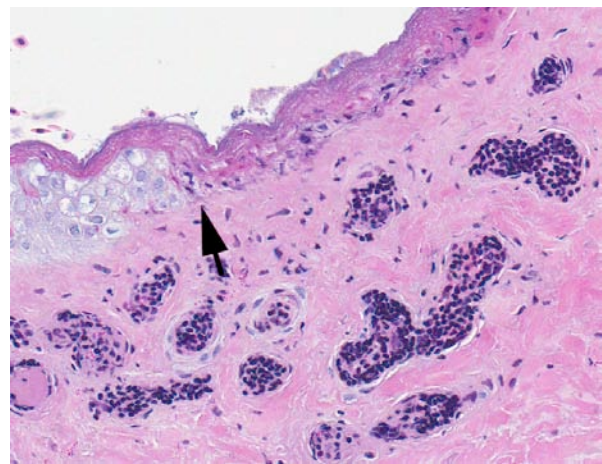
Gross Pathology: Four of five turkeys had necrosis and edema of areas of skin on the head, and/or snood, and/or wattles.

Laboratory Results: *Pasteurella multocida* was isolated from skin lesions.

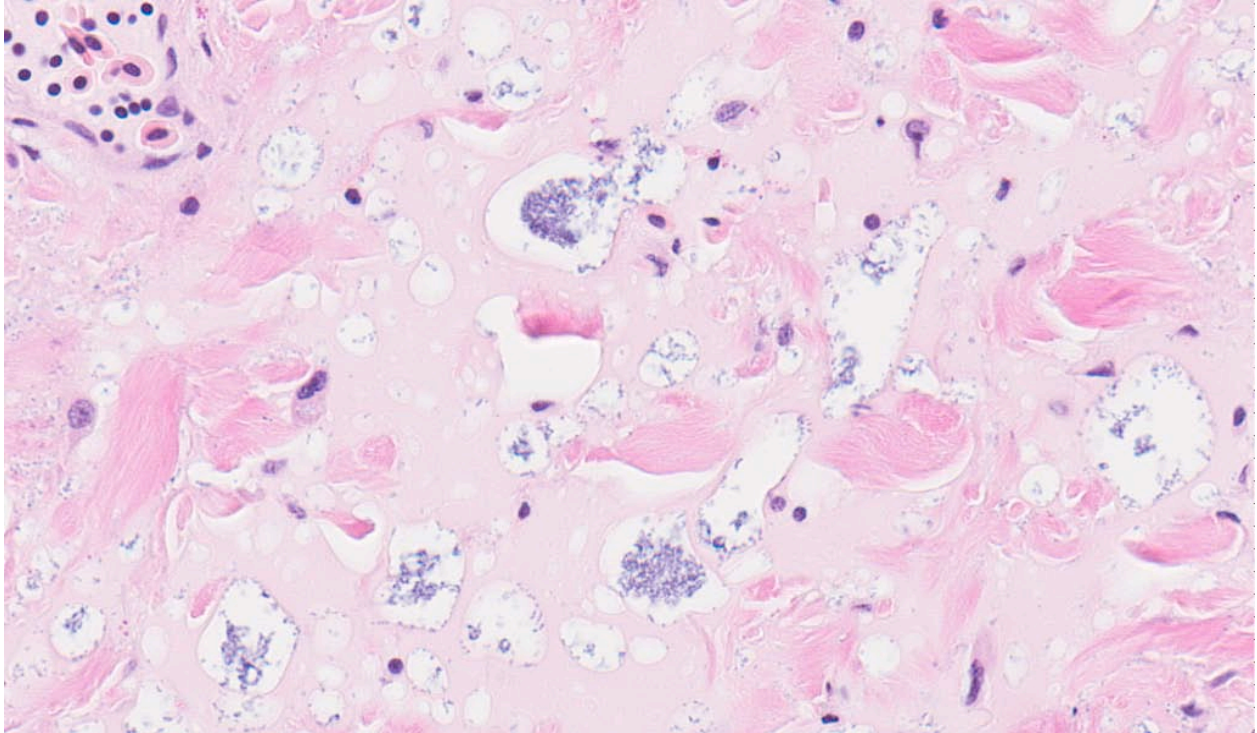
Contributor's Histopathologic Description: Specimens of skin, largely without feather follicles, came from the wattles of several birds. The microscopic lesions are a mixture of infarction and inflammation. Several areas of acute necrosis of the epidermis and dermis are mixed with areas of severe heterophilic infiltration, edema, fibrin, epidermal necrosis, ulceration and numerous bacterial colonies. A small region of early fibroblast proliferation,



1-1. Non-feathered skin, turkey: There are multiple well-demarcated areas of infarction within the dermis and subcutis, outlined by a dense band of inflammatory cells and cellular debris. Intervening areas are markedly expanded by edema. (HE 9X)



1-2. Non-feathered skin, turkey: Areas of epidermal necrosis at the edges of infarcts are very well demarcated (arrow). (400X)



1-3. Non-feathered skin, turkey: The deep dermis contains numerous colonies of 2-4 μm rod-shaped bacilli. (HE 400X)

capillary proliferation and predominantly histiocytic inflammation occurs in some sections. No gross or histologic lesions were observed in internal organs, and affected skin was the only site of *Pasteurella* isolation and visceral tissues was unaffected.

Several ascarids were found in one cross section of jejunum (not shown).

Contributor's Morphologic Diagnosis: Severe necrotizing dermatofasciitis, with focal infarcts, and bacterial colonies.

Contributor's Comment: *Pasteurella multocida* is the etiology of severe necrotizing dermatitis in turkeys that can affect the skin of the carcass as well as of the face.¹ Outbreaks can be recurrent and cutaneous lesions may predominate over systemic infection in affected flocks.² Lesions of internal organs are uncommon although septicemia has resulted from experimental infection via scarification.

It is suspected that dermal manifestations such as this are a result of generalized Schwartzman reaction, and occur in the context of subclinical or previous infection. In turkey poults sensitized to *P. multocida* LPS, inflammation was observed in turkeys challenged with epidermal LPS, whereas inflammation and vasculitis resulted when intradermal and intravenous injections were given.³

Pasteurella multocida produces a spectrum of disease in turkeys. Lesions may be minimal in the peracute cases. Consolidation of the lungs, often extensive, is the most common manifestation of acute infection, with fibrinous exudates in the coelomic cavity. *Erysipelas* and colibacillosis are the major differential etiologies. Because surviving birds become carriers, depopulation may be necessary to stop recurrence.⁴

Serotyping of the *Pasteurella* isolate, conducted at NVSL, revealed that it was different from the organism previously used for vaccines and also differed from previous pathogenic isolates from this particular facility.

JPC Diagnosis: Unfeathered skin: Dermatitis and cellulitis, necrotizing and heterophilic, diffuse, severe, with numerous intracellular and extracellular bacilli and multifocal skeletal muscle necrosis and mineralization.

Conference Comment: In the moderator's experience, this is an unusual presentation of fowl cholera in a turkey, and is more typical of chronic fowl cholera in male broiler chickens, which develop caseous necrosis of the subcutaneous tissues of the wattle. Female broilers or breeders typically present with acute egg yolk peritonitis, and chronic cases in female turkeys and chickens produce caseous to fibrinonecrotic salpingitis. Fowl cholera in turkeys usually presents with a triad of lesions that include

fibrinous pericarditis, splenic necrosis, and fibrinonecrotic pneumonia and air sacculitis, and occasionally multifocal necrotizing hepatitis or fibrinocaseous arthritis. Infections are generally acquired from oculonasal secretions which contaminate drinkers, and the reservoir in poultry is pigs, cats, and raccoons. The differential diagnosis includes *Erysipelothrix rhusiopathiae*, which is the best option and causes cutaneous hemorrhage and infarction with thrombosis and cyanosis, “paintbrush” hemorrhages, systemic hemorrhage and necrosis, and splenomegaly; as well as colibacillosis caused by *E. coli*, or frostbite with secondary bacterial infection.⁴

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References:

1. Glass SE, Panigrahy B. Dermal necrosis caused by *Pasteurella multocida* infection in turkeys. *Avian Diseases*. 1990;34:491-494.
2. Frame DD, Clark FD, Smart RA. Recurrent outbreaks of a cutaneous form of *Pasteurella multocida* infection in turkeys. *Avian Diseases*. 1994;38:390-392.
3. Mendes S, Carmichael KP, Nunnally JC, et al. Lesions resulting from attempted Shwartzman reaction in turkey poults inoculated with *Pasteurella multocida* lipopolysaccharide. *Avian Diseases*. 1994;38:790-796.
4. Charlton BR, et al. *Avian Disease Manual*. 6th edition. American Association of Avian Pathologists. 2006:77, 81, 84-7.

CASE II: N09-168 (JPC 3166501).

Signalment: Fourteen male and female sexually immature five to six-week-old bobwhite quail (*Colinus virginianus*).

History: These birds were a representative sampling of a flock with a reported history of spiked mortality, depression and respiratory distress. On physical exam quails had ruffled feathers, white watery diarrhea, and a few were non-ambulatory and in lateral recumbency.

Gross Pathology: All birds were in poor body condition as determined by prominent keel bones and inadequate musculature. Eight of the fourteen birds were alive. All live birds and five of six dead birds were necropsied. Four of the 13 birds had cloudy air sacs interpreted as mild airsacculitis. The lumen of two ceca and one ileum had a whitish-yellow caseous cores. The mucosal and serosal surfaces of the intestinal tracts of five birds were dark red (congestion and hemorrhage). There were multiple depressed areas (ulcerations) in the duodenum of two birds and the ileum of a third bird. Mucosal scrapings from the intestines of multiple birds revealed numerous thin-walled, circular protozoa (*Eimeria* spp.) within enterocytes.

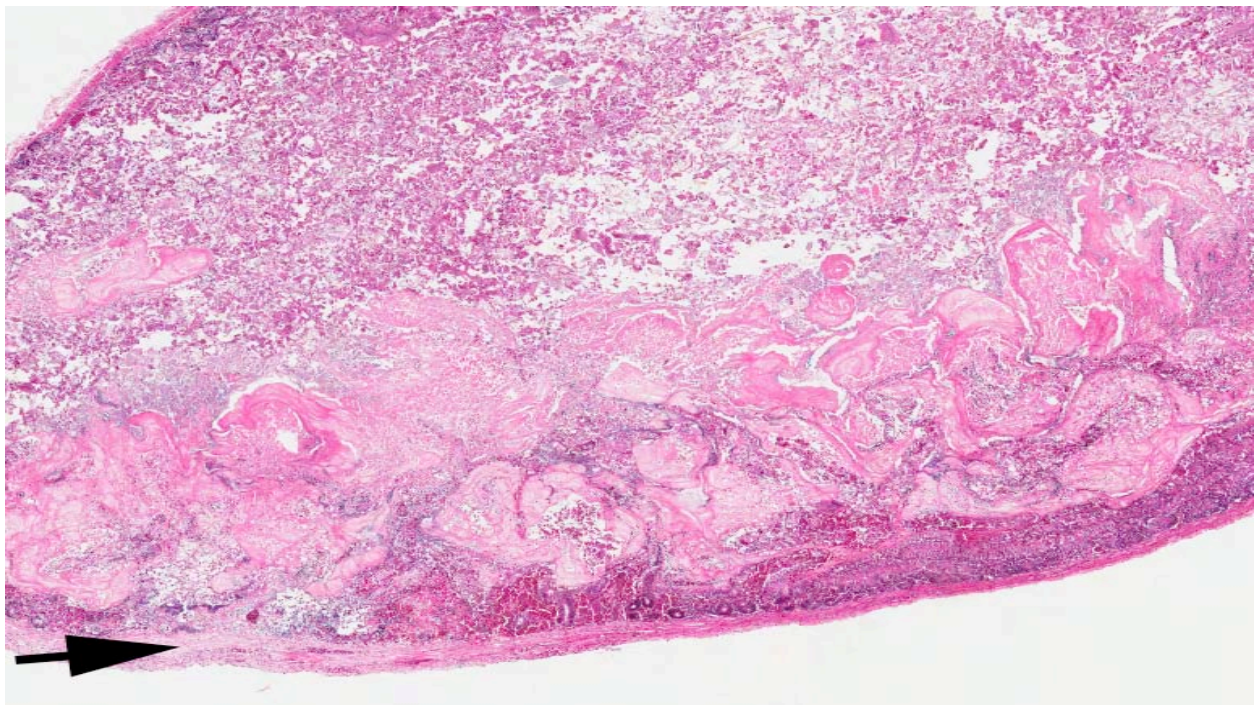
Contributor's Histopathologic Description: In some sections, the small intestinal mucosa is replaced by large, well-demarcated bands of abundant eosinophilic cellular debris with loss of differential

staining and cells with pyknotic nuclei and eosinophilic, vacuolated cytoplasm. In some sections, these necrotic bands extend into the submucosa and muscularis. The mucosa and submucosa in less severely affected sections contain numerous plasma cells, moderate lymphocytes, and few extravasated erythrocytes. The muscularis is expanded by increased clear spacing and ectatic lymphatics (edema), and blood vessels are engorged with blood. Moderate to numerous rod-shaped basophilic structures (bacteria) are randomly distributed throughout the mucosa. Randomly distributed throughout intestinal villi, affecting up to 30% of enterocytes, there are numerous coccidia at variable stages of development (sporozoites, schizonts containing merozoites, and gamonts).

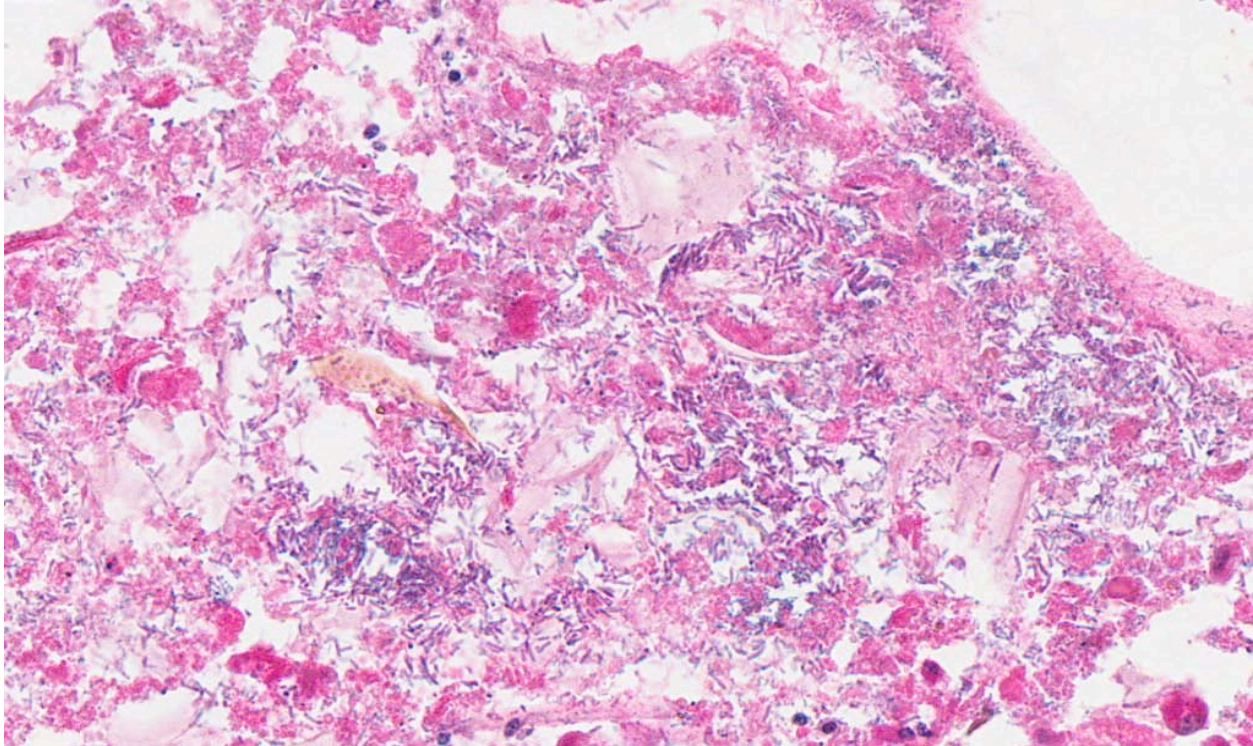
Contributor's Morphologic Diagnosis: Intestine: Enteritis, necrotizing, diffuse, severe, subacute with intralésional coccidia.

Intestine: Enteritis, necrotizing and ulcerative, diffuse, severe, acute with intralésional bacteria.

Contributor's Comment: *Clostridium colinum*, first named and described in 1974, has been established as the causative agent of ulcerative enteritis.^{1,2} *C. colinum* is an anaerobic, gram-positive, spore-forming, fastidious, bacterial rod that affects multiple gallinaceous species, particularly captive game birds, young poults, and young chickens.³ *Colinus virginianus*, the bobwhite quail, is the most susceptible



2-1. Small intestine, bobwhite quail: There is multifocal to coalescing areas of mucosal necrosis, which is focally transmural at one point (arrow). (HE 15X)



2-2. Small intestine, bobwhite quail: Robust, rod-shaped, 1x4 μm bacilli (*C. colinum*) abound within necrotic mucosal tissue. (400X)

species while *Coturnix coturnix japonica*, the Japanese quail, is resistant.^{1,3} *C. colinum* produces spores that persist in the environment for years and is transmitted from the droppings of acutely ill or recovered carrier birds.^{2,3} The typical clinical presentation can range from classic signs of depression (ruffled feathers, listlessness, drooped wings) to acute death. In quail, mortality can reach 100% in days.³

Fecal smears and histopathologic tissue sections were positive for abundant protozoal organisms consistent with coccidia of the genus *Eimeria*. Eimeriosis is thought to be a major predisposing factor for the development of ulcerative enteritis.³ Often it is not possible to determine whether mortality can be attributed to the Eimeriosis or the Clostridial infection.³ Attempts to culture the *C. colinum* in this case were not successful but the presence of gram positive bacterial rods on histopathologic evaluation along with the classic deep and coalescing intestinal ulcers are sufficient for a presumptive diagnosis of ulcerative enteritis.

In some southern states, quail hunting is a \$30 million a year or greater industry. As the industry continues to grow in popularity, increasing numbers of small scale brood-rearing/hunting operations have appeared.⁴ With the rise of these operations, increased incidences of quail disease outbreaks can be expected. Ulcerative enteritis is a quail disease that is common and

potentially devastating to new and unprepared quail operations.

JPC Diagnosis: Small intestine: Enteritis, fibrinonecrotizing, transmural, multifocal to coalescing, severe, with numerous robust bacilli and rare coccidia.

Conference Comment: Ulcerative enteritis, or “quail disease”, often causes up to 100% mortality in quail and usually occurs in young birds. Additional histologic lesions include centrilobular or diffuse pinpoint coagulative hepatic necrosis with abundant bacteria and variable splenic necrosis. The intestinal necrosis is often severe enough to penetrate the intestine and cause peritonitis.³ The differential diagnosis for ulcerative enteritis includes coccidiosis, which is often seen with quail disease but does not cause focal hepatic necrosis or splenomegaly; *Histomonas meleagridis*, a flagellated amoeboid protozoan which produces similar caseous cecal cores and targetoid hepatic necrosis and can look grossly similar; and *Heterakis gallinarum*, a cecal worm which also produces cecal and hepatic necrosis.

There was some slide variation, and some sections were of small intestine with transmural, well-demarcated coagulative necrosis bordered by bacilli, which demonstrated the histologic effects of the diffusion of clostridial toxin into tissue. Other sections

included pancreas which exhibited variable vacuolation of the exocrine epithelial cells, which was interpreted as autolysis by conference participants. Most conference participants found it difficult to identify the coccidia, and discussed the role of coccidians, as well as ascarid migration, hemorrhagic enteritis in turkeys caused by avian adenovirus type 2, and severe salmonellosis, as a predisposing factor for necrotic enteritis of chickens and turkeys caused by *Clostridium perfringens* type A or C, which does not affect quail. Necrotic enteritis mostly affects broiler chickens, and grossly presents with necrotic intestinal mucosa with a pseudomembrane. In the moderator's experience, eimeriosis is not necessary as a predisposing factor for ulcerative enteritis.³

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References:

1. Berkhoff H. *Clostridium colinum* sp. nov., nom. rev., the Causative Agent of Ulcerative Enteritis (Quail Disease) in Quail, Chickens, and Pheasants. *International Journal of Systematic Bacteriology*. 1985;35 (2)::155-159.
2. Brown J, Dawe D. Antibiotic Treatment of Ulcerative Enteritis of Bobwhite Quail. *Journal of Wildlife Diseases*. 1970; Vol 6. January..
3. Charlton BR, et al. *Avian Disease Manual*. 6th edition. American Association of Avian Pathologists. 2006:102-3, 120-121.
4. Flanders A, McKissick J. Economic Impacts of Alabama Quail Hunting. 2008.
5. The University of Georgia. Center for Agribusiness and Economic Development College of Agricultural and Environmental Sciences. Center Report: CR-08-21.

CASE III: AFIP 2010 #1 (JPC 3167238).

Signalment: Adult male chicken, *Gallus domesticus*.

History: This chicken was a member of a backyard flock. The chickens of this flock demonstrated difficulty breathing, coughing with the expectoration of plugs of mucus, swelling of the tissues of the head, and chalky droppings. There was mortality.

Gross Pathology: The bird had mild pectoral muscle atrophy. Petechiae were present along the length of the tracheal mucosa, which was covered by watery mucus containing yellow debris.

Laboratory Results: Immunohistochemistry for Infectious Laryngotracheitis Virus antigen: positive in trachea and air sac.

Contributor's Histopathologic Description:

Trachea: The luminal surface of the mucosa is covered by a thick pseudomembrane comprised of fibrin and protein which are admixed with large numbers of heterophils with erythrocytes, macrophages, lymphocytes, plasma cells, scattered necrotic epithelial cells and occasional syncytial cells with eosinophilic intranuclear inclusion bodies. There is diffuse and marked erosion of the respiratory epithelium with focal ulcerations, and the mucosa is occasionally lined by attenuated epithelial cells, few of which form syncytia and have amphophilic to eosinophilic intranuclear inclusion bodies and marginated chromatin. The lamina propria and, to a lesser extent, the submucosa are diffusely infiltrated by moderate to large numbers of macrophages, lymphocytes plasma cells and heterophils. There is

congestion of blood vessels in the lamina propria and submucosa.

Cranial Air Sac (not present on every slide): The air sac is folded and its connective tissue stroma is markedly expanded by edema, fibrin, karyorrhectic debris, and an infiltrate of heterophils, histiocytes and lymphocytes, with congestion and foci of hemorrhage. Along the luminal surface is an exudate of degenerate heterophils, detached and necrotic epithelial cells, protein and fibrin, and rafts of syncytial cells. Syncytial cells have as many as seventeen nuclei, which frequently possess eosinophilic intranuclear inclusion bodies and marginated chromatin.

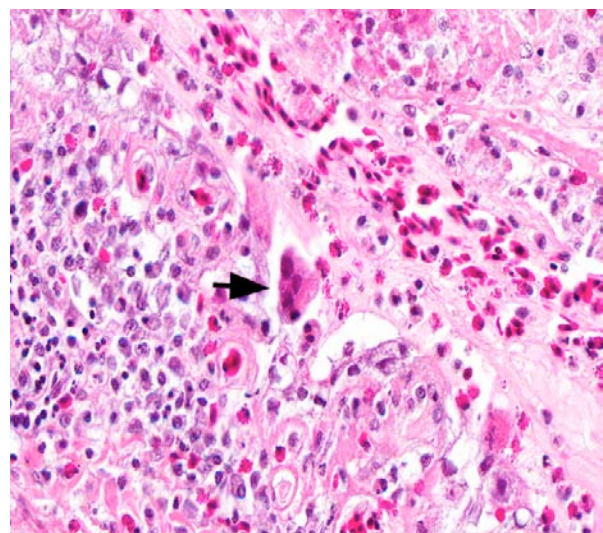
Contributor's Morphologic Diagnosis: Trachea. Marked, diffuse, fibrinonecrotizing and heterophilic, tracheitis with pseudomembrane formation, epithelial syncytia and intranuclear inclusion bodies, etiology consistent with Gallid herpesvirus-1.

Air Sac. Marked, diffuse, fibrinoheterophilic and exudative air sacculitis, with epithelial syncytia and intranuclear inclusion bodies, etiology consistent with Gallid herpesvirus-1.

Contributor's Comment: Gallid herpesvirus 1 (GaHV-1) is the sole member of the Iltovirus genus within the alphaherpesvirinae subfamily and the cause of avian laryngotracheitis, or "LT" as it is known in the poultry industry. The virus naturally infects chicken and pheasants, and it circulates widely in the field and worldwide. Young turkeys can be experimentally infected, producing mild clinical signs. The virus has a narrow tropism that is limited to the upper and lower respiratory epithelia as well as the conjunctival epithelium. GaHV-1 has also been detected in feather



3-1. Trachea, chicken: The tracheal mucosa is circumferentially necrotic and replaced by a pseudomembrane composed of fibrin, hemorrhage and inflammatory cells. (HE 8X)



3-2. Trachea, chicken: Remaining epithelial cells occasionally form syncytia which contain round, eosinophilic intranuclear inclusion bodies which peripheralize nuclear chromatin (arrow). (HE 400X)

shafts.² There is no evidence of a viremic phase, but it can be transported to the trigeminal ganglion. Like other alphaherpesviruses, GaHV-1 can remain latent and can be reactivated several months later, for instance, after relocation of chickens or the onset of laying.⁶ Vaccination of layer chickens (well before onset of production) is commonly practiced in areas of intensive poultry production if there is endemic disease. The epidemiology of the disease is complicated by the variable virulence of field strains, clinically inapparent carriers and circulation of vaccine strains.^{6,7} Strains that are closely related genetically to vaccine strains have been isolated from outbreaks and have produced severe respiratory signs in experimentally inoculated chickens.⁵ This phenomenon is a recognized problem for the poultry industry and has been termed "vaccinal laryngotracheitis".⁵

Although histopathology is not the most sensitive method available for recognizing the presence of the virus compared to molecular methods,⁶ infectious laryngotracheitis is diagnosable by histopathology, as the virus causes fusion and desquamation of epithelial cells (syncytial cells) with basophilic to eosinophilic intranuclear inclusions.

JPC Diagnosis: Trachea and air sac: Tracheitis and air sacculitis, fibrinonecrotic and heterophilic, circumferential, severe, with occasional epithelial syncytia and numerous intranuclear viral inclusion bodies.

Conference Comment: Gross lesions with Gallid herpesvirus 1 (GaHV-1) include severe laryngotracheitis with necrosis, hemorrhage, ulceration and occlusive pseudomembranous or caseous plugs in the trachea. In milder forms there is sinusitis, conjunctivitis, congestion of mucous membranes and seromucoid discharge. The lungs and air sacs are rarely affected, although syncytia and intranuclear viral inclusion bodies are readily apparent in the section of air sac in this case.^{1,4} The differential diagnosis for GaHV-1 includes:

- Newcastle disease, a Paramyxovirus that causes high mortality, although the surface epithelium is seldom lost
- Fowl pox, which has intracytoplasmic viral inclusion bodies (Bollinger bodies), no syncytial cells, epithelial proliferation, and similar caseous exudates which can plug the trachea
- Infectious bronchitis caused by a Coronavirus which presents with no viral inclusion bodies and readily affects the lower respiratory tract
- Avian influenza, an Orthomyxovirus with similar lesions and a lack of viral inclusion bodies
- *Mycoplasma gallisepticum* which causes severe air sacculitis, has no pseudomembranes and has a lack of extensive epithelial necrosis
- Infectious coryza caused by *Avibacterium paragallinarum* which causes facial edema and usually involves the lower respiratory tract and other organs
- *Trichomonas gallinae*, the cause of frounce in raptors and canker in columbids, which presents with caseous material in the trachea and esophagus
- Vitamin A deficiency which presents with hyperkeratotic lesions primarily in the mouth and tongue¹

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References:

1. Charlton BR, et al. Avian Disease Manual. 6th edition. American Association of Avian Pathologists. 2006:15, 43, 46, 52, 55, 90, 93, 154, 165.
2. Davidson I, Nagar S, Ribshstein I, et al. Detection of wild and vaccine-type avian infectious laryngotracheitis virus in clinical samples and feather shafts of commercial chickens. *Avian Dis.* 2009;53(4): 618-23.
3. Dufour-Zavala L. Epizootiology of infectious laryngotracheitis and presentation of an industry control program, *Avian Dis.*2008;52:1-7.
4. Guy JS, Garcia M. Laryngotracheitis. In: Saif YM, ed. *Diseases of Poultry*. Ames, IA: Blackwell Publishing; 2008:137-52.
5. Oldoni I, Rodriguez-Avila A, Riblet SM, et al. Pathogenicity and growth characteristics of selected infectious laryngotracheitis virus strains from the United States. *Avian Pathol.*2009;38:47-53.
6. Guy JS, Garcia M. Laryngotracheitis. In: *Diseases of Poultry*. 12th ed. Ames, IA: Blackwell Publishing Professional; 2008:137-144.
7. Kirpatrick NC, Mahmoudian A, Colson CA, et al. Relationship between mortality, clinical signs and tracheal pathology in infectious laryngotracheitis. *Avian Pathol.*2006;35:449-53.

CASE IV: 62555 (JPC 4002939).

Signalment: Adult male American Singer Canary (*Serinus canaria*), Avian.

History: An adult male American Singer Canary presented for scaly proliferation of unknown duration on both legs. The bird was singly housed since acquired on 5/7/2010, and was periodically treated with topical Scalax (0.03% pyrethrin and 0.3% piperonyl butoxide) following the manifestation of lesions. Given the severity and progression of the lesions despite treatment, the animal was euthanized.

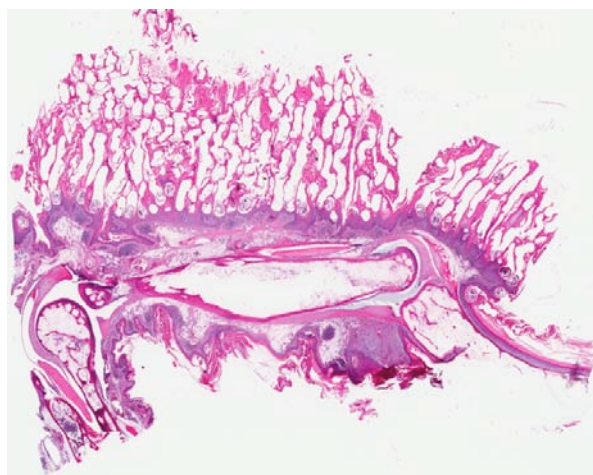
Gross Pathology: On external examination of the hind limbs, the skin was markedly thickened and contained many frond-like keratinized projections (hyperkeratosis). Dried blood was present on the left foot, and all toenails were severely overgrown. No fractures were palpated, and the remainder of the exam was unremarkable.

Contributor's Histopathologic Description: Legs and feet including skin, underlying bone and musculature: Diffusely there is marked orthokeratotic hyperkeratosis of the overlying stratum corneum, with proliferation of anastomosing parallel plates radiating from the epidermis and creating a complexity of keratinized tunnels. Within these anastomosing tunnels and adjacent to the epidermis, are multiple cross and oblique sections of parasitic arthropods that are approximately 100 to 250 µm in diameter. Characteristic features of these arthropods include a spiny, chitinous exoskeleton, highly chitinized segmented appendages, striated skeletal muscle, distinct reproductive and digestive tracts, yolk glands, and occasional observation of mouthparts. Scattered throughout the anastomosing tunnels are numerous,

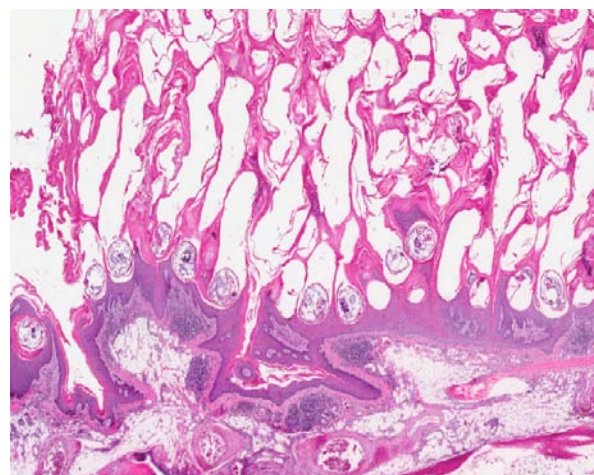
20-40 µm, spherical to ovoid developing ova. Rare, granular to amorphous, pigmented material is also present within these tunnels, depicted as mite excrement. Mild, multifocal acanthosis is noted within the underlying epidermis with prominent, rarely anastomosing rete pegs. The dermis is multifocally and markedly infiltrated with large numbers of inflammatory cells consisting predominantly of macrophages, and fewer lymphocytes and plasma cells. The inflammation multifocally extends into the underlying subcutis.

Contributor's Morphologic Diagnosis: Skin (feet and lower legs), dermatitis, proliferative, diffuse, chronic active, marked, with marked orthokeratotic hyperkeratosis, mild acanthosis, marked histiocytic lymphoplasmacytic dermatitis, intracorneal parasitic arthropods, and developing ova.

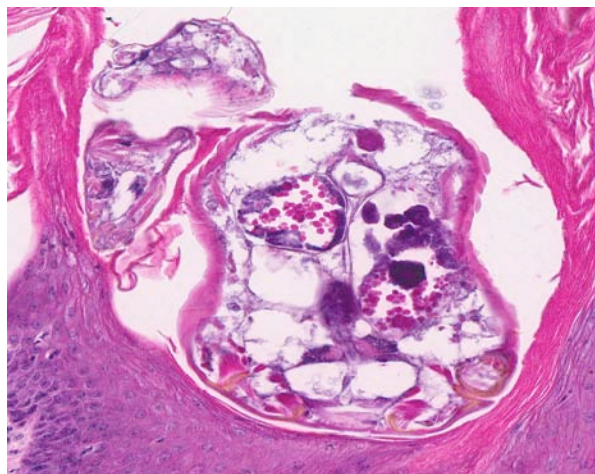
Contributor's Comment: *Knemidokoptes* is a common ectoparasitic burrowing mite that causes proliferative skin lesions in a variety of avian species. First described in budgerigars, over 15 *Knemidokoptes* spp. have now been reported³ in a variety of avian species including galliforms (chickens, turkeys, etc.), passerines (canaries, finches, robins, etc), psittacines (budgerigars, parrots, cockatiels, parakeets, etc), and anseriforms (ducks, geese). Additionally, infection has been identified in both free-ranging and captive bird species, with lower prevalence in wild birds.⁷ Mainka et al. reported that approximately 0.05-3% of wild passerines examined in Hong Kong were infected with *Knemidokoptes* mites.⁷ Despite these findings, additional studies have suggested that the prevalence of subclinical infection in both captive and wild birds may be largely undocumented.⁹ These findings suggest that *Knemidokoptes* spp. may serve as an opportunistic pathogen and manifest with stress or



4-1. Cross section of phalanx, canary: At subgross inspection, there is profound orthokeratotic hyperkeratosis (top) with moderate epidermal hyperplasia. Longitudinal section of the bones of the phalanx is seen at the bottom of the section. (HE 7X)



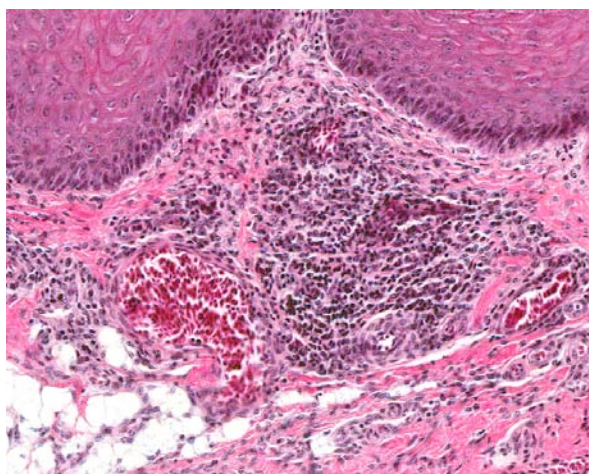
4-2. Cross section of phalanx, canary: Tortuous clear spaces within the keratin scale (mite tunnels) contain cross sections of adult mites. (HE 20X)



4-3. Cross section of phalanx, canary: Cross sections of adult mites within the keratin scale contain a brown serrated chitinous cuticle, jointed appendages with skeletal muscle, gonads, and a rudimentary nervous system. (HE 192X)

secondary to other predisposing conditions, however this has not been confirmed.

Knemidokoptes spp. are often referred to as scaly leg, tassel leg, or scaly face mites due to the mite's predilection sites and characteristic lesions. Infected birds generally present with slowly progressive, proliferative, crusting lesions on unfeathered regions of the body, particularly the legs, feet, cere, and periocular regions.^{1,3} Proliferative lesions can also be observed in feathered parts of the body⁸, but are less common and may be associated with chronicity or vary by mite species. Left untreated, severe infections may progress to thickened, frond-like keratinized protuberances that can reduce flexibility, cause lameness, and inability to perch.³ Additional gross lesions include necrosis of the digits¹², restriction secondary to leg bands, and overgrown nails. Hyperplastic lesions surrounding the cere may obstruct the nasal openings. Generally, pruritis is not observed, with the exception of *K. gallinae*, which causes an intense irritation, feather plucking, and self-mutilation.^{1,6} Diagnosis can be made by skin scraping¹⁰, tape preparations⁸, or histopathology. As the *Knemidokoptes* spp. life cycle takes place entirely on the host, transmission generally occurs as a result of direct or indirect contact from an infected to an uninfected host.^{3,6} Histopathologic lesions include severe, diffuse hyperkeratosis with the formation of tunnels in the stratum corneum and intracorneal mites.^{9,10} Characteristic features of the mites include a spiny, chitinous exoskeleton, striated epidermis, uninterrupted dorsal striations, segmented appendages, striated skeletal muscle, and distinct reproductive and digestive tracts.^{1,6} Female adults are generally 0.5 mm in length and contain short legs that lack pretarsi¹; males are smaller, contain longer legs that include pretarsi, thus making them morphologically similar to



4-4. Cross section of phalanx, canary: Dermal vessels are frequently surrounded by a cellular infiltrate composed predominantly of lymphocytes with fewer plasma cells and histiocytes. (HE 2X)

Sarcoptes spp.¹ Finally, underlying dermal inflammation consisting of histiocytes, lymphocytes, plasma cells, and heterophils is also commonly observed.⁹

Common species of *Knemidocoptes* that produce lesions in avian include *K. jamaicensis*, the scaly leg mite of passerines, particularly canaries and finches, but has been observed in other species.^{5,9} *K. mutans* is observed on the legs, combs and wattles of gallinaceous birds.⁶ *K. gallinae*, the depluming mite, causes severe irritation and self mutilation in chickens secondary to burrowing, resulting in decreased egg production and weight loss.⁶ Finally, *K. pilae* causes "scaly face mange" in psittacine birds.¹¹ Secondary lesions include beak overgrowth and deformity, with consequential dysphagia and grooming impairment.

JPC Diagnosis: Feathered skin: Orthokeratotic hyperkeratosis, diffuse, severe, with numerous intracorneal adult mites and eggs, epidermal hyperplasia, and marked lymphoplasmacytic perivascular dermatitis.

Conference Comment: This case provides an excellent example of mites in tissue section. The cuticle, mouth parts, striated muscles, thickened cuticle at their point of attachment, and eggs inside and outside of the female mites are well demonstrated. Although the diagnosis is readily evident histologically, the differential diagnosis for the gross finding of scaly hyperkeratotic encrustations on the featherless areas of the face and legs include dermatophytosis, the dry version of fowl pox, and cutaneous papilloma virus causing papillomas on the feet of European goldfinches.^{2,4}

Other mites which typically affect caged layers (but do not result in severe hyperkeratosis like *Knemidocoptes* spp.) include *Dermanyssus gallinae*, the red mite which feeds on host blood and causes anemia and mortality in heavy infestations and has also been reported to transmit fowl cholera and spirochetosis; and *Ornithonyssus sylviarum*, the northern fowl mite which can transmit fowl pox and Newcastle disease. Grossly, both mites appear as red or black specks on the feathers and skin.²

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References:

1. Bowman DD. Georgis' Parasitology for Veterinarians. 6th ed. Philadelphia, PA: W.B. Saunders Co.; 1999:61-64.
2. Charlton BR, et al. Avian Disease Manual. 6th edition. American Association of Avian Pathologists. 2006:43, 130, 141.
3. Dabert J, Mihalca AD, Sándor D. The first report of *Knemidocoptes intermedius* Fain et Macfarlane, 1967 (Acari: Astigmata) in naturally infected European birds. *Parasitol Res*, web: n. pag, 2011 Apr 19.
4. Dorrestein GM. Passerines. In: Ritchie BW, Harrison GJ, Harrison LR, eds. *Avian medicine: principles and application*. Lake Worth, FL: Wingers Pub; 2004:884.
5. Latta SC, O'Connor BM. Patterns of *Knemidokoptes jamaicensis* (Acari: Knemidokoptidae) infestations among eight new avian hosts in the Dominican Republic. *J Med Entomol*. 2001;38:437-440.
6. Loomis EC. External Parasites. In: Hofstad MS, Barnes JH, Calned BW, Reid WM, Yoder HW, eds. *Diseases of Poultry*. 8th ed. Ames, Iowa: Iowa State University Press; 1984:606-608.
7. Mainka SA, Melville DS, Galsworthy A, et al. *Knemidocoptes* sp. On wild passerines at the Mai Po Nature Reserve, Hong Kong. *J Wildl Dis*. 1994;30:254-6..
8. Miller DS, Taton-Allen GF, Campbell TW. *Knemidokoptes* in a Swainson's Hawk, *Buteo swainsoni*. *J Zoo Wildl Med*. 2004;35:400-402.
9. Pence DB, Cole RA, Brugger KE, et al. Epizootic Podoknemidokoptiasis in American Robins. *J Wildl Dis*. 1999;35:1-7.
10. Schmidt RE, Reavill DR, Phalen DN. Gastrointestinal system and pancreas. In: *Pathology of Pet and Aviary Birds*. Ames, Iowa: Iowa State Press; 2003:43.

11. Toparlak M, Tüzer E, Gargili A, Gülanber A. Therapy of *Knemidocoptic* mange in Budgerigars with spot-on application of moxidectin. *Tr. J. of Veterinary Animal Sciences*. 1999;23:173-174..
12. Morishita TY, Johnson G, Johnson G, et al. Scaly-leg mite infestation associated with digit necrosis in Bantam Chickens (*Gallus domesticus*). *J Avian Med Surg*. 2005;19:230-233.