

Latin Comparative Pathology Group

The Latin Subdivision of the CL Davis Foundation

Diagnostic Exercise

Case #: 13 Month: September Year: 2011

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Clinical History: Four turkey hens (Broad Breasted White), 11-week-old, were submitted for necropsy with a history of increased mortality in the flock. The production ranch had 50,000 birds with 7,500 in the affected house, out of which approximately 20 % were sick. Mortality in the house reached 0.3 % daily. The affected birds were described as not eating, with watery yellow tinged feces and low activity level.

Laboratory Findings: *No pre-mortem laboratory analysis available.*

Necropsy Findings: Overall the birds were dehydrated and had a mild ascites. Figure 1 shows the two organs where the most significant lesions were found; all birds had similar lesions. Figure 2 displays microscopic images of the organs shown in Fig 1.

Follow-up questions:

- 1) Describe briefly the gross findings in the two organs (A and B) shown in Fig. 1. Give a morphological diagnosis for both of them.
- 2) Name the main histological findings in the microscopic sections (at least two per organ).
- 3) Name the agent involved and the etiological diagnosis. What is the common name of the disease?

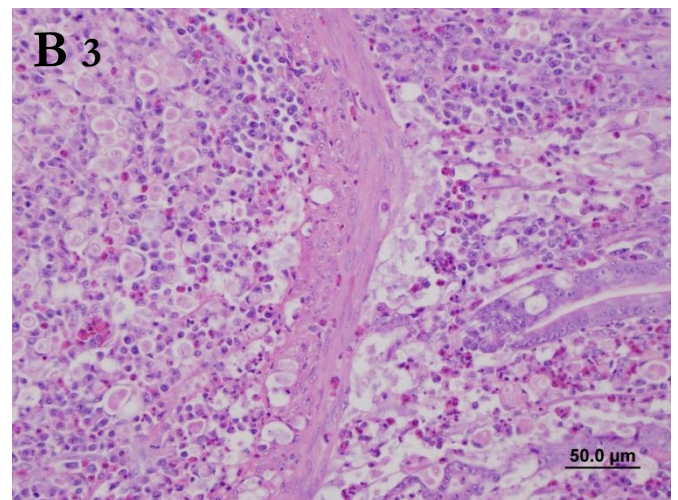
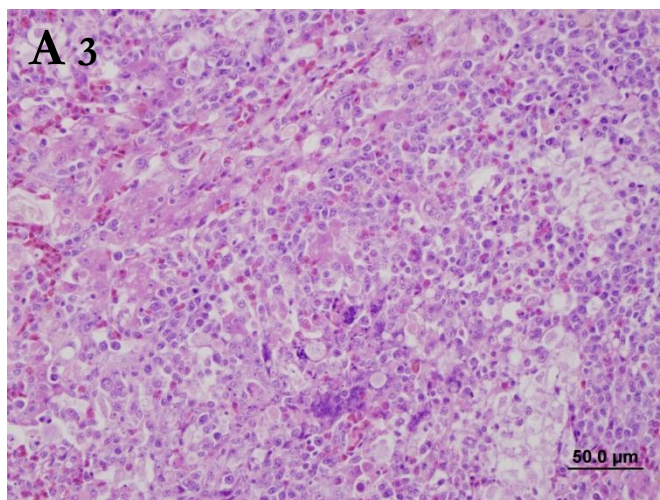
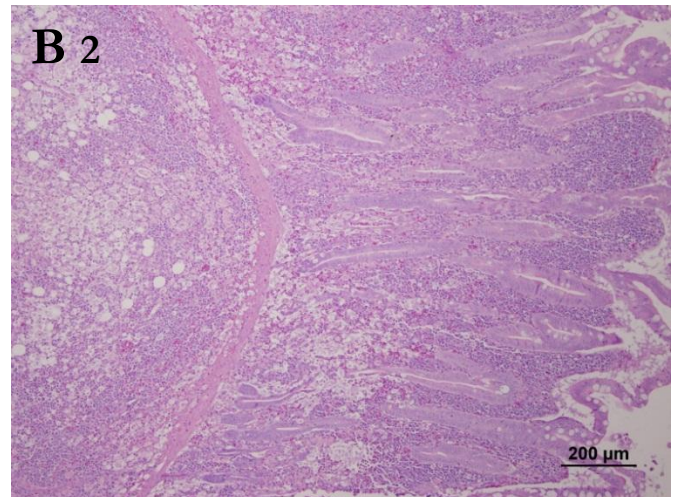
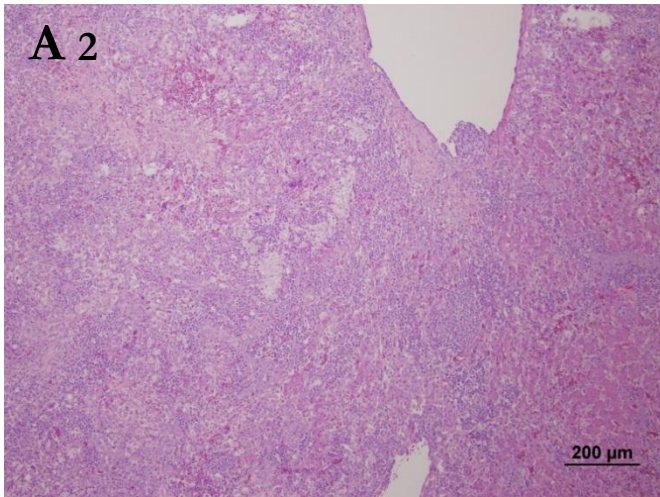
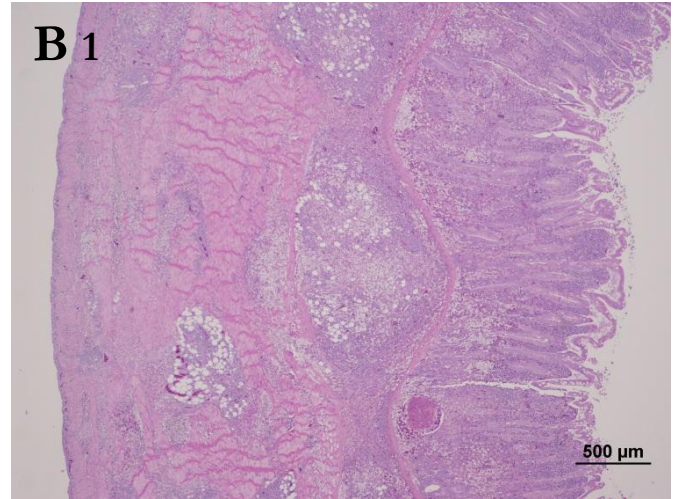
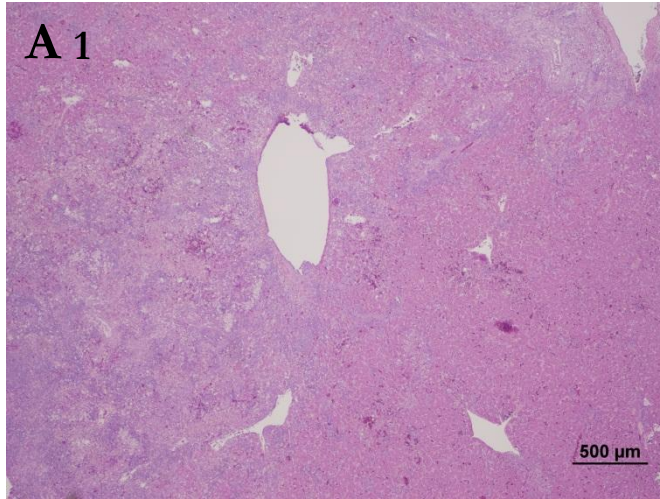
Gross (Figure 1) and microscopic images (Figure 2):

Fig. 1



Gross image of two organs (A and B), from a bird with representative lesions.

Fig. 2



Microscopic images are labeled as A or B accordingly to gross images of organs A and B on Fig. 1.

Different magnifications of representative fields are shown here; 1 lowest (4x), 2 (10x) and 3 highest (40x), H/E.

Answers:

1. Liver: Multiple, round, variable sized, poorly limited foci; composed of a dark, depressed center, surrounded by a white, broad ring (target-like lesions).

Ceca: Thickened walls, slightly reddened serosa, mucosa irregularly covered with a patchy yellow fibrinous coat, and containing a luminal, caseous-necrotic core.

Liver: Severe, multifocal (random), necrotizing hepatitis, acute.

Ceca: Severe, diffuse, fibrinous-necrotizing typhlitis, acute.

2. Liver: Multifocal to coalescing necrotizing hepatitis, diffuse heterophilic inflammatory infiltrates, scattered giant cells. Numerous intralesional extracellular organisms present (protozoa). Organisms are eosinophilic, round to ovoid (10 to 20 micrometers in diameter), and surrounded by a clear halo.

Ceca: Diffuse, heterophilic inflammatory infiltrates in lamina propria, pleocellular inflammatory aggregates in submucosa with multiple intralesional organisms similar to the ones described in liver. Occasional necrotic foci in crypts lumens are also present.

3. Agent: *Histomonas meleagridis*; Etiologic diagnosis: Histomoniasis; Disease: Blackhead.

Notes on Histomoniasis¹

Histomoniasis is caused by a protozoan that infects the ceca, and later the liver, of turkeys, chickens, and occasionally other galliform birds. In turkeys, most infections are fatal; in other birds, mortality is less common.

The protozoan parasite *Histomonas meleagridis* is transmitted most often in embryonated eggs of the cecal nematode *Heterakis gallinarum*, and sometimes directly by contact with infected birds. Outbreaks spread quickly through flocks by direct contact. A large percentage of chickens harbor this worm, and histomonads have been located in adult worms of both sexes. Three species of earthworms can harbor *H. gallinarum* larvae containing *H. meleagridis*, which are infective to both chickens and turkeys. *H. meleagridis* survives for long periods within *Heterakis* eggs, which are resistant and may remain viable in the soil for years. Histomonads are released from *Heterakis* larvae in the ceca a

few days after entry of the nematode and replicate rapidly in cecal tissues. The parasites migrate into the submucosa and muscularis mucosae and cause extensive and severe necrosis. Histomonads reach the liver either by the vascular system or via the peritoneal cavity, and rounded necrotic lesions quickly appear on the liver surface. Histomonads interact with other gut organisms, such as bacteria and coccidia, and depend on these for full virulence.

Traditionally, histomoniasis has been thought of as affecting turkeys, while doing little damage to chickens. However, outbreaks in chickens may cause high morbidity, moderate mortality, and extensive culling. Liver lesions tend to be less severe in chickens, but morbidity can be especially high in young layer or breeder pullets. Tissue responses to infection may resolve in 4 weeks, but birds may be carriers for another 6 weeks.

Signs are apparent 7–12 days after infection and include listlessness, drooping wings, unkempt feathers, and yellow droppings. The origin of the name “blackhead” is obscure. Young birds have a more acute disease and die within a few days after signs appear. Older birds may be sick for some time and become emaciated before death.

The primary lesions are in the ceca, which exhibit marked inflammatory changes and ulcerations, causing a thickening of the cecal wall. Occasionally these ulcers erode the cecal wall, leading to peritonitis and involvement of other organs. The ceca contain a yellowish green, caseous exudate or, in later stages, a dry, cheesy core. Liver lesions are highly variable in appearance; in turkeys, they may be up to 4 cm in diameter and involve the entire organ. The liver and cecal lesions together are pathognomonic. However, the liver lesions must be differentiated from those of tuberculosis, leukosis, avian trichomoniasis, and mycosis. In some cases, especially in chickens, histopathologic examination is helpful. Histomonads are intercellular, although they may be so closely packed as to appear intracellular. The nuclei are much smaller than those of the host cells, and the cytoplasm less vacuolated. Scrapings from the liver lesions or ceca may be placed in isotonic saline solution for direct microscopic examination; *Histomonas* spp must be differentiated from other cecal flagellates.

Suggested reading:

1. Merck Veterinary Manual, available at: <http://www.merckvetmanual.com>
2. Diseases of Poultry, 12th Edition, Y. M. Saif (Editor-in-Chief), Aly M. Fadly (Associate Editor), John R. Glisson (Associate Editor), Larry R. McDougald (Associate Editor), L. K. Nolan (Associate Editor), David E. Swayne (Associate Editor), 2008, Wiley-Blackwell. Chapter 28, pages 1095–1100.

Please send your comments/questions to the whole LCPG list by hitting "reply to all".

A final document containing this material with answers and a brief discussion will be posted on the C. L. Davis website by the end of the current month (http://www.cldavis.org/lcpg_english.html).