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Case Study

Pathological Investigation of a Histomoniasis Outbreak in Turkeys

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Abstract

Histomoniasis causes severe, necrotizing typhlitis and hepatitis with up to 100% mortality in turkeys. A tom turkey flock in Western Canada, exhibiting clinical signs including depression and sulfur-yellow feces, was suspected for histomoniasis. Characteristic lesions in ceca and liver on necropsy strongly favored a presumptive diagnosis of histomoniasis. Microscopical identification of *Histomonas* trophozoites confirmed the disease.

Keywords: *Histomonas meleagridis; Histomonas trophozoites; Histomoniasis; Histomonosis; Blackhead Disease; Cecal Cores; Necrotic Hepatitis*

Introduction

Histomoniasis (histomonosis or blackhead disease), is caused by *Histomonas meleagridis* [2] and causes severe intestinal and systemic disease in turkeys with up to 100% mortality [4]. Histomoniasis field outbreaks have been documented worldwide [6]. Infected *Heterakis gallinarum* eggs, a cecal nematode of poultry and an earthworm intermediate host, are an important route of disease transmission [3,4]. Worms, flies and insects also act as mechanical vectors to transmit the disease [4]. Clinical signs include dullness and depression with characteristic "sulfur-yellow droppings" [4]. *Histomonas meleagridis* lesions initially target the ceca and spread through the blood stream to the liver [1]. Pathognomonic lesions in ceca and liver are most common, while systemic dissemination of the disease can cause lesions in other organs including the spleen, lungs, kidneys, pancreas, and bursa of Fabricius [7].

A histomoniasis outbreak was noticed in a conventional tom turkey flock (n = 10,000) located in Western Canada. This barn had a previous history of histomoniasis. The histomoniasis outbreak occurred in 5-week-old-poults, and 60% mortality was reported over a period of two weeks. A field investigation was conducted two weeks later. Dull and depressed poults with ruffled feathers were observed. On necropsy, affected birds had severe typhlitis with cecal cores (Figure 3) and multifocal necrotic hepatitis (Figure 1). Liver and cecal samples were collected in 10% neutral buffered formalin for histology.

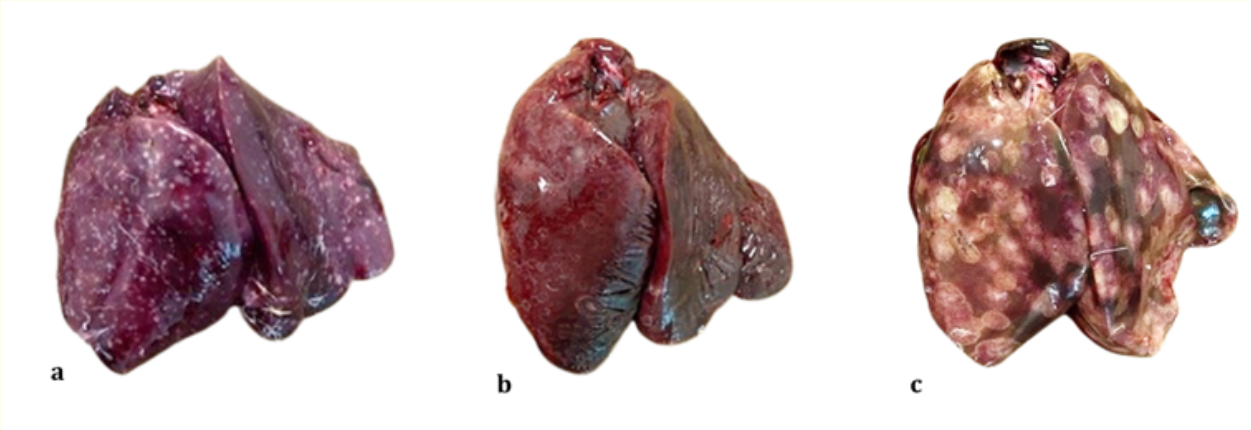

Figure 1: *Liver from seven-week-old turkeys from histomoniasis affected flock. A: Numerous pin-point white necrotic foci, b: Necrotic foci with red center and circumscribed pale white periphery (bulls-eye lesions), c: Extensive liver discoloration with circular, pale tan, necrotic foci and round, shallow depression (saucer shaped lesions)*

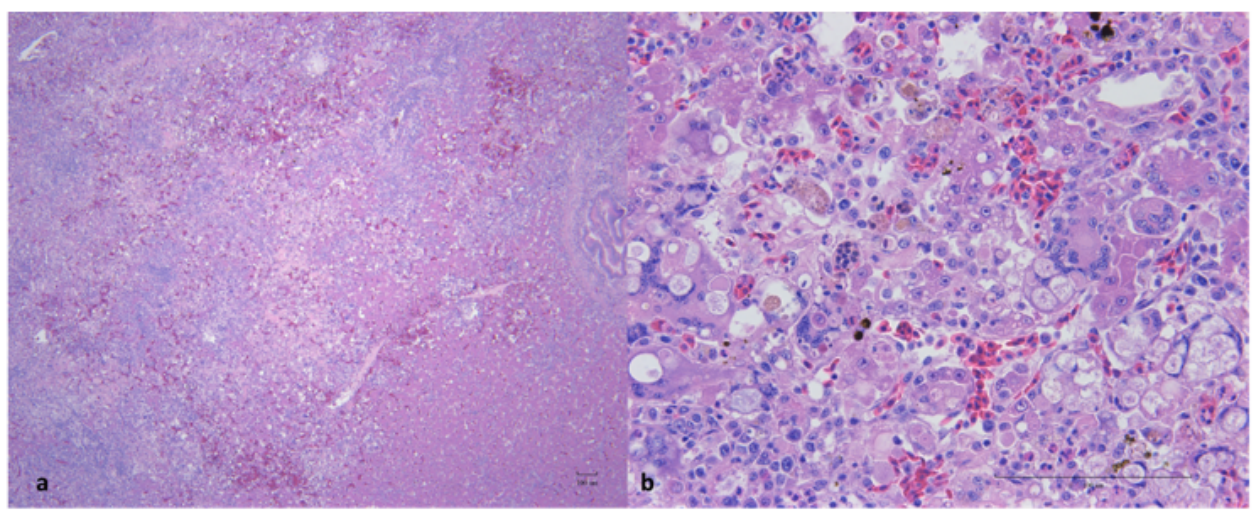

Figure 2: *Photomicrographs of turkey liver stained with hematoxylin and eosin. a: Multifocal to coalescing necrotizing hepatitis, b: Predominantly histiocytic inflammation, loss of hepatic cords, abundant histomonads infiltrate hepatic sinusoids, and hepatocytes with additional trophozoites.*

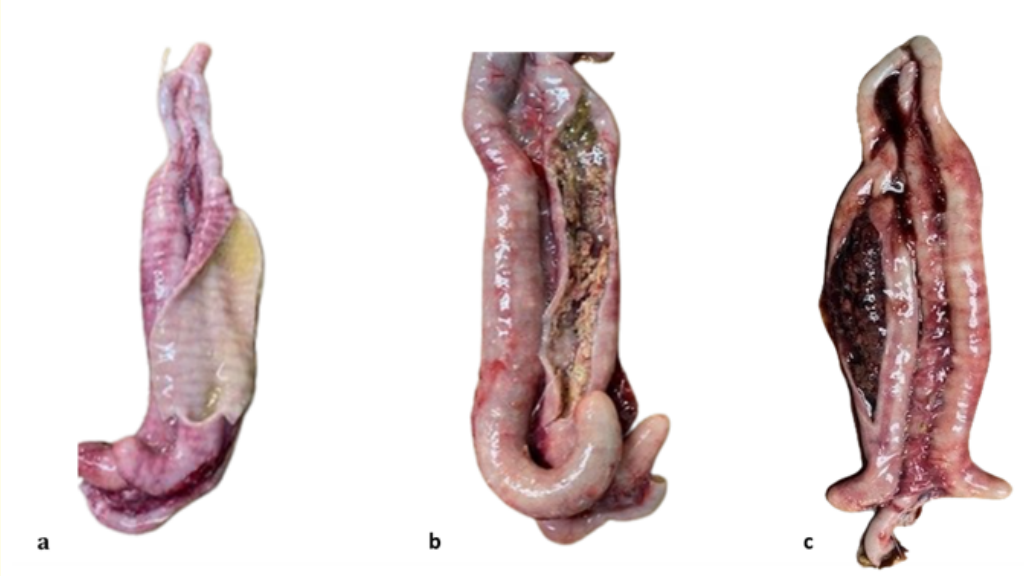

Figure 3: *Ceca from seven-week-old turkeys from histomoniasis affected flock. a: Hyperemia and thickening of cecal wall, b: Ceca engorged with fibrinonecrotic cecal core, c: Cecal core covered with blood.*

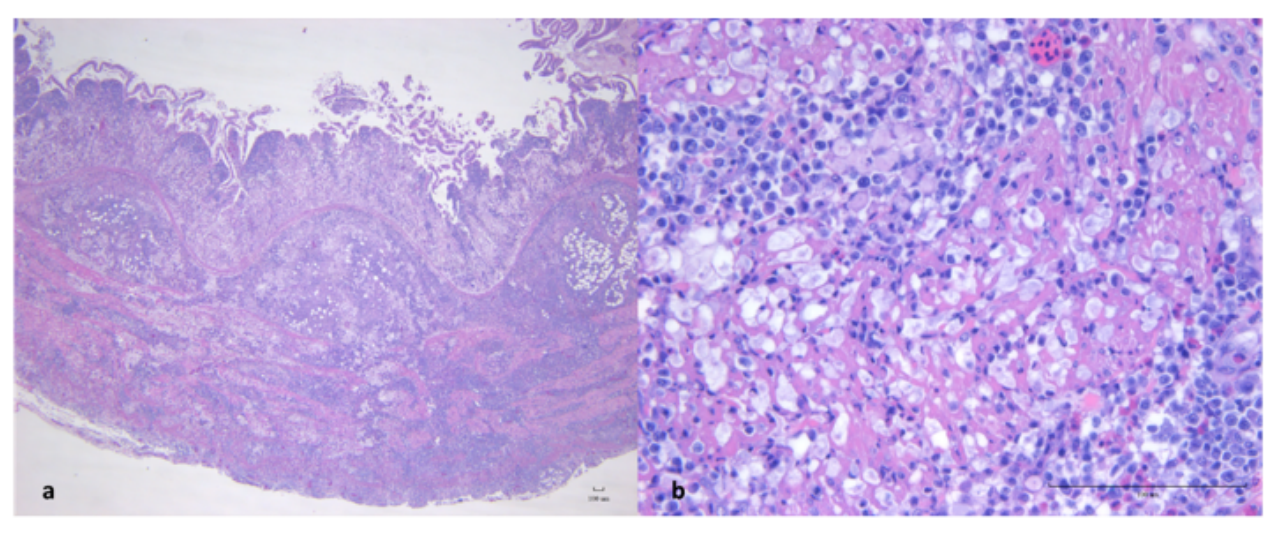

Figure 4: *a: Transmural cecal necrosis associated with abundant histomonads and histiocytic to pyogranulomatous inflammation. b: Predominantly histiocytic inflammation, abundant histomonads, and fewer neutrophils with tunica muscularis necrosis.*

Discussion

Histomonas meleagridis and *Tetratrichomonas gallinarum* cause similar lesions in ceca and liver [4,5]. *Eimeria*, *Salmonella* and *Trichomonas* cause similar cecal lesions while *E. coli*, *Campylobacter*, *Mycobacterium, Ascaridia*, and Turkey hepatitis virus (picornavirus) cause similar liver lesions. In this study, the liver lesions range from numerous pin-point white necrotic foci (Figure 1a) to numerous necrotic foci with red center and circumscribed pale white periphery resembling "bulls-eye lesions" (Figure 1b), to extensive liver discoloration with circular, pale tan, necrotic foci and round, shallow depressions (Figure 1c). On histology, the liver has random, locally extensive, multifocal to coalescing, liquefactive necrosis (Figure 2a) associated with heterophilic and histiocytic inflammation, including abundant epithelioid macrophages, multinucleated giant cells, abundant *Histomonas* trophozoites, lymphocytes and plasma cells, debris-laden Kupffer cells, hemorrhage, and edema. There is variable bile duct hyperplasia, fibrin and cellular thrombi within blood vessels, and extensive loss of sinusoidal architecture (Figure 2b). Similar inflammation, hemorrhage, and necrosis frequently obstructs bile ducts within portal triads. Severe damage to the liver and bile ducts may contribute to sulfur-yellow droppings.

The cecal lesions range from hyperemia and thickening of cecal wall (Figure 3a), ceca engorged with fibrinonecrotic cecal core (Figure 3b) and cecal core covered with blood (Figure 3c). The cecal mucosa is extensively effaced and expanded by abundant heterophilic and histiocytic inflammation, hemorrhage, liquefactive necrosis, thrombosis of mucosal vessels, and moderate numbers of *Histomonas* trophozoites expand the apical to mid villi enterocytes (Figure 4a, b). The crypt lumens and lamina propria are expanded and effaced by heterophilic cellular debris and the crypt epithelium is variably attenuated to ulcerated contributing towards partial to diffuse loss of crypt architecture. The tunica muscularis, submucosa, and subserosal stroma are moderately to extensively infiltrated and effaced by similar inflammation, protozoa, fibrin, and granulation tissue. The mixture of inflammatory exudates and necrotic cellular debris fills the cecal lumen resulting in the formation of a solid core. Severe ulceration and hemorrhage lead to blood in the cecal lumen. The submucosa, tunica muscularis, and subserosal stroma has similar inflammation along with *Histomonas* trophozoites, fibrin, and granulation tissue. The serosal surface is thickened by thin coagulum of inflammatory cells, fibrin, and mesothelial cell hypertrophy. Lymphoid tissue is occasionally intact and has moderate lymphoid hyperplasia, sinus histiocytosis, and multifocal aggregates of trophozoites associated with pyogranulomatous inflammation. All these factors contributed to the gross thickening and hyperemia of the serosal layer of the ceca. Thus, microscopic evaluation of the affected tissues provided confirmation results along with more insights in understanding the pathogenesis of the disease.

Conclusion

In this field outbreak, a presumptive diagnosis was made based on distinctive gross pathology lesions in liver and ceca. On histopathology, necrotizing, pyogranulomatous typhilitis and hepatitis with abundant *Histomonas* trophozoites were observed and confirmed the presumptive diagnosis.

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