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Histomoniasis outbreak in free-range chickens in semiarid Paraíba, Brazil

Surto de histomoníase em frangos caipiras no semiárido da Paraíba, Brasil

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Abstract

We describe an outbreak of histomoniasis in free-range chickens (*Gallus gallus domesticus*) in the semiarid region of Paraíba State, Brazil. Juvenile chickens aged approximately 48 days showed disease over a 15-day period. Seven of 16 chickens showed yellow, watery stool, anorexia, lethargy, and death within 3-5 days of clinical illness. The mortality rate was 43.7%. The chickens were raised in a backyard with 12 adult birds that remained healthy. None of the chickens were vaccinated or dewormed. A necropsy on one deceased bird showed lesions confined to the cecum and liver. The left cecum was distended and had yellow, slightly elevated areas on the serosa. The liver was reddened with centrally depressed areas surrounded by a pale halo, which were distributed throughout the capsular surface and extended deep throughout the parenchyma. Microscopically, typhlitis and pyogranulomatous necrotizing hepatitis associated with numerous intralesional *Histomonas* trophozoites was observed. The birds were administered metronidazole and albendazole, which effectively controlled the histomoniasis. Although histomoniasis is rare in free-range poultry, it can occur and cause high mortality and significant economic losses.

Key words: *Histomonas meleagridis*, parasitic hepatitis, poultry protozoa

Resumo

Descreve-se um surto de histomoníase em frangos caipiras (*Gallus gallus domesticus*) na região semiárida da Paraíba. A doença ocorreu em pintos de aproximadamente 48 dias de idade durante um período de 15 dias. De um total de 16 pintos, sete apresentaram fezes amareladas liquefeitas, anorexia, letargia e morreram com 3 a 5 dias de evolução clínica. A taxa de mortalidade foi de 43,7%. Os pintos eram criados soltos em quintal de chão batido com outras 12 aves adultas que não adoeceram. Não eram vacinados e nem vermifugados. Na necropsia de uma ave as lesões eram restritas ao ceco e ao fígado. O ceco esquerdo estava distendido e na serosa havia áreas amareladas e discretamente elevadas. No fígado havia áreas avermelhadas e deprimidas centralmente, circundadas por halo pálido, distribuídas na superfície capsular e ao corte por todo o parênquima. Microscopicamente verificou-se tiflite e hepatite piogranulomatosa necrosante associada a miríades de *Histomonas* intralesionais. O tratamento das aves com metronidazol e albendazol foi eficaz no controle da histomoníase. Apesar de ser uma doença rara em frangos caipiras, ela pode ocorrer causando elevada taxa de mortalidade e perdas econômicas significativas.

Palavras-chave: *Histomonas meleagridis*, hepatite parasitária, protozoários de aves

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Introduction

Histomoniasis is a disease caused by the protozoan *Histomonas meleagridis* and affects the liver and cecum of domestic and wild birds, triggering degenerative and necrotizing inflammation (TRINDADE et al., 2011). It is transmitted primarily by direct contact when a bird ingests contaminated feces or eggs from the helminth *Heterakis gallinarum* containing the protozoan (SENTÍES-CUÉ et al., 2009).

This disease is frequently diagnosed in the United States primarily in turkeys (NORTON et al., 1999; SENTÍES-CUÉ et al., 2009), domestic chickens (CORTES et al., 2004) and several wild bird species (REIS JÚNIOR et al., 2009; MCDOUGALD et al., 2012). In Brazil, the disease has been described in turkeys from the Rio de Janeiro state (BRENER et al., 2006) and in peacocks from the Rio Grande do Sul state (TRINDADE et al., 2011), but it has not been reported in chickens. Therefore, the aim of this work was to describe the epidemiological, clinical, and pathologic features of a histomoniasis outbreak in free-range chickens in the semiarid region of Paraiba state, Northeastern Brazil.

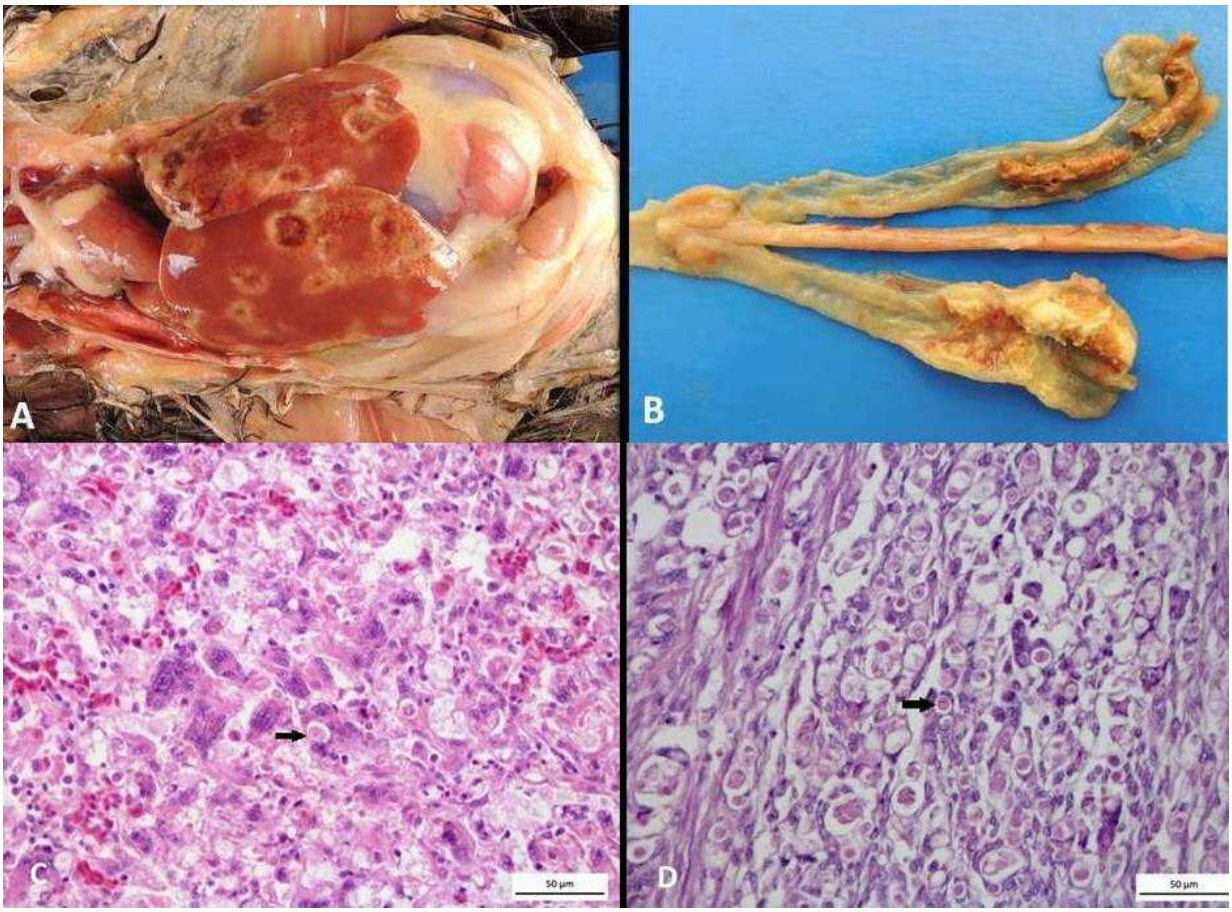
Case Report

The outbreak occurred at a small private breeding facility in the city of Patos, Paraiba state. Of 16 total chicks aged 48 days, seven birds presented with watery yellow stool, anorexia, lethargy, and death after 3 to 5 days of clinical signs. The death rate was 43.7%, and the deaths occurred over a 15-day period. The chicks were raised free in a backyard together with 12 adult birds that remained healthy. One of the deceased chicks was referred to the Animal Pathology Laboratory of the Veterinary Hospital of the Federal University of Campina Grande (UFCG) for a necropsy and histopathology examination. The coelomic cavity organs and central nervous system were sampled, fixed in 10% buffered formalin, and embedded in paraffin. The samples were then sliced into 4-µm sections and stained with Hematoxylin and Eosin (HE). Select cecum and liver samples were also stained using Periodic-acid Schiff stain (PAS).

Results and Discussion

Grossly, the lesions were restricted to the liver and cecum. The liver was slightly enlarged with reddened multifocal to coalescing areas, which measured from 1.0 to 3.0 cm in diameter and showed a central depression surrounded by a pale halo. The lesions were distributed throughout the capsular surface (Figure 1A) and extended deep into the parenchyma. The left cecum was distended and had multifocal, slightly elevated, yellow lesions on the serosa. The mucosa contained multifocal yellow lesions containing friable caseous material. Adjacent to these mucosal lesions, the mucosa was reddened with irregular thickening (Figure 1B). Microscopically, pyogranulomatous necrotizing hepatitis and severe typhlitis associated with numerous intralesional *Histomonas* trophozoites was observed. The liver also showed multifocal to coalescing, periportal pyogranulomas, as well as random multifocal areas of marked hepatocyte necrosis associated with numerous *Histomonas* trophozoites. Degenerated trophozoites were also identified in the necrotic areas. The pyogranulomas comprised a central area of necrosis, cellular debris, and fibrin strands, surrounded by a numerous multinucleated giant cells containing a moderate amount of intracytoplasmic trophozoites (Figure 1C), epithelioid macrophages, heterophils, rare lymphocytes, and sparse plasma cells interspersed with discrete connective tissue. The necrotic areas were often associated with hemorrhage, and in some lesions, the necrosis extended to the Glisson capsule. The hepatic sinusoids were distended with trophozoites and discrete inflammatory cells. Fibrinoid necrosis of the vessel wall was also observed.

Figure 1. Histomoniasis in chickens. A – Increased liver volume, with the multifocal to coalescing areas of necrosis, reddish and centrally depressed, surrounded by pale halo in the capsular surface. B – In the mucosa of the left cecum, multifocal areas of yellow, friable caseous material can be observed. C – Necrosis of hepatocytes, granulomatous infiltrate, and large number of multinucleated giant cells, some of them showing trophozoites of *Histomonas* in the cytoplasm (Arrow). D – The submucosal layer had necrosis, high amount of giant cells, some of them phagocyting *Histomonas* trophozoites (Arrow) and epithelioid macrophages.



In the cecum, a focal but extensive necrotic region was identified on the mucosal surface containing multiple areas of mineralization associated with cellular debris. In some areas, the epithelium was partially lost, and the lamina propria was distended by inflammatory infiltrates predominantly comprising mononuclear cells and heterophils associated with numerous spherical to oval structures and occasionally extending into the submucosa (Figure 1D). These ovoid structures measured between 5-15 µm in diameter, had a prominent central nucleus, and slightly granular eosinophilic cytoplasm surrounded by a clear halo and thin cell wall, all characteristics of *Histomonas* trophozoites. The muscular layer showed multifocal areas of necrosis associated with high quantities of multinucleated giant cells, with some observed phagocytizing protozoa; epithelioid macrophages were also present. Several areas showed fibrinoid necrosis in the vessel wall. On PAS samples, the trophozoites stained mildly positive.

The chicks were diagnosed with histomoniasis based on epidemiological data, clinical signs, and primarily by the lesions restricted to the cecum and liver observed during the necropsy and confirmed

by histopathology. Both juvenile and adult birds were administered metronidazole (110 mg/kg) once daily for 10 days and albendazole (10 mg/kg) for three days, which controlled further mortality. Only one chick died after beginning treatment.

A clinical diagnosis of histomoniasis is supported by a history of yellow stool in juvenile birds residing in mixed age groups including adult birds, or a lack of response to antimicrobial therapy (BACK, 2010). However, forming a clinical diagnosis in this fashion can be uncertain, as there are other diseases affecting birds that cause similar clinical manifestations. The presence of necrotic lesions in the large intestine, cecum, and especially in the liver on necropsy are suggestive of histomoniasis. In this case, the recognition of the morphological characteristics of this parasite on HE staining was the primary tool used in the etiologic diagnosis. On histopathology, necrotizing typhlitis and pyogranulomatous hepatitis associated with intralesional trophozoites was morphologically diagnosed, findings that are similar to those described by other authors (HU et al., 2006; SENTÍES-CUÉ et al., 2009; MCDOUGALD et al., 2012).

The etiologic diagnosis of histomoniasis in chickens can be made by histological examination based on the morphology of the trophozoites and their characteristic staining, especially in cases where the agent cannot be cultured (SENTÍES-CUÉ et al., 2009). This requires that the examiner be able to recognize the trophozoites of this agent, which helps to differentiate the infection from other protozoal poultry diseases, particularly coccidiosis. More sensitive molecular techniques are currently used to detect and identify *H. meleagridis* such as polymerase chain reaction (PCR), which allows the parasitic DNA to be amplified and aids in differentiation from infections by *Tetratrichomonas gallinarum* and *Blastocystis* spp. (HUBER et al., 2005; GRABENSTEINER; HESS, 2006).

In this study, the classic form of the disease was observed, which is the most commonly diagnosed. However, the disseminated form of the disease, characterized by the distribution of lesions in multiple organs beyond the liver and cecum, can also occur (SENTÍES-CUÉ et al., 2009; MCDOUGALD et al., 2012). Furthermore, only chicks were affected, which was likely due to their increased susceptibility compared to the adult birds. The high mortality rate (43.7%) observed in this outbreak may reflect management failures, namely the mixed-age flock and lack of deworming procedures. Raising young and adult birds in a single facility is considered a risk factor for the onset of infection; the disease primarily occurs when juveniles roam freely throughout the facility and shared an environment with adult carrier birds (BACK, 2010).

Histomoniasis outbreaks are commonly observed in poultry 3 to 7 weeks of age (HU et al., 2006). Outbreaks with high mortality rates sometimes occur in young chickens, but field studies report a moderate or low mortality in this species, indicating that chickens are less susceptible to disease (VAN DER HEIJDEN; LANDMAN, 2011). Adult birds usually acquire immunity against this parasite and can become natural hosts of the *H. meleagridis* protozoan; adults generally do not exhibit clinical signs (REIS JÚNIOR et al., 2009).

In the present case, once treatment was established, the outbreak was controlled, though one additional bird still died, possibly because it already had severe disease. Metronidazole is an orally administered chemotherapeutic that can effectively control histomoniasis when combined with albendazole. The curative effect is thought to be due to the destruction of the transmitting vector or to direct killing of *Histomonas* protozoa normally found in the cecal lumen, a mechanism that contrasts its low efficacy against the amoeboid form of the parasite that resides in tissues (HEGNGI et al., 1999). Despite the rare incidence of histomoniasis in chickens, it can cause high mortality rates and significant economic losses, especially when associated with failures in production and health management.

References

BACK, A. *Manual de doenças de aves*. 2. ed. Cascavel: Editora Integração, 2010. 311 p.

BRENER, B.; TORTELLY, R.; MENEZES, R. C.; MUNIZ-PEREIRA, L. C.; PINTO, R. M. Prevalence and pathology of the nematode *Heterakis gallinarum*, the trematode *Paratanaisia bragai*, and the protozoan Histomonas meleagridis in the turkey, *Meleagris gallopavo*. *Memórias do Instituto Oswaldo Cruz*, Rio de Janeiro, v. 101, n. 6, p. 677-681, 2006.

CORTES, P. L.; CHIN, R. P.; BLAND, M. C.; CRESPO, R.; SHIVAPRASAD, H. L. Histomoniasis in the bursa of Fabricius of chickens. *Avian Diseases*, Madison, v. 48, n. 3, p. 711-715, 2004.

GRABENSTEINER, E.; HESS, M. PCR for the identification and differentiation of *Histomonas meleagridis*, *Tetratrichomonas gallinarum* and *Blastocystis* spp. *Veterinary Parasitology*, Amsterdam, v. 142, n. 4, p. 223-230, 2006.

HEGNGI, F. N.; DOERR, J.; CUMMINGS,T. S.; SCHWARTZD, R. D.; SAUNDERS, G.; ZAJAC, A.; LARSEN, C. T.; PIERSON, F. W. The effectiveness of benzimidazole derivatives for the treatment and prevention of histomonosis (blackhead) in turkeys. *Veterinary Parasitology*, Amsterdam, v. 81, n. 1, p. 29-37, 1999.

HU, J.; FULLER, L.; ARMSTRONG, P. L.; MCDOUGALD, L. R. *Histomonas meleagridis* in chickens: attempted transmission in the absence of vectors. *Avian Diseases*, Madison, v. 50, n. 2, p. 277-279, 2006.

HUBER, K.; CHAUVE, C.; ZENNER, L. Detection of *Histomonas meleagridis* in turkeys cecal droppings by PCR amplification of the small subunit ribosomal DNA sequence. *Veterinary Parasitology*, Amsterdam, v. 131, n. 3, p. 311-316, 2005.

MCDOUGALD, L. R.; ABRAHAM, M.; BECKSTEAD, R. B. An outbreak of blackhead disease (*Histomonas meleagridis*) in farm-reared bobwhite quail (*Colinus virginianus*). *Avian Diseases*, Madison, v. 56, n. 4, p. 754-756, 2012.

NORTON, R. A.; CLARK, F. D.; BEASLEY, J. N. An outbreak of histomoniasis in turkeys infected with a moderate level of *Ascaridia dissimilis* but no *Heterakis gallinarum*. *Avian Diseases*, Madison, v. 43, n. 2, p. 342-348, 1999.

REIS JÚNIOR, J. L.; BECKSTEAD, R. B.; BROWN, C. C.; GERHOLD, R. W. *Histomonas meleagridis* and capillarid infection in a captive chukar (*Alectoris chukar*). *Avian Diseases*, v. 53, n. 4, p. 45-46, 2009.

SENTÍES-CUÉ, G.; CHIN, R. P.; SHIVAPRASAD, H. L. Systemic histomoniasis associated with high mortality and unusual lesions in the bursa of fabricius, kidneys, and lungs in commercial turkeys. *Avian Diseases*, Madison, v. 53, n. 2, p. 231-238, 2009.

TRINDADE, M. M.; SCHENEIDERS, G.; CORREA, I. M. O.; FLORES, M.; KOMMERS, G. D. Histomoníase em pavão (*Pavo cristatus*). *A Hora Veterinária,* Porto Alegre, v. 31, n. 184, p. 56-58, 2011.

VAN DER HEIJDEN, H. M. J. F.; LANDMAN, W. J. M. High Seroprevalence of *Histomonas meleagridis* in dutch layer chickens. *Avian Diseases*, Madison, v. 55, n. 2, p. 324-327, 2011.