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Histomoniasis in a Captive Great Rhea (*Rhea americana*)

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Histomoniasis caused by *Histomonas meleagridis* and commonly called blackhead, has been reported in turkeys (Cushman, 1893, Agr. Expt. Sta., Rhode Island St. Coll. of Agri. Mech. A&T Bull. 25: 89–123) and other gallinacious birds such as quail, pheasants, guinea hens, peafowl and grouse (Desowitz, 1951, J. Comp. Pathol. 61: 231–236; Dhillon et al., 1980, Avian Dis. 24: 510–516). Outbreaks of blackhead occur occasionally in chickens which are considered natural hosts of *Heterakis gallinarum*, the nematode vector of *Histomonas meleagridis*. Turkeys, chickens, partridges and ruffed grouse (Lund and Chute, 1972, J. Parasitol. 58: 940–945) are reported to be affected severely by histomoniasis. This report describes histomoniasis in a young captive greater rhea.

Two 1-day-old greater rheas were raised until 6 wk of age in Issaquah, Washington and were subsequently purchased by the present owner who lives in Bothel, Washington. Other birds kept at the Issaquah facility included white leghorn and Rhode Island red chickens, wild turkeys (*Meleagris gallopavo*), ring-necked pheasants (*Phasianus colchicus*), waterfowl (*Anas Platyrhynchos* and *Anser anser*) and peafowl (*Pavo cristatus*). The rheas were raised in a pen that was 1.2 to 1.8 meters from the chickens and 36.4 meters from the turkeys, pheasants and waterfowl. The present owner raised chickens in addition to the rheas. The two birds were fed a ratite ration that was formulated commercially (Anderson Feed Company, Maunee, Ohio 43537, USA). Both were thin and had ruffled feathers. Two weeks after being moved to Bothel, Washington one bird died and was submitted for post mortem examination. The second bird died a week later and was buried without being examined.

Gross morphologic alterations included thickened hyperemic cecal wall and diphtheretic necrotic cecal mucosa that separated multifocally from the wall forming a hemorrhagic caseated core in the lumen. Ulceration of the cecal wall was not present. The cecae were full of foul-smelling fecal material. No alterations were seen in the liver. There was a partial impaction of the gizzard with fibrous strands of plant material and hair. Selected tissues were fixed in neutral buffered 10% formalin and sections were cut for light microscopy by routine paraffin embedding procedures. Sections were stained with hematoxylin and eosin.

Histopathologic alterations were of typhlitis, characterized by excoriation of ulcerated cecal mucosa forming an amorphous red-tinged core composed of sloughed epithelium, fibrinous proteinaceous material, erythrocytes, lymphocytes, and macrophages with predominant heterophils. Pale, slightly bluish, 8–20 μm organisms were present occasionally. The autolytic changes present in the ulcerated excoriated cecal mucosa made morphologic evaluation difficult. However, the sizes of the organism resembled histomonads and the gross pathology of the ceca was consistent with blackhead disease. Mucosa still attached to the cecal wall had hypercellularity, however, no organisms were seen. No microscopic alterations were present in the liver and other tissues examined.

Bacteriologic examination of the ceca revealed moderate numbers of *Escherichia coli*, but no *Salmonella*. No bacterial organisms were isolated from liver specimens. Specimens collected from ceca on embryo inoculations were negative for the presence of histomonads; autolysis may have made isolation unsuccessful.

The occurrence of blackhead was not reported in the wild turkeys, pheasants or peafowl in the Issaquah facility where the birds were reared from 1 day to 6 wk of age. There is a possibility that carrier birds transmitted the infection to the rheas.

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