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Histomoniasis in Turkeys

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Abstract

Histomoniasis caused by protozoa *Histomonas meleagridis* mainly affects turkeys. Chickens are subclinical carriers of infection. The disease in turkeys is characterized by yellow feces, drowsiness, dropping of wings, head down and high mortality. *Histomonas meleagridis* is transmitted in the embryonated eggs of cecal worm of poultry, *Heterakis gallinarum*. Lesions of the disease in liver are seen as multifocal, circular, depressed areas of necrosis of varying size circumscribed by a raised ring. There is thickening, congestion and ulceration of cecal walls and presence of cecal cores in chronic cases. Disease is diagnosed on the basis of gross and histopathological lesions, microscopic identification of histomonads, PCR assays, *in situ* hybridization and immunohistochemistry. As there is no treatment and vaccine available, prevention is the only available tool. Disease can be prevented by quarantine and avoidance of contact between turkeys and the poultry, frequent changing of wet litter and practice of strict biosecurity measures on farm.

Introduction

Histomoniasis caused by protozoan *Histomonas meleagridis* affects wide range of gallinaceous birds. Turkey is the most susceptible species where the disease causes high mortality rate reaching up to 80-100%. This high mortality rate frequency leads to loss of entire flocks. Chickens are typically subclinical carriers. Histomoniasis has no public health significance as the disease is confined to birds only. Histomoniasis causes disease with the existing intestinal bacteria, such as *E. coli.* Disease is characterized by ulceration and inflammation of the cecal walls, engorgement of ceca with large caseous casts, inflammation of mesenteries and extensive multi focal necrosis of liver. Cecal worm, *Heterakis gallinarum*

acts as an intestinal host and earthworms as accessory hosts. Histomoniasis is also called as Black Head disease as the head of the bird becomes cyanotic.

Etiology

Disease is caused by protozoa *Histomonas meleagridis.* There are two forms of the parasite viz. Cecal lumen form of the parasite having a single flagellum and Tissue form of the parasite which usually lack flagella and exist in 3 forms viz. 1. Parasite 2. Vegetative stage 3. Degenerating form

Transmission

Histomonas meleagridis is commonly transmitted in the embryonated eggs of common cecal worm of poultry, *Heterakis gallinarum*. Turkeys and other gallinaceous birds harbour this worm which acts as a reservoir. Earthworms can act as vectors for cecal worm larvae containing *H. meleagridis*. Transmission within the turkey flock occurs readily by direct contact between susceptible and infected birds and contamination of feed and water by the feces of infected birds and does not require an infected intermediate host.

Life cycle

Life cycle of *Histomonas meleagridis* is complex. The protozoa multiply in the cecum of the infected bird. Organism move to the intestine of the bird, the site where the roundworm *H. gallinarum* resides. The round worms ingest the protozoa and the egg of roundworm gets infected with the protozoa. The birds excrete the protozoa infected *H. gallinarum* eggs in its droppings. Healthy birds get infected by the ingestion of contaminated feed and water by the infected feces and by invertebrates (earthworm).

Clinical Signs

Signs of Histomoniasis in turkeys include yellow feces, drowsiness, drooping of wings, stilted gait, closure of eyes, head down and close to the body or tucked under a wing and anorexia. Sick birds tend to huddle together. Birds become emaciated. Although the mortality in turkeys reach up to 80-100 % but outbreaks with low mortality are also reported. Mortality can decline to normal after the acute phase of the disease is over but the stress of movement may sometimes cause another peak of mortality.

Pathology

Gross Pathology

The primary lesions of Histomoniasis in turkeys are seen in cecum and liver. Lesions have also been observed in other organs such as the spleen, bursa of Fabricius, pancreas and kidneys indicating a widespread dissemination of pathogen within the host. First lesions are observed in ceca. After tissue invasion by histomonads, cecal walls become thickened and

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congested. Serous and hemorrhagic exudate from the mucosa fills the lumen of ceca and distends the walls with a caseous or cheesy core. Ulceration of the cecal wall may lead to its perforation resulting in generalized peritonitis. Liver lesions in turkeys are often apparent after few days of infection and are highly variable in size, number and appearance. Most common lesions are multifocal, circular, depressed areas of necrosis with varying size circumscribed by a raised ring typically resembling a Saucer. In rare cases of recovery, lesions leave purulent scars on the surface of the liver. Lesions in lung, kidney, spleen, pancreas and mesenteries are sometimes recognized as white, rounded areas of necrosis.

Histopathology

There is congestion and heterophilic infiltration in the cecal wall due to combined response to invasion of cecal wall by bacteria, histomonads and heterakid juveniles. Within 5-6 days post infection, numerous histomonads are visible as pale, weakly staining, ovoid bodies within the lacunae in the mucosa of ceca. Cecal cores are composed of sloughed epithelium, fibrin, erythrocytes, leucocytes along with the trapped cecal ingesta. By 12-16 days post infection, gaint cells appear in the tissue of cecum. Coagulative necrosis and histomonad invasion extends well into the muscular layer, extending nearly to the serosa. In survivors, histomoads are scarce within the tissue and are seen by 17-21 days post infection and they are mostly concentrated near the serosal layers. Large number of giant cells form and may appear grossly as granulomas bulging upon the serosal aspect of the ceca. Old lesions, after recovery are characterized by lymphoid cells infiltrated throughout the cecal tissue. Microscopic lesions of liver are visible by 6-7 days post infection and consist of small clusters of heterophils, lymphocytes and monocytes near portal vessels. Histomonads are difficult to visualize in these areas. After 10-14 days post infection, the lesions are enlarged, becoming confluent in some areas. There is extensive lymphocytic and macrophagic infiltration and heterophils are present in moderate numbers. There are degeneration and necrosis of hepatocytes in centre of lesions. Many individual/clustered histomonads are visible in lacunae near the periphery of the lesions. From 14-21 days post infection, there is increase in severity of necrosis. Histomonads at this stage are present mostly as small bodies in macrophages. In the recovered birds, foci of lymphoid cells along with areas of fibrosis and regenerating hepatocytes are seen.

Diagnosis

Most experienced poultry workers make a field diagnosis in turkeys on the basis of gross appearance of lesions. Laboratory confirmation is sometimes necessary to rule out concurrent infections with other agents that affect the cecum or liver. Identification of

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histomonads by microscopy adds confirmation to the diagnosis. The organisms can be observed with warm phase contrast microscopy in fresh specimens. The parasites can be isolated and cultured *in vitro*. In recent years, various PCR methods have been described, some of which are used to confirm the presence and quantification of the histomonads in fecal or tissue samples. For routine diagnosis by histopathology, hematoxylin and eosin or periodic acid-Schiff stains may be used. Excellent cytologic preparations have been made from fresh cultures using Hollande cupric picroformal and a protein silver stain. Tissue forms are unambiguously identified by *in situ* hybridization and immunohistochemistry.

Prevention and Control

As there are no chemotherapeutic preparations available for treatment and there is no commercial vaccine for Histomoniasis, control measures are mainly focused on prevention. The primary reservoir of infection is the cecal worm eggs. Thus, prevention is largely based on quarantine and avoidance of contact of susceptible birds with sources of cecal worm eggs. For the protection of turkeys, exclusion of chickens from any contact is essential, because chickens may often harbor large number of egg laying cecal worms. Outdoor turkey ranges can become contaminated with Heterakid eggs, resulting in reoccurrence of Histomoniasis in turkey flocks for years. Rearing turkeys indoors tends to reduce outbreaks as hygiene increases, but exacerbates the extent and severity of outbreaks. Wet litter may also support the spread of Histomonads which would explain the beneficial effect of changing the litter after the first clinical signs are noticed.

Conclusion

Histomoniasis mainly affects turkeys leading to high mortality thereby causing loss of entire flock many times resulting in huge economic losses to the turkey farmer. Presently, as the disease has neither any cure nor any vaccine is available to prevent the disease, prevention of disease is the only way out available. Prevention of the disease can be done by practicing strict biosecurity and quarantine measures on the farms in the endemic areas. Focused research on development of therapeutics and prophylactics against *Histomonas* should be undertaken on the priority basis. A positive development in these areas can bring a big relief to the turkey farmers by reducing their economic losses.

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