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An Outbreak of Concurrent *Histomonas meleagridis* and *Enterococcus fecalis* Infection in Ducks

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Abstract: Seventy four young ducks in a flock of about 300 birds, died of acute hemorrhagic enteritis within 15 days of appearance of the disease. Clinical signs were severe bloody diarrhea, weakness and dehydration. Fecal smears stained with Giemsa's stain showed presence *Enterococcus* spp. and red blood cells. Gram stain showed the *Enterococcus* spp. occurred singly, in pairs and in short chains and were Gram positive. Postmortem examination showed patchy hemorrhages and ulceration in the small and large intestines as well as the caeca. No worms were seen in the caeca. The liver was enlarged and dark in colour. Histopathological examination showed haemorrhages and mucosal erosion and ulceration in the small and large intestines as well as liver cell necrosis. The intestine mucosa and submucosa contained *Histomonas meleagridis*. The liver also contained *Histomonas meleagridis*, occurring singly or in clusters, with its characteristic (double-eyed) structure. It was concluded that the severity of infection of this outbreak was attributed to the concurrent effect of both *Enterococcus* and *Histomonas* infections.

Key words: Concurrent infection, histomoniasis, *Enterococcus* infection, ducks

INTRODUCTION

Enterococcus fecalis is a gram positive bacteria that occurs singly, in pairs or short chains (Saif, 2001). It is a common inhabitant of the small intestine of ducks and very resistant to macrolide and lincosamide antibiotics (Saikia *et al.*, 1995). It causes a wide syndrome of clinical signs and in the acute disease may cause lesions in the intestine, heart, liver and airsacs in ducks (Sandhu, 1988).

Histomonas meleagridis is usually transmitted by the caecal worm *Heterakis gallinae* (Saif, 2001). However direct transmission can occur. McDougald and Fuller (2005) confirmed that histomoniasis can be transmitted readily from directly exposed young turkeys to other in absence of the caecal worm vector. Direct transmission of *Histomonas meleagridis* through a flock has also been proved to occur through normal contact between uninfected birds and infected ones in the total absence of caecal worms by Hue and McDougald (2003).

Concurrent infections have been reported to augment the clinical effect of *Histomonas* infection. It has been shown that and 6% increase in mortality rate and 11% decrease in egg production in free-range layer hens was attributed to concurrent infection with *Histomonas meleagridis* and *Brachyspiralis*-like bacteria (Esquenet *et al.*, 2003). McDougald and Hue (2001) showed that blackhead disease (caused by *Histomonas meleagridis*) was aggravated in broiler chickens by concurrent infection with caecal coccidiosis. They concluded that this dual exposure may contribute to increase clinical outbreaks of blackhead disease in chickens under field conditions.

We report an outbreak farm characterized by high mortality in young birds with concurrent infection with *Histomonas meleagridis* and *Enterococcus* spp. occurred in a duck farm at Qassim, central Saudi Arabia in absence of the known intermediate host *Heterakis gallinae*.

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MATERIALS AND METHODS

Ten ducks that died of acute haemorrhagic enteritis were presented to the Pathology Department, Qassim University at Buraydah, Saudi Arabia to investigate the cause of death. The disease occurred during December 2005. Fecal samples were examined for presence of worm parasites. Smears from feces were also stained with Giemsa and Gram stains.

Organs showing lesions were fixed in 10% formol-saline and processed for routine histopathology.

Smears were made from feces of clinically ill birds and stained with Giemsa and Gram stains. The bacterium was identified as described by Cowman (1985).

RESULTS

The farm contained about 300 ducks of different ages. They reared on a semi-intensive system of production and fed a commercially prepared food. Water is offered *at lib*.

The disease appeared suddenly with profuse bloody diarrhea, prostration and dehydration. Death occurred suddenly in some birds but most affected ducks died within a period of 1-10 days from appearance of symptoms.

Examination of wet mounts of feces showed no worm parasites. Fecal smears stained with Giemsa's stain showed red blood cells and enterococci occurring singly, in pairs or short chains of 3-5 cocci (Fig. 1a). Fecal smears stained with Gram stain showed gram positive enterococci. The enterococci were almost in pure form and stained positive with Gram stain (Fig 1b).

Postmortem examination showed the extensive ulcerative haemorrhagic enteritis. The liver is dark in colour and swollen. There was congestion of kidneys and haemorrhages in the heart. The lungs were normal.

Histopathological examination showed haemorrhagic enteritis and shedding of the intestinal mucosa. *Histomonas meleagridis* had been shown to infect almost all the in the intestinal mucosa and submucosa (Fig. 2). *Histomonas meleagridis* was also seen in the liver as singly or in aggregates (Fig. 3). The liver parenchyma was largely destroyed and coagulative necrosis is evident on those remaining cells. *Histomonas meleagridis* in the intestine and liver had the typical diagnostic feature known as double-eyed lesion. The liver cells were necrotic and fatty degenerated and the parenchyma was infiltrated with inflammatory cells.

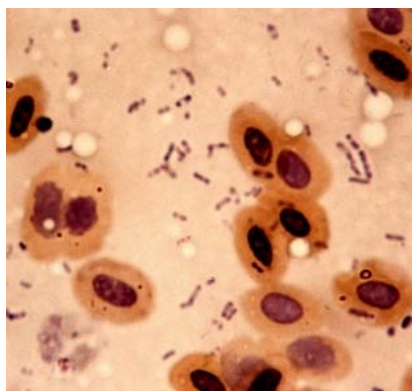


Fig. 1a: Fecal smear showing red blood cells and single, pairs and short chains of *Enterococcus* spp. Giemsa stain x1000

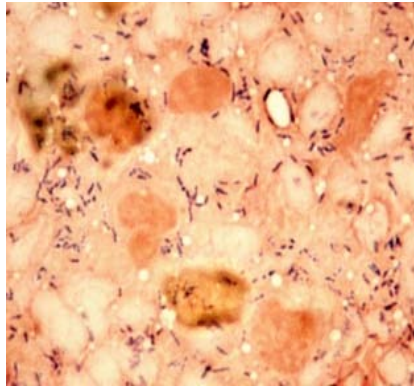


Fig. 1b: Fecal smear showing Gram positive single, pairs and short chains of *Enterococcus* spp. Gram stain x1000

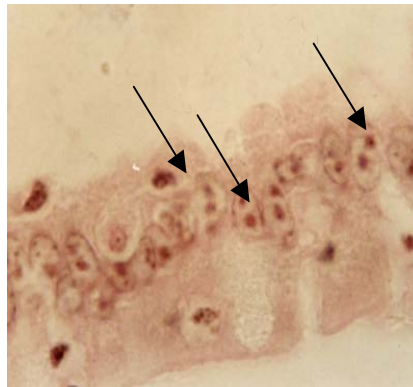


Fig. 2: Section of infected intestine showing *Histomonas meleagridis* inside enterocytes. H and E x 400

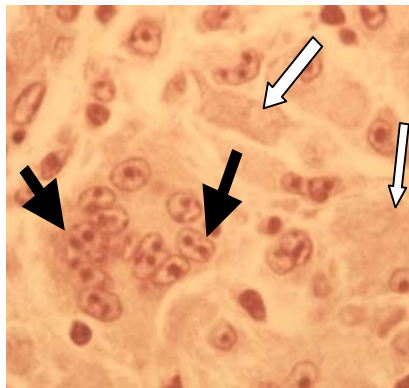


Fig. 3: Section of liver showing *Histomonas meleagridis* (black arrows) and necrotic liver cells (white arrows). H and E x 400

DISCUSSION

The researchers are not aware of a previous report describing concurrent *Histomonas* and *Enterococcus* infection in ducks. The high mortality experienced in the above reported outbreak was attributed to the concurrent *Enterococcus* infection of the intestine and *Histomonas* infection of intestine and the liver.

Concurrent infections in the avian species have been reported to aggravate the main clinical disease and increase mortality. Previous reports have shown that the clinical effects of *Histomonas* infection has been aggravated by concurrent infection with the protozoan *Eimeria* (McDougald and Hue, 2001) and by infection with a pathogenic bacterium (Esquenet *et al.*, 2003).

The rapid spread of the above disease in the duck flock and the high incidence of mortality denoted a direct transmission of infection. Transmission of *Histomonas meleagridis* is known to occur through the worm *Heterakis gallinarum*. In the above outbreak we have not seen this caecal worm in any of the postmortemed ducks. Many recent studies showed that both Histomoniasis and Enterococcosis could be transmitted by direct contact (McDougald and Fuller, 2005; Saif, 2001).

In conclusion the present research shows that concurrent infection of *Histomonas* spp. and *Enterococcus* spp. can result in a severe disease in ducks. Histopathological examination of infected liver and intestine is to be supported by microbiological investigation to give a diagnosis for the mixed infection.

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