MORTALITY IN MUSCOVY DUCKS (Cairina moschata) CAUSED BY Haemoproteus INFECTION

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MORTALITY IN MUSCOVY DUCKS *(Cairina moschata)* CAUSED BY *Haemoproteus* INFECTION

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Abstract: Haemoproteus infection was confirmed in white Pekin ducks *(Anas platyrhynchos domesticus)* following placement in a pond where severe mortality from a respiratory condition apparently caused by schizonts of *Haemoproteus* had occurred in muscovy ducks *(Cairina moschata).* Blood from one infected white Pekin duck was experimentally inoculated into five muscovy ducks; two of the five developed large numbers of schizonts in the endothelial cells and died. Illness did not occur in the white Pekin ducks.

INTRODUCTION

A variety of haematozoa *(Plasmodium, Leucocytozoon, Haemoproteus,* and *Lankesterella)* have been identified in the blood of wild ducks and other birds in North America.[1,3,4,5] These protozoa appear to have a commensal relationship with many of their hosts and are pathogenic only under special circumstances.[2,6] *Haemoproteus* sp. generally are reported as being nonpathogenic. This article reports mortality from *Haemoproteus* infection in a species of domestic duck.

CASE HISTORY

On 23 September, 1970, one ill and two dead, five-month-old muscovy ducks *(Cairina moschata)* were submitted to the Veterinary Services Laboratory at Brighton, Ontario, by their owner. The history indicated that 21 of 26 ducks of this age had died during the previous four days. A fourth duck was submitted two days later. Within a few days, the three remaining ducks died. The adults (one drake and eight females) remained healthy.

The young ducks had been housed with their mothers in an open shed and small fenced yard about 30 m from a 14-ha marshy, spring-fed pond. About three weeks before mortality began, they had been allowed access to the pond. The pond was the nesting and feeding site for a large number of free-ranging wild ducks and other birds. Turtles, snakes, fish, muskrats, beaver and other wildlife also inhabited the area. No evidence of mortality was noted in the free-ranging wild ducks or other species. Mosquitoes, blackflies and other insects were very numerous in the vicinity of the barnyard and pond. Domestic ducks have not been kept on these premises since 1972.

The owner described affected ducks as dying quickly, and many were not seen ill. Clinical signs in ill ducks were lameness a day or two before death and terminal respiratory distress. Ducks submitted alive to the laboratory appeared bright, but had laboured breathing; they died shortly after admission.

Necropsy indicated ducks were dying from respiratory failure due to edema in the lungs. Upon removal of the edematous lungs, the thoracic cavity filled with clear fluid. Other changes included increased pericardial fluid and hemorrhage in the heart. Liver, kidney, and spleen were swollen and firm.

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Blood smears from live and dead ducks did not reveal any significant abnormalities. No blood parasites were present.

Aerobic culture from tissues and organs was negative. Injection of ground lung into baby chicks did not produce disease or lesions in tissue or organs.

Histopathologic examination revealed large numbers of heterophil granulocytes, mononuclear cells, and erythrocytes in the lung. Air capillaries were occluded and obliterated by inflammatory cells (heterophils, monocytes and lymphocytes), edema and by enlarged endothelial cells which contained pseudocysts or schizonts and which were also blocking the blood capillaries of the lungs (Figure 1). Schizonts varied in size from 5-25 μm diameter, but the individual organisms (merozoites) were quite uniform in size (spherical and less than 1 μm diameter). Schizonts were so numerous that they appeared to be causing the congestion and edema by mechanical interference with circulation. Schizonts were common in endothelial cells in the spleen (Figure 2), heart and skeletal muscle, and smaller numbers were present in endothelial cells in blood vessels of other organs, including the brain. A moderate inflammation was present in the liver, spleen and heart. Heterophils and lymphocytes were present around portal triads in the liver (Figure 3) and considerable yellow pigment was present. Heart muscle fibres were separated by focal accumulations of heterophils, lymphocytes and macrophages (Figure 4).

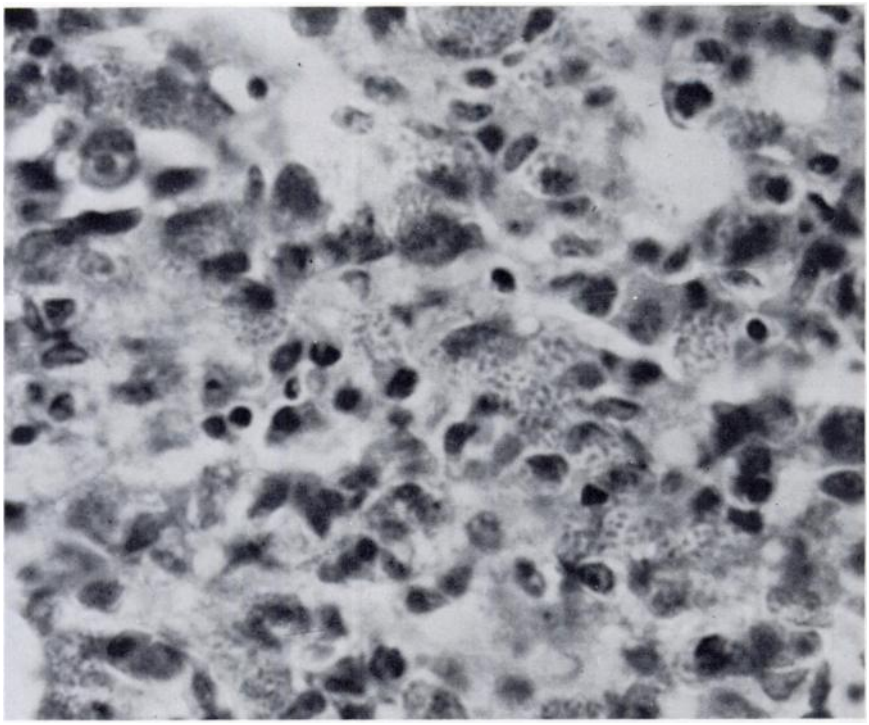

FIGURE 1. Severe pneumonitis and clusters of small coccoid organisms (schizonts) in the lung. × 1250.

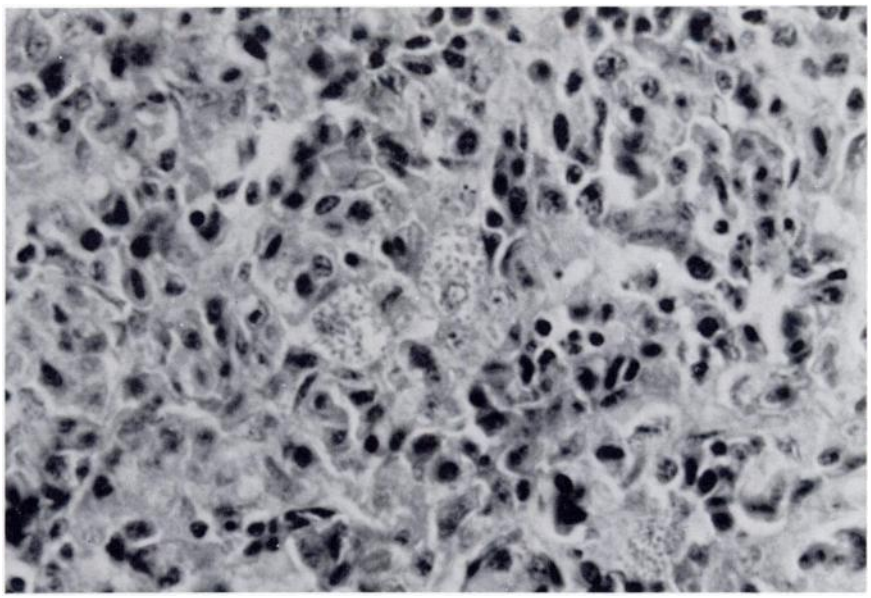

FIGURE 2. Schizonts in the reticuloendothelial cells of the spleen. × 630.

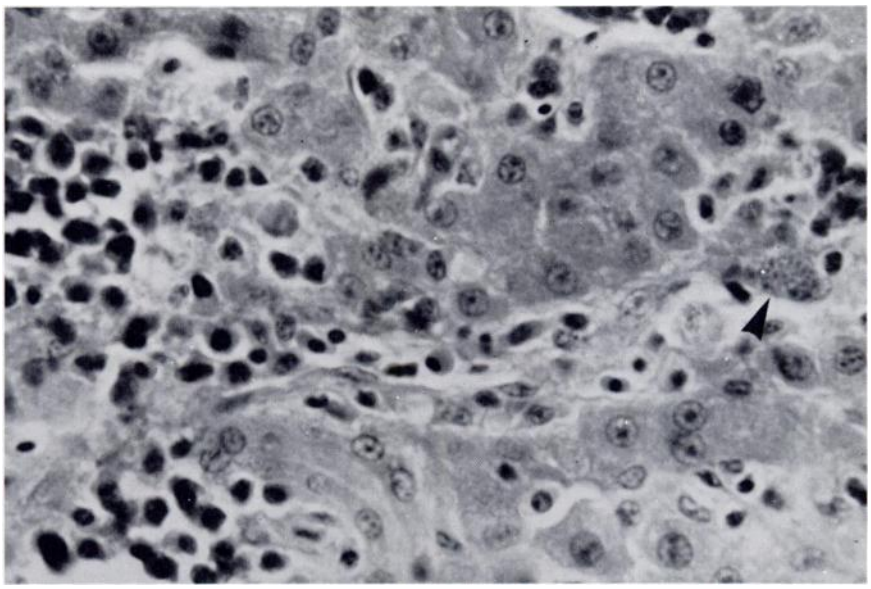

FIGURE 3. Pigment in Kupffer cells in the liver with foci of heterophils and lymphocytes around portal triads. Schizont present in endothelial cell (arrow). × 630.

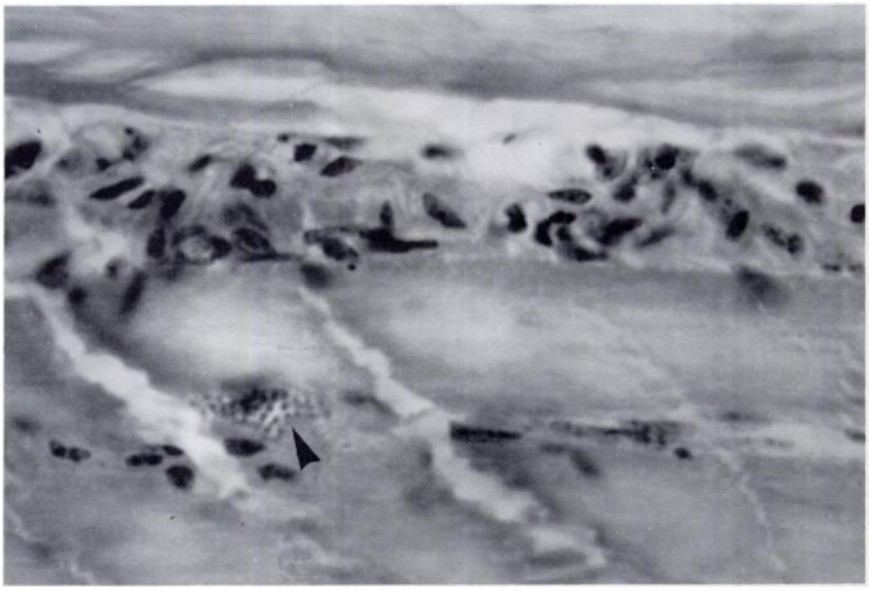

FIGURE 4. Heart muscle fibres separated by leucocytes. Small schizont present (arrow). × 630.

The following year, a similar outbreak of disease occurred on the same premises. Young ducks were kept housed until the middle of October and then allowed to go to the pond. Mortality started on 7 November 1971, and the ducks were housed again; however, seven died. In May, 1972, the 20 survivors were given access to the pond. Mortality started about four weeks later, and the ducks were housed. Five ducks died on that occasion. The ducks were allowed access to the pond three weeks later and there was no further mortality.

Lameness and dyspnea were noted in the ill ducks in both of these outbreaks. Ducks were submitted to the laboratory on both occasions. Necropsy and histopathological results were identical to the original outbreak.

In September, 1974, a similar mortality occurred at a separate location (90 km distant) in a group of muscovy ducks on a pond in association with free-ranging wild ducks. Necropsy results were similar to the outbreak described with identical organisms present in the tissues.

Death from respiratory failure has been reported in a duck in another part of Canada, and histopathologic examination revealed similar organisms in the lungs and other organs. (Riddell, C. University of Saskatchewan, Saskatoon, Sask. Pers. Commun.).

In 1970, 1971 and 1972, tissues from typical cases were examined by a number of general, avian and wildlife pathologists,[2] but none could identify the clusters of organisms although some

[2] Bennett, G. F. Memorial Univ. of Newfoundland, St. John's, Nfld.; Carlson, H. C. Univ. of Guelph, Guelph, Ont.; Fallis, A. M. Univ. of Toronto, Ont.; Griner, L. A. San Diego Zoological Garden, San Diego, Ca.; Herman, C. M. Patuxent Wildlife Research Center, Laurel, Md.; Karstad, L. H. Univ. of Guelph, Guelph, Ont.; van Dreumel, A. A. Ontario Ministry of Agric. & Food, Guelph, Ont. Pers. Commun.

reported having seen similar lesions and organisms in ducks dying from unknown causes (Mathey, W. J. Washington State Univ., Puyalleys, Wa.; Riddell, C. Univ. of Saskatchewan, Saskatoon, Sask. Pers. commun.). *Leucocytozoon, Plasmodium, Toxoplasma, Haemoproteus and Parahaemoproteus,* or bacterial infection were suggested as possibilities. Because the cause of the condition had not been determined, an investigation into the possible etiology seemed warranted.

EXPERIMENTAL

Eight two-week-old white Pekin ducks were obtained from a hatchery and on 9 July 1972, were released on the pond where the original outbreak had occurred. Stained blood smears were examined and packed cell volumes were performed on each duck before release and at 4- to 6-day intervals thereafter for two months. On 2 August (day 24), gametocytes of a *Haemoprotus*-like organism were present in the erythrocytes of several of these ducks. The organisms were small when they were first found and were about the length of the erythrocyte nucleus. They had a small nucleus and clear cytoplasm. On day 28, most organisms were much larger, displacing the erythrocyte nucleus and forming a crescent around the distal extremity of the nucleus. The cytoplasm contained yellow, refractile, rod-shaped pigment granules distributed evenly through the organisms. No trophozoites were found in the blood cells.

Parasites were found in all eight white Pekin ducks and persisted for over two months in the cells of a few. These ducks remained healthy; there was no evidence of anemia or illness throughout the investigation. Blood from several muscovy ducks raised on the farm was examined each time the white Pekins were sampled but no gametocytes were found.

One white Pekin duck that continued to have a high level of circulating gametocytes was returned to the laboratory, and one ml of its blood was injected intraperitoneally into an adult muscovy duck and each of her four one-month-old ducklings three times a week starting 1 September 1972. These ducks had not been to the pond or had contact with free-ranging wild ducks, and they were held at the laboratory throughout the test. Blood from these ducks was examined prior to the test and three times a week thereafter.

Following injection of blood from the white Pekin on 17 September, the adult female muscovy appeared ill. She died within four hours. Examination revealed she had bled to death from a ruptured liver. The liver and the spleen were enlarged and firm. Histopathologic examination revealed a moderate hepatitis with infiltration of heterophils around portal triads. Schizonts similar to those seen in the natural outbreak were found in the lung, heart muscle and other tissues.

Two days later, one duckling was found dead. Necropsy and histopathologic examination revealed lesions identical to those in the muscovy ducks dying from the natural disease; large numbers of organisms were present in the lung, spleen and other organs. The remaining ducklings showed no evidence of illness, and circulating gametocytes were not found over the following 30-day period.

DISCUSSION

The organism developing in the erythrocytes of the white Pekin ducks has been identified as *Haemoproteus (Parahaemoproteus) nettionis* (Bennett, G. F. Memorial Univ. of Newfoundland, St. John's, Nfld. Pers. Commun.), which is considered to be a non-pathogenic blood parasite and, in 1973, the organisms in the endothelial cells were tentatively identified as schizonts of *Haemoproteus* (Garnham, P. C. C. Imperial College Field Stn., Ashurst Lodge, Ascot, Berks., U.K. Pers. Commun.).

Gametocytes did not appear in the blood of muscovy ducks but the length of time between infection and the development of schizonts was similar to the development of gametocytes in the white Pekin ducks. Also, the disease was produced by injection of large quantities of blood (which might have contained very occasional merozoites (the gametocytes are not infective)) from a *Haemoproteus*-infected duck. Therefore, it is likely that the disease in the muscovy ducks is due to an increase of schizonts of *Haemoproteus* in endothelial cells and is, in fact, a lethal form of *Haemoproteus* infection in muscovy ducks.

The response of the muscovy ducks to *Haemoproteus* is similar to the response of zoo penguins to *Plasmodium*.[2] In *Plasmodium*, circulating gametocytes appear simultaneously with exoerythrocytic schizonts; this did not occur in the ducks. *Leucocytozoon* infection was eliminated on morphological grounds, and *Toxoplasma* on morphology and failure to infect baby chicks.

If the disease in the muscovy ducks is due to an increase of exoerythrocytic schizonts of *Haemoproteus*, muscovy ducks apparently have a different reaction from other ducks and free-ranging wild birds to *Haemoproteus*. Muscovy ducks are in the genus *Cairina* and it may be that there is a generic interference with development of gametocytes. It would be of interest to determine if other species of this genus are refractory to *Haemoproteus* gametocyte development.

It is difficult to say why *Haemoproteus* does not develop the gametocyte stage even when muscovys do not die from the acute disease. This study indicates that there is a build-up of schizonts but that further development to the gametocyte stage does not occur.

LITERATURE CITED

1. BENNETT, G.F., A.D. SMITH, W. WHITMAN and M. CAMERON. 1975. Haematozoa of the *Anatidae* of the Atlantic flyway. II. The Maritime Provinces of Canada. J. Wildl. Dis. 11: 280-289.
2. GRINER, L.A. 1968. *Plasmodium* infection in zoo penquins. Bull. Wildl. Dis. Ass. 4: 132-135.
3. HERMAN, C.M., J.O. KNISLEY and G.D. KNIPLING. 1971. Blood parasites of wood ducks. J. Wildl. Manage. 35: 119-122.
4. KARSTAD, L.H. 1965. A case of leucocytozoonosis in a wild mallard. Bull. Wildl. Dis. Ass. 1: 33.
5. KEYMER, I.F. 1969. Parasitic diseases. In: *Diseases of Cage and Aviary Bird*, Petrak, M.L., Ed. Lea & Febiger, Philadelphia, Pa., pp. 400-408.
6. MARKUS, M.B. and J.H. OOSTHUIZEN. 1972. Pathogenicity of *Haemoproteus columbae*. Trans. Royal Soc. Trop. Med. Hyg. 66: 186-187.

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