Atoxoplasmosis in Canaries

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Introduction

Atoxoplasmosis is a parasitic disease of canaries and other passerine birds caused by the coccidian parasite *Atoxoplasma serini* (previously known as *Isospora serini* and *Lankersterella garnhami*). Coccidia are single celled parasites that cause intestinal disease. Unlike other coccidia which complete their life cycle within the intestines, Atoxoplasma spread via the blood to internal organs and, therefore, cause more serious disease.

With support from the AFA (American Federation of Aviculture), we studied this disease in a group of naturally infected canaries that were generously donated by a concerned aviculturist. The main objectives of our investigations were to: 1) Characterize the clinical signs in live birds and the findings at necropsy to aid avian veterinarians in recognizing this disease, 2) Investigate methods of diagnosing infection in live birds, 3) Determine how long infected birds shed infectious eggs, and 4) Attempt to treat infected birds. These investigations took place during a four year study period.

Transmission

Atoxoplasma is spread when infected birds shed oocysts (eggs) in their droppings and these are consumed by other birds. We found that infected birds may intermittently shed large numbers of eggs over a long period of time. We counted oocysts in feces collected during 24 hour periods on a weekly basis and found that between 10 and 30,000 oocysts may be shed daily by a single bird. A group of infected birds maintained in suspended wire cages (to reduce the chance of reinfection) continued to shed large numbers of oocysts for over eight months, and then smaller numbers for several years. This is in contrast to other coccidial infections where the birds usually become immune to the parasite and stop shedding within months unless reinfected.

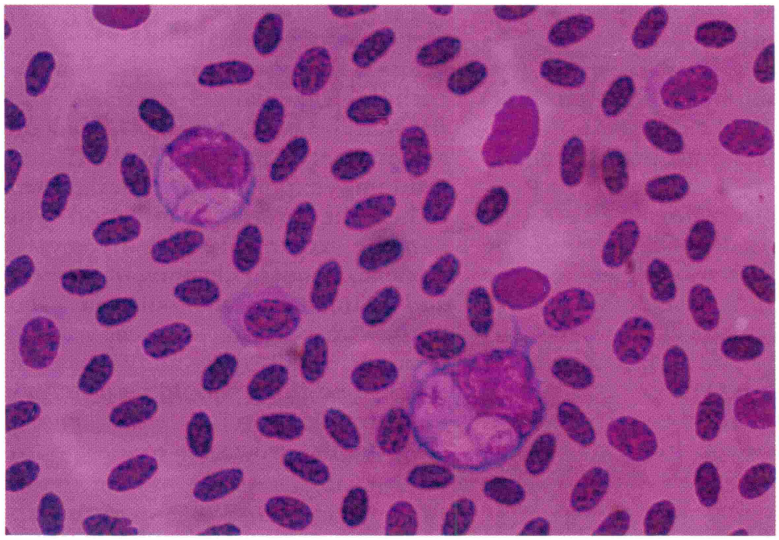
Atoxoplasma are common parasites of other passerine birds as well as canaries. E. Box[1] found that 95% of the house sparrows she tested in Texas were infected with Atoxoplasma. Oocysts from sparrows could not be transmitted to canaries, indicating that Atoxoplasma are probably highy host-specific and wild sparrows are probably not a significant risk for canary collections. We were unable to infect cockatiels with Atoxoplasma eggs collected from our canaries.

Clinical and Postmortem Signs

Atoxoplasmosis is commonly recognized in European canaries, but has rarely been reported in the United States, possibly because the parasite may be missed on routine examination of necropsy specimens. Illness and death are most pronounced in juvenile birds between two and nine months of age and may result in mortality of up to 80% of exposed birds[2] Clinical signs are nonspecific and include diarrhea and general signs of illness (e.g., decreased appetite, inactivity, weight loss, and ruffled feathers). Rarely are neurological signs observed. On physical exam, liver enlargement and dilated loops of bowel may be observed through the thin skin of the abdomen. Adults are commonly asymptomatic but shed infectious eggs (oocysts) in their feces. Adult birds may develop clinical signs following stress or reinfection after exposure to large numbers of oocysts.

The severity of the infection is deter-

Photo courtesy of H. John Barnes



Mononuclear white blood cells containing multiple atoxoplasma organisms in the cytoplasm.

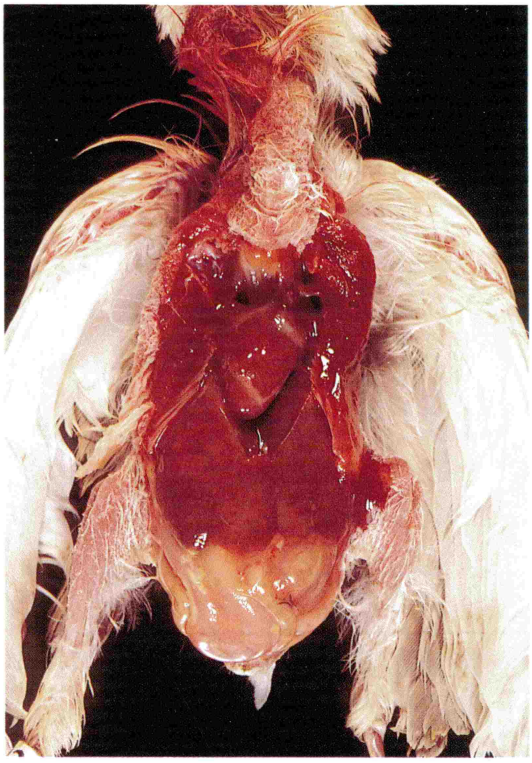


Photo by Keven Flammer, D.V.M.

Canary infected with atoxoplasmosis at necropsy demonstrating enlarged liver and swollen, fluid-filled intestines.

mined by the number of infectious eggs that are consumed[3] Gross necropsy signs include enlargement and variegated coloring of the liver and spleen, and swollen, fluid filled intestines (see Fig. 1). Histopathological changes also vary with the severity of infection[4] The most prominent changes are liver and spleen necrosis combined with a marked lymphocytic infiltration. This change is similar to what is seen in the disease lymphoid leucosis and in several cases birds infected with Atoxoplasma have been misdiagnosed as having lymphoid leucosis.

Diagnosis

Diagnosis in live birds. Atoxoplasmosis should be included in the differential diagnosis of canaries showing nonspecific, generalized signs of il-

lness, especially when signs are present in juvenile birds. Suspicions become stronger if Isospora-like oocysts (containing two gametocytes) are found on fecal examination. Atoxoplasma oocysts must be distinguished from those of another canary coccidian, *Isospora conaria.* This is best accomplished by measuring the oocysts with a microscope micrometer and determining the length/width ratio. Other differences between *Atoxoplasma serini* and *Isospora conaria* have been described[5,6]

We investigated the use of blood smears to diagnose infection. Specifically, we centrifuged the blood to separate the white blood cell layer (buffy coat) and smeared and stained it. We then counted 500 mononuclear blood cells and counted the number of Atoxoplasma organisms in these cells. In only five of 12 birds known to be infected could infection be identified by this method, and even in positive birds, only one to eight cells out of 500 contained the parasite. Therefore, it is possible to diagnose the infection via a blood test, but many positive birds will be missed.

Postmortem Diagnosis. This parasite may be missed with routine histopathology and confused with the disease lymphoid leukosis because of the large number of lymphocytes that invade infected livers and spleens. The most reliable way to confirm diagnosis is to examine Giemsa-stained impression smears from the liver and spleen. The parasite forms a reddish cytoplasmic inclusion in the mononuclear cells which accompany the inflammatory reaction to this parasite (see Fig. 2).

Treatment and Control

No drug has successfully eliminated Atoxoplasma from infected birds. Use of tetracyclines[4] and sulfas[2] may reduce the shedding of oocysts but the tissue infection persists. We tried treating infected canaries with two separate regimens and monitored treatment success by counting Atoxoplasma oocysts in the droppings before and after treatment. The first regimen used a combination of amprolium (2 ml/gallon drinking water — five days on, two days off, five days on for four treatment periods) and the antimalarial drugs chloroquine and primaquine given orally daily for three days and then every two to three days for a total of ten treatments. A separate group of birds was treated with spiramycin (150 mg/kg orally once daily for seven days). Neither treatment regimen stopped oocyst shedding.

Since the severity of infection and pathological changes correlate with the number of oocysts consumed and the number of parasites in the tissues, reducing exposure to oocysts will reduce the severity of infection. This can be accomplished by better hygiene, frequent cleaning of the nest, and maintaining the birds in cages with wire bottoms so feces can fall through. Finally, new additions to an established flock should be tested via fecal examination for the presence of Isospora-like oocysts.

Summary

Atoxoplasmosis should be considered as a possible diagnosis when canaries become sick with nonspecific signs, particularly when juvenile mortality is high. Suspicions become stronger if a large liver and dilated intestines are seen on physical exam and coccidia-type eggs are demonstrated on a fecal flotation of feces. In the live bird, a confirmed diagnosis is difficult but can be attempted by measuring the size of the parasite eggs or by identifying the parasite in lymphocyte blood cells in a buffy coat smear. In the dead bird, it is important to collect impression smears of the liver and spleen and examine these in addition to fixed tissues. If only fixed tissues are examined, this disease may be misdiagnosed as lymphoid leukosis. There is no treatment at this time, but infected flocks can be managed by improving hygiene and treating with sulfa drugs to decrease oocyst shedding. New birds should be carefully quarantined and their feces examined for parasites prior to entry into an established collection.

References

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