Journal of Zoo and Wildlife Medicine 20(3): 354–359, 1989

CEREBROSPINAL NEMATODIASIS IN MACAWS DUE TO *BAYLISASCARIS PROCYONIS*

Douglas L. Armstrong, D.V.M., Richard J. Montali, D.V.M., A. R. Doster, D.V.M. and Kevin R. Kazacos, D.V.M., Ph.D.

Abstract: Seven out of 10 birds in a mixed group of blue and gold macaws (*Ara arauna*), scarlet macaws (*A. macao*), and hybrid macaws developed ataxia, torticollis, and depression after being placed on an island exhibit accessible to raccoons (*Procyon lotor*). Larvae of *Baylisascaris procyonis* were found in cerebrospinal tissue from six of the macaws.

Key words: Blue and gold macaw, *Ara arauna,* scarlet macaw, *Ara macao,* cerebrospinal nematodiasis, *Baylisascaris procyonis.*

INTRODUCTION

Cerebrospinal nematodiasis has been reported in various mammalian and avian species in North America.[9,11] Larvae of the raccoon ascarid, *Baylisascaris procyonis,* have been incriminated in most cases of the disease.[11] The disease has been seen as an insidious problem in pheasants (*Phasianus colchicus*),[16] as outbreaks with high morbidity and mortality in chickens and quail (*Colinus virginianus*),[23,24] and as sporadic cases involving the chukar partridge (*Alectoris graeca*),[25] brush turkey (*Alectura lathami*),[13] emu (*Dromiceius novae-hollandiae*),[18] cockatiel (*Nymphicus hollandicus*),[22] and several songbird species.[3]

Few reports of this disease have involved animals from zoological collections,[18] despite the fact that free-ranging raccoons (*Procyon lotor*) are a common problem on zoo premises. One might expect that the presence of raccoons would result in cases of cerebrospinal disease involving zoo animals. The present report describes cerebrospinal nematodiasis due to *B. procyonis* affecting a mixed-species group of macaws at the Henry Doorly Zoo, Omaha, Nebraska.

From the Henry Doorly Zoo, 3701 South 10th Street, Omaha, Nebraska 68107-2200, USA (Armstrong); the Department of Pathology, National Zoological Park, Smithsonian Institution, Washington, D.C. 20008, USA (Montali); the Veterinary Diagnostic Center, University of Nebraska–East Campus, Lincoln, Nebraska 68583, USA (Doster); and the Department of Veterinary Pathobiology, Purdue University, West Lafayette, Indiana 47907, USA (Kazacos).

CASE REPORT

In 1986, a mixed group of three male and one female blue and gold macaws (*Ara arauna*), two male and two female scarlet macaws (*A. macao*), and two hybrid macaws of undetermined sex were placed on an island exhibit located in a pond at the Henry Doorly Zoo. The birds were placed on the island over an 8-day period in late June and early July 1986. All birds had been managed indoors at the zoo for more than 12 mo prior to the new introduction. The birds were of different ages and in apparent good health when transferred. Their diet consisted of a commercial seed and grain parrot food (Kay Tee Products, Inc., Chilton, Wisconsin 53014, USA) supplemented with bananas, oranges, apples, carrots, and a vitamin mix (American Breeder Supply Corp., P.O. Box 594, Omaha, Nebraska 68101, USA). The diet was available in a metal pan attached to one of the mulberry trees on the island.

The island exhibit was 22.9 m long and averaged 4.6 m in width. At its near point, the island was 3.0 m from the shoreline of the pond. The island substrate was dirt. The flora principally consisted of two medium-sized mulberry trees and numerous small willow trees. The island had not previously been used as an exhibit but was accessible to a variety of waterfowl exhibited on the pond as well as to feral wildlife.

Seven birds from the group developed

various symptoms of ataxia, torticollis, and depression over a 9-mo period. Onset was acute and severe in the first three birds, which developed clinical signs 35, 42, and 43 days after being introduced to the island. A variety of treatments were administered to the three initial cases including antibiotics, anti-malarials, anti-fungals, corticosteroids, oral and i.v. fluids, electrolytes, and dextrose. No bird showed improvement with or following any of the treatments. All three initial cases continued to eat although those with impaired coordination experienced some weight loss. The progress of the disease in these three birds varied. One bird became progressively ataxic and depressed over a 20-day period and died. Another bird survived for 29 days without weight loss and with little change in its neurological signs after initial onset of the disease. This bird was euthanized.

Hematological parameters and serum chemistry evaluations for all 10 birds were normal or difficult to interpret because of wide variations in values among individual birds. Blood parasites were not detected. Serum samples from seven birds were negative for *Chlamydia psittaci,* paramyxovirus, and eastern, western, and Venezuelan equine encephalitis viruses (National Veterinary Services Laboratory, Ames, Iowa 50010, USA). Lead level determinations performed on liver and kidney samples from two birds were between 0.30 and 0.58 ppm. Cerebrospinal fluid analysis on one bird revealed 1,025 WBC/mm^3 with reactive lymphocytes and macrophages, but no eosinophils were observed.

Complete necropsies were performed on the three birds. Gross necropsy of the macaws was unremarkable with the exception of reduced pectoral muscle mass and reduced body fat. Significant histopathologic lesions were limited to the brain and spinal cord. The lesions consisted of degenerative foci with accumulations of heterophils in the neuropil, perivascular lymphocytic cuffing, and glial cell proliferation, all with an asymmetric pattern (Fig. 1). Serial sections of the hindbrain in one of the first three birds revealed cross sections of a large (65 μm diameter) nematode larva identified as an ascarid (Fig. 2). A free-ranging peacock chick (*Pavo cristatus*) from the zoo grounds that developed neurologic signs similar to the macaws was euthanized and had similar lesions on histopathologic examination, although no larvae were found. This bird probably had the same infection.

The seven remaining birds were removed from the island to indoor holding cages 42 days after initial introduction. Following identification of the ascarid larva in tissues from one of the first three macaws affected, these seven remaining birds were treated with ivermectin (MSD AGVET, P.O. Box 2000, Rahway, New Jersey 07065, USA) at 0.4 mg/kg i.m. 76 days after removal from the island. Four of the seven birds went on to develop severe neurologic signs at 4, 10, 18, and 68 days after anthelminthic treatment. In these four birds, the initial onset of ataxia and depression was more subtle than that in earlier cases but was often followed by acute, severe exacerbation of clinical signs. The acute exacerbations occurred up to 9 mo after placement on the island and 7.5 mo after removal from the island. Three macaws of the group developed only very subtle or no clinical signs of infection. Of these three birds, one died because of surgical complications, one died following a cloacal prolapse, and one bird has survived 2 yr with no apparent clinical problems.

Complete necropsies were performed on the six birds that died or were euthanized from this chronically affected group. In addition to routine gross and histological examination of tissue, five birds of this group were examined for total numbers of larvae and larval location by compressing 1-g pieces of fresh brain and spinal cord tissue between glass plates and examining them for larvae under a light transmission dissecting microscope. Various skeletal muscles, visceral organs, and the eyes were examined for larvae. Pieces of tissue (25–60 g) were ground in a

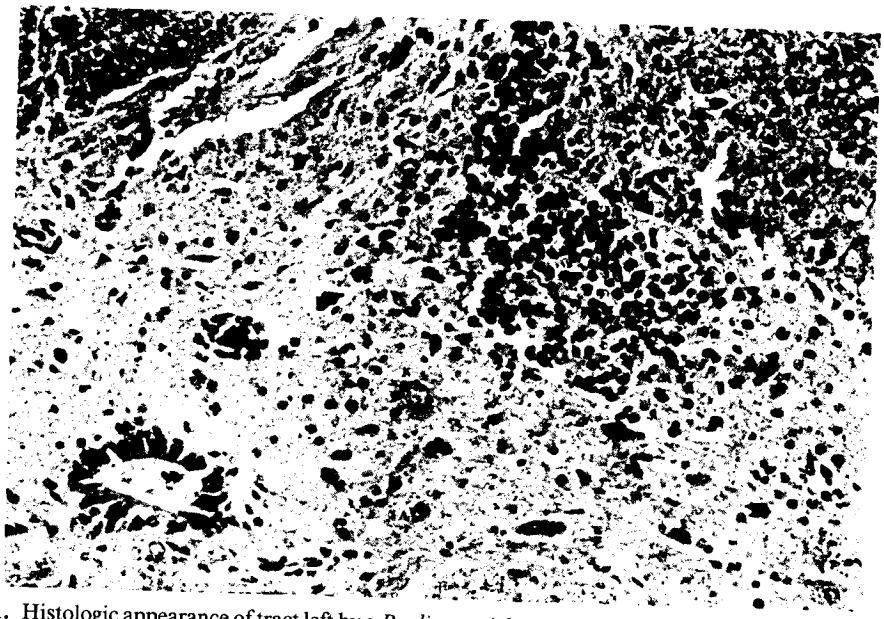

Figure 1. Histologic appearance of tract left by a *Baylisascaris* larva in spinal cord of macaw with cerebrospinal nematodiasis. Cells are primarily heterophils accumulated in a tract in the dorsal horn (upper right); central canal is lower left. H&E, ×80.

blender and then agitated in a digestion solution consisting of 1% pepsin and 1% hydrochloric acid in 0.85% saline for 1–3 hr at 37°C. Sediment was examined for larvae with a dissecting microscope.

Soil samples from the island exhibit, debris collected from the crotches of trees on the island, and material from the macaw food pan were examined for the presence of ascarid eggs using a centrifugal wash-flota-

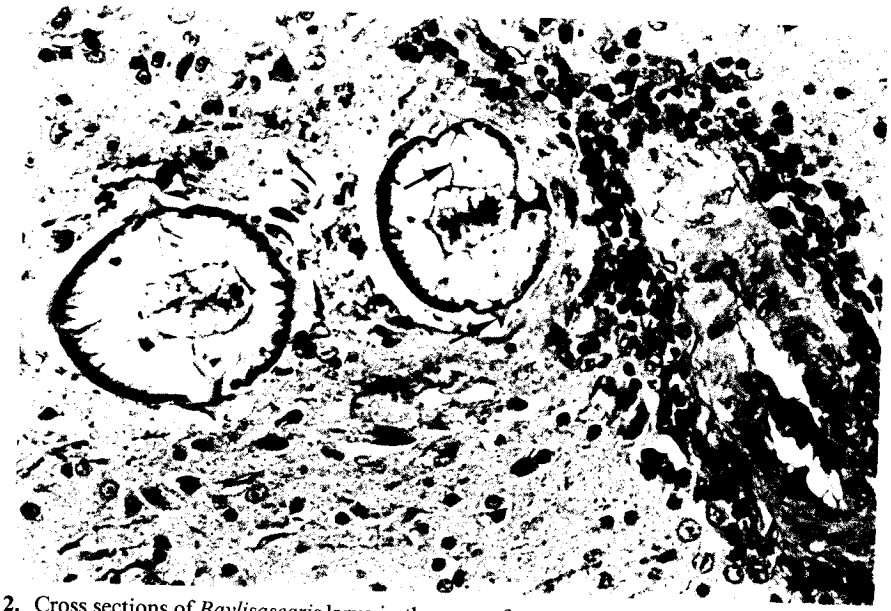

Figure 2. Cross sections of *Baylisascaris* larva in the pons of a macaw with cerebrospinal nematodiasis. Note lateral alae (lower arrow), prominent excretory gland cells (upper arrow) typical of mammalian ascarid larvae, and perivascular lymphoplasmacytic infiltrates at right. H&E, ×320.

tion method.[8] Four yearling raccoons captured on the zoo grounds were examined for *B. procyonis* at necropsy.

RESULTS

A single ascarid larva was found in the brain of each of four birds examined for larvae; these larvae were located in the cerebrum (two birds), cerebellum, and medulla, respectively. The fifth bird had two larvae located in the cerebrum. All of the birds except the most mildly affected one had evidence of larval migration in other areas of the brain. All of the larvae were alive despite prior treatment of the macaws with ivermectin. The larvae were recovered and fixed in hot alcohol–formalin–acetic acid fixative. No larvae were recovered from the skeletal muscles, visceral organs, or eyes.

The larvae averaged 1.67 mm in length and 69 μm in greatest width (range: 1.58–1.75 mm and 65–74 μm, respectively). They had prominent single lateral alae, a smoothly rounded anterior end, and a flexed tail tip with a capped appearance. In midbody cross sections, they had large alae and paired lateral excretory columns that were roughly triangular and smaller in diameter than the prominent, centrally located intestine (Fig. 2). Based on these characteristics, they were identified as a *Baylisascaris* species.[1,4,5,10,24]

The debris collected from the forks of trees and from the elevated macaw food pan contained small pieces of weathered feces. This material and several soil samples were positive for *Baylisascaris* eggs, all of which contained motile infective larvae. *Baylisascaris procyonis* adults were recovered from the small intestines of three of the four raccoons trapped on the zoo grounds.

DISCUSSION

This report describes a natural occurrence of cerebrospinal larval nematodiasis in macaws due to a *Baylisascaris* species. The species involved was considered to be *B. procyonis* based on the epidemiology and circumstances of the infection. *Baylisascaris* species of similar appearance and life cycles are present in the raccoon, skunk (*Mephitis mephitis*), and badger (*Taxidea taxus*),[11] and are capable of larva migrans infections in aberrant hosts. Skunks and badgers have been observed on the zoo property very rarely, whereas raccoons are common. Several feral animals, including raccoons, opossums (*Didelphis marsupialis*), squirrels (*Sciurus niger*), and rats, had been observed on the island in past years. Neither skunks nor badgers have been observed on the island. The presence of *Baylisascaris* eggs in the elevated food pan and in the forks of several trees on the island is consistent with the climbing ability and defecation habits of raccoons. Raccoons preferentially establish latrines in the forks of trees, at the bases of trees, and on raised horizontal surfaces.[11] The principle attractions to the island for feral mammals were the abundance of ripe mulberries in the trees and the macaw diet, which included some fresh fruit. These items would be very attractive to raccoons and consistent with their feeding habits.

The absence of larvae and/or lesions in extraneural tissues of the macaws is consistent with previous reports of this infection in birds.[3,13,16,18,19,22,25] This is different from the situation in mammals in which larvae are also commonly found in granulomas in skeletal muscles and visceral organs.[9,11] Considering the large size of the macaws, it is noteworthy that only one or two larvae were found in the brains of the five chronically affected birds. However, single *B. procyonis* larvae in the brain are known to kill small mammals and birds,[9–11] and a dose–response pattern for the production and duration of clinical disease has been seen in experimentally infected chickens.[19] Therefore, prolonged migration of one or two *Baylisascaris* larvae in the brain could have produced the chronic but progressive disease that was seen. The chronically affected macaws developed symptoms as much as 7.5 mo after the last possible exposure to infective eggs. Higher numbers of larvae may have entered the brain of the first three birds,

which were affected more acutely and severely, or the larvae may have entered a more "critical" area of the brain earlier in their random migration in these birds.

Central nervous system disease due to *Baylisascaris* species should be of significant concern to zoological gardens because of the wide range of animal species susceptible to this disease.[9,11] In addition to the avian species described previously in this paper, numerous mammal species may be affected including primates,[6,17,20] canids,[21,26] rodents, and other species.[2,7,12,15] The trend to develop more open, natural exhibit areas that are also more accessible to feral mammals enhances the probability of exposure. Zoos are frequently located in wooded urban areas where certain feral wildlife, particularly raccoons, are a common occurrence and often a nuisance. It is often difficult to control such animals or to prevent their access to zoo exhibits. Particular attention should be paid to raccoon latrine sites established in or near areas where susceptible species are also kept. These sites are a primary source of *Baylisascaris* infection. *Baylisascaris* eggs in such areas may remain viable and infective for years.[9,11]

Raccoons or skunks kept on exhibit or as pets pose a more direct threat of infection because of localized fecal contamination and the increased possibility of contact with infective eggs.[9,11] Extremely large numbers of *Baylisascaris* eggs may build up in and around cages, enclosures, and other areas where these animals are kept and have led to fatal infections in other species.[9,11] In one such case, infected skunks kept in a natural exhibit with several species of marmosets (*Callithrix* sp.) resulted in death of most of the marmosets from *B. columnaris* infection.[6] Humans are also susceptible to *Baylisascaris* infection, and *B. procyonis* has produced ocular and visceral larva migrans and central nervous system disease in humans.[4,5,9–11,14] Because of this, zoo personnel and others should take appropriate precautions around *Baylisascaris*-infected mammals and potentially contaminated areas. Recommendations for preventing *Baylisascaris* infections in humans and other animals and for decontaminating egg-contaminated areas have been detailed elsewhere.[9,11]

LITERATURE CITED

1. Bowman, D. D. 1987. Diagnostic morphology of four larval ascaridoid nematodes that may cause visceral larva migrans: *Toxascaris leonina, Baylisascaris procyonis, Lagochilascaris sprenti,* and *Hexametra leidyi.* J. Parasitol. 73: 1198–1215.

2. Dade, A. W., J. F. Williams, A. L. Trapp, and W. H. Ball. 1977. Cerebral nematodiasis in captive nutria. J. Am. Vet. Med. Assoc. 171: 885–886.

3. Evans, R. H., and B. Tangredi. 1985. Cerebrospinal nematodiasis in free-ranging birds. J. Am. Vet. Med. Assoc. 183: 1089–1090.

4. Fox, A. S., K. R. Kazacos, N. S. Gould, P. T. Heydemann, C. Thomas, and K. M. Boyer. 1985. Fatal eosinophilic meningoencephalitis and visceral larva migrans caused by the raccoon ascarid *Baylisascaris procyonis.* N. Engl. J. Med. 312: 1619–1623.

5. Huff, D. S., R. C. Neafie, M. J. Binder, G. A. DeLeon, L. W. Brown, and K. R. Kazacos. 1984. The first fatal *Baylisascaris* infection in humans: an infant with eosinophilic meningoencephalitis. Pediatr. Pathol. 2: 345–352.

6. Huntress, S. L., and T. Spraker. 1985. *Baylisascaris* infection in the marmoset. Proc. Annu. Meet. Am. Assoc. Zoo Vet. P. 78.

7. Jacobson, H. A., P. F. Scanlon, V. F. Nettles, and W. R. Davidson. 1976. Epizootiology of an outbreak of cerebrospinal nematodiasis in cottontail rabbits and woodchucks. J. Wildl. Dis. 12: 357–360.

8. Kazacos, K. R. 1983. Improved method for recovering ascarid and other helminth eggs from soil associated with epizootics and during survey studies. Am. J. Vet. Res. 44: 896–900.

9. Kazacos, K. R. 1983. Raccoon Roundworms (*Baylisascaris procyonis*). A Cause of Animal and Human Disease. Bull. 422, Purdue Univ. Agric. Exp. Station, West Lafayette, Indiana.

10. Kazacos, K. R. 1986. Raccoon ascarids as a cause of larva migrans. Parasitol. Today 2: 253–255.

11. Kazacos, K. R., and W. M. Boyce. 1989. *Baylisascaris* larva migrans. J. Am. Vet. Med. Assoc. In press.

12. Kazacos, K. R., and E. A. Kazacos. 1984. Experimental infection of domestic swine with *Baylisascaris procyonis* from raccoons. Am. J. Vet. Res. 45: 1114–1121.

13. Kazacos, K. R., E. A. Kazacos, J. A. Render, and H. L. Thacker. 1982. Cerebrospinal nematodiasis and visceral larva migrans in an Australian (Latham's) brush turkey. J. Am. Vet. Med. Assoc. 181: 1295–1298.

14. Kazacos, K. R., L. A. Raymond, E. A. Kazacos,

and W. A. Vestre. 1985. The raccoon ascarid. A probable cause of human ocular larva migrans. Ophthalmology 92: 1735–1743.

15. Kazacos, K. R., W. M. Reed, E. L. Kazacos, and H. L. Thacker. 1983. Fatal cerebrospinal disease caused by *Baylisascaris procyonis* in domestic rabbits. J. Am. Vet. Med. Assoc. 183: 967–971.

16. Kazacos, K. R., W. M. Reed, and H. L. Thacker. 1986. Cerebrospinal nematodiasis in pheasants. J. Am. Vet. Med. Assoc. 189: 1353–1354.

17. Kazacos, K. R., W. A. Vestre, and E. A. Kazacos. 1982. Experimental ocular larva migrans and cerebrospinal nematodiasis due to *Baylisascaris procyonis* in subhuman primates. Proc. 5th Int. Congress Parasitol. Pp. 261–262.

18. Kazacos, K. R., R. W. Winterfield, and H. L. Thacker. 1982. Etiology and epidemiology of verminous encephalitis in an emu. Avian Dis. 26: 389–391.

19. Kazacos, K. R., and W. L. Wirtz. 1983. Experimental cerebrospinal nematodiasis due to *Baylisascaris procyonis* in chickens. Avian Dis. 27: 55–65.

20. Kazacos, K. R., W. L. Wirtz, P. P. Burger, and K. S. Christmas. 1981. Raccoon ascarid larvae as a cause of fatal central nervous system disease in subhuman primates. J. Am. Vet. Med. Assoc. 179: 1089–1094.

21. Larson, D. J., and J. H. Greve. 1983. Encephalitis caused by *Baylisascaris* migration in a silver fox. J. Am. Vet. Med. Assoc. 183: 1274–1275.

22. Myers, R. K., W. E. Monroe, and J. H. Greve. 1983. Cerebrospinal nematodiasis in a cockatiel. J. Am. Vet. Med. Assoc. 183: 1089–1090.

23. Reed, W. M., K. R. Kazacos, A. S. Dhillon, R. W. Winterfield, and H. L. Thacker. 1981. Cerebrospinal nematodiasis in bobwhite quail. Avian Dis. 25: 1039–1046.

24. Richardson, J. A., K. R. Kazacos, H. L. Thacker, A. S. Dhillon, and R. W. Winterfield. 1980. Verminous encephalitis in commercial chickens. Avian Dis. 24: 498–503.

25. Sass, B., and E. J. Gorgacz. 1978. Cerebral nematodiasis in a chukar partridge. J. Am. Vet. Med. Assoc. 173: 1248–1249.

26. Snyder, D. E. 1982. Fatal cerebrospinal nematodiasis and visceral larva migrans in dogs experimentally infected with *Baylisascaris procyonis*. Proc. 34th Annu. Midwest Conf. Parasitol. P. 14.

Received for publication 3 April 1989.