Full Recovery from *Baylisascaris procyonis* Eosinophilic Meningitis

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Infection by *Baylisascaris procyonis* is an uncommon but devastating cause of eosinophilic meningitis. We report the first case-patient, to our knowledge, who recovered from *B. procyonis* eosinophilic meningitis without any recognizable neurologic deficits. The spectrum of illness for this organism may be wider than previously recognized.

Eosinophilic meningitis is defined by >10 eosinophils/mm^3 in the cerebrospinal fluid (CSF) or >10% eosinophils of the total CSF leukocyte count. In the United States, the most common cause of eosinophilic meningitis is the presence of a ventriculoperitoneal shunt, but worldwide it is infection by *Angiostrongylus cantonensis* (*1*). Other infectious causes of eosinophilic meningitis include *Toxocara* spp., *Gnathostoma spinigerum*, neurocysticercosis, and *Baylisascaris procyonis*. The latter is an intestinal roundworm endemic to the US raccoon population (*2*). *B. procyonis* has not been described in New Orleans but is known to occur in raccoons in northern Louisiana (D. Bowman, pers. comm.) and in the nearby states of Texas, Georgia, Oklahoma (*2,3*), and Mississippi (C. Panuska, unpub. data).

Previously, all reported human cases of *B. procyonis* eosinophilic meningitis have resulted in death or severe neurologic sequelae. We describe a patient with clinically apparent *B. procyonis* eosinophilic meningitis, who appears to have completely recovered. This patient's recovery supports the notion that a wide spectrum of clinical disease exists for this parasite and that it may be a more common human infection than previously recognized.

The Case

A 4-year-old boy from New Orleans, Louisiana, who had sickle cell disease and a history of a splenectomy, was admitted to our hospital with 1 day of headache, right arm pain, and emesis. He was alert and oriented; his oral temperature was 38°C; and physical examination found only mild upper extremity tremors, dysmetria, and bilateral extensor plantar response. His blood leukocyte count was 16,000/mm^3, with 12% eosinophils; blood cultures yielded no growth. During the next 3 days, his headache and vomiting worsened and ataxia developed. Magnetic resonance imaging of the brain, performed on day 5 of illness, demonstrated cerebellar edema (Figure). CSF analysis showed the following: 5 erythrocytes, 1,734 leukocytes/mm^3 (55% of which were eosinophils), protein 290 mg/dL, and glucose 53 mg/dL. The boy was given 1 dose of mannitol (0.25 g/kg) and a loading dose of dexamethasone (1 mg/kg), followed by 0.25 mg/kg dexamethasone every 6 hours for 2 weeks. On day 9, a parasitic infection was suspected, and albendazole (10 mg/kg every 12 hours for 5 days) was begun. The boy's history was negative for travel outside the United States, raw food consumption, household pets, developmental disability, or pica. His mother reported household rodent infestation and fecal droppings on the patient's bed; raccoons had been seen in the neighborhood, but the boy had not been directly exposed to them. Three days after corticosteroid therapy began, headache and vomiting stopped
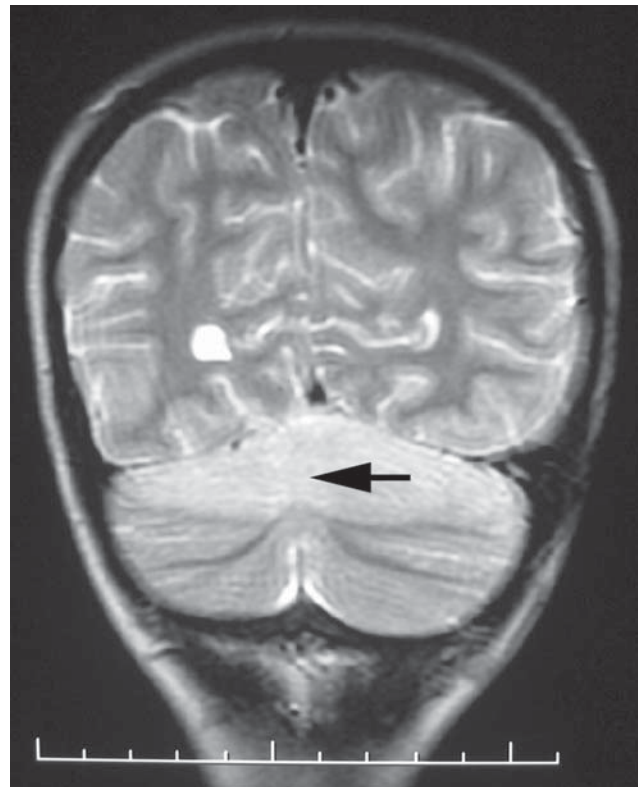

Figure. Coronal T2-weighted magnetic resonance imaging of the brain in a 4-year-old child with *Baylisascaris procyonis* eosinophilic meningitis. Arrow shows diffuse edema of the superior cerebellar hemispheres. Scale bar increments = cm.

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and only mild ataxia remained. Repeat magnetic resonance imaging and lumbar puncture on day 12 showed improvement in cerebellar edema and CSF eosinophilia (107 leukocytes/mm³ with 6% eosinophils). Upon discharge (day 16), the patient's only abnormal finding was mild dysmetria of the right hand. One month later he was asymptomatic, and results of a neurologic examination were within normal limits; 24 months later he remained well.

Serologic test results for *Toxocara canis* (Centers for Disease Control and Prevention, Atlanta, GA, USA), *Treponema pallidum*, *Mycoplasma*, Epstein-Barr virus, and lymphocytic choriomeningitis virus were all negative. Acute-phase and convalescent-phase serum specimens (obtained 10 days and 7 weeks after illness onset, respectively) were negative for *A. cantonensis* (Western blot performed at Mahidol University, Bangkok, Thailand) (*4*); both were positive for *B. procyonis* (ELISA performed at Purdue University, West Lafayette, IN, USA) with optical density (OD) values of 0.547 and 0.976, respectively (positive cut-off OD ≥0.250; K. Kazacos, pers. comm.). CSF (acute specimen only) was negative for *A. cantonensis* and *B. procyonis* (OD = 0.006). A stool sample was negative for ova and parasites.

Conclusions

Comprehensive reviews of the epidemiology and clinical features of *B. procyonis* infection have been recently published (*2,3,5,6*). The definitive host for this roundworm is the raccoon (*Procyon lotor*), which each day sheds millions of eggs in feces, which heavily contaminate parks and neighborhoods (*7*). Humans are infected by ingesting the eggs; the median age of infected humans is 13 months, reflecting the propensity of young children to explore the environment orally (*2,3*). Subclinical infection has been suggested by a study in Chicago, which found 30 (8%) of 389 children 1–4 years of age were seropositive for *B. procyonis,* although none had experienced symptoms (*8*). After *B. procyonis* larvae hatch, they penetrate the intestinal wall and disseminate; the ensuing manifestations depend on infecting dose, location of migrating larvae, and degree of inflammation produced (*2,3*). The larvae are not neurotropic, but some may reach the central nervous system and, because of their large size (1.5–2.0 mm), cause major tissue damage (*2,3*). All previously described 13 patients who had clinically apparent *B. procyonis* eosinophilic meningitis died or were left with severe neurologic sequelae (Table); to our knowledge, our patient represents the first to fully recover. *B. procyonis* could cause low-level infections with mild and nonspecific clinical manifestations, similar to covert toxocariasis, (*9,10*) that can go unrecognized. Our patient underwent an extensive evaluation because of his underlying sickle cell disease and the suspicion for a cerebrovascular event; otherwise, his eosinophilic meningitis may have been missed.

Table. Reported human cases of eosinophilic meningoencephalitis caused by *Baylisascaris procyonis**

Year of onset	Location	Age	Sex	Blood eos/ mm³, n (%)†	CSF eos/ mm³, n (%)†	Diagnostic method	Outcome
1975	Missouri	18 mo	Female	5,139 (30)	209 (80)	Serologic (cross-reacting)	Persistent weakness and spastic right arm and leg
1980	Pennsylvania	10 mo	Male	4,698 (27)	63 (68)	Autopsy, serologic	Death
1984	Illinois	18 mo	Male	3,700 (37)	220 (80)	Autopsy, serologic	Death
1990	New York	13 mo	Male	6,513 (39)	75 (60)	Serologic	Neurologic deficits, cortical blindness, brain atrophy
1993	Michigan	9 mo	Male	Unknown	Unknown	Serologic	Neurologic deficits, cortical blindness
1993	California	13 mo	Male	12,780 (45)	Not done	Brain biopsy, serologic	Neurologic deficits, blindness, seizures, brain atrophy
1996	Illinois	6 y	Male	605 (5)	2 (<1)	Serologic	Neurologic deficits, seizures
1996	Minnesota	13 mo	Male	7,035 (35)	3 (54)	Serologic	Death
1997	Minnesota	19 mo	Male	2,232 (18)	5 (4)	Serologic	Death
1998	California	11 mo	Male	3,111 (17)	1 (7)	Serologic	Neurologic deficits, seizures, profound visual impairment
2000	Illinois	2.5 y	Male	5,880 (28)	26 (32)	Serologic	Neurologic deficits, blindness, generalized spasticity
2000	California	17 y	Male	2,385 (15)	7 (37)	Brain biopsy, serologic	Death
2002	California	11 mo	Male	Unknown	Unknown	Serologic	Neurologic deficits, cortical blindness, seizures
2004‡	Louisiana	4 y	Male	1,920 (12)	954 (55)	Serologic	Full recovery

*Modified from references (*5*) and (*6*), with permission.
†Highest within first 5 days of admission; eos, eosinophils; CSF, cerebrospinal fluid.
‡Case reported in this article.

Confirmation of *B. procyonis* infection requires identification of the larvae in tissues. A biopsy specimen was not obtained from our patient because of his benign clinical course; his case thus remains probable, rather than confirmed. Probable cases can be ascertained by a rise in serum or CSF antibody, as detected by an ELISA (*11*) performed at the Department of Comparative Pathobiology, Purdue University. Our patient's acute-phase serum sample was moderately positive and the convalescent-phase titer increased, which supports *B. procyonis* infection. Stool testing, however, is not useful because in humans the parasite does not complete its life cycle and thus does not produce eggs (*3*).

Treatment with albendazole after egg ingestion but before the onset of symptoms can prevent development of clinical baylisascariasis (*2,10*). However, after central nervous system symptoms appear, whether albendazole confers clinical benefit or instead worsens outcome due to parasite death and the resultant inflammatory response is unclear. Because of the latter possibility, corticosteroids should be used (*3*). Whether our patient's favorable outcome was due to a low burden of infection (as suggested by mild symptoms and modest serum antibody levels), host factors, or prompt initiation of therapy (mannitol, corticosteroids, and albendazole) is not known.

In conclusion, eosinophilic meningitis is a syndrome with many causes; in the United States, an important cause to consider is *B. procyonis*. All previously reported cases of eosinophilic meningitis resulted in death or severe neurologic disability. We report the likely first case-patient who fully recovered from *B. procyonis* eosinophilic meningitis. Although the potential for long-term sequelae in this patient is unknown, his short-term recovery has been excellent, and clinical and radiologic improvements suggest resolution of the disease process. This case demonstrates that milder presentations of *B. procyonis* infection are possible and suggests that infection by this parasite may be more frequent than previously recognized.

Acknowledgments

Dr Pai is a fellow in pediatric hematology-oncology at Duke University in Durham, North Carolina, USA. Her main research interest is infections in immunocompromised persons.

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