Raccoon Roundworm (Baylisascaris procyonis) Encephalitis: Case Report and Field Investigation

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ABSTRACT. Baylisascaris procyonis is a common and widespread parasite of raccoons in the United States and Canada. With large raccoon populations occurring in many areas, the potential risk of human infection with B procyonis is high. We report a case of severe raccoon roundworm (B procyonis) encephalitis in a young child to illustrate the unique clinical, diagnostic, and treatment aspects, as well as public health concerns of B procyonis infection. Acute and convalescent serum and cerebrospinal fluid samples from the patient were tested for antibodies against B procyonis to assist in documenting infection. An extensive field survey of the patient’s residence and the surrounding community was performed to investigate raccoon abundance and to determine the extent of raccoon fecal contamination and B procyonis eggs in the environment. The patient evidenced serologic conversion, and the field investigation demonstrated a raccoon population far in excess of anything previously reported. There was abundant evidence of B procyonis eggs associated with numerous sites of raccoon defecation around the patient’s residence and elsewhere in the community. Because B procyonis can produce such severe central nervous system disease in young children, it is important that pediatricians are familiar with this infection. The public should be made aware of the hazards associated with raccoons and B procyonis to hopefully prevent future cases of B procyonis infection. Pediatrics 2000;106(4). URL: http://www.pediatrics.org/cgi/content/full/106/4/e56; larva migrans, eosinophilic meningoencephalitis, raccoons, Baylisascaris procyonis, leukoencephalopathy.

ABBREVIATIONS. CNS, central nervous system; VLM, visceral larva migrans; OLM, ocular larva migrans; NLM, neural larva migrans; ED, emergency department; MRI, magnetic resonance imaging; CSF, cerebrospinal fluid; DUSN, diffuse unilateral subacute neuroretinitis.

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Baylisascaris procyonis, the raccoon ascarid, is inherently pathogenic and is likely to produce severe central nervous system (CNS) disease. It is emerging as a significant cause of visceral larva migrans (VLM), ocular larva migrans (OLM), and neural larva migrans (NLM).1,2 In particular, pediatricians should be familiar with B procyonis because of its ability to produce devastating CNS disease in young children. We present a child with severe B procyonis encephalitis. This case illustrates the unique clinical, diagnostic, and treatment aspects as well as the public health concerns of B procyonis infection.

CASE REPORT

In August 1998, a previously healthy 11-month-old boy developed irritability and behavioral regression. Three days later, his parents brought him to the local emergency department (ED) because of progressive irritability and decreased activity. Except for minor irritability, there were no findings on examination, and the patient was discharged with a diagnosis of a viral syndrome. Two days later, the patient presented to the ED again. He had developed increased lethargy and markedly decreased interactions with his family. Notable findings included hypertonia, extensor posturing of his extremities, and lateral deviation of his right eye.

The patient lived in the suburban area of Pacific Grove, California, with his father, mother, and 5-year-old sister, who were all well. They had no pets. The parents noted that a neighborhood cat often frequented their property, and that the patient had played with a litter of 1-week-old puppies –2 weeks before presentation. In addition, many deer and at least 20 raccoons populated their property and surroundings. He had never been bitten or scratched by any of these animals. The patient often sat playing outside and had been observed to occasionally put stones in his mouth.

After 2 days at a local hospital, the patient was transferred to our institution for more extensive evaluation and management. On admission his general examination was notable for a temperate of 38.2°C, irritability, and lethargy. Ophthalmologic and neurologic signs were remarkable. He had a left gaze preference superimposed on an intermittent right exotropia with an afferent pupil defect on that side. Funduscopic examination revealed unilateral choriotretinal scarring with discrete focal lesions in a linear track-like configuration and mild optic atrophy of the right optic nerve (Fig 1). Motor examination revealed pleiotropic upper motor neuron signs including cortical thumbing and bilateral Babinski responses.

Laboratory tests were performed at the local hospital and at our institution (Table 1). Initial head computed tomography was unremarkable. A head magnetic resonance image (MRI) revealed small foci of enhancement at the left temporal lobe and left periventricular region frontally, along with overall patchy white matter abnormalities and decreased myelination for age. An electroencephalogram was abnormal with diffuse slow activity.

The patient was initially placed on ceftriaxone, erythromycin, and acyclovir. These were discontinued when blood and cerebrospinal fluid (CSF) cultures remained negative after 72 hours and CSF polymerase chain reactions for varicella and herpes simplex virus were negative. The ophthalmologic evaluation as above was...
consistent with diffuse unilateral chorioretinitis of at least 2 weeks duration. Because of this finding, the patient’s clinical presentation, and the significant raccoon exposure history, treatment with high doses of methylprednisolone (20 mg/kg/day) and albendazole (40 mg/kg/day) was begun on hospital day 4 for presumed *B. procyonis*. Both *Toxocara* and *Coccidioides* serologies were confirmed negative by hospital day 10. Extensor hypertonia was treated with various agents, including baclofen, clonazepam, and dantrolene. During the next several weeks, the patient’s clinical condition progressed to opisthotonic posturing with diffuse hypertonia and rigidity. Additional head MRIs during hospitalization demonstrated progressive white matter disease (Fig 2). Repeated ophthalmologic examinations revealed no change from the initial evaluation. Albendazole was continued for 4 weeks, and the steroid therapy was tapered over the 5 weeks of hospitalization.

One month after presentation, our patient manifested severe irritability and frequent extensor spasms. He exhibited dystonic posturing of the right side and frequent extensor posturing. He required a gastrostomy tube to feed. Two months after onset, he could turn toward his mother’s voice and fixate on objects. He could take feedings from a bottle, would intermittently hold up his head, and would occasionally smile. Four months after onset, the patient developed focal and myoclonic seizures, although his head control was improving, as were hypertonia and dystonia. He had become more active and would smile, laugh, or babble.

One and one half years later, the patient continues to have incomplete seizure control. He remains encephalopathic with improving responses to visual and auditory stimulation. He has up to moderate spasticity in his extremities with poor trunk and neck control, although he demonstrates slow progress. His ophthalmologic examination demonstrates profound visual impairment. There is light perception in the right eye and 20/100 vision in the left. There is a constant right exotropia with optic atrophy and unchanged chorioretinal scarring.

**METHODS**

**Baylisascaris Serology**

Serum and CSF samples were tested for antibodies against *B. procyonis* by indirect immunofluorescence using cryostat-sectioned third-stage larvae as antigen. Fourfold dilutions of serum (1:16–1:4096) or CSF (undiluted: 1:1024) were tested. Sections were blocked with 1:10 normal goat serum and reacted first with patient serum or CSF and then with 1:200 fluorescein isothiocyanate-conjugated affinity-purified goat antihuman immunoglobulin G (H + L) with minimal cross-reactivity to bovine, horse, and mouse serum proteins (Jackson ImmunoResearch, Inc, West Grove, PA).

All washes were performed using phosphate-buffered saline, and rinses with deionized water. Sections were examined using a Nikon Labophot-2 (Nikon Inc, Melville, NY) or Olympus BX-60 fluorescent microscope (Olympus America Inc, Melville, NY).

**Field Investigation**

A field investigation of the patient’s residence and of the surrounding community was conducted. Assessments were made of the raccoon population and of the extent of raccoon fecal contamination, especially the presence and location of raccoon latrines (sites of defecation). Raccoon fecal samples collected from latrine and several other sites were examined for *B. procyonis* eggs using a modified detergent wash-flotation procedure. *B. procyonis* eggs were identified based on their size and morphologic characteristics. Raccoons from the property were trapped, humanely euthanized, and examined postmortem for *B. procyonis* infection.

**RESULTS**

**Serology Results**

Serologic results are reported in Table 2. Serum from 2 positive controls, titered to 1:1024 and 1:4096. Serum from a negative control was negative, with weak, dull, uniform staining at 1:16 and no reaction at 1:64.

**Patient Residence**

Extensive evidence of raccoon activity and fecal contamination, including 21 latrine sites, were identified on the patient’s property and the adjacent vacant lot.

**Latrine and Soil Samples**

All fecal samples collected from the latrine sites on the patient’s property and the adjacent lot contained *B. procyonis* eggs. Many infective *B. procyonis* eggs were recovered from the soil sample from the child’s
swing set play area and from soil associated with several raccoon latrines. In addition, 27 raccoon latrine sites elsewhere in the community were sampled and 12 (44%) were positive for *B procyonis* eggs.

**Raccoons**

Eleven raccoons were necropsied, and all were found to be infected with adult or immature *B procyonis*. Subsequent to the case investigation, many raccoons were trapped and removed from the patient’s property and the adjoining lot. The existing raccoon latrines were cleaned up. A year later there were ongoing raccoon problems with over a dozen newly established raccoon latrines in the vacant lot.

**DISCUSSION**

*B procyonis* is a well-recognized cause of larva migrans, having produced fatal or severe CNS disease in over 90 species of mammals and birds in North America.6 The parasite is widely distributed in raccoons in North America, with the highest prevalence of infection (68%–82%) occurring in the Midwest, Northeast, and West Coast of the United States.5,6 In the San Francisco Bay Area, 60% to 70% of raccoons are infected.2,6 Infected raccoons shed millions of *B procyonis* eggs daily in their feces, and at warm temperatures these eggs can reach infectivity in as few as 11 to 14 days. The eggs are very resistant to environmental degradation and, given adequate moisture, can remain viable and infective for years.1,2,5,6

The primary risk factors for *B procyonis* infection are pica, particularly geophagia, and exposure to raccoons or contaminated environments. Children 1 to 4 years old are at the greatest risk of heavy infection.1,2,5,6,9–13 Larvae hatch in the small intestine and migrate via first the portal circulation and then the systemic circulation to multiple organ systems, including the liver, lungs, heart, eyes, and brain. The CNS may be invaded by 5% to 7% or more of larvae from ingested eggs.1,5 Larval migration occurs rapidly, as demonstrated in murine studies, which detected larvae in the eyes, brain, and somatic tissues in 3 days; in subhuman primates, they were observed in the eyes as early as 7 days.6,14,15 In addition to causing traumatic damage and necrosis, the larvae incite a potent inflammatory reaction, in which eosinophils play a major role. Migration halts when the immune system overtakes the larvae and encapsulates them within eosinophilic granulomas.1,2,5,9,10,16

NLM is the most apparent and significant form of *B procyonis* infection and results in an eosinophilic meningoencephalitis.1,2,5 The extent and severity of *B procyonis* NLM depend on the number of infective *B procyonis* eggs ingested and the severity of migration damage and inflammation in the brain. Human infection varies from asymptomatic or mild infection to severe disease with marked clinical signs, including sudden onset of lethargy, irritability, loss of motor coordination, weakness, and generalized ataxia, which can progress to opisthotonus, stupor, coma, and death.5,6,9–13 Severe NLM may be rapidly fatal and at the very least neurologically devastating.5,6,9–13 Clinical signs may develop as early as 2 to 4 weeks postinfection. OLM or diffuse unilateral subacute neuroretinitis (DUSN) causing unilateral visual loss may occur with CNS disease in heavy infections.6,12,14,15

*Baylisascaris* eggs or larvae are not passed in the feces of infected humans. Definitive diagnosis can only be made by identification of *B procyonis* larvae in tissues, although antemortem biopsies are rarely justified.2,6 Diagnosis is based on a combination of

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**TABLE 2. Serological Results**

<table>
<thead>
<tr>
<th>Samples</th>
<th>Hospital Day Collected</th>
<th>Titer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum</td>
<td>3</td>
<td>1:64</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>1:1024</td>
</tr>
<tr>
<td></td>
<td>158</td>
<td>1:1024</td>
</tr>
<tr>
<td>CSF</td>
<td>2</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>1:64</td>
</tr>
</tbody>
</table>

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Fig 2. Axial T2-weighted MRIs (TR: 2500 msec; TE: 80 msec) showing patchy T2 hyperintensity in the central white matter, particularly in the corona radiata. This abnormality progresses dramatically between the initial scan (top row: 8/31) and the follow-up 10 days later (bottom row: 9/10). TE indicates echo time; TR, repetition time.
history, clinical findings, neuroimaging features, and serologic testing. Signs of progressive CNS disease with peripheral eosinophilia, CSF eosinophilic pleocytosis, MRI white matter changes, and positive serology are most important to the diagnosis. Ophthalmologic examination may demonstrate migration tracks and other lesions of OLM/DUSN, and occasionally an intraocular larva, which can be identified morphologically. Baylisascaris procyonis infection is a possibility wherever raccoons reside. Raccoons are able to adapt to man’s environment and can be found in both rural and urban settings. When we reviewed the exposure history with our patient’s parents, they mentioned the many raccoons on their property. On further investigation, extensive raccoon fecal contamination was discovered on their property. Raccoons typically defecate in common areas (latrines), which, in forested areas, are usually found at the base of trees, in raised crotches of trees, and on large logs, stumps, rocks, and tree limbs. In urban/suburban areas, they also occur on woodpiles, decks, and other accessible sites. In this instance, numerous latrines were found on or adjacent to our patient’s property and elsewhere in the community, a further indication of the presence of large numbers of raccoons.

During our field investigation, we noted up to 30 raccoons per one quarter acre, far in excess of anything previously reported in the available scientific literature. To date, the maximum population densities of raccoons reported from other areas of the United States and Canada have been ~1 raccoon per .6 to 1 acre, with most reports documenting much lower densities than this (1 per 4–60+ acres). Factors contributing to the large raccoon population are the widespread availability of pet food inadvertently and even intentionally left outdoors, the presence of large numbers of denning sites, abundant water, a mild climate, and the absence of predators or epizootic disease outbreaks (eg, distemper or rabies) that would naturally reduce the population.

All animals, including pets, can harbor zoonotic pathogens, but in the case of raccoons and B procyonis, the danger is especially high. Few other wild animal species have the propensity to interact with and live freely in such close association with hu-

### Table 3. Summary of Baylisascaris procyonis NLM Cases

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Location</th>
<th>Presentation</th>
<th>Treatment</th>
<th>Disposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>*18 mo/F</td>
<td>Missouri</td>
<td>Irritability, hemiplegia</td>
<td>Piperazine citrate (65 mg/kg/d for 2 d)</td>
<td>Residual Weakness/spasticity</td>
</tr>
<tr>
<td>10 mo/M</td>
<td>Pennsylvania</td>
<td>Encephalitis</td>
<td>None</td>
<td>Deceased</td>
</tr>
<tr>
<td>18 mo/M</td>
<td>Illinois</td>
<td>Encephalitis</td>
<td>Thiaabendazole (50 mg/kg/d for 5 d)</td>
<td>Cortical blindness, hemiparesis, DD</td>
</tr>
<tr>
<td>13 mo/M</td>
<td>New York</td>
<td>Encephalitis</td>
<td>Thiaabendazole (50 mg/kg/d for 7 d); prednisone (2 mg/kg/d for 7 d); ivermectin (175 µg/kg for 1 d)</td>
<td>Deceased</td>
</tr>
<tr>
<td>*21 y/M</td>
<td>Oregon</td>
<td>Abnormal behavior, CNS disease; history of DD, geophagia/pica</td>
<td>None</td>
<td>Unknown</td>
</tr>
<tr>
<td>13 mo/M</td>
<td>Northern California</td>
<td>Encephalitis</td>
<td>None</td>
<td>Residual deficits, significant DD</td>
</tr>
<tr>
<td>11 mo/M</td>
<td>Northern California (this case)</td>
<td>Encephalitis</td>
<td>Albendazole (40 mg/kg/d for 28 d; tapering steroid combo (refer to case)</td>
<td>Residual deficits, significant DD</td>
</tr>
</tbody>
</table>

DD indicates developmental delay.

* Suspected case of Baylisascaris.
mans. The best method to control the raccoon popula-
tion is yet to be known. Parents and communities
should be aware of the increasing reports of large
raccoon populations in the United States and Cana-
da 8 and should be vigilant with children and
should take measures to prevent food sources (gar-
bage and pet food) from being readily available to
artificially support raccoons. Physicians should be
aware of the zoonotic infection risk these animals
pose, especially for very young children, to hopefully
help prevent future cases of B procyonis infection.

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