



## **Raccoon Roundworm Encephalitis --- Chicago, Illinois, and Los Angeles, California, 2000**

*Baylisascaris procyonis* (BP), a common roundworm found in the small intestine of raccoons, causes severe or fatal encephalitis (neural larva migrans [NLM]) in a variety of birds and mammals, including humans (1--8). BP also can cause human ocular and visceral larva migrans (1,2,9). Humans become infected with BP by ingesting soil or other materials (e.g., bark or wood chips) contaminated with raccoon feces containing BP eggs (2). Young children are at particular risk for infection as a result of behaviors such as pica and geophagia and placing potentially contaminated fingers and other objects (e.g., toys) into their mouths. This report describes two cases of BP encephalitis in residents of Chicago and Los Angeles and illustrates the importance of reducing exposure to raccoons and their feces in U.S. urban areas.

### **Chicago**

During July 2000, a boy aged 2½ years with a history of iron deficiency anemia and pica was admitted to a Chicago hospital with a low-grade fever of 8 days duration and increasing lethargy, irritability, and ataxia during the 3 days preceding admission. A diagnosis of encephalitis was made based on the clinical presentation and laboratory findings on admission, including peripheral eosinophilia (28% of 21,000 white blood cells/mm<sup>3</sup>), cerebrospinal fluid (CSF) eosinophilic pleocytosis (32% of 80 white blood cells/mm<sup>3</sup>), and diffuse slow waves on an electroencephalogram. Less than 24 hours after admission, the patient lapsed into a coma with opisthotonus and decerebrate posturing; magnetic resonance imaging (MRI) revealed abnormalities in the deep white matter of both cerebellar hemispheres. Other possible causes of encephalitis (e.g., herpes simplex; arboviruses and enteroviruses; lymphocytic choriomeningitis; measles; and bacterial, fungal, and parasitic infections [e.g., toxocariasis and cysticercosis]) were excluded based on direct examination, culture, serology, and polymerase chain reaction (PCR) testing of blood and CSF. Antibodies to BP were detected in CSF and serum specimens

by indirect immunofluorescence assay (IFA) (6,8) with titers increasing several fold and reaching high levels (1:1,024 in CSF and 1:4,096 in serum specimens) during the 4 weeks following admission. The child was treated with albendazole and corticosteroids, but his condition did not improve. After 4 weeks of hospitalization, he was transferred to a rehabilitation center where he stayed for several months. He then was sent home where he remains profoundly neurologically disabled and in need of continuous nursing care.

Eighteen days before admission, the child's parents had observed that he had dirt on his mouth while playing beneath a cluster of trees in a nearby yard in a Chicago suburb where raccoons are common. A field study conducted in September 2000 revealed several sites of raccoon fecal contamination positive for BP eggs in the yard. Infective BP eggs were recovered from soil and debris at the base of the tree cluster; mice infected with these eggs developed fatal encephalitis as a result of NLM.

### **Los Angeles**

In January 2000, a boy aged 17 years with an 8-year history of severe developmental disabilities and geophagia was admitted to a Los Angeles hospital comatose and with generalized hypertonia and hyperreflexia. His mouth was tightly clenched, his eyes wandered rapidly, and he responded only to painful stimuli. Two days before admission, he had a low-grade fever, drowsiness, and problems with coordination. Laboratory findings on admission included peripheral eosinophilia (15% of 15,900 white blood cells/mm<sup>3</sup>) and a CSF eosinophilic pleocytosis (37% of 19 white blood cells/mm<sup>3</sup>). He was treated with antibacterial, antiviral, antifungal, antiparasitic (albendazole), and antiinflammatory agents, but his condition did not improve. Tests on CSF and blood failed to identify an infectious agent. On examination by a pathologist, a brain biopsy revealed sections of a nematode consistent with *Baylisascaris* species. *Baylisascaris* IFA tested strongly positive with titers of 1:256 in CSF and 1:4,096 in serum specimens. The patient's condition deteriorated and he had progressive, deep white matter abnormalities of the brain on MRI. After a 2-month hospitalization, he was transferred to a long-term-care facility where he remained comatose until he died a year later.

The patient had resided in a group home for developmentally handicapped adolescents and adults in Los Angeles County. In February 2000, a field study conducted in the yard in which the patient regularly played revealed several sites containing raccoon feces; a sample of sandbox soil was positive for BP eggs. Multiple sites in the adjoining yard, to which he also had access, contained raccoon feces with BP eggs.

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### **Editorial Note:**

Including the two cases in this report, at least 12 cases of severe or fatal BP encephalitis have been identified since 1981 in the United States (California, Illinois, Michigan, Minnesota, New York, Oregon, and Pennsylvania) (2--8). Ten of the 12 cases occurred in children aged 9 months--6 years; eight of the children were aged <19 months. Cases of BP ocular larva migrans also have been identified (2,9).

Raccoons infected with BP inhabit most of the United States; the highest prevalence of BP infection in raccoons (68%--82%) occurs in the Midwest, Northeast, and on the West coast (1,2). Infected raccoons commonly shed millions of BP eggs daily in their feces, and the eggs usually embryonate to the infective stage in 2--4 weeks. The eggs are resistant to most environmental conditions and with adequate moisture can survive for years.

Humans become infected by ingesting infective eggs; from the gastrointestinal tract, the larvae migrate to various somatic tissues, viscera, the eyes, and the central nervous system (CNS). The severity of neurologic disease in humans varies depending on the number of eggs ingested and the number of larvae migrating in the CNS (1,2). Larvae in the CNS cause inflammatory reactions and tissue damage and can become encapsulated within granulomas.

A diagnosis of BP encephalitis should be considered in persons, especially children, with sudden onset of eosinophilic encephalitis and a history of potential exposure (e.g., possible ingestion of raccoon feces or contaminated soil). Diagnostic findings include CSF eosinophilic pleocytosis, peripheral eosinophilia, deep white matter abnormalities on MRI, and positive titers on serologic testing of CSF and serum. Because CNS damage can occur before symptom onset, treatment of symptomatic patients with antihelminthic or antiinflammatory drugs often will not improve outcome. Antihelminthic treatment (albendazole, 25--50 mg/kg/d for 10 days) started in 1--3 days of possible infection might prevent clinical disease by killing larvae before they enter the CNS (2). Immediate treatment is recommended in cases of probable infection.

The risk for BP infection is greatly reduced by avoiding direct contact with raccoons and their urban habitats, by removing access to food and potential denning sites, and by limiting exposure to areas and materials that might be contaminated by raccoon feces. Raccoons typically defecate at the base of or in raised forks of trees or on raised horizontal surfaces such as fallen logs, stumps, or large rocks. Raccoon feces also can be found on woodpiles, decks, rooftops, and in attics, garages, and haylofts. Feces usually are dark and tubular, have a pungent odor, and often contain undigested food items.

To eliminate BP eggs, feces and contaminated material should be removed carefully and burned, buried, or sent to a landfill, and care should be taken to avoid contamination of hands and clothes. Decks, patios, and other surfaces can be treated with boiling water. Newly deposited eggs take at least 2--4 weeks to become infective; therefore, prompt removal and destruction of raccoon feces will reduce risk for exposure and infection. Additional information about raccoon roundworm is available at <http://www.cdc.gov/ncidod/dpd/parasites/baylisascaris/default.htm>.

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