

## Raccoon Roundworm Infection Associated with Central Nervous System Disease and Ocular Disease — Six States, 2013–2015

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*Baylisascaris procyonis*, predominantly found in raccoons, is a ubiquitous roundworm found throughout North America. Although raccoons are typically asymptomatic when infected with the parasite, the larval form of *Baylisascaris procyonis* can result in fatal human disease or severe neurologic outcomes if not treated rapidly. In the United States, *Baylisascaris procyonis* is more commonly enzootic in raccoons in the midwestern and northeastern regions and along the West Coast (1). However, since 2002, infections have been documented in other states (Florida and Georgia) and regions (2). Baylisascariasis is not a nationally notifiable disease in the United States, and little is known about how commonly it occurs or the range of clinical disease in humans. Case reports of seven human baylisascariasis cases in the United States diagnosed by *Baylisascaris procyonis* immunoblot testing at CDC are described, including review of clinical history and laboratory data. Although all seven patients survived, approximately half were left with severe neurologic deficits. Prevention through close monitoring of children at play, frequent handwashing, and clearing of raccoon latrines (communal sites where raccoons defecate) are critical interventions in curbing *Baylisascaris* infections. Early treatment of suspected cases is critical to prevent permanent sequelae.

Despite expansion of the geographic distribution of *Baylisascaris procyonis* in the last 14 years (2) and probable increasing human exposure, baylisascariasis is likely an under-reported disease: only 22 documented cases were reported in the United States during 1973–2010 (3,4). Laboratory and clinical diagnosis can be challenging: there is no commercially available serologic test in the United States, and although identification of larvae in tissue or specimens is confirmatory, this is not always possible or practical (3). If it is not considered in the differential diagnosis, baylisascariasis can be missed.

*Baylisascaris procyonis* eggs are passed in raccoon feces and become infectious after weeks to months in the environment. Infection occurs when soil or materials contaminated with feces containing infectious *Baylisascaris procyonis* eggs are ingested. Young children are at particular risk for infection if they place fecally contaminated objects or fingers into their mouths or have syndromes such as pica or geophagia. Once ingested, larvae migrate through the brain (neural larva migrans), eye (ocular larva migrans), and other organs (visceral larva migrans) (1).

Because there is no commercially available serologic test for baylisascariasis, a history of raccoon exposure and a high index of clinical suspicion are necessary to make the diagnosis. Serologic testing at CDC using a recombinant antigen rBpRAG1 immunoblot assay can aid in diagnosis by detecting *Baylisascaris procyonis* antibody in cerebrospinal fluid (CSF) and serum (5); however, because the assay detects immunoglobulin G antibodies, it is not possible to distinguish current from previous *Baylisascaris* infection, and detected antibody might represent an earlier exposure. Diagnosis of *Baylisascaris* ocular larva migrans is based on visualization of an appropriately sized larva in the eye, with or without serologic testing.

Neuroimaging studies can aid in the diagnosis of baylisascariasis. Although not diagnostic, the presence of diffuse deep white matter changes on magnetic resonance imaging (MRI) have been suggested as characteristic of baylisascariasis (6). A definitive diagnosis of neural larva migrans is based on compatible clinical findings and positive antibody test for *Baylisascaris*.

*Baylisascaris* serologic testing results were reviewed for serum, CSF samples, or both, and submitted to CDC during 2013–2015. Physicians of patients in whom antibody to *Baylisascaris procyonis* had been detected in either serum or CSF provided clinical summaries, imaging data, and laboratory data. Because of the overlap in geographic distribution, pathogenesis and clinical presentation of *Baylisascaris procyonis* and *Toxocara spp.*, a common roundworm of cats and dogs, all samples were also tested for *Toxocara spp.* antibodies.

During May 2013–December 2015, six cases of *Baylisascaris* neural larva migrans and one case of *Baylisascaris* ocular larva migrans were identified among patients from six states (California, Ohio, Oklahoma, [recurrent or persistent infection later diagnosed in Arkansas], Massachusetts, Minnesota, and Virginia), whose serum or CSF samples tested positive by the rBpRAG1 *Baylisascaris procyonis* immunoblot assay (Table). All patients tested negative for *Toxocara* and all survived, although some were left with severe neurologic deficits.

### Case Summaries

**Oklahoma and Arkansas.** In May 2013, a female, aged 31 years with a history of Chiari malformation, and who

**TABLE. Year and state of diagnosis, age and sex, clinical and laboratory\* findings, and suspected exposure in seven cases of *Baylisascaris procyonis* infection — United States, 2013–2015**

Year	State	Age	Sex	<i>Baylisascaris procyonis</i> rBpRAG1 Ab result (source)	Clinical finding	Suspected exposure	Outcome	Toxocara Ab result
2013	Oklahoma, Arkansas	31 yr	Female	05/2013: positive (serum); 05/2013: positive (CSF); 07/2014: positive (serum); 09/2014: positive (CSF)	Eosinophilic meningitis	Pet raccoon	Survived; neurologic deficits	Negative
2014	Ohio	15 mo	Male	05/2014: positive (serum); 08/2014: positive (serum); 08/2014: negative (CSF)	Eosinophilic meningitis	Patient's father hunted raccoons and sold pelts	Survived; full recovery	Negative
2014	Massachusetts	32 yr	Female	12/2014: positive (serum)	Eosinophilic meningitis	Hiking in raccoon-prevalent area	Survived; full recovery	Negative
2015	California	63 yr	Male	06/2015: positive (serum); 06/2015: positive (CSF)	Eosinophilic meningitis	Working in raccoon-prevalent areas	Survived; neurologic deficits	Negative
2015	California	3 yr	Male	08/2015: positive (Serum); 08/2015: positive (CSF)	Eosinophilic meningitis	Living in raccoon-prevalent area	Survived; neurologic deficits	Negative
2015	Virginia	10 mo	Female	08/2015: positive (serum); 08/2015: positive (CSF)	Eosinophilic meningitis	Geophagia in raccoon-prevalent area	Survived; neurologic deficits	Negative
2015	Minnesota	7 yr	Male	12/2015: positive (serum)	Ocular larva migrans	Living in raccoon-prevalent area	Survived; full recovery	Negative

**Abbreviations:** Ab = antibody; CSF = cerebral spinal fluid; rBpRAG1 = recombinant *Baylisascaris procyonis* RAG1 protein.

\**Baylisascaris procyonis* rBpRAG1 Ab testing and *Toxocara* Ab testing done at CDC.

owned a pet raccoon, was evaluated in Oklahoma for headaches and generalized pain. Analysis of CSF was consistent with eosinophilic meningitis (60% eosinophils), and *Baylisascaris procyonis* antibody was detected in both blood and CSF. The pet raccoon was removed from the home, and the patient was treated with albendazole and corticosteroids for 3 weeks and clinically improved; however, 16 months later, she was reevaluated in Arkansas for worsening headaches, nausea, photophobia, and sensory deficits. *Baylisascaris procyonis* antibody was again detected in CSF and serum, and the patient was retreated with albendazole and corticosteroids. It was unclear if this episode was caused by a recurrent or persistent infection. A ventriculoperitoneal shunt was placed to correct increased intracranial pressure. She survived with persistent neurologic deficits including intermittent headaches, sensory changes on her right side, diplopia, and gait disturbances.

**Ohio.** In May 2014, a male, aged 15 months from rural Ohio was brought to the hospital with lethargy and seizures and was found to have eosinophilic meningitis based on CSF testing. Despite the family's report that the child had no exposure to raccoons, clinical suspicion for baylisascariasis was high, based on the elevated CSF eosinophil count, and the fact that the family lived in an area where raccoons were prevalent. CSF samples were negative for *Baylisascaris procyonis* antibody; however, a serum sample was positive. The child was treated with albendazole and corticosteroids and survived without neurologic complications. It was later discovered that the patient's father hunted raccoons and stored raccoon pelts in a garage, where the child often played.

**Massachusetts.** In December 2014, a female, aged 32 years from rural Massachusetts was admitted to the hospital with a

history of rapidly progressive right-sided sensory and motor deficits and decreased ability to concentrate. CSF contained 15% eosinophils but was negative for *Baylisascaris procyonis* antibody; however, serum was positive. All other microbiologic testing on CSF and serum was negative. The patient was hospitalized for several weeks. Large enhancing brain lesions found on MRI completely resolved with corticosteroid treatment alone (4). The patient showed a marked clinical improvement thereafter. Although no direct exposure to raccoons or raccoon feces was ever reported, the patient was known to hike in rural areas where raccoons are commonly found.

**California.** In June 2015, a male, aged 63 years from northern California was evaluated at a local hospital for a 2-week history of progressive memory impairment, loss of motor function, fatigue and confusion. He was found to have both peripheral blood and CSF eosinophilia (8% and 34%, respectively). He worked as a contractor, and his family reported that he rarely washed his hands before meals. Raccoons had been observed under his home and at his rural jobsite. *Baylisascaris procyonis* antibody was detected in both blood and CSF. The patient was treated with albendazole and corticosteroids for 6 weeks; 4 months after his diagnosis, he demonstrated partial recovery of cognitive and motor function (7).

In August 2015, a male, aged 3 years from California with a history of spastic paraplegia was admitted to the hospital with altered mental status and hypotonia and developed seizures while hospitalized. CSF analysis revealed 88% eosinophils and MRI findings were consistent with meningitis. Although no direct contact or evidence of feces ingestion was observed or reported, the child lived in a neighborhood where raccoons were common. Results of testing on both serum and

CSF detected *Baylisascaris procyonis* antibody. The patient was treated with albendazole and corticosteroids and was discharged to rehabilitation after a prolonged hospitalization. The patient suffered persistent neurologic deficits, including right-sided paralysis, incontinence, dysphagia, and apraxia.

**Virginia.** In August 2015, a female, aged 10 months from rural Virginia was found to have eosinophilic meningitis after being evaluated for altered mental status and seizures. The patient lived on a farm with her family and several dogs. There had been a witnessed episode of geophagia. *Baylisascaris procyonis* antibody was detected in both serum and CSF. She was treated with albendazole and corticosteroids and survived with substantial neurologic impairment. Although no specific raccoon exposure was reported, raccoons infected with *Baylisascaris procyonis* have been reported in Virginia (1).

**Minnesota.** In December 2015, a male, aged 7 years from rural Minnesota was evaluated at an ophthalmology clinic for a 1-week history of worsening vision in his right eye. Ocular examination and retinal imaging revealed a larva compatible in size with *Baylisascaris procyonis*. Routine blood tests showed no evidence of eosinophilia, and the patient had no neurologic abnormalities. He lived in an area where raccoons were common. Serum tests detected *Baylisascaris procyonis* antibody. CSF was not tested. The patient was treated with albendazole, and corticosteroids, and retinal photocoagulation laser therapy, and had near total recovery of his vision upon completion of therapy.

### Discussion

The geographic distribution, clinical presentations, exposure history, and sequelae reported in this case series are similar to those previously reported (3,8); however, unlike most case reports of *Baylisascaris procyonis* infection, there were no deaths in this series. The patients in this series all lived in areas where raccoons are common. In all six patients with neural larva migrans, substantial eosinophilia was detected in the CSF, which is often found in *Baylisascaris procyonis* central nervous system disease (8). In the single case of ocular larva migrans, a larva compatible in size with *Baylisascaris procyonis* was visualized on retinal imaging. In all cases, antibody to *Baylisascaris procyonis* was detected by rBpRAG1 *Baylisascaris* immunoblot assay in a serum sample, a CSF sample, or both. All patients were treated with recommended regimens for baylisascariasis and all seven survived, although four were left with serious neurologic complications as a consequence of their infection.

The findings in this report are subject to at least two limitations. First, detection of *Baylisascaris procyonis* antibody is not a confirmation of current or active infection, but rather reflects a history of exposure to the parasite at some point. Other disease processes might have caused or contributed to the

### Summary

#### What is already known about this topic?

*Baylisascaris procyonis*, predominantly found in raccoons, is a ubiquitous roundworm found throughout North America. Infection can result in fatal human disease or severe neurologic outcomes if it is not treated rapidly. Only 22 documented cases were reported in the United States during 1973–2010; little is known about its actual prevalence or varied clinical presentation.

#### What is added by this report?

During May 2013–December 2015, seven cases of baylisascariasis not already described in the literature were identified among patients in the United States through testing at CDC, including six cases of central nervous system disease and one of ocular disease. Laboratory and clinical information for each patient was gathered and reviewed in a case series to contribute to knowledge about *Baylisascaris procyonis* infection. All seven patients survived, although approximately half had residual neurologic sequelae.

#### What are the implications for public health practice?

Prevention of baylisascariasis through close monitoring of children at play, frequent handwashing, especially after working or playing outdoors, and clearing of raccoon latrines, remain essential intervention strategies in curbing *Baylisascaris procyonis* infections. Prompt treatment after possible exposure to *Baylisascaris procyonis* is critical in preventing the devastating sequelae of infection. Communities should be aware of raccoons living in their area and take precautions to avoid accidental infection with *Baylisascaris procyonis*.

clinical presentation of the patients described here. However, in the absence of alternative, confirmed diagnoses, and given a history of raccoon exposure with compatible symptoms and response to baylisascariasis treatment, the patients presented were most likely infected with *Baylisascaris procyonis*. Second, all illnesses in this series were identified from specimens tested at CDC, and might represent an underestimate of the actual number of illnesses. Additional, undiagnosed illnesses, or illnesses diagnosed with testing conducted elsewhere were not collected in this series.

Because of the severity of most recognized illnesses associated with this disease, prevention and treatment of *Baylisascaris procyonis* infection are critical. Transmission can be prevented by avoiding contact with raccoons and their feces. Handwashing after contact with soil or outdoor play, and educating children not to put soil in their mouths are important prevention strategies. In addition to raccoons, other species of animals, such as kinkajous (9) and skunks that might be kept as pets can serve as *Baylisascaris* hosts and shed eggs in their feces. Wild animals should not be kept as pets, and susceptible exotic animal hosts should be tested and treated by a veterinarian if indicated. Although uncommon, infected dogs can also shed *Baylisascaris procyonis* eggs, therefore regular testing and

deworming of dogs by a veterinarian is important. Avoiding and safely clearing raccoon latrines can minimize the risk for accidental infection as well.

Treatment after a possible exposure to *Baylisascaris procyonis*, such as ingestion of raccoon feces, oral exposure to objects contaminated with raccoon feces or playing or working near raccoon latrines, is crucial to prevent the devastating sequelae of *Baylisascaris procyonis* infection (3). Physicians caring for patients with suspected or confirmed ingestion of raccoon feces should consider preemptive treatment to inhibit larval migration before development of clinical manifestations. Treatment should begin as soon as infection is considered, even if consideration is based on clinical suspicion or epidemiologic history alone. Patients with suspected exposure to *Baylisascaris procyonis* should immediately be started on a course of 20–50 mg/kg oral albendazole per day for 10–20 days, while further diagnostic investigations are being conducted (6). Additional information on *Baylisascaris procyonis* prevention, diagnosis and treatment, and sending samples to CDC for testing is available at <http://www.cdc.gov/parasites/baylisascaris/>.

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#### References

1. Kazacos KR. *Baylisascaris procyonis* and related species. In: Samuel WM, Pybus MJ, Kocan AA, eds. Parasitic diseases of wild mammals, 2nd ed. Ames, IA: Iowa State University Press; 2001:301–41.
2. Blizzard EL, Yabsley MJ, Beck MF, Harsch S. Geographic expansion of *Baylisascaris procyonis* roundworms, Florida, USA. *Emerg Infect Dis* 2010;16:1803–4. <http://dx.doi.org/10.3201/eid1611.100549>
3. Graeff-Teixeira C, Morassutti AL, Kazacos KR. Update on baylisascariasis, a highly pathogenic zoonotic infection. *Clin Microbiol Rev* 2016;29:375–99. <http://dx.doi.org/10.1128/CMR.00044-15>
4. Chun CS, Kazacos KR, Glaser C, Bardo D, Dangoudoubiyam S, Nash R. Global neurologic deficits with baylisascaris encephalitis in a previously healthy teenager. *Pediatr Infect Dis J* 2009;28:925–7. <http://dx.doi.org/10.1097/INF.0b013e3181a648f1>
5. Rascoe LN, Santamaria C, Handali S, et al. Interlaboratory optimization and evaluation of a serological assay for diagnosis of human baylisascariasis. *Clin Vaccine Immunol* 2013;20:1758–63. <http://dx.doi.org/10.1128/CVI.00387-13>
6. Murray WJ, Kazacos KR. Raccoon roundworm encephalitis. *Clin Infect Dis* 2004;39:1484–92. <http://dx.doi.org/10.1086/425364>
7. Langelier C, Reid MJ, Halabi C, et al. *Baylisascaris procyonis*-associated meningoencephalitis in a previously healthy adult, California, USA. *Emerg Infect Dis* 2016;22:1480–4. <http://dx.doi.org/10.3201/eid2208.151939>
8. Gavin PJ, Kazacos KR, Shulman ST. Baylisascariasis. *Clin Microbiol Rev* 2005;18:703–18. <http://dx.doi.org/10.1128/CMR.18.4.703-718.2005>
9. CDC. Raccoon roundworms in pet kinkajous—three states, 1999 and 2010. *MMWR Morb Mortal Wkly Rep* 2011;60:302–5.