

Spinal cord involvement in a child with raccoon roundworm (*Baylisascaris procyonis*) meningoencephalitis

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Abstract A 14-month-old previously healthy boy developed progressively worsening neurological symptoms secondary to eosinophilic meningoencephalitis with myelitis caused by raccoon roundworm (*Baylisascaris procyonis*) infection. MRI demonstrated T2 hyperintensity and enhancement of the cerebral white matter, cerebellum and spinal cord. Prior case reports have described signal abnormality within the brains of patients with raccoon roundworm neural larva migrans (NLM). This is a unique case in which spinal cord involvement was established by imaging. Knowledge of this combination of imaging findings expands the known imaging phenotype of this noteworthy infection.

Keywords Raccoon roundworm · Meningoencephalitis · Myelitis

Introduction

Baylisascaris procyonis is an intestinal roundworm endemic to the raccoon population in North America. *B. procyonis* can cause neurological disease in numerous animals, including humans, known as *B. procyonis* neural larva migrans (NLM) [1]. We treated a progressively ill toddler who had developed eosinophilia and fever as well as white matter, cerebellar and spinal cord changes caused by *B. procyonis* infection. This case is notable for the unique spinal cord involvement, as demonstrated by MRI.

Case report

A previously healthy 14-month-old boy was admitted to the hospital for progressive motor weakness over the course of 3 weeks and fever prior to admission. He suffered transient episodes of unsteadiness of gait along with slumping to one side, intention tremors of the hands and periods of staring. His symptoms progressed, lethargy developed, and he was admitted to the hospital. On admission, the toddler had meningismus, mental status changes, mild spastic paraparesis and a faint reticular rash. There was no evidence of chorioretinitis. His CBC showed marked eosinophilia and his CSF demonstrated pleocytosis with eosinophilia. The MRI demonstrated patchy T2 hyperintensity within the periventricular white matter and within the corpus medullaris of the cerebellum (Figs. 1 and 2). Enhancement was seen of the periventricular white matter, centrum semiovale (Fig. 3), cerebellum (Fig. 4) and leptomeninges. The MRI

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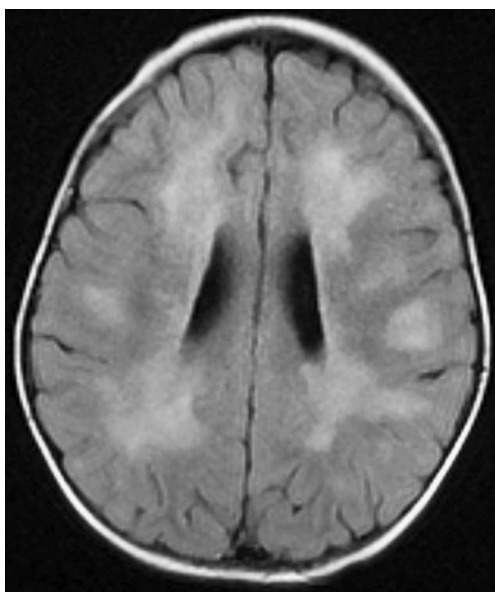


Fig. 1 Axial T2-W FLAIR MR image shows extensive T2 signal prolongation within the periventricular white matter and centrum semiovale

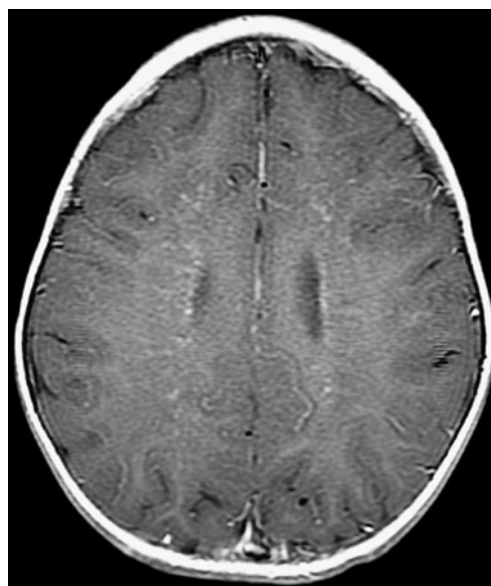


Fig. 3 Axial T1-W post-gadolinium MR image demonstrates both punctate and linear enhancement of the deep white matter of both cerebral hemispheres and mild leptomeningeal enhancement

of the spine demonstrated two areas of T2 signal hyperintensity within the cervical and thoracic spinal cord, respectively, without spinal cord enlargement (Fig. 5). There was mild enhancement of the involved area within the thoracic cord (Fig. 6) and nerve roots of the cauda equina. Based on the clinical findings and laboratory results, as well as epidemiological findings suggesting environmental rac-

coon exposure, he was presumptively diagnosed with eosinophilic meningoencephalitis, likely caused by the raccoon roundworm *B. procyonis*. He was treated promptly with corticosteroids and albendazole and discharged from the hospital 12 days later. Serological testing with ELISA and Western blotting at Purdue University subsequently confirmed infection with *B. procyonis*.

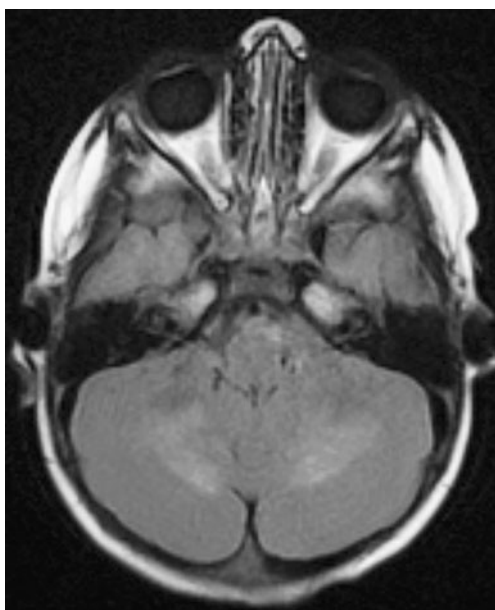


Fig. 2 Axial T2-W FLAIR MR image shows T2 signal prolongation within the corpus medullaris of the cerebellum

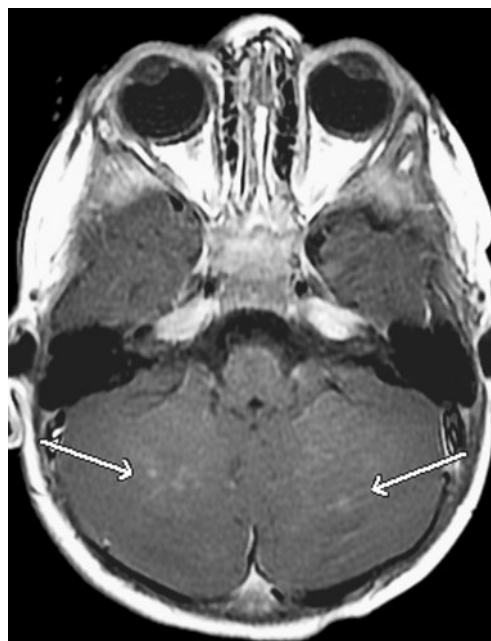


Fig. 4 Axial T1-W post-gadolinium MR image demonstrates punctate enhancement within the cerebellum (arrows)



Fig. 5 Sagittal T2-weighted MR image of the spinal cord demonstrates two areas of T2 signal prolongation centered at C4 and T5, respectively (arrows). No spinal cord enlargement is present

Discussion

B. procyonis is endemic in North American raccoons, with a reported prevalence of 68–82% in the Midwest, Northeast

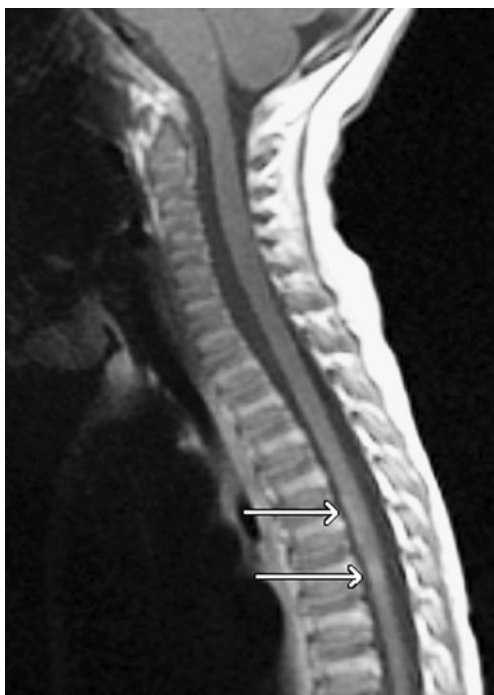


Fig. 6 Sagittal T1-W post-gadolinium MR image of the spine demonstrates mild enhancement of the spinal cord at T5 (arrows) and no enhancement within the cervical cord

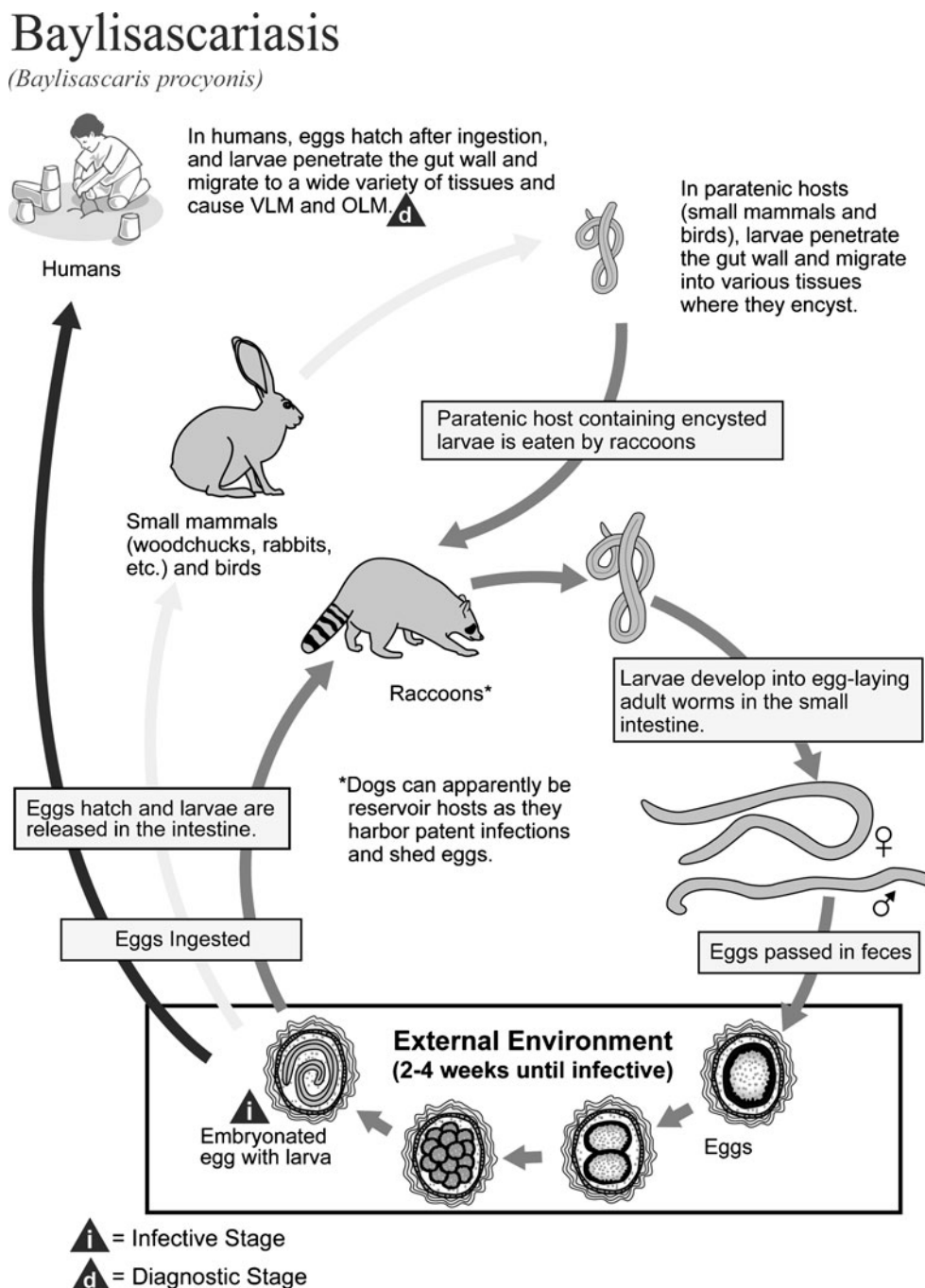
and West Coast populations [1, 2]. Infected raccoons can shed millions of *B. procyonis* eggs in their stool each day and these eggs are resistant to environmental degradation. Given adequate moisture, infective eggs can last several years in soil [1, 2]. Infected soil is associated with communal raccoon defecation sites termed “latrines” located on or in logs, sandboxes, wood piles and barns [1, 2]. Humans become accidental paratenic hosts of *B. procyonis* by ingestion of infective eggs. Upon ingestion, infective *B. procyonis* eggs release larvae that migrate to various viscera within the human body including the central nervous system (CNS) and eyes (Fig. 7). Infants are at the greatest risk for severe infection because of their potential for accidental ingestion of a large number of eggs, either by direct ingestion of raccoon feces or infected soil or by oral exploration of contaminated toys and other materials [1–7]. Older children are also at risk from outdoor play in contaminated areas [1, 2].

B. procyonis NLM can affect both pediatric and adult populations. However, most cases involve very young children, usually younger than 2 years [1–7]. Patients with *B. procyonis* NLM typically present with CSF and peripheral eosinophilia, encephalitis and sometimes neuroretinitis [1–7]. In our case, myelitis was also observed. The clinical presentation of NLM disease is variable and is related to the dose of eggs ingested as well as the extent and location of migrating larvae within the CNS and the degree of ensuing inflammation, degeneration and necrosis within the brain and spinal cord [1, 2, 4]. Most reported cases of *B. procyonis* NLM have described fatal outcomes or survival with severe neurological sequelae [1–4, 7]. More recent reports suggest a better outcome in patients promptly treated with albendazole and steroids [5, 6].

Diagnosis of *B. procyonis* NLM is based on the clinical CNS presentation, laboratory findings of peripheral and CSF eosinophilia, MR imaging, epidemiological findings of raccoon exposure and positive serology. Biopsy with identification of larvae in a CNS specimen is considered definitive [1, 2].

The neuroradiology findings in this case are in keeping with known imaging manifestations of *B. procyonis* meningoencephalitis, characterized by T2 signal hyperintensity within the periventricular white matter and cerebellum. Enhancement of the white matter and cerebellum was seen in our patient, a feature that has been variably reported in prior cases [5] and might be related to the timing of imaging relative to initiation of steroid treatment. Our patient was imaged prior to receiving steroid therapy. The MRI findings of the spinal cord were consistent with myelitis. The signal alterations seen in the spinal cord were presumably caused by migrating larvae. In a study of squirrel monkeys inoculated with infective *B. procyonis* eggs, numerous larvae and migration tracks were demon-

Fig. 7 This illustration depicts the life cycle of *Baylisascaris procyonis* (picture courtesy of the U.S. Centers for Disease Control, Alexander J. da Silva, PhD and Melanie Moser)



strated within the brain and spinal cord on necropsy [8]. Autopsy findings in a fatal case of *B. procyonis* in a toddler revealed larvae in the spinal cord with and without inflammation [3]. The leading diagnostic consideration for a patient from the northeastern United States with CSF and peripheral eosinophilia and meningoencephalomyelitis is a parasitic infection from either *B. procyonis* or *Toxocara canis*. *Coccidioides immitis* and *Gnathostoma spinigerum* can cause meningoencephalitis with spinal cord involvement, but imaging features differ from those observed in our case. Coccidioidomycosis is a fungal infection that is not

commonly seen in immunocompetent patients in the northeastern part of the USA [4]. CNS imaging of *Coccidioides immitis* meningitis usually reveals dense basal enhancement, hydrocephalus, brain infarctions and occasionally subarachnoid hemorrhage. Infection secondary to *Gnathostoma spinigerum*, an intestinal parasite of cats and dogs endemic to southeast Asia, China and Japan [4], is characterized by intracerebral and subarachnoid hemorrhage, myelitis and radiculitis, features not observed in our case.

In summary, *B. procyonis* is a noteworthy parasitic infection endemic to the United States and Canada that can

severely affect the central nervous system, especially in children. Previous reports elucidate the intracranial MRI findings of *B. procyonis* meningoencephalitis. This is the first case that also describes spinal cord involvement on MRI. Awareness of this combination of findings expands the known imaging phenotype and suggests that spinal imaging should be considered in cases of suspected parasitic meningoencephalitis.

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