**CASE REPORT**

**A child with raccoon roundworm meningoencephalitis: A pathogen emerging in your own backyard?**

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Raccoons are common in North America, and often live in close proximity to people, particularly in cities such as Toronto, Ontario, where their density reaches over 100 raccoons per square kilometre (1). Approximately 50% of raccoons in Ontario carry the roundworm *Baylisascaris procyonis* asymptptomatically in their intestinal tract and shed millions of eggs in their stool (2,3). These eggs are extremely resistant to disinfection and remain infective in the environment for years. If eggs are accidentally ingested by a human, or another susceptible host, larval infection can lead to devastating neurological disease and death (2,4). We describe the clinical presentation of a Canadian child with *B procyonis* meningoencephalitis and ocular larva migrans. The case is noteworthy in demonstrating neurological improvement in association with early administration of albendazole and corticosteroids.

**CASE PRESENTATION**

A previously healthy, active and communicative seven-year-old boy with autism presented to hospital in Toronto with increasing lethargy and anorexia. His symptoms began insidiously two weeks before admission. Two days before admission, when the patient was seen in the emergency department, he was drowsy, but easily arousable. A marked peripheral eosinophilia (white cell count 11.6×10\(^9\)/L; eosinophils 2.78×10\(^9\)/L) was the only laboratory abnormality identified. He was treated for dehydration and discharged home. The following day, he returned to hospital with increasing lethargy and was refusing anything by mouth. He was incontinent of urine and appeared to have abdominal pain. He had no fever, headache, convulsions or rash. His Glasgow Coma Scale score was 8 (eyes = 3, verbal = 2, motor = 3). He exhibited decorticate posturing, hyperreflexia, up-going plantar reflexes bilaterally, and a right gaze preference. He had no meningismus or fever. The peripheral eosinophilia had increased (eosinophils 4.23×10\(^9\)/L). Cerebrospinal fluid (CSF) analysis revealed a white blood cell count of 22×10\(^9\)/L with 30% eosinophils. The CSF protein and glucose levels were normal. Magnetic resonance imaging (MRI) of the brain demonstrated abnormal enhancement in the cortex and cerebellum (Figure 1). Fundoscopy revealed small bilateral subretinal infiltrates, more prominent in the left eye (Figure 2).

There was no history of recent travel outside the country, and the child had no pets. However, the parents reported the presence of raccoons and raccoon latrines in their backyard. The child often played in an open sandbox in the backyard and had a habit of putting his hands in his mouth.

Given a strong suspicion of *B procyonis* infection, albendazole 25 mg/kg/day and methylprednisolone 30 mg/kg/day were initiated within 48 h of presentation. *B procyonis* ELISA was strongly positive in both serum and CSF (Table 1). The convalescent CSF titre increased fourfold (Table 1). The patient’s serum was also positive by Western blotting and strongly positive in a recombinant antigen ELISA for *B procyonis*, confirming the diagnosis. Toxocara serology was negative and Ascaris serology was indeterminate. A field investigation revealed two latrines with raccoon excrement in the child’s backyard. Fecal samples from these latrines and soil from the sandbox contained *B procyonis* eggs.

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The patient improved with treatment. Within days, he became more alert and his gaze deviation resolved. After five days, methylprednisone was changed to oral prednisone 2.5 mg/kg/day. By one month, he was able to tolerate oral nutrition and weight bear with assistance. The eosinophil count had normalized. Albendazole was discontinued at that time, and prednisone was gradually tapered. At a four-month follow-up visit, the patient had made significant improvement, and was ambulatory and interactive. He regained his baseline level of function, with the exceptions that he no longer used speech to communicate, had cortical visual impairment and a seizure disorder. A repeat MRI documented postinflammatory atrophic changes, with no acute disease (Figure 1).

**DISCUSSION**

The present case illustrates the importance of early clinical suspicion of raccoon roundworm meningoencephalitis. Neurological improvement was observed in our patient following treatment with albendazole and corticosteroids. This patient’s recovery from a coma to being independently mobile, communicative but nonverbal, and interactive was significant.
Despite the remaining deficits, the outcome was significantly better when compared with most previously reported in the literature. Other patients with similar presentations have been characterized by more severe neurological sequelae or death (4). We speculate that early intervention with both albendazole and steroids may have contributed to this patient’s partial recovery. Albendazole is active against migrating larvae of B procyonis (2), while steroids may have a role in suppressing the host inflammatory response (4).

B procyonis is being recognized with increasing frequency as a cause of human disease. There have been 14 reported cases of human infection (2,4-7), with over a dozen unpublished cases (Kazacos, personal communication). Most of the previously affected individuals were male and young or had developmental disabilities. Geophagia was a frequent risk factor. Initial clinical features may be subtle, with fever and meningismus frequently absent, but subsequent neurological deterioration is often dramatic (4-6). Approximately one-third of reported cases have been fatal, with the remainder having severe residual deficits including cognitive impairment, seizures, paralysis and blindness (4). The full spectrum of human disease is unknown but appears to range from subclinical infection to rapidly progressive disease (4,5,7). Recently, a mild case of suspected B procyonis infection was reported, with apparent early complete response after cerebellar edema was treated with early corticosteroids and later albendazole (7). Until an optimum therapeutic strategy is available, the principle of early treatment of this aggressive infection with both albendazole and steroids should be considered.

Ocular disease was reported in one-third of cases of B procyonis meningoencephalitis (4). In our patient, fundoscopic examination revealed patchy bilateral subretinal infiltrates that resolved following therapy. Larval migration in the subretinal space can cause localized ocular damage leading to diffuse unilateral subacute neuroretinitis (8). Diffuse unilateral subacute neuroretinitis has been previously reported in Canada, unilateral subacute neuroretinitis (8). Diffuse unilateral subacute neuroretinitis has been previously reported in Canada, and although rare, is important as a preventable cause of childhood blindness (9). Ocular larva migrans is most often unilateral; however, bilateral disease has been reported (10).

Eosinophilia is an important diagnostic clue of invasive helminthic infection. Although peripheral eosinophilia has a very broad differential diagnosis, eosinophilic meningitis has a much more limited differential diagnosis (11). In North America, infection with B procyonis must be considered as a cause of eosinophilic meningitis and investigated (4-6). If a peripheral eosinophilia and/or CSF eosinophilia is found in a child with meningitis, in combination with risk factors such as young age, developmental delay and possible contact with raccoon feces, specific treatment and testing for B procyonis of both serum and CSF should be initiated. In our patient, antibodies to B procyonis were detected and strongly positive in both serum and CSF (Table 1). The serum level was well above the putative cut-off value of the assay, and confirmatory testing was positive. B procyonis serology is performed by ELISA from the Department of Comparative Pathobiology at Purdue University in West Lafayette, Indiana. ELISA for Baylisascaris antibodies uses larval excretory-secretory antigens and has a putative optical density cut-off value for serum of 0.250 or greater. CSF and ocular fluid should have no antibody present. Neuroimaging is nonspecific and may be normal initially. As seen in our patient, suggestive features on MRI include patchy periventricular and cerebellar white matter lesions accompanied by later atrophic changes (4,5).

Ideally, homeowners, particularly those with children, should recognize the risk posed by raccoon feces and avoid raccoon latrines. However, we share our urban environment with raccoons, and avoiding all contact is not possible. An option when contact has been made with raccoon feces is postexposure prophylaxis with albendazole. In animal models it has been documented that the administration of albendazole shortly after ingestion of B procyonis eggs can prevent clinical disease, while the response to treatment is very limited once larval invasion of the brain has occurred and neurological symptoms have developed (4). Albendazole is a relatively safe medication with few side effects or contraindications. Given the potential severity of infection following accidental ingestion of raccoon feces, prompt administration of a prophylactic course of albendazole should be considered (4,12). The decision to provide prophylaxis should not require the identification of B procyonis eggs in the environment.

Increasing awareness of the risk posed by raccoon feces and contact with raccoon latrines is critical in preventing this serious central nervous system infection. Preventive measures can minimize the risk that children may be exposed to B procyonis in the backyards of our cities and towns. Equally important is a high index of suspicion on the part of physicians, who should consider the diagnosis, be aware of the implications of eosinophilia and elicit a history of raccoon exposure in all cases of encephalitis. Empirical therapy with albendazole and steroids should be started based on this suspicion, and not be delayed while waiting serological or other testing, to limit the damage by this life-threatening parasite.

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REFERENCES