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Case Report

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Baylisascaris procyonis infection of the pediatric brain and spine: Neuroimaging findings

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Abstract

Baylisascaris procyonis is a type of intestinal roundworm that may infect the Central Nervous System (CNS) resulting in severe and lasting neurologic symptoms and possibly death. Few reports have presented the CNS imaging findings in children. This case report presents the initial, acute and follow up brain and spine imaging findings in an 18 month old boy with confirmed *Baylisascaris procyonis* infection.

Introduction

Up to 82% of the American raccoon population is infected with *Baylisascaris procyonis*, a type of intestinal roundworm proven to cause severe and lasting neurologic disease in humans due to its aggressive somatic migration, larval invasion of the Central Nervous System (CNS), and capability for continued growth within the host [1-8]. Young children and persons with development delay are most likely to be infected with *B. procyonis* due to increased hand-mouth behaviors [8]. In cases of Neural Larva Migrans (NLM), children may present with the following symptoms: low grade fever, ataxia, increasing lethargy, somnolence, periods of increased irritability, extensor posturing, increasing spasticity with hemi- or quadriparesis, ocular or cranial involvement, uncontrollable seizures, and deterioration in neurologic status [1-16]. The CNS is not the only system that can be affected by *B. procyonis*; visceral organs are oftentimes also impacted [1,2]. *B. procyonis* infection in humans is very rare and diagnosis is therefore often delayed because the symptoms are nonspecific. However, early treatment is benificial [1-16].

Diagnosis is made based upon clinical presentation, serology, Cerebro-Spinal Fluid (CSF) puncture, and neuroimaging. The goal of this manuscript is to alert and familiarize radiologists and neuroradiologists about the anatomical and advanced neuroimaging findings in this rare disease.

Case presentation

In December 2021, a previously healthy 18-month-old boy presented to the Emergency Department (ED) of our tertiary children's hospital with a 1-2 week history of fevers, fussiness, axial hypotonia, appendicular hypertonia, trismus, and diffuse upper motor neuron signs with elevated eosinophils both in serum and CSF. His social history was remarkable for potential access to chemical substances. The patient grew up on a farm and resided in a rural community with known zoonotic exposure and possible chemical exposures.

An initial Magnetic Resonance Imaging (MRI) of brain/spine with and without intravenous contrast application as well as a Magnetic Resonance Venography (MRV) showed no major acute intracranial abnormalities (Figure 1). However, slight dural thickening and enhancement along the posterior tentorium, right greater than left, was noted of uncertain significance. Spine MRI showed mild increased enhancement and thickening of lumbosacral anterior nerve roots extending into the cauda

equina (Figure 2). The findings were suggestive of an inflammatory process including a Guillain-Barre syndrome. Additional considerations included an infectious process such as acute flaccid myelitis or West Nile virus. Carcinomatosis was less likely given the absence of a known primary neoplasm and the relatively smooth nature of the contrast enhancement.

A repeat brain/spine MRI with and without contrast 6 days later showed discrete leptomeningeal enhancement along the infratentorial and supratentorial brain, most likely representing meningitis. Additional findings included a mild increase in the size of the supratentorial ventricular system and minimal widening of the subarachnoid spaces compared to the prior study. Subtle, non-expansile intramedullary T2 hyperintensity was noted mainly centrally in the cervical spine and to a lesser extent in the thoracic spine. This may have been secondary to myelitis. Redemonstration of mild ventral more than dorsal enhancement of the cauda equina was also noted, similar to the prior study. A repeat MRI of the brain 16 days post initial MRI showed progressing enlargement of the ventricles and interval development of diffuse, ill-defined T2 and FLAIR hyperintensity of the hemispheric cerebral and cerebellar white matter bilaterally (Figure 3). Furthermore, the central gray matter (basal ganglia and thalami) appeared hyperintense on T2-weighted MR imaging as well as on Diffusion Weighted Imaging (DWI). On the matching apparent diffusion coefficient (ADC) maps, minimally restricted diffusion is noted of the central gray matter. On the contrast-enhanced T1-weighted sequence, subtle, ill-defined contrast enhancement was seen within the cerebral and cerebellar white matter as well as minimal persisting increased contrast enhancement of the leptomeninges.

Alongside repeat anatomical and advanced MR imaging, the child had an extensive work-up involving consultation from infectious disease, genetics, and neurology. The majority of his workup was negative, excluding leukocytosis with peripheral eosinophilia (absolute eosinophils 11000) and CSF pleocytosis with eosinophilic predominance. He was started on treatment for eosinophilic meningitis. A Baylisascaris antibody study came back positive indicating a nematode infection 22 days after the initial MRI study. Following this finding, the patient completed a 28-day course of albendazole and intravenous methylprednisolone, with dramatic improvement in eosinophil count after initiation. Additionally, EEG did not show any seizures and no Anti-Epileptic Drugs (AED) were started during the hospitalization.

Upon completion of the steroid course, the patient did however go on to develop seizures with fair seizure control despite multiple antiepileptic drugs. The patient also developed developmental delay on follow up.

Discussion

Infection with *B. procyonis*, or raccoon roundworm, is a rare disease that most often affects young children between the ages of 0-24 months [2]. Infection occurs through the ingestion of infective *B. procyonis* eggs, which are commonly found in raccoon feces. Raccoon latrines, which are often found at the base of trees, woodpiles, roofs, decks, and sandboxes pose a high risk of being contaminated by *B. procyonis* [16]. Children with pica, geophagia, or otherwise excessive hand-mouth behaviors are most likely to be infected as a result of their interacting with the environment and should therefore be carefully watched [1-16].

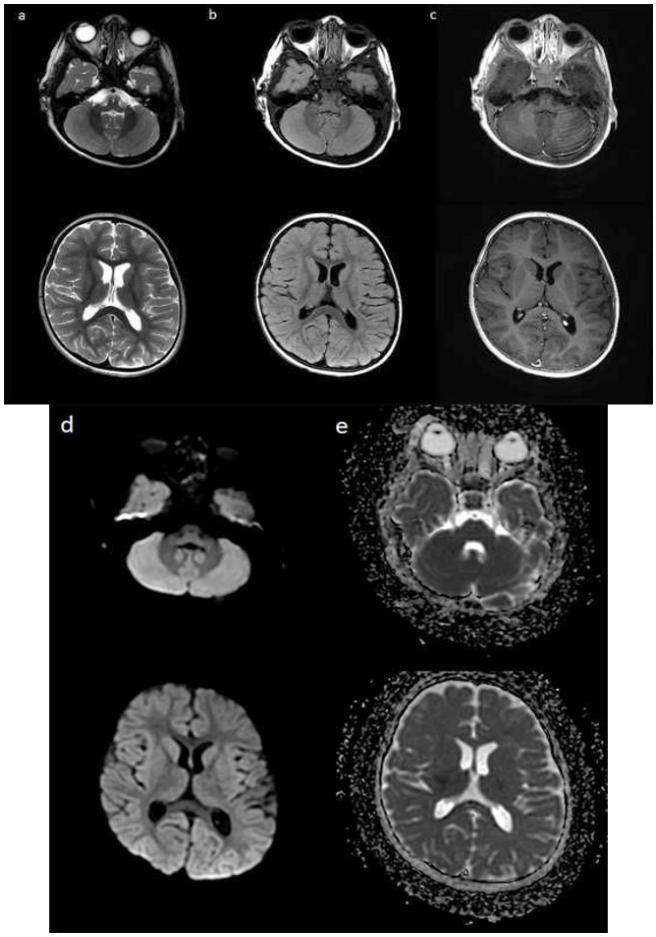

Figure 1: Axial T2-weighted **(a)**, FLAIR **(b)** and contrast-enhanced T1-weighted MR images **(c)** at the first day of presentation to our hospital shows no focal parenchymal abnormality. Minimal increased leptomeningeal enhancement was noted (not shown). Diffusion weighted imaging **(d)** including apparent diffusion coefficient (ADC) maps **(e)** are unremarkable.

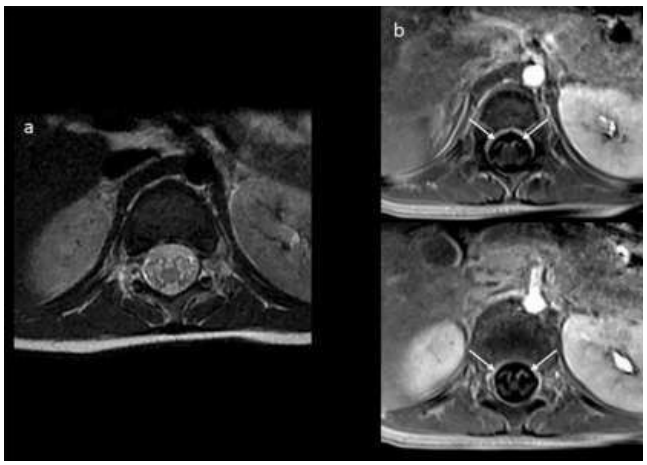

Figure 2: Axial T2-weighted **(a)**, and axial **(b)** contrast enhanced T1-weighted MR images of the lumbosacral spine show an abnormal enhancement of the anterior nerve roots (arrows) of the lumbosacral spinal cord and cauda equina nerve roots. The posterior nerve roots show no significant enhancement.

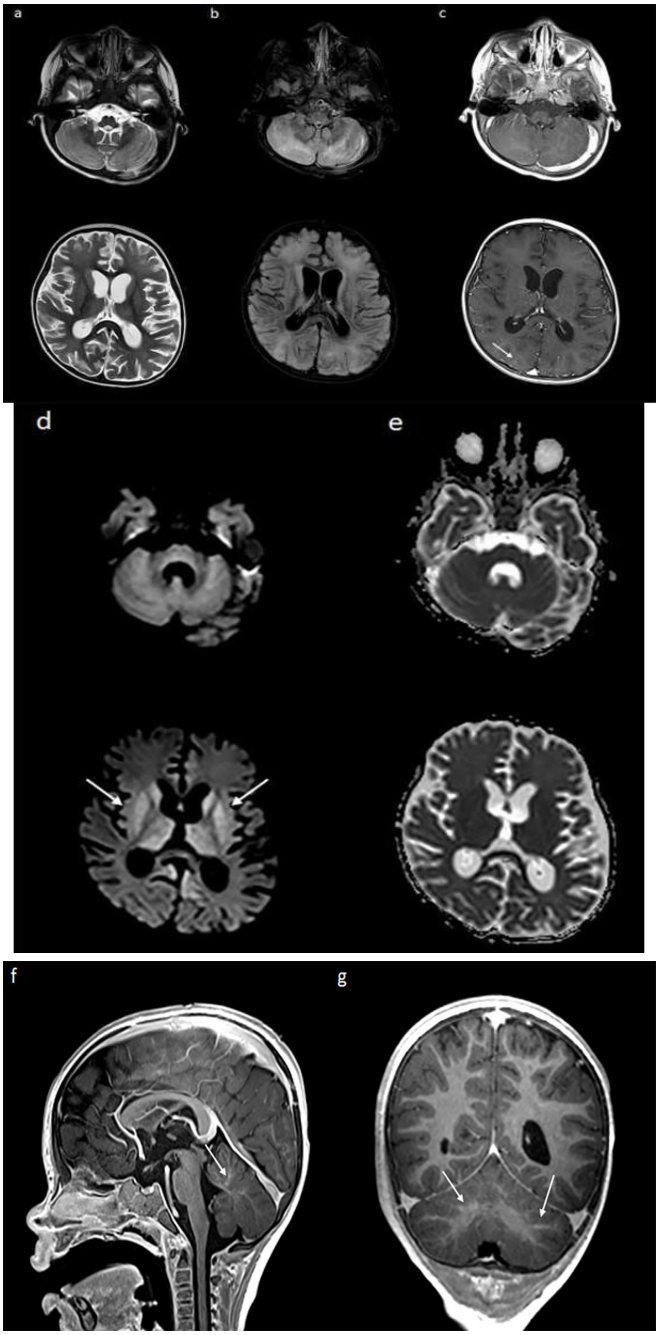

Figure 3: Follow up MR 16 days after the initial MRI show marked overall volume loss of the cerebellar and cerebral hemispheric white matter with e vacuo enlargement of the ventricles and subarachnoid spaces. In addition, thinning of the cortical gray matter and mild volume loss of the central gray matter. On T2-weighted **(a)** and FLAIR MR images **(b)** the white matter appears diffusely hyperintense. On axial contrast enhanced T1-weighted MR images **(c)** ill-defined faint contrast enhancement (arrow) is noted of the white matter. On diffusion weighted MR **(d)** the central gray matter appear DWI-hyperintense (arrows) with matching minimal restricted diffusion on the ADC maps **(e)**. The sagittal **(f)** and coronal **(g)** contrast enhanced T1-weighted MR images display a mildly increased enhancement of the tentorium cerebelli and multiple areas of faint white matter enhancement (arrows).

The clinical presentation of *B. procyonis* infection varies, however it usually presents with the following symptoms: low grade fever, ataxia, increasing lethargy, somnolence, periods of increased irritability, extensor posturing, increasing spasticity with hemi- or quadriparesis, ocular or cranial involvement, uncontrollable seizures, and deterioration in neurologic status [1-16]. Death may occur in severe cases. The clinical symptoms are typically nonspecific; however, they suggest an infectious disease involving the CNS. In addition to a clinical examination, blood work up, and CSF analysis, neuroimaging plays an essential role in diagnosing the severity and degree of CNS involvement. In the case of *B. procyonis* infection, early and accurate diagnosis is essential to initiate adequate treatment in a timely manner to prevent or limit complications.

There are few reports in the literature which describe the neuroimaging features of B. procyonis infection [1-16]. In particular, no data has been reported on diffusion weighted MR imaging and spinal cord involvement. Our case demonstrated mild meningeal enhancement indicating meningitis without obvious parenchymal enhancement/infection early in the disease process. Follow up MR imaging showed diffuse white matter T2 hyperintensity without diffusion restriction, indicating significant white matter involvement. This may be secondary to parainfectious, possibly auto-immune mediated white matter demyelination versus direct infection.

Moreover, spinal MRI showed increased enhancement of the anterior nerve roots of the lumbo- sacral spinal cord and cauda equina mimicking Guillain-Barre disease. Consequently, our observations in this child suggest that in *B. procyonis* disease, next to the primary infectious pathogenesis, an inflammatory component may also contribute to the overall CNS injury. This combination of both an infectious and inflammatory pathogenesis is well known from several other CNS infections (tuberculosis or herpes simplex virus).

Conclusion

In conclusion, neuroimaging features of a *B. procyonis* CNS infection include subtle meningeal and multifocal white matter enhancement, T2- and FLAIR hyperintensity of the cerebral and cerebellar white matter, and increased enhancement of predominantly the anterior spinal nerve roots. The goal of this manuscript is to alert the radiologist to include *B. procyonis* CNS infection in the differential diagnosis if a child presents with symptoms of infectious or inflammatory CNS disease, including eosinophilia, especially when they grow up on a farm or reside in a rural community with known zoonotic exposure and possible chemical exposures.

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