"Owl's Eye" Sign in Acute Flaccid Paralysis

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A 4-year-old boy presented with asymmetric acute flaccid paralysis (AFP) of his right arm and both legs. He was alert with no oculobulbar weakness or incontinence. He had fever and diarrhea 5 days earlier. He was fully immunized with no travel history.

MRI of his spine performed 6 days after onset of his infectious symptoms revealed an "owl's eye" sign. Axial T2-weighted imaging (T2WI) showed symmetric hyperintense signal in the spine corresponding to the location of anterior horn cells (Figure 1). Sagittal T2WI noted the linear hyperintensities to extend from the cervicomedullary junction to the conus with mild expansion of the cervical cord observed.

The MRI findings raised several diagnostic considerations. Infectious etiology was clinically suspected given his prior infectious symptoms. Viral pathogens have been linked to AFP including poliomyelitis, polio-like viruses (enterovirus [EV], coxsackievirus, and echovirus)[1], and more recently flaviviruses (West Nile virus [WNV]).[2] Acute anterior spinal artery infarct can show a similar MRI appearance in children who have had an axial load with resulting fibrocartilaginous embolism.[3] Inflammatory disorders may show linear MRI abnormality, but it is more common for a larger cross-sectional area to be involved in acute transverse myelitis; for a more central location in neuromyelitis optica;[4] or for a peripheral/dorsal location in multiple sclerosis.[5] Compressive myelopathies such as Hirayama syndrome[6] or neurodegenerative disorders such as amyotrophic lateral sclerosis[7] should be considered in an adolescent or adult.

Cerebral spinal fluid (CSF) studies were performed 5 days after symptom onset and identified a lymphocytic pleocytosis (178×10^6/L cells; 79% lymphocytes). CSF RBC, protein, and glucose were normal and CSF cultures were negative. Testing was negative for EVs, including poliovirus, in CSF, stool and nasopharyngeal swab. Serology was negative for WNV IgM/IgG, Powassan virus IgG, Epstein–Barr virus viral capsid antigen IgM, cytomegalovirus IgM/IgG, and Lyme IgM/IgG.

Nerve conduction studies completed 3 weeks later noted robust sensory responses and low motor amplitudes in his right arm and legs consistent with a disorder of motor neurons.

Several viral pathogens (above) may cause AFP due to an infectious myelitis. Wild-type poliovirus type 1 remains endemic in Pakistan, Afghanistan, and Nigeria, with periodic outbreaks documented after travel from an endemic country. Polio-like viruses, including EV71, EV68, and coxsackievirus can cause infectious myelitis and be difficult to confirm in some patients.

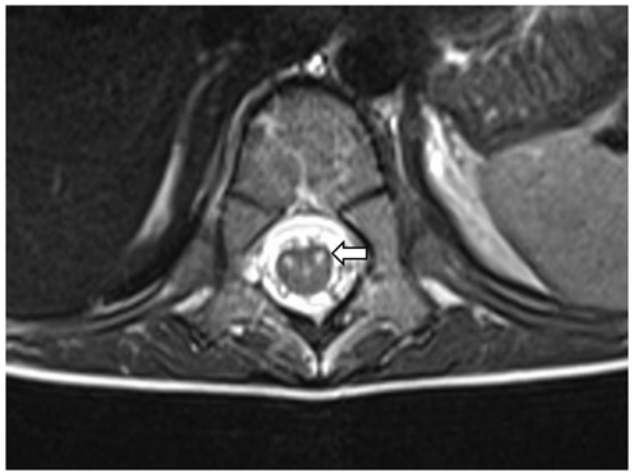

Figure 1: *MRI of the lumbar spine (T2WI, axial view) reveals an "owl's eye" sign reflecting symmetrical hyperintensities in the anterior horn cells. No gadolinium enhancement was noted.*

Other viruses have been linked with AFP due to Guillain–Barre syndrome.[1]

In 2014, a cluster of AFP was reported in 25 Canadian children.[8] CSF testing for EV and rhinovirus by polymerase chain reaction was negative in all cases, despite 72% showing CSF lymphocytic pleocytosis. All but one child had a nasal swab with 14/24 (58%) testing positive for EV68, EV71, coxsackie, or rhinovirus. Overall, 44% of children with AFP did not have a confirmatory pathogen identified.

This boy is one of over 250 cases of polio-like AFP reported in North America in 2018 (US Center for Disease Control: 210 cases,[9] Canadian Surveillance: 49 confirmed cases[10]). Similar to other patients in this recent cluster, no clear infectious etiology has been identified, underscoring the importance of complete neurological workup including neuroimaging and electrodiagnostic testing to assist with localization and guide comprehensive workup.

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Conflict of Interest

The authors have no conflict of interest to report.

Statement of Authorship

JZD: manuscript conception, drafting, and editing.
HM: manuscript conception, drafting, and editing.

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