

EXTENSIVE MYELITIS AFTER ORAL POLIO VACCINATION: MRI FEATURES

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A 7-year-old boy presented with fever and ataxia 20 days after oral polio vaccination. Magnetic resonance imaging showed extensive myelitis, involving both anterior and posterior horns of the gray matter. Complete posttreatment recovery was evident. Postvaccinal myelitis after oral polio vaccination, of either infectious or immune mediated etiology, is very rare entity that should be promptly recognized in order to initiate adequate treatment.

Key-word: Viruses.

The term poliomyelitis derives from Greek words “polios”, meaning gray and “myelos”, meaning marrow, referring to the inflammation of the spinal cord gray matter. The viral invasion affects the motor neurons of the anterior horn cells, responsible for the movement of the limbs, trunk and intercostal muscles. On very rare occasions, the attenuated virus in oral polio vaccine reverts into a form that can cause severe clinical manifestation of poliomyelitis that need to be treated promptly (1). Although patients with primary immunodeficiencies have a much higher risk of the disease, vaccine-associated poliomyelitis or vaccine-induced transverse myelitis may also occur in healthy recipients. Public concerns regarding potential postvaccinal adverse effects in all prophylactic inoculations are present. Replacement of oral vaccine with inactivated poliomyelitis vaccine is growing (2). The aim of our report is to stress the role of imaging in detecting the potential involvement of the spinal cord, as a consequence of oral polio vaccination.

Case report

A 7-year-old previously healthy boy, presented with fever (38.5C), severe neck pain, ataxia, muscle pain and weakness. Neurological examination revealed normal mental status and cranial nerves, the presence of increased deep tendon reflexes and positive Romberg and Mingazzini signs. Evaluation of cere-

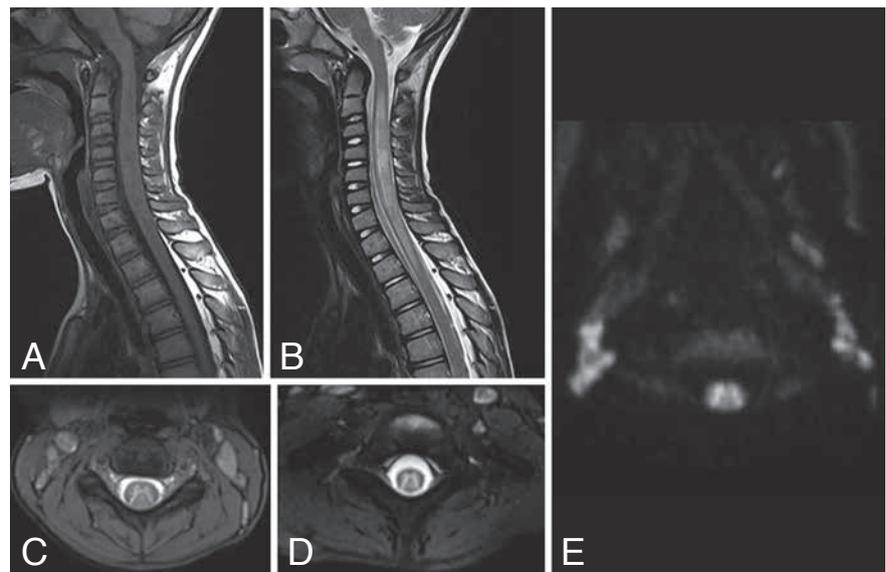


Fig. 1. — Enlargement of the spinal cord on T1W sagittal images (A), associated with extensive inflammatory process of the spinal cord evident on T2W sagittal images (B), mainly involving the anterior and posterior horns of the gray matter, best appreciated on T2W axial images in cervical (C) and thoracic level (D) and diffusion-weighted images (E).

brospinal fluid revealed the presence of 30 white blood cells, mostly lymphocytes, normal glucose and protein levels. The child received oral polio vaccine 20 days before initial symptoms. Cerebellitis was suspected and brain magnetic resonance imaging (MRI) was requested. No abnormalities were found on brain MRI. However, cervical spine MRI revealed the presence of extensive myelitis, involving both anterior and posterior horns of the gray matter,

associated with diffusely increased T2W signal affecting the posterior column of the spinal cord white matter, most consistent with inflammation and edema (Fig. 1). No enhancement of the lesions was noted after intravenous contrast administration. Diffusion restriction of both anterior and posterior horns of the gray matter was compatible with either postvaccinal poliomyelitis or vaccine-induced transverse myelitis. No restricted diffusion of the spinal cord white matter tracts was evident. Intravenous immunoglobulin and corticosteroid therapy and oral antibiotics were promptly initiated. General improvement with gradual regression of the neurological signs was evident in the following weeks. No residual symptoms and signs were present on neurological examination 10 months later, and follow-up MRI revealed no residual abnormalities in the spinal cord (Fig. 2).

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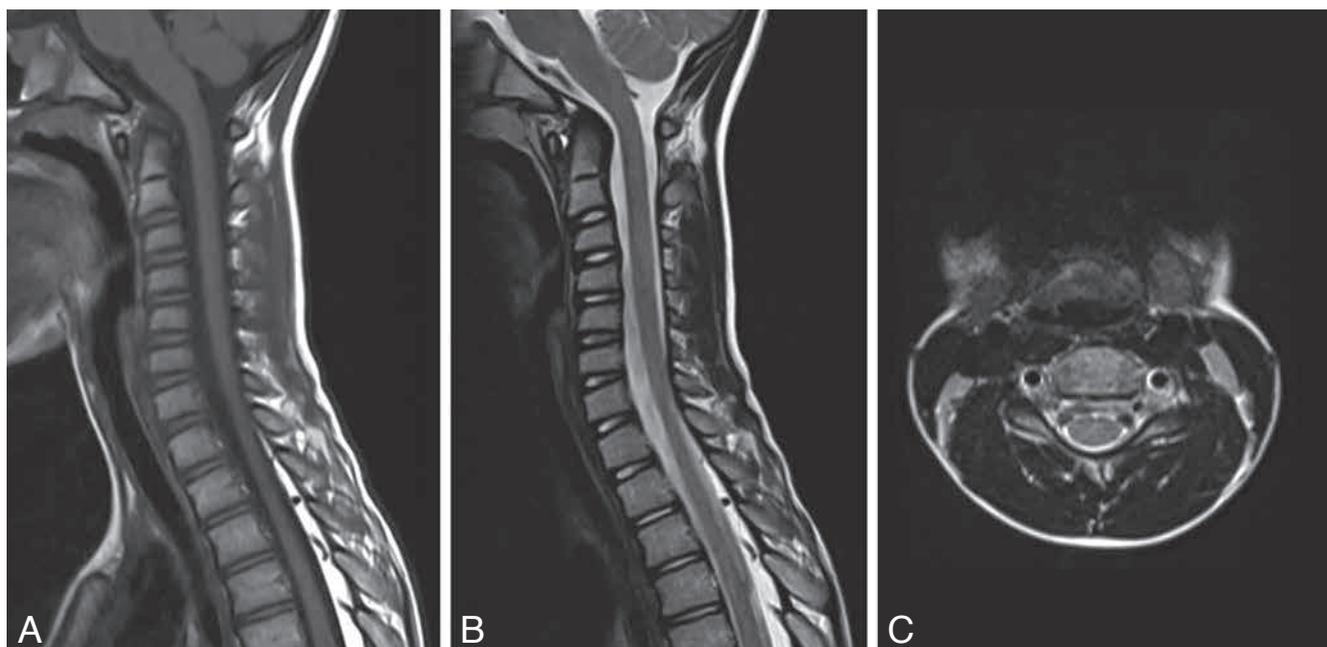


Fig. 2. — Complete regression of poliomyelitis with normal spinal cord morphologic characteristics on T1W sagittal (A), T2W sagittal (B) and turbo spin-echo T2W axial (C) images.

Discussion

Increased T2 signal involving the anterior horns of the spinal cord gray matter has been described on MRI in patients with poliomyelitis (3). Oishi et al. reported postvaccinal poliomyelitis affecting the anterior horn and showing postcontrast enhancement, that did not improve on intravenous immunoglobulin treatment (4), while Choudhary et al. reported poliomyelitis with both brain and spinal cord affection, presented with subacute onset of flaccid paralysis, associated with bilateral changes in substantia nigra and anterior horns of the spinal cord gray matter (5). However, the involvement of both anterior and posterior horns of the spinal cord gray matter in our patient were clear on axial T2-weighted images and diffusion-weighted sequences. To the best of our knowledge, no radiological contributions exist, confirming the involvement of both anterior and posterior horns of the spinal cord gray matter in patients with poliomyelitis or oral polio vaccine induced transverse myelitis. Histopathologically proven invasion of the posterior horns was reported in 1951 by Harrer and Niedermeyer (6). In 2013, Kosaka et al. reported the presence of post-polio syndrome in a 77-year-old who presented with neuromuscular problems many years after acute poliomyelitis, mimicking

amyotrophic lateral sclerosis. Pathologically, neuronal loss and gliosis were extending outside the anterior horns, affecting the intermediate zone (7). Blondel et al. also reported that affection of intermediate zone and even the posterior gray column could be present in severe cases (8). The posterior horn receives several types of sensory information from the body including proprioception and vibration. This is the most likely reason why our patient presented altered proprioception, clinically confused by infectologist with ataxia and possible cerebellitis.

Increased T2 signal of the anterior horns is not only pathognomonic for poliomyelitis. Hopkins syndrome, also associated with flaccid paralysis, and Lyme neuroborreliosis affect anterior horns, presented with elevated signal on T2-weighted images (9, 10). Kraushaar et al. reported a serologically proved West Nile virus flaccid paralysis of right upper extremity associated with striking T2 hyperintensity in the anterior horns of the cervical spinal cord, similar to those seen in cases of poliomyelitis (11).

The main limitation of this report is the fact that polymerase chain reaction was not available at the institution where the child had been initially admitted. It remained unclear if the spinal cord inflammation was compatible with infectious pro-

cess caused by attenuated strains of vaccinal polio virus, or with immune-mediated transverse myelitis. Both entities are extremely rare. Literature search reveals numerous studies confirming the presence of postvaccinal acute disseminated encephalomyelitis. However, only 37 patients have been reported in the literature having isolated postvaccinal transverse myelitis (12). Majority of these patients had a history of recently administered hepatitis B, measles-mumps-rubella, diphtheria-tetanus-pertussis and other vaccines. Only 3 out of 37 patients had a history of recent oral polio vaccination.

Transverse myelitis is an acute inflammatory condition, of idiopathic, postinfectious, postvaccinal or disease-associated etiology, typically involving thoracic segment of the spinal cord. MRI usually reveals a long, centrally located intramedullary lesion, extending over 3 vertebral segments, and involving complete or the most of the cross section of the cord (13).

Our patient recovered completely with regression of all clinical symptoms. Recovery in patients with poliomyelitis is usually completed in six to eight months. Paralysis remaining after one year is likely to be permanent, although modest recoveries of muscle strength are possible 12 to 18 months after infection (14). Strebel et al. reported that

the risk of vaccine-associated paralytic poliomyelitis increased with tissue injury, such as intramuscular injections, within one month of immunization with oral polio vaccination (15). It is suggested that skeletal muscle injury induces retrograde axonal transport of poliovirus, facilitating viral invasion of the central nervous system and consequent spinal cord damage (16).

In 2000, the American Academy of Pediatrics changed their policy to recommend only inactivated poliovirus vaccine for routine childhood immunization, since they considered that vaccine associated poliomyelitis would not be eliminated unless the use of oral polio vaccination was stopped (17). Therefore, some countries, including USA and Germany, have changed their national policies replacing the oral polio vaccine with inactivated poliovirus vaccine (18).

High resolution MRI scanners, in appropriate clinical context, might be considered as quite reliable, or even confirmatory diagnostic modality for detecting even early stages of spinal involvement in poliomyelitis, crucial for prompt initiation of most adequate treatment.

References

1. Racaniello V.R.: One hundred years of poliovirus pathogenesis. *Virology*, 2006, 344: 9-16.
2. Frolov A.F., Sel'nikova O.P., Zadorozhnaia V.I., Moiseeva A.V., Demchishina I.V., Bondarenko V.I., et al.: Immunoprophylaxis of poliomyelitis in Ukraine with the use of inactivated vaccine. *Zh Mikrobiol Epidemiol Immunobiol*, 2005: 49-53.
3. Haq A., Wasay M.: Magnetic resonance imaging in poliomyelitis. *Arch Neurol*, 2006, 63: 778.
4. Oishi M., Kawai M., Ogasawara J., Koga M., Kanda T.: Vaccine-associated paralytic poliomyelitis showing biphasic motor paresis. *Rinsho Shinkeigaku*, 2012, 52: 744-749.
5. Choudhary A., Sharma S., Sankhyan N., Gulati S., Kalra V., Banerjee B., Kumar A.: Midbrain and spinal cord magnetic resonance imaging (MRI) changes in poliomyelitis. *J Child Neurol*, 2010, 25: 497-499.
6. Harrer G., Niedermeyer E.: Case report on lesions in the posterior horn in poliomyelitis. *Klin Med Osterr Z Wiss Prakt Med*, 1951, 6: 57-61.
7. Kosaka T., Kuroha Y., Tada M., Hasegawa A., Tani T., Matsubara N., et al.: A fatal neuromuscular disease in an adult patient after poliomyelitis in early childhood: consideration of the pathology of post-polio syndrome. *Neuropathology*, 2013, 33: 93-101.
8. Blondel B., Duncan G., Couderc T., Delpeyroux F., Pavio N., Colbere-Garapin F.: Molecular aspects of poliovirus biology with a special focus on the interactions with nerve cells. *J Neurovirol*, 1998, 4: 1-26.
9. Arita J., Nakae Y., Matsushima H., Maekawa K.: Hopkins syndrome: T2-weighted high intensity of anterior horn on spinal MR imaging. *Pediatr Neurol*, 1995, 13: 263-265.
10. Charles V., Duprez T.P., Kabamba B., Ivanoiu A., Sindic C.J.: Poliomyelitis-like syndrome with matching magnetic resonance features in a case of Lyme neuroborreliosis. *BMJ Case Rep*. 2009, 2009, pii: bcr07.2008.0527. doi: 10.1136/bcr.07.2008.0527.
11. Kraushaar G., Patel R., Stoneham G.W.: West Nile Virus: a case report with flaccid paralysis and cervical spinal cord: MR imaging findings. *AJNR Am J Neuroradiol*, 2005, 26: 26-29.
12. Agmon-Levin N., Kivity S., Szyper-Kravitz M., Shoenfeld Y.: Transverse myelitis and vaccines: a multi-analysis. *Lupus*, 2009, 18: 1198-204.
13. Hodel J., Outteryck O., Jissend P., Zins M., Leclerc X., Pruvo J.P.: MRI of myelitis. *JBR-BTR*, 2012, 95: 270-276.
14. Neumann D.A.: Polio: its impact on the people of the United States and the emerging profession of physical therapy. *J Orthop Sports Phys Ther*, 2004, 34: 479-492.
15. Strebel P.M., Ion-Nedelcu N., Baughman A.L., Sutter R.W., Cochi S.L.: Intramuscular injections within 30 days of immunization with oral poliovirus vaccine – a risk factor for vaccine-associated paralytic poliomyelitis. *N Engl J Med*, 1995, 332: 500-506.
16. Gromeier M., Wimmer E.: Mechanism of injury-provoked poliomyelitis. *J Virol*, 1998, 72: 5056-5060.
17. Prevention of poliomyelitis: recommendations for use of only inactivated poliovirus vaccine for routine immunization. Committee on Infectious Diseases. American Academy of Pediatrics. *Pediatrics*, 1999, 104: 1404-1406.
18. Alexander L.N., Seward J.F., Santibanez T.A., Pallansch M.A., Kew O.M., Prevots D.R., Strebel P.M., Cono J., Wharton M., Orenstein W.A., Sutter R.W.: Vaccine policy changes and epidemiology of poliomyelitis in the United States. *JAMA*, 2004, 292: 1696-1701.