Acute Disseminated Encephalomyelitis Associated with Oral Polio Vaccine

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Abstract

A 27-year-old woman presented with acute paresis after taking an oral polio vaccine (OPV). Deep tendon reflexes were preserved, needle electromyography showed no neurogenic changes, and there were no lesions on spine magnetic resonance imaging (MRI), suggesting that motor neurons of the spinal cord were not affected. Brain MRI showed abnormal lesions in the tegmentum of the upper pons, left cerebral peduncles, truncus of the corpus callosum, and right parietal lobe. Cerebrospinal fluid revealed mild pleocytosis. The most probable diagnosis was acute disseminated encephalomyelitis associated with OPV.

Key words: oral polio vaccine, magnetic resonance imaging, acute disseminated encephalomyelitis

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Introduction

Polio is caused by polivirus infection, and is characterized by impairment of motor neurons in the spinal cord, brain, and brainstem (1). Since the introduction of the oral polio vaccine (OPV), infection from the wild type poliovirus has not been reported in Japan since 1981 (2). However, OPV can cause vaccine-associated paralytic poliomyelitis (VAPP) in rare cases (3), and acute disseminated encephalomyelitis (ADEM) associated with OPV has also been described in one report (4). We describe a patient with acute paresis and brain MRI abnormalities after OPV intake.

Case Report

A 27-year-old woman and her 6-month-old son received OPV in May 2005. Seven days later, she noticed weakness in her right upper extremity. Ten days later, she experienced muscular stiffness in her right extremities, and subsequently suffered from a transient loss of consciousness while in her bath. She then felt weakness in her lower extremities that progressed slowly. She was referred to our hospital 20 days later. On examination, her body temperature was 37.3°C, blood pressure was 122/72 mmHg, and heart rate was 60 beats/min with regular sinus rhythm. The cranial nerves were intact. Manual muscle testing revealed mild weakness in the lower extremities: iliopsoas (4/4+), quadriceps (5-/5), hamstrings (4/4+) using the Medical Research Council rating scale. Mingazzini sign was positive in the right lower extremity. The sensory system and coordination were intact. Deep tendon reflexes were normal, and the plantar responses were flexor. Gowers’ sign was positive. Blood counts, renal and liver function tests, glucose, C-reactive protein, rheumatoid factor, and antinuclear antibody were normal. The serum poliovirus antibody titers (NT) were measured: type 1 (1:128), type 2 (1:256), and type 3 (1:32) on the 23th day after OPV; and type 1 (1:64), type 2 (1:128), and type 3 (1:32) on the 48th day. The titers for influenza A and B, measles, herpes simplex virus, varicella-zoster virus, cytomegalovirus, Epstein-Barr virus, mycoplasma, echovirus 6, 7, 9, 11, 30, coxsackievirus A 4, 7, and B1-5 were not significantly elevated. The cerebrospinal fluid (CSF) revealed pleocytosis (13.6 /mm³), normal protein (23 mg/dl), IgG index (0.62), and glucose (60 mg/dl). No myelin basic protein or oligoclonal bands were detected. Poliovirus was not detected in viral cultures of CSF or stools. Nerve conduction studies were normal. Needle electromyography (EMG) revealed no
neurogenic findings in the right vastus lateralis, tibialis anterior and flexor digitorum longus on the 28th day after OPV. Electroencephalogram, electrocardiogram, visual, motor and short somatosensory evoked potentials, and brainstem auditory evoked potentials were normal. Diffusion-weighted imaging (DWI), T2-weighted magnetic resonance imaging (MRI), and fluid-attenuated inversion recovery (FLAIR) on the 28th day after OPV intake showed high intensity signals in the tegmentum of the upper pons, left cerebral peduncle, and truncus of the corpus callosum (Fig. 1). A second DWI, T2-weighted MRI, and FLAIR on the 38th day after OPV intake revealed high intensity signals in the right parietal lobe in addition to the pons and corpus callosum (Fig. 2). Apparent diffusion coefficient was not decreased in the truncus of the corpus callosum. Gadolinium enhancements were present in the small parts of the corpus callosum and in the right parietal lobe of the cortex. Cervical and thoracic MRIs showed no abnormal findings of the anterior horn cells of the spinal cord. We treated the patient with intravenous methylprednisolone (1000 mg per day for 3 days) twice. The first methylprednisolone therapy was started on the 35th day, and the second one on the 42nd day after OPV intake. No oral corticosteroid therapy was given afterwards. The clinical symptoms improved gradually with a decrease of pleocytosis and brain MRI abnormalities, and her symptoms completely disappeared in 2 months.

Discussion

We report a patient with acute paresis and brain MRI abnormalities after OPV intake. OPV has almost eradicated poliomyelitis, but it can cause VAPP in rare cases with a morbidity incidence as low as one case per a few million (3). While our patient showed asymmetric paresis and CSF pleocytosis, her deep tendon reflexes were preserved, needle EMG showed no neurogenic changes, and there were no lesions of the spinal cord MRI, suggesting that motor neurons in the spinal cord were not affected, and acute poliomyelitis was ruled out.

Unexpectedly, brain MRI showed several lesions including the upper pons, left cerebral peduncle, corpus callosum, and the right parietal lobe of the cerebral cortex. In poliomyelitis, contrast enhancement in the anterior horn of the spinal cord has been described on MRI (5). With respect to the brainstem, Wasserstrom et al described a patient with bulbar poliomyelitis in whom postmortem pathological tissue sections reflected changes seen on MRI of the midbrain and medulla oblongata (6). Interestingly, an abnormal high intensity signal was described in the cerebral peduncles of the midbrain as seen in the present patient. Muscle weak-
Figure 2. MRI on the 38th day after OPV intake. Axial FLAIR images show high intensity lesions in the right parietal cerebral lobe (A) and the truncus of the corpus callosum (C). Abnormal enhancements were observed in the right parietal cerebral lobe (B) and in the truncus of the corpus callosum (D) on gadolinium-enhanced T1-weighted images.

In our patient, weakness in the right lower extremity may be attributed to the lesion in the left cerebral peduncle. We also observed an abnormal high intensity signal mainly in the truncus of the corpus callosum. A reversible lesion in the splenium of the corpus callosum has been reported in encephalopathy, encephalitis, ADEM and epilepsy, and two possible mechanisms have been postulated: intramyelinic edema and inflammatory infiltrates (7). In our study, the lesion in the tegmentum of the upper pons was unusual, and seemed to correspond to decussation of the superior cerebellar peduncle. On the second MRI, the new lesion in the right parietal cortex appeared later, and contrast enhancements were observed in small regions of the corpus callosum and right parietal cortex. These findings suggested that our patient might have had inflammatory infiltrates in the lesions.

Why did our patient show various lesions on MRI? One possibility was that encephalitis had occurred by direct poliovirus infection associated with OPV. Poliovirus was not detected in viral cultures of CSF or in the stool in our patient. However, no virus was recovered from 24% of VAPP patients (8), and we could not deny direct poliovirus infection in our patient. In some VAPP patients, hyperactive reflexes have been reported, and other authors speculated involvement of the pyramidal tracts (8). Another possibility was that post-infectious encephalitis had developed by immune-mediated mechanisms. ADEM has been described in one patient, and was associated with OPV (4). This 6-year-old female patient showed spastic palsy, sensory disturbances, visual deterioration, urinary incontinence, increased myelin basic protein, and lesions of the spinal cord on MRI. Our patient showed symptoms of upper motor neuron involvement, and the myelin basic protein or oligoclonal IgG bands were not detected in CSF. Our patient’s neurophysiological examinations were normal. However, lesions in the corpus callosum, upper pons, and cerebral peduncle might be explained by demyelination of the white matter. Though multiple sclerosis must be considered as a differential diagnosis, there are no new symptoms after recovery from her illness. Various white matter lesions and no detection of poliovirus in CSF suggest that she most likely had ADEM associated with OPV.

In conclusion, we reported a patient with acute paresis and brain MRI abnormalities after OPV intake. There was no evidence of poliomyelitis, and the most probable diagnosis is ADEM associated with OPV. ADEM has not been reported as a complication of inactivated polio vaccine (IPV). OPV can also cause VAPP. While the incidence rate of complications associated with OPV is very rare, the introduction of IPV may be necessary in Japan.
References


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http://www.naika.or.jp/imindex.html