International Medical Case Reports Journal

Dovepress
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CASE REPORT

Acute Transverse Myelitis in a Child Following Oral Live Polio Vaccine Administration

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Background: Transverse myelitis is an urgent medical problem all over the world. Acute transverse myelitis is more common in children than in adults. Postvaccinal transverse myelitis is a rare complication of vaccination. In vaccine-associated paralytic poliomyelitis, the virus mosaically infects the motor neurons of the anterior horns of the spinal cord, resulting in asymmetric paralysis of predominantly proximal muscles.

Case Presentation: The case of acute transverse myelitis in a 12-year-old child corresponds to the existing definition of a vaccine-associated paralytic poliomyelitis case: a temporal relationship between the onset of flaccid paralysis and the administration of oral polio vaccine and the duration of paralysis. The child developed flaccid paralysis 18 days after (August, 2024) the administration of oral polio vaccine and persisted for more than 60 days from the onset of the disease. Vaccine virus type 3 was isolated from the feces. However, the child received 2 doses of inactivated oral polio vaccine and 1 dose of bivalent (types 1 and 3) oral polio vaccine before the disease. The child did not have an increase in the titer of antibodies in paired sera to polioviruses types 1 and 3. Spinal cord magnetic resonance imaging revealed an intramedullary focus with hyperintense MR signal on 2WI, 2FS at the level of the spinal cord cone (Th_{11}-L_2), which spread across the entire diameter and unevenly accumulated paramagnetic. These changes were characteristic of acute transverse myelitis.

Conclusion: This article presents a clinical case of Acute transverse myelitis in a child after administration of live oral polio vaccine. We aimed to emphasize the importance of differential diagnosis of myelitis in a child who received OPV. This is important from the point of view of epidemiological surveillance of poliomyelitis and possible adverse reactions after vaccination.

Keywords: acute transverse myelitis, oral polio vaccine, inactivated oral polio vaccine, vaccine-associated paralytic poliomyelitis, spinal cord, paralysis

Introduction

Transverse myelitis is a major medical problem worldwide. It affects approximately from 1 to 8 million people annually,[1] or one in three per 100,000 population.[2] Acute transverse myelitis (ATM) is five times more common in children than in adults.[3]

After the disease, approximately 33% of patients recover completely, 33% of patients have long-term mild disorders, and 33% of patients remain with persistent neurological disorders leading to disability.[4] The prognosis in children is somewhat better: almost 50% of children with acute myelitis recover within 2 years, and 10–20% of children have long-term severe disorders.[3,5]

The etiology of ATM has not been finally established. In some patients, the development of myelitis may be associated with the influence of various bacterial and viral infections (Lyme disease, syphilis, tuberculosis, Mycoplasma pneumonia, COVID-19, herpes virus and enterovirus infections, poliomyelitis, etc)., injuries, autoimmune

Received: 4 March 2025
Accepted: 8 July 2025
Published: 18 July 2025

diseases, multiple sclerosis, vasculitis and vaccine preparations.[1,6] When the cause of myelitis cannot be established, it is called idiopathic or primary myelitis, the frequency of which ranges from 30% to 60% of cases.[7]

According to the literature, among children with ATM, 66% had an infectious disease and 28–30% received vaccination within a month before the appearance of the first manifestations of the disease.[3,5,8]

Postvaccinal transverse myelitis is a rare complication of vaccination.[8] There are no separate statistics on ATM associated with vaccination. According to the literature from 1970 to 2009, only 43 cases of postvaccinal ATM were described after the administration of vaccines against viral hepatitis B, measles-mumps-rubella, diphtheria-tetanus-pertussis, rabies, tetanus, poliomyelitis, influenza, typhoid fever, and cholera.[9,10] In most cases, ATM developed within a few days to 3 months after the vaccine administration.[11]

The literature sources there is information about 11 cases of transverse myelitis per 33 million doses of the Janssen/Johnson&Johnson (J/J&J) COVID-19 vaccine administered, which is 3 cases per 10 million doses. Therefore, ATM has been recognized as a possible, very rare adverse event after the J/J&J vaccine, but a causal relationship has not been definitively established.[12] The Netherlands Pharmacovigilance Center Lareb has registered 48 reports of ATM after vaccination against COVID-19. ATM has been identified as a rare adverse event of the COVID-19 vaccines from AstraZeneca and Janssen, but a relationship between ATM and other COVID-19 vaccines (Pfizer/BioNTech and Moderna) has not been established.[13]

The mechanism of vaccine-mediated damage to the nervous system has not been fully established. There is a hypothesis of "molecular mimicry", which states that immunization can lead to autoimmune disease due to structural similarity between a viral antigen (or other vaccine component) and an auto antigen.[14,15] This similarity may trigger an autoimmune reaction. The hypothesis of a neuroinflammatory disorder is also proposed, when a cross-reaction of antibodies and T-cells in epitopes of the central and peripheral nervous system is created after vaccination.[15]

Cases of polio vaccine-associated paralysis are closely monitored worldwide. Oral polio vaccine (OPV) contains attenuated vaccine poliovirus, which in rare cases can re-acquire virulence and cause vaccine-associated paralytic poliomyelitis (VAPP) in the vaccine recipient or in contacts.[11,16] The incidence of VAPP is 1 case per 2.6 million doses of vaccine, after the first dose of OPV the incidence of VAPP reaches 1 case per 520 thousand doses and decreases with repeated administration of the vaccine (on average 1 case per 12.3 million doses).[17] In European countries, the WHO has determined the annual incidence of VAPP at 0.4–3.0 cases per million vaccinated children, with significant differences between different countries.[8] In Romania, the incidence of VAPP was significantly higher, reaching one case per 183,000 doses of OPV,[10] which is associated with the frequent intramuscular injections in this region. According to the literature, intramuscular injections within 30 days after OPV administration are one of the risk factors for VAPP.[11,18,19] Individuals with primary immunodeficiency have an almost 3000 time higher risk of developing VAPP than the general population.[20] Recognized risk factors for VAPP after OPV include impaired B-cell immunity in children and primary immunodeficiency.[19]

In populations with low immunization rates, persistently circulating vaccine-derived poliovirus (cVDPV) can cause paralytic poliomyelitis, similar to wild-type strains. Among cVDPV, type 2 (cVDPV2) is the most common: by 2015, more than 90% of cVDPV cases were associated with cVDPV2. According to WHO, since 2000, more than 10 billion doses of OPV have been administered to almost 3 billion children worldwide. During this time, 24 outbreaks of cVDPV have been reported in 21 countries, resulting in 760 cases of paralysis. WHO attributes this to low polio immunization coverage and emphasizes that if the population is fully immunized, it will be protected against both vaccine-derived viruses and wild polioviruses.[16]

In single cases, people with rare immunodeficiency disorders have experienced prolonged replication of the virus in the intestines with prolonged release into the environment after OPV administration. This variant is called vaccine-associated immunodeficiency poliovirus (iVDPV). Since 1962, 111 cases of iVDPV have been reported worldwide. Most of these patients either ceased shedding the virus within six months or died.

VAPP occurs due to mutation reversion in the viral genome of vaccine strains of poliovirus with the acquisition of virulence.[21] For example, a U to C reversion at nucleotide 472 is observed in the virus isolated from patients with VAPP caused by vaccine virus type 3. After the administration of OPV, the virus multiplies mainly in the intestinal mucosa, then enters the mesenteric lymph nodes and then into the blood. In 99% of cases, the immune system response (mainly due to

interferon alpha/beta (IFNα/β)) limits poliovirus replication.[12] However, in 1–2% of people, this response may be insufficient, as a result of which the virus reaches the central nervous system and can cause paralysis.[21]

The median time between OPV administration and the onset of VAPP symptoms was usually 2 weeks.[19] VAPP usually presents with acute flaccid paralysis (AFP). Specific symptoms of VAPP described in the literature include irritability, muscle weakness, hypotension, atrophy, and absence of tendon reflexes in the absence of sensory deficits.[19,22] In addition to VAPP, one case of postvaccinal flaccid monoparesis after the first dose of OPV has been described, which presented with muscle weakness, hypotension, and atrophy of the lower limb.[22]

In VAPP, complete recovery occurs in approximately 15% of cases. According to Suares J.E. (2024) among the registered cases of VAPP, fatal outcomes were in 6.15% of cases, and permanent disability was in 36.9% of cases.[19]

Inactivated oral polio vaccine (IPV) does not carry a risk of VAPP, and many countries have therefore abandoned the use of OPV. No case of VAPP after IPV administration has been described in the available literature, even when OPV vaccination was continued.[11,23]

Rare neurological complications associated with OPV include encephalitis, seizures, transverse myelitis, and Guillain-Barré syndrome (GBS).[24]

The cause-and-effect relationship between the administration of OPV and VAPP/ATM is proven by determining the neurovirulence of selected viruses in a mouse model in vivo. Neurovirulence is expressed in PD (50). PD (50) is the dose of virus that causes paralysis in 50% of mice. The PD (50) for wild-type poliovirus was 3.83, for the attenuated vaccine strain was 7.63, for the virus from a patient with transverse myelitis was 4.81, and for the poliovirus that caused VAPP was 4.96.[25]

Molecular biology studies of polioviruses isolated from feces and the central nervous system of patients with VAPP confirmed the vaccine origin of the isolates and revealed genomic modifications that enhance their neurovirulence. However, similar genomic modifications have been found in strains isolated from healthy OPV recipients and healthy contacts. This indicates that susceptibility and immune response of the host is also involved in the development of VAPP.[24]

Case Presentation

A 12-year-old child has become acutely ill. Symptoms appeared suddenly while fishing. The boy squatted down, suddenly felt numbness and weakness first in the left, and then in the right leg and lower third of the torso, lower back pain and acute urinary retention appeared. 3–4 days before that, the child felt pain in the legs and a feeling of "tingles running".

According to the immunization record, the child was vaccinated with IPV at the age of 6 months and 8 months and OPV at the age of 1 year and 3 months. Revaccination against poliomyelitis at the age of 6 years, according to the vaccination calendar in the country, was not carried out. The child has not been vaccinated against COVID-19.

Eighteen days before the onset of the disease (at the age of 12 years), the child was revaccinated against polio with OPV.

During hospitalization in the infectious disease department, the child's general condition was severe. Tone in the lower extremities was reduced. Tendon reflexes: from the hands – normotonic; from the feet – knee, Achilles were not evoked; cremasteric and abdominal reflexes were absent. There were no active movements in the lower limbs. All types of deep and superficial sensitivity were absent. Sensitivity disorders by conductive type from level T −10. Muscle tone in the hands was preserved, in the legs was absent. Does not control the work of the pelvic organs, disorders of the central type. Consciousness was clear, according to the Glasgow Coma Scale (GCS) – 15 points. Eyes were symmetrical, eye movement was full, photoreaction was lively, symmetrical. Cranial nerves without pathological changes. Meningeal symptoms were absent.

A magnetic resonance imaging (MRI) scan of the spine (spinal cord) was performed.

The result of the MRI: Local uneven expansion of the central spinal canal at the levels of Th_3-Th_9. Limbus vertebra (bone tubercle) of the L_2 vertebra. MR signs of myelopathy at the level of the spinal cord cone. MR picture corresponds to transverse myelitis (Figure 1A and B).

Serological examination of blood was performed using polymerase chain reaction (PCR) for herpesvirus DNA: HSV ½. Epstein-Barr, CMV – not detected.

Virological study of feces on cell culture was conducted and intratypic differentiation for polioviruses in the reference laboratory of the Center for HIV/AIDS Diagnostics, Viral and Particularly Dangerous Pathogens with confirmation in the WHO regional reference laboratory. Vaccine poliovirus type 3-Sabin-3 (PV-SL3) was detected in the first sample. Serological examination of blood by the paired serum method: antibody titers to poliovirus PV1 – 1:32 and PV type 3–1:16; in the dynamics of the study after 3 weeks, antibody titers to poliovirus PV1 – 1:32 and PV type 3–1:16.

Due to the lack of positive dynamics, the child was directed to the State Institution "Institute of Neurosurgery" (Kyiv) to clarify the diagnosis and determine further treatment tactics.

On the 18-th day after the onset of acute lower paraplegia there were complaints about the inability to make active movements in the legs, loss of all types of sensitivity in the legs, lower abdomen and lower lumbar area, perineum area; uncontrolled work of the pelvic organs and complete absence of sensations during urination and defecation. On the heels – initial signs of bedsores. Clearly expressed symptoms of oral automatism: proboscis, naso-labial. Periosteal and tendon reflexes from the hands were lively, symmetrical, from the legs were absent. Abdominal reflexes: upper was evoked, torpid, middle and lower were absent. Cremasteric reflexes were absent. Absence of all types of sensitivity (deep and superficial) in the legs and lower parts of the trunk (lower abdomen, lumbar zone, perineum) – from the level T 10 by conductive type. Muscle strength in the hands was preserved, absent in the legs. Muscle tone in the hands was preserved, normal, absent in the legs. No pathological foot signs were detected. Finger-nose test in the supine position was performed clearly. Did not sit independently, can sit for some time with support (the end of the bed is raised). Did not control the work of the pelvic organs. Acute lower paraplegia was diagnosed. Transverse myelitis Th_{11}-L_5. Isolation of vaccine poliovirus type 3.

A spinal tap was performed, revealing: cytosis 3 cells per μL, protein 0.99 g/l; RNA/DNA of cerebrospinal fluid enteroviruses, EBV, CMV, herpes viruses of the group – 5,2, 6, 8 types were not detected. Due to the fact that the child did not urinate on his own, a urinary catheter was installed. Urinary tract infection was diagnosed. An MRI of the spine (spinal cord) was performed in dynamics (Figure 2).

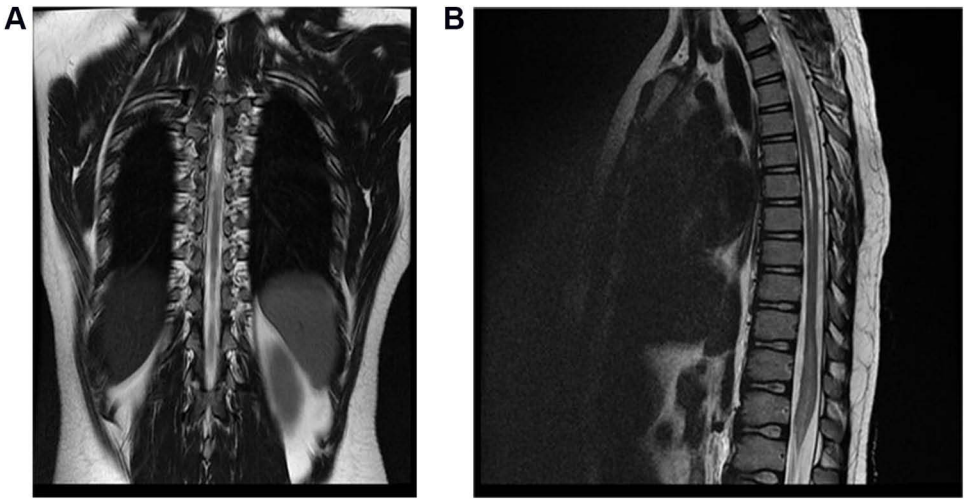

Figure 1 (**A**) A magnetic resonance imaging (MRI) scan of the spine (spinal cord) of the patient (frontal projection). (**B**) A magnetic resonance imaging (MRI) scan of the spine (spinal cord) of the patient (sagittal projection).

The spinal cord is located in the center of the spinal canal. In comparison with the previous MR study (Figure 2), there is a moderate positive MR dynamics in relation to previously detected changes at the level of the spinal cord cone (Th_{II}-L_2) in the form of a moderate decrease in the volume of the lesion due to a moderate weakening of edematous changes, a decrease in the intensity of the accumulation of paramagnetic in this area. The zone of the changed MR signal, as before, extended to the entire diameter of the spinal cord, is accompanied by a restriction of diffusion and an increase in the MR signal on TIWI, which may be due to diffuse hemorrhagic infiltration. The degree of hydromyelia at the level of Th_6-Th_9 has somewhat decreased, now the width of the central canal is approximately 0.27 cm (previously it was 0.38 cm). In the anterior cords of the spinal cord, more to the right, at the level of Th_7-Th_9, small foci of hyperintense MR signal on 2WI are determined, which were not clearly visualized during the previous study. At the level of Th_{11}, there is moderate local thinning of the spinal cord.

Considering the clinical data, anamnesis, and MRI dynamics of the process, the indicated intramedullary changes at the level of the spinal cord cone and at the level of Th_6-Th_9 may be due to a combination of two processes: transverse myelitis and impaired spinal blood circulation.

On the 27-th day after the onset of acute lower paraplegia the consciousness was clear. Emotionally labile: episodic euphoria alternates with episodes of depression. Could sit independently for some time (up to 5 min), but could not sit up on his own. Minimal active efforts in the lower extremities appeared: with passive bending of the legs in the knee joints in the supine position – could tilt the legs to the right and left. The face was symmetrical. Full eye movement. Photoreaction was lively, symmetrical. Bulbar disorders were absent. Periosteal and tendon reflexes from the hands were lively, symmetrical, reflexogenic zones were not disturbed; from the feet were absent. Meningeal symptoms were absent. No pathological foot signs were found. Abdominal and cremaster reflexes were absent.

Partial restoration of vibration and temperature sensitivity at the level of Th_{11}-Th_{12} was noted; complete absence of all types of sensitivity according to the conductive type from the level of L_1 and mosaic violation of all types of sensitivity at the level of T_{10}-T_{12}. Involuntary muscle contractions – fasciculation and fibrillation appeared in the paralytic limbs. Pronounced symptoms of oral automatism were preserved: proboscis, naso-labial and palmar-chin reflexes. The work of the pelvic organs is not controlled by the control; violation of the work of the pelvic organs according to the central type. When examining the amplitude of passive movements in the lower limbs, initial signs of restriction were found, which might be a prerequisite for the formation of contractures.

Another MRI of the spine (spinal cord) was performed in dynamics with intravenous contrast (Figure 3).

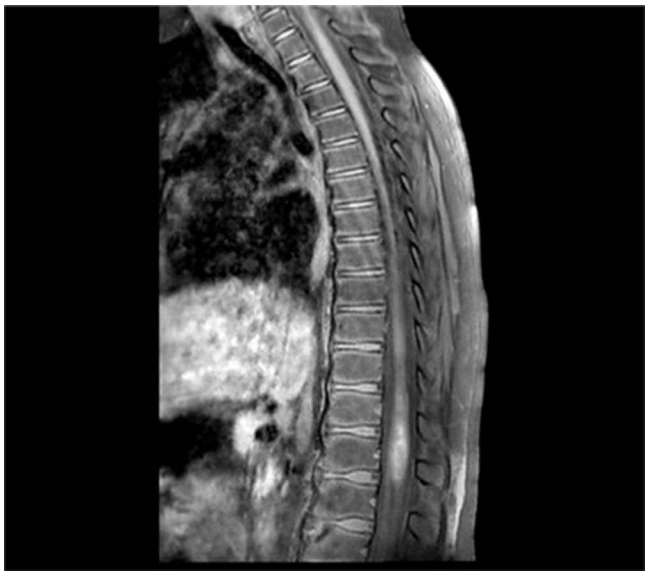

Figure 2 An MRI of the spine (spinal brain) in dynamics.

https://doi.org/10.2147/IMCRJ.S526156

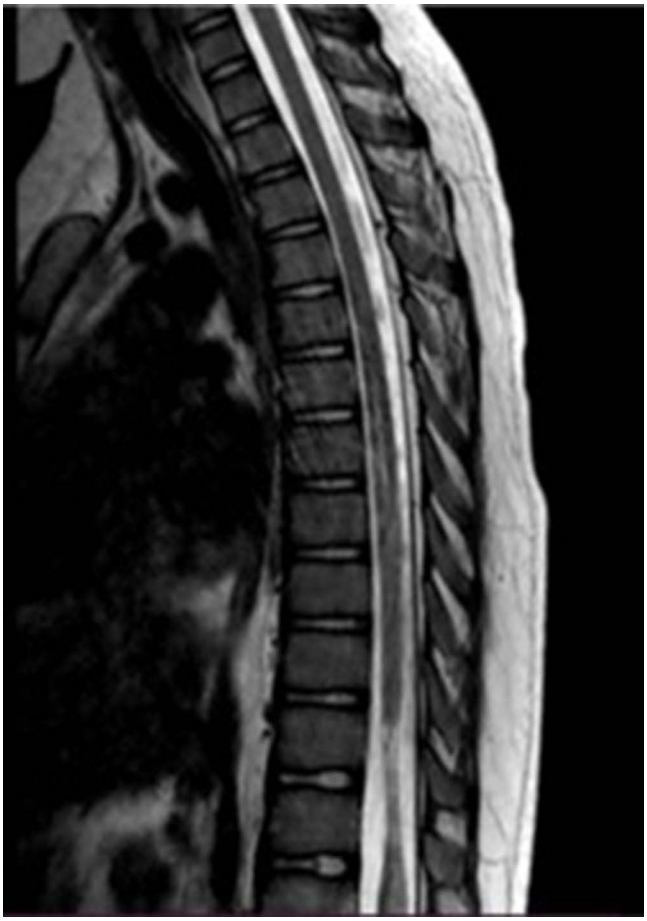

Figure 3 An MRI of the spine (spinal brain) in dynamics with intravenous contrast.

The result of the MRI (Figure 3): In comparison with the previous MR study, atrophic changes in the spinal cord were observed at the level of Th_{11}-L_1 in the form of a decrease in volume, which extends over its entire diameter, the MR signal from its structure was heterogeneously increased on T2WI, STIR. After pre-contrast, areas of paramagnetic accumulation were observed. At the level of the spinal cord cone, as before, a diffuse increase in the MR signal on T1WI was observed, which might be due to hemorrhagic infiltration. Objectively analyzing the dynamics of the disease, the MRI data could not be assessed as positive. Against the background of a decrease in edema, extensive areas of atrophy and gliosis were visualized in numerous segments of the spinal cord, and as a result, the increase in hydromyelia. Clear correlations could be noted between the dramatic MRI indicators of the spine, pathological neurological status and pessimistic development of the clinical picture.

For further treatment and rehabilitation, the child was referred to the neurological department at his place of residence.

Discussion

A case of VAPP is defined as a case of acute flaccid paralysis with residual paralysis (consistent with paralytic poliomyelitis) lasting at least 60 days and occurring in an OPV recipient between 4 and 40 days after vaccine administration.[19,26] On the other hand, it can develop in a person who had known contact with a vaccine recipient between 7 and 60–75 days after a dose of OPV.

Isolation of vaccine-associated poliovirus from any stool specimen and absence of wild poliovirus isolation is often used as VAPP diagnosis criteria, but virus isolation alone cannot be the basis for the diagnosis.[27]

In VAPP, the virus mosaically infects the motor neurons of the anterior horns of the spinal cord, resulting in asymmetric paralysis of predominantly proximal muscles. VAPP is clinically indistinguishable from poliomyelitis caused by the wild poliovirus strain. Impaired sensitivity and function of the pelvic organs is not typical for VAPP.[10,16]

Clinical manifestations of ATM associated with OPV vaccination, include motor, sensory and autonomic dysfunctions, often with pelvic organ dysfunction.[1,10,28]

In ATM, regardless of etiology, spinal MRI reveals a widespread, centrally located intramedullary lesion extending into 3 vertebral segments, predominantly thoracic, and involving all or most of the cord diameter.[29] Involvement of smaller number of segments is more typical of transverse myelitis associated with multiple sclerosis, while involvement of more than three vertebral segments increases the likelihood of idiopathic ATM or neuromyelitis optica.[10]

The case of ATM in a 12-year-old child described by us corresponds to the existing definition of a VAPP case: a temporal relationship between the onset of flaccid paralysis and the administration of OPV and the duration of paralysis. The child developed flaccid paralysis 18 days after the administration of OPV and persisted for more than 60 days from the onset of the disease. Vaccine virus type 3 was isolated from the feces. However, the child received 2 doses of IPV and 1 dose of OPV before the disease. It is known from the literature that the use of IPV allows to level the development of VAPP with the sequential use of IPV-OPV.[23,30] The child did not have an increase in the titer of antibodies in paired sera to polioviruses types 1 and 3. These facts minimize the causal relationship between vaccination and ATM.

The patient had sensory and motor disorders in the distal and proximal leg muscles with pelvic organ dysfunction, which was not typical for VAPP. But this fact was often found in ATM of various etiologies.

Spinal cord MRI revealed an intramedullary focus with hyperintense MR signal on 2WI, 2FS at the level of the spinal cord cone (Th_{11}-L_2), which spread across the entire diameter and unevenly accumulated paramagnetic. These changes were characteristic of ATM. In addition, the first images revealed local uneven expansion of the central spinal canal at the levels of Th_3-Th_9. In the dynamics of spinal cord MRI, atrophic changes were detected at the level of Th_{11}-L_1, which spread across the entire diameter, the MR signal was heterogeneously increased on T2WI, STIR with accumulation of paramagnetic after contrast. At the level of the spinal cord cone, a diffuse increase in the MR signal on T1WI was observed, which might be due to hemorrhagic infiltration.

The clinical picture and MRI data were described: sensory, motor and autonomic disorders below the Th_{11} level, pelvic organ dysfunction, spinal cord lesions throughout the entire diameter, hyperintense intramedullary lesions on T2WI, STIR at the level of the spinal cord cone (Th_{11}-L_2) with paramagnetic accumulation after contrast, meeting the criteria for ATM.

The case was considered by an expert commission of specialists. An expert commission emphasized that the presence of prior administration of 2 doses of IPV, one dose of OPV, the absence of an increase in the titer of antibodies to type 3 poliovirus isolated from feces, did not make it possible to unequivocally consider ATM in a child as a vaccine-associated complication of OPV.

By presenting this clinical case, we aimed to emphasize the importance of differential diagnosis of myelitis in a child who received OPV. This is important from the point of view of epidemiological surveillance of poliomyelitis and possible adverse reactions after vaccine administration.

The presented study has certain limitations. First of all, this is a clinical case that described the condition of one child and does not represent a sample of a certain cohort of patients. We also have some limitations in conducting laboratory studies, such as genotyping and studying the virus for neuropathogenicity due to the war in our country. But this does not eliminate the need to conduct differential diagnostics of myelitis. In further cases, conducting a detailed laboratory study, virological sequencing, and a comprehensive immunological assessment is desirable.

Abbreviations

ATM, acute transverse myelitis; OPV, oral polio vaccine; IPV, inactivated oral polio vaccine; VAPP, vaccine-associated paralytic poliomyelitis; cVDPV, circulating vaccine-derived poliovirus; iVDPV, vaccine-associated immunodeficiency poliovirus; GCS, Glasgow Coma Scale; AFP, acute flaccid paralysis; MRI, magnetic resonance imaging.

Data Sharing Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed.

Ethical Approval

Ethical approval is not required to publish the case details in accordance with local or national guidelines.

Consent for Publication

Written informed consent was obtained from the parents of the minor patient, including for the publication of the article and its content.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study did not receive any external funding.

Disclosure

The authors report no conflicts of interest for this work.

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