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PEDIATRIC ACUTE FLACCID PARALYSIS: ENTEROVIRUS D68–ASSOCIATED ANTERIOR MYELITIS

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☐ **Abstract—Background: Enteroviral infections can cause acute flaccid paralysis secondary to anterior myelitis. Magnetic resonance imaging (MRI) is important in the diagnosis of this potentially devastating pediatric disease. Before the 2014 outbreak of Enterovirus D68 (EV-D68), the virus was considered a relatively benign disease. Case Report: A fully immunized 8-year-old boy was brought to the emergency department complaining of a cough, headache, neck pain, and right arm pain and weakness. Deep tendon reflexes in the weak arm could not be elicited. MRI of the brain and cervical spine revealed anterior myelitis of the cervical spine. The patient was given intravenous antibiotics, acyclovir, and methylprednisolone with no initial improvement. He was then given intravenous immunoglobulin over 3 days with improvement in symptoms. Nasal swab polymerase chain reaction revealed EV-D68. Despite medical management, the child was left with long-term motor disability in the effected extremity. Why Should an Emergency Physician Be Aware of This?: Acute flaccid paralysis is a potential devastating complication of enteroviral infections. Extremity complaints in the clinical setting of central nervous system infection should raise concern for encephalomyelitis. MRI is extremely helpful in establishing this diagnosis. Prevalence of non-polio enteroviral paralytic events is increasing in the United States. Potential EV-D68 cases should be reported to local health departments. Emergency medicine providers should consider this complication in the child with acute, unexplained significant respiratory illness with new neurologic complaints. Published by Elsevier Inc.**

☐ **Keywords—AFP; acute flaccid paralysis; enterovirus; anterior myelitis; pediatric; EV-D68**

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INTRODUCTION

Acute flaccid paralysis (AFP) is a specific clinical syndrome causing focal neurologic disease. AFP is related to infectious, traumatic, metal toxicity, or post-infectious autoimmune conditions (1,2). Focal neurologic diseases in pediatric patients create a unique diagnostic and management challenge. In contrast to the adult population, neurovascular events are rare and thorough evaluations for infectious, traumatic, and oncologic etiologies are often required. The ability to make timely, accurate diagnoses and initiate appropriate therapy in these circumstances can often be a challenge. The importance of awareness of

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AFP is important given the increasing number of reported Enterovirus D68 (EV-D68) cases in the United States from 2014 to 2016 (3).

Enteroviral infections can cause aseptic meningitis, encephalitis, or AFP secondary to anterior myelitis (4). Non-poliomyelitis enteroviral infections have historically caused regional epidemics of brainstem encephalitis and AFP secondary to anterior myelitis (5–7). Patients with AFP classically have motor abnormalities, such as muscle weakness and altered deep tendon reflexes, but tend not to have sensory dysfunction (8,9). Magnetic resonance imaging (MRI) has classic findings for infectious anterior myelitis and can assist in establishing the diagnosis (10). We report an 8-year-old boy who presented with unilateral upper-extremity flaccid paralysis secondary to EV-D68.

CASE REPORT

A fully immunized 8-year-old boy was brought to the emergency department (ED) complaining of 7 days of cough, headache, neck pain, and right arm pain. He otherwise had no significant medical history. The symptoms began after a local coastal camping trip where he was bitten by several mosquitos. His parents initially sought help at an urgent care clinic 4 days after his symptoms began. At the urgent care clinic he was started on azithromycin after an abnormal chest radiograph was suggestive of community-acquired pneumonia. Over the next 3 days, his neck pain became progressively worse with flexion and he also began to experience right upper arm pain. On day 7, he presented to the ED with arm weakness, abdominal pain, and anorexia.

His vital signs in the ED included blood pressure 123/76 mm Hg, pulse 57 beats/min, temperature 98.6°F, SpO_2 100%, and respiration 20 breaths/min. Physical examination was notable for significantly uncomfortable-appearing child with pain upon passive motion of the right arm or neck. Flexion of the neck per the child was debilitating and sent a sensation down the arm and upper back. Deep tendon reflexes (DTRs) in the weak arm were not able to be elicited and caused significant pain. Remaining neurologic examination was normal, including strength and DTRs in other extremities.

A repeat chest radiograph revealed stranded opacity at the right lung base, suggestive of subsegmental atelectasis. His chemistry panel, complete blood count, urinalysis, creatinine kinase, and C-reactive protein levels were within normal limits. Liver function testing revealed elevation of aspartate aminotransferase to 222 U/L (normal range: 15–40 U/L) and alanine aminotransferase to 147 U/L (normal range: 10–35 U/L). A lumbar puncture was performed and the findings revealed white blood cell count (WBC) of 250/μL (neutrophils 23%, lymphocytes 63%, mononuclear cells 14%, eosinophils undetected), red blood cell count of 455/μL, protein 42 mg/dL (15–45 mg/dl), and glucose 50 mg/dL (40–70 mg/dL). Enterovirus polymerase chain reaction (PCR) was negative. Cerebrospinal fluid (CSF) gram stain was negative. Opening pressure was not recorded.

The overall picture was concerning for an infectious encephalitis of unknown etiology and the child was started on 100 mg/kg ceftriaxone and 10 mg/kg acyclovir and 2 mg/kg solumedrol before being transferred to the pediatric intensive care unit. Although poorly cooperative, he showed allodynia of the neck and significant weakness in the proximal right arm with relative preservation of strength in the right hand. Glasgow Coma Scale score declined to 12 during initial 12 h of admission. The remainder of his examination was normal, including a normal cardiac examination, a mildly tender nonsurgical abdomen, a nonpalpable liver edge, and no skin or mucosal lesions.

Due to worsening right arm pain and depressed mental status, MRIs of the brain and cervical spine were obtained, which revealed central intramedullary T2 hyperintensity and swelling of the cervical spine that extended to the cervicomedullary junction. There was subtle involvement of the dorsal pons, midbrain, periaqueductal region, and the cerebellum around the margins of the fourth ventricle bilaterally (Figure 1).

Antibiotics and acyclovir were continued until bacterial cultures were negative and herpes simplex virus (HSV) PCR results were finalized. Based on degenerative MRI findings and a concern for post-infectious etiology, the patient was started on 30 mg/kg/d methylprednisolone shortly after admission. With no improvement in neurologic deficits after 3 days, the patient was given 2 g/kg intravenous immunoglobulin (IVIG) over 3 days. Four days after admission, the patient showed improvement in mental status, which returned to baseline by hospital day 14. His neurologic examination, however, demonstrated a persistent and unimproved proximal right upper extremity weakness with 0/5 to 1/5 strength proximally and 4/5 distally.

The patient's nasal respiratory swab was negative for all viral studies except for enterovirus. The local department of health confirmed typing of EV-D68. A repeat lumbar puncture showed a normal opening pressure, WBC 12/μL, negative bacterial cultures, and negative HSV and enterovirus PCR. All remaining laboratory values were normal, including California encephalitis and West Nile viruses. A repeat MRI after completion of his steroid course showed a near-complete resolution of spinal cord lesions, although this did not correspond with complete clinical improvement (Figure 2).

The patient was discharged after a 2-week hospital stay with a referral for outpatient neurorehabilitation.