

## Correspondence



## Poliomyelitis Due to West Nile Virus

*To the Editor:* Poliomyelitis is a clinical syndrome defined by the presence of fever, meningitis, and flaccid paralysis. In the United States, this syndrome was historically associated with infection by poliovirus but is now more commonly seen with other enteroviruses. We describe a case of poliomyelitis in a patient infected with West Nile virus, a flavivirus.

A 50-year-old woman from Louisiana had a headache on the day before she traveled to Georgia for the July 4 holiday. After she arrived, her headache worsened, and she had severe myalgia. Two days after the onset of headache, weakness developed, and the patient was admitted to the hospital. She was febrile (temperature, 39.5°C) but was awake, alert, and fully cognizant. She had moderate bifacial and appendicular weakness (Medical Research Council grade 4–5), with a normal sensory examination and retained deep-tendon reflexes. Lumbar puncture showed 54 white cells per cubic millimeter (22 percent neutrophils), with a normal glucose level and a protein level of 110 mg per deciliter (normal level, <45 mg per deciliter).

She remained febrile through the eighth day. Her weak-

ness progressed, and she required intubation on the sixth day after the onset of headache, when she could no longer lift her head or move her arms or legs against gravity. Her cognition, sensation, and reflexes remained normal. Magnetic resonance images of the complete spinal cord were normal. Serial electrodiagnostic findings are presented in Table 1.

On day 12 of hospitalization, a cerebrospinal fluid specimen tested positive for antibodies against West Nile virus (IgM titer, 1:256; IgG titer, 1:128). Stool cultures and polymerase-chain-reaction studies for the presence of enteroviruses were negative. Two months after the onset of weakness, the patient remains in a rehabilitation facility and requires respiratory assistance.

This unusual clinical presentation of paralytic poliomyelitis is distinguished from Guillain-Barré syndrome, the most common cause of acute flaccid paralysis in the United States, by the presence of fever, pleocytosis, and retained tendon reflexes. Electrodiagnostic studies during the acute phase of the illness confirmed the pure motor nature of her illness, excluded demyelination as a pathogenic mechanism, and identified the anterior horn cell as the site of pathology.

Previous reports of West Nile virus, including those from the New York City outbreak of 1999, described patients with weakness.<sup>1,2</sup> The mechanism underlying weakness in West Nile virus has not been clearly established, and in some cases, this symptom has been attributed to Guillain-Barré syndrome. Our data and those of previous studies are consistent with effects at the level of the anterior horn cell. Autopsies in four patients did not include spinal cord.<sup>3</sup>

Poliovirus attacks motor neurons by attaching to the poliovirus receptor on the anterior horn cell. Flaviviruses other than West Nile virus are not known to cause poliomyelitis. These findings raise the possibility that these viruses can attack motor neurons directly.

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TABLE 1. ELECTRODIAGNOSTIC DATA.

| VARIABLE                                 | DAY 4           | DAY 11   | DAY 18   |
|--|-----------------|--|--|
| Sensory amplitudes                       | Normal          | Normal   | Normal   |
| Motor amplitudes                         | Normal          | 25–50%<br>of normal                              | 25–50%<br>of normal                              |
| Motor distal latencies                   | Normal          | Normal   | Normal   |
| Conduction velocities                    | Normal          | Normal   | Normal   |
| Spontaneous activity on electromyography | None            | None   | Profuse in proximal and distal muscles           |
| Motor units and recruitment              | Patient sedated | Normal motor units, severely reduced recruitment | Normal motor units, severely reduced recruitment |

1. Gadoth N, Weitzman S, Lehmann EE. Acute anterior myelitis complicating West Nile fever. *Arch Neurol* 1979;36:172-3.

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3. Sampson BA, Armbrustmacher V. West Nile encephalitis: the neuropathology of four fatalities. *Ann N Y Acad Sci* 2001;951:172-8.

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