

Correspondence



A Poliomyelitis-like Syndrome from West Nile Virus Infection

To the Editor: Muscle weakness is a common finding and an important predictor of death in patients with West Nile virus encephalitis.^{1,2} Yet this important sign does not have a defined pathological basis. In monkeys, horses, and birds, West Nile virus causes poliomyelitis.³⁻⁵ Our clinical and electrodiagnostic findings in three consecutive patients with confirmed West Nile virus infection suggest that the virus also attacks the spinal cord in humans.

Patient 1, a 56-year-old man, presented with fever, chills, night sweats, myalgias, and confusion. Weakness gradually developed in his arms, along with flaccid paralysis in his right leg, areflexia, bladder dysfunction, and acute respiratory distress. He reported that he had no pain or paresthesias. Sensory examination was normal. Suspected diagnoses included stroke, Guillain-Barré syndrome, and inflammatory myopathy, for which he received anticoagulation therapy and intravenous immune globulin and underwent muscle biopsy. Cerebrospinal fluid showed 3 white cells per cubic millime-

ter, a glucose level of 54 mg per deciliter, and a protein level of 89 mg per deciliter. Magnetic resonance imaging (MRI) of the brain and cervical spine was normal. In an enzyme-linked immunosorbent assay, the ratio of IgM antibodies against West Nile virus (the ratio of the reactivity of the patient's serum to West Nile virus antigen to the reactivity of control serum to the same antigen) was 14.78:1 (reference ratio, <2.00:1). Electrodiagnostic studies showed reduced motor responses, preserved sensory responses, and scattered denervation without evidence of myopathy or polyneuropathy (Table 1).

Patient 2, a 57-year-old man, presented with fever, chills, nausea, vomiting, and headache. Asymmetric flaccid paralysis developed, involving the distal portion of the left leg, the right thigh, and the right arm, as well as areflexia, dysphagia, urinary incontinence, and acute respiratory distress requiring mechanical ventilation. The patient reported having no altered sensation. Sensory examination was normal except that there was a slight decrease in vibratory sensation in the toes bilaterally. Cerebrospinal fluid showed 80 white cells per cubic millimeter, a glucose level of 99 mg per deciliter, and a protein level of 196 mg per deciliter. The virus-specific IgM ratio was 24.48:1. MRI of the brain was normal. Electrodiagnostic studies showed reduced motor responses, normal sensory responses, widespread denervation, and neurogenic recruitment (Table 1).

Patient 3, a 50-year-old man, presented with severe nausea, vomiting, headache, and diarrhea, but no fever. He was given a diagnosis of food poisoning, but flaccid paralysis developed, together with areflexia limited to the right arm,

TABLE 1. MOTOR AND SENSORY AMPLITUDES.*

NERVE	STIMULATION SITE	RECORDING SITE	PATIENT 1		PATIENT 2		PATIENT 3		NORMAL VALUES
			RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	
Median motor nerve (mV)	Wrist	Abductor pollicis brevis	3.9	—	3.8	—	2.0	11.4	≥5.0
Median sensory nerve (μV)	Wrist	Second digit	20.4	—	34.4	—	33.4	35.4	≥20.0
Ulnar motor nerve (mV)	Wrist	Abductor digiti minimi	5.0	—	4.4	—	1.6	7.5	≥4.5
Ulnar sensory nerve (μV)	Wrist	Fifth digit	18.8	—	17.8	—	28.8	27.6	≥15.0
Musculocutaneous motor nerve (mV)	Erb's point	Biceps	2.4	—	—	—	0.2	7.6	≥4.0
Musculocutaneous sensory nerve (μV)	Elbow	Forearm	10.6	—	17.2	—	27.1	32.6	≥10.0
Axillary motor nerve (mV)	Erb's point	Deltoid	1.7	—	0.3	—	0.4	5.3	≥4.0
Radial sensory nerve (μV)	Forearm	Dorsum of hand	23.2	—	48.6	—	31.9	31.8	≥15.0
Peroneal motor nerve (mV)	Ankle	Extensor digitorum brevis	NR	4.2	1.2	0.1	—	—	≥2.0
		Knee	0.2	2.8	—	0.4	—	—	≥4.0
Peroneal sensory nerve (μV)	Leg	Dorsum of foot	2.2	3.1	6.6	7.1	—	—	≥5.0
Tibial motor nerve (mV)	Ankle	Abductor hallucis	2.5	7.6	2.8	3.3	—	—	≥3.0
Sural sensory nerve (μV)	Posterior leg	Ankle	8.4	9.7	13.3	14.8	—	—	≥8.0

*NR denotes no response.

without pain or paresthesias. Sensory examination was normal. He received anticoagulation therapy for suspected stroke, although MRI of the brain was normal. The ratio of IgM against West Nile virus was 2.08:1. No spinal tap was performed. After the patient was transferred to a rehabilitation hospital, the IgM ratio was 25.74:1. Electrodiagnostic studies showed markedly reduced motor responses in the monoplegic limb, with normal sensory responses (Table 1).

Asymmetric flaccid paralysis and areflexia developed in all three patients, and two had bladder dysfunction and acute respiratory distress. Electrodiagnostic findings confirmed involvement of anterior horn cells or motor axons. These clinical and electrodiagnostic findings are classic features of poliomyelitis and strongly suggest that in humans, as in animals,³⁻⁵ the spinal cord gray matter is a target of West Nile virus. The poliomyelitis-like presentation in our cases warrants a reevaluation of other cases of West Nile virus with similar presentation that have previously been attributed to Guillain-Barré syndrome or axonal polyneuropathy.^{1,2} Awareness that muscle weakness with West Nile virus infection may be of spinal origin should help to eliminate mis-

diagnoses and inappropriate treatment, and it should encourage pathological study of the spinal cord.

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