Sensory and autonomic involvement in West Nile virus-associated acute flaccid paralysis

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Practical Implications

Consider West Nile virus neuroinvasive disease in the differential diagnosis of acute flaccid paralysis even if there is clinical evidence of associated sensory and autonomic dysfunction or signal abnormality on MRI that extends beyond the spinal cord to involve the nerve roots or plexus.

West Nile virus (WNV) is an arthropod-borne flavivirus that rarely causes acute flaccid paralysis, classically presenting as a pure motor neuronopathy without sensory abnormalities.¹ We describe a patient with WNV-associated acute flaccid paralysis and clinicoradiographic disease involvement beyond the anterior horn cell, with sensory and autonomic dysfunction as well as signal abnormality of the spinal cord, nerve roots, and lumbosacral plexus on MRI.

Case

A 27-year-old healthy man acquired mosquito bites while visiting Mississippi in September 2016. Two weeks after returning to Ontario, he experienced fevers, myalgias, and a morbilliform rash, followed by progressive right leg weakness over 1 month, prompting neurology assessment on November 1, 2016. On history, he reported right leg weakness and dysesthesia, as well as urinary hesitancy. On examination, he was afebrile. Cognition was intact. There was no meningismus. Cranial nerve examination and motor, sensory, and coordination testing of the asymptomatic extremities was normal. There was diffuse weakness of the right leg graded 1-2/5, with reduced tone, depressed reflexes, and atrophy (figure 1). Sensory examination identified decreased pinprick appreciation in the right L5 distribution, and mottling of the distal right leg was noted. His presentation was compatible with a lower motor neuron syndrome causing motor, sensory, autonomic bladder, and vasomotor dysfunction. He had no orthostasis, constipation, or erectile dysfunction to indicate more severe autonomic involvement. Nerve conduction studies on November 2, 2016, showed reduced compound muscle action potentials of the right common peroneal nerve to tibialis anterior and tibial nerve to abductor hallucis, with normal conduction velocity of the right common peroneal nerve across the fibular head. Right superficial peroneal and sural sensory nerve action potentials were normal. Needle EMG of proximal and distal right leg muscles found fibrillations and positive sharp waves, as well as reduced motor unit recruitment. Gadolinium-enhanced lumbosacral spine MRI showed central T2 hyperintensity of the conus medullaris, right-sided nerve root enhancement, and T2 hyperintensity of the right lumbosacral plexus (figure 2). Serum WNV immunoglobulin M (IgM) enzyme immunoassay (EIA) and flavivirus IgG EIA on November 3, 2016, were negative. CSF on November 3, 2016, revealed 5 leukocytes/ μ L (93% lymphocytes), elevated protein at 546 mg/L, normal glucose, elevated immunoglobulin G (IgG) index, 5 oligoclonal bands not present in serum, and positive WNV IgM EIA. Repeat serum WNV IgM EIA and flavivirus IgG EIA on November 10, 2016, were both positive, with a plaque reduction neutralization test (PRNT) that identified WNV-specific neutralizing

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Figure 1 Muscle atrophy in West Nile virus-associated acute flaccid paralysis

Profound atrophy of the right anterior thigh is seen, 1 month after onset of right leg weakness.

antibodies at a titer of 1:160 (normal <1:10). All WNV testing was performed by Public Health Ontario. The patient was diagnosed with neuroinvasive WNV disease and underwent intensive rehabilitation. When reassessed on March 10, 2017, right leg strength had improved to 4/5 proximally but dorsiflexion remained 1/5. His right leg dysesthesia, mottling, and urinary hesitancy had markedly improved.

DISCUSSION

We describe a patient with WNV-associated acute flaccid paralysis and clinical evidence of sensory and autonomic dysfunction. Lumbosacral spine MRI revealed signal abnormality not only in the spinal cord but also the nerve roots and lumbosacral plexus, suggesting disease involvement beyond the anterior horn cell. Electrodiagnostic testing was consistent with a motor neuronopathy, which is typical of WNV-associated acute flaccid paralysis. The normal sensory nerve action potentials indicated a lesion proximal to the dorsal root ganglia causing sensory symptoms in our patient, and was compatible with disease involvement of the right L5 nerve root leading to corresponding sensory loss and root enhancement on MRI. Normal sensory responses would not suggest a plexopathy, making it unclear if our patient had mild lumbosacral plexus disease involvement that was only identified radiographically. Clinicians should be aware that this more extensive clinicoradiographic disease presentation can occur, and atypical neuromuscular manifestations of neuroinvasive WNV infection such as radiculitis, plexitis, and Guillain-Barré syndrome have been rarely reported.²⁻⁵ Regarding diagnostic testing, positive WNV IgM in CSF indicates neuroinvasive WNV disease.⁶ Our case underscores the value of paired serum and CSF testing, since initial serology was negative but positive WNV IgM in CSF was diagnostic; whether this was due to an initial serum false-negative or delayed seroconversion is unclear. Serologic cross-reactivity occurs between WNV and other flaviviruses, so PRNT may be performed to confirm WNV infection.⁷ Intriguingly, our patient had an elevated CSF IgG index with positive oligoclonal bands, indicating intrathecal antibody synthesis and suggesting that immunologic mechanisms may have contributed to his disease extension beyond the anterior horn cell. Although no antiviral therapy or immunotherapy for WNV-associated acute flaccid paralysis has been

Figure 2 Gadolinium-enhanced MRI in West Nile virus-associated acute flaccid paralysis

Axial T2-weighted imaging shows T2 hyperintensity of the conus medullaris centrally (A, arrows), which is also seen on sagittal images (B, arrows). Axial T1-weighted imaging postgadolinium shows enhancement of the right L5 and S1 nerve roots (C, arrows). Coronal T2-weighted imaging shows T2 hyperintensity of the right lumbosacral plexus (D, oval).

proven effective,⁷ making the diagnosis is critical to avoid extraneous investigations and ensure accurate disease reporting.

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AUTHOR CONTRIBUTIONS

A. Budhram reviewed the imaging and drafted the manuscript. M. Sharma reviewed the imaging and edited the manuscript for intellectual content. B. Shettar edited the manuscript for intellectual content. S.M. Hosseini-Moghaddam edited the manuscript for intellectual content. A. Khaw reviewed the imaging and edited the manuscript for intellectual content.

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