**CLINICAL VIGNETTE**

# West Nile Virus Presenting with Rash and Hand Weakness

Michael Estes, MD and Peter Lefevre, MD

A 52-year-old male presented with a four-day history of myalgias and migratory arthralgias. The joints that were primarily involved included bilateral shoulders, ankles, wrists, and knees. He complained of pain both at rest and with motion. He noted no erythema or warmth over the joints. He also complained of myalgias involving the back, neck and lower extremities. He noted generalized malaise but denied fever and chills. There was no throat pain, cough, or changes to bowel movements. He denied any headache, nausea or vomiting.

His history was otherwise significant for a recent increase in activity due to lifting of moving boxes. Additionally, he had traveled to Hawaii three weeks prior to the onset of symptoms. He reported swimming in the ocean without any fresh water exposure. He remembered no insect bites. At initial presentation, his physical exam was notable for normal vital signs, no joint effusions, non-focal neurologic exam, and a fine reticular rash involving the palms and the plantar aspect of the feet. Additionally, he had macular lesions of the soft palate. A presumptive diagnosis of hand foot and mouth disease was made and supportive care was recommended.

Two days after presentation, the patient contacted the office for increased tremor and weakness in the left upper extremity. He also noted difficulty with ambulation due to vertigo. He had 4 episodes of emesis. His symptoms progressed to the point that he was unable to write with his left hand and was unable to ambulate without assistance. He was directed to the emergency department for further evaluation.

In the emergency department, his clinical presentation was as above, with the addition of intermittent diplopia as well as urinary retention requiring Foley catheterization. Evaluation included MRI of the brain and C-spine as well as lumbar puncture. Radiology revealed no abnormality and his CSF showed a meningitis with a lymphocyte dominant pleocytosis with a WBC of 97 with normal glucose and mildly elevated protein. Initial CSF serologies were negative, however, he tested positive on serology for West Nile virus IgG and IgM. Supportive care was undertaken.

Due to ongoing ataxia and urinary retention, the patient was discharged to a skilled nursing facility where his neurologic symptoms began to improve. He was discharged home after 2 weeks with discontinuation of the Foley catheter and improvement in his ambulation. Four weeks following discharge, the patient's hand weakness had improved to the point that he was able to write and he no longer required an assistive device for ambulation.

***Discussion***

West Nile virus (WNV) is an RNA virus from the *Flavivirus* genus. It generally cycles between birds and mosquitos but since the 1930s has been known to cause infections in humans. The first identified cases in the United States took place in New York City in 1999 when 62 patients presented with meningoencephalitis.[1] The most common presentation (~ 20%) is an acute illness with fever, nausea, rash and arthralgias lasting 3-10 days. The far less common neuroinvasive disease, which will be discussed in detail, is found in less 1% of infected individuals. According to recent CDC data, between 1999 and 2017 there were 48,183 cases of west nile virus disease and 22,999 cases of neuroinvasive disease in the United States.[2] The incidence of disease is highest during warm summer months when mosquito populations are greatest. Risk of infection goes up with age, immunosuppression, malignancy, renal disease and male gender.

The rarer presentation of West Nile virus infection is neuroinvasive disease. Fewer than 1/100 patients will manifest nervous system involvement. The three well-characterized presentations include meningitis, encephalitis and acute flaccid paralysis. Meningitis presents with fever, neck stiffness, headache and photophobia whereas encephalitis also includes the spectrum of altered mental status to coma. Acute flaccid paralysis is a polio-like syndrome where the virus infects the anterior horn neurons of spinal cord and causes limb weakness with EMG demonstrated denervation.[3] Other conditions that can similarly present include plexopathies, cranial nerve palsies, myoclonus, seizures, sensory loss, ataxia, diplopia, parkinsonism, paresis and plegia.

West Nile neuroinvasive disease is diagnosed with cerebrospinal fluid analysis (CSF). As with most viral illnesses, the CSF generally shows pleocytosis, normal glucose and elevated protein. The diagnosis is confirmed with elevated WNV IgM antibody in either serum or CSF.[4] Patients can also develop thrombocytopenia, hyponatremia, transaminitis, and creatinine kinase elevations. Imaging is typically not helpful as it is commonly normal. Differential diagnoses are secondary syphilis, Japanese encephalitis, bacterial or TB meningitis, HSV and Guillaine-Barre syndrome. To date, treatment remains supportive care. Several therapies have been studied but there is no

compelling statistical evidence to support their use. Most studies are small case series or clinical trials with few patients. Interferon, ribavirin, polyclonal IVIG, IVIG with high titers of WNV-specific antibodies (Omr-IgG-am), recombinant WNV monoclonal antibody (MGAWN1) and corticosteroids have all been tried with minimal success.[5,6] Most patients are cared for acutely in the hospital followed by a period of recovery in a rehabilitation facility.

While West Nile virus neuroinvasive disease represents an extremely small number of symptomatic infections. These can have lasting consequences. Recovery is variable and a small percentage of cases result in coma and death. Patients can report muscle weakness, sleep disturbances, mood changes, dizziness, pain and gait impairment one year post infection.[3] A small study conducted in southern California found that as many as 80% of patients had residual symptoms at follow up.[7] Risk factors for persistent sequela include advanced age, male gender, and severe initial presentation such as coma.[8]

Finally, prevention programs remain the cornerstone of the fight against West Nile Virus. Community wide pest control entails reduction in breeding sites, larvicide and insecticide use. Individuals can address areas of freestanding water on their properties, use insect repellants and specialized clothing. Despite past and present research, there is no approved WNV vaccine for human use.

**REFERENCES**


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