



BMH Med. J. 2026;13(1):21-24. **Case Report**

A lethal virus that led to a dead end

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Abstract

West Nile virus (WNV) is a neurotropic Flavivirus transmitted primarily through mosquito bites, with *Culex* species being the main vectors. While most WNV infections remain asymptomatic or present with mild flu-like symptoms, neuroinvasive disease occurs in approximately 1 in 150 cases, leading to meningitis, encephalitis, or acute flaccid paralysis.

Brainstem encephalitis is a rare but severe manifestation of WNV neuroinvasive disease, characterized by cranial nerve dysfunction, ophthalmoplegia, dysphagia, ataxia, and respiratory failure. Brainstem involvement can lead to life-threatening complications, necessitating early recognition and supportive management.

Here, we present a case of West Nile virus infection which had all the dreadful complications of this virus including multiple cranial nerve palsies, movement disorder and autonomic dysfunction that finally resulted in cardiac and respiratory failure. The relevance of this case is to highlight on its diagnostic challenges, and management, with a focus on its distinct neurological features.

Keywords: brainstem encephalitis, West Nile virus (WNV), Flavivirus

Introduction

West Nile virus (WNV) is a mosquito-borne flavivirus that has emerged as a significant cause of viral encephalitis worldwide. First identified in Uganda in 1937, WNV has since spread across multiple continents, leading to sporadic outbreaks in Africa, Europe, the Middle East, and the Americas. In the United States, the virus became a major public health concern following its introduction in 1999, causing thousands of cases of neuroinvasive disease [1]. WNV is primarily transmitted to humans through bites from infected *Culex* mosquitoes, with birds serving as the main reservoir hosts. While most infections remain asymptomatic or present with mild flu-like symptoms, a small proportion progress to severe neuroinvasive disease, including West Nile encephalitis (WNE), meningitis, and acute flaccid paralysis. Elderly individuals and immunocompromised patients are at higher risk of developing severe complications.

The pathogenesis of WNE involves viral entry into the central nervous system (CNS), where it triggers an inflammatory response leading to neuronal injury and dysfunction. Clinically, patients with WNE may present with fever, headache, altered mental status, seizures, and focal neurological

deficits. Diagnosis is primarily confirmed through detection of WNV-specific IgM antibodies in cerebrospinal fluid (CSF) via enzyme-linked immunosorbent assay (ELISA). Despite advances in supportive care, no specific antiviral treatment or licensed vaccine exists for WNV encephalitis, making prevention through vector control measures crucial.

Case report

Middle aged male with history of systemic hypertension presented with complaints of low grade fever, rhinitis, dry cough of 3 days, followed by high grade fever associated with severe dysphagia, dysarthria and tremors of both hands of 1 day duration. In the previous 2 weeks, he had history of recurrent travels to remote forest areas. There was no history of similar illness in the family.

On arrival to emergency department of our hospital, he was conscious, oriented with a GCS of E4V5M6, tachypneic and normotensive. Neurological examination revealed resting tremors in both hands, facial deviation to the right, pooling of saliva in the floor of the mouth and bilaterally absent palatal movements. He had no limb weakness and all reflexes were preserved. Other system examinations were normal. Owing to his neurological findings, a possibility of brainstem involvement was considered and he was shifted to Neurology ICU. Within a few hours he developed hypoxia with periodic respiration and immediately went into respiratory arrest. He was intubated and mechanically ventilated. His POCUS showed global LV hypokinesia and severe LV dysfunction. Trop I and NTproBNP were elevated, 4297ng/ml and 25,000 pg/ml respectively.

In view of recent history of travel to remote forest areas, possibility of tropical fever was considered. Tropical fever panel ruled out dengue, scrub typhus and leptospirosis. Peripheral smear for malarial parasite was negative. HIV, HbsAg, AntiHCV were negative.

On day 2 of hospital stay, he developed multiple focal seizures that were controlled with IV antiepileptics. MRI brain with contrast performed on the day of admission showed subtle increase in leptomeningeal enhancement involving bilateral frontoparietal regions with no evidence of exudates. MR Angiogram and MR Venogram were normal. EEG showed evidence of diffuse cerebral dysfunction. CSF done on day 1 of admission showed high protein (94.1mg/dl), Sugar-149mg/dl (corresponding blood sugar 255mg/dl), Total WBC-200 (Neutrophil 80% Lymphocytes 20%). He was started on Intravenous antibiotics (Inj.Ceftriaxone), Inj Acyclovir, Inj Methylprednisolone and other supportive measures.

CSF meningoencephalitis panel which included testing for Ecoli, H Influenza, Listeria, Neisseria, streptococcus, CMV, Enterovirus, HSV 1 and 2, HHV 6, Varicella zoster virus were negative. CSF autoimmune encephalitis panel also tested negative. CSF, blood and urine culture remained sterile.

His general condition deteriorated and GCS dropped to E1VTM1 by day 3 of admission. A provisional diagnosis of seronegative autoimmune encephalitis was made and he was also started on IVIG. His repeat CSF study showed high protein (48.4mg/dl), sugar (139 mg/dl), T.WBC-100, Neutrophil-100 %.

In view of history of travel and rapid deterioration of symptoms, rare viral infections causing Brainstem encephalitis caused by Nipah, Rabies, Japanese B encephalitis and West Nile virus were considered. Of these IgM antibody to West Nile virus turned out to be positive in the CSF sample. So a final diagnosis of Acute brainstem encephalitis with severe cardiac and respiratory failure due to West Nile virus infection was made. He was hence started on Oral Ribavirin and was continued on IV Acyclovir, IV steroids (Inj Methylprednisolone) and other supportive measures. Repeat MRI showed same findings along with subtle hyperintensities in bilateral basal ganglia and thalamus-suggestive of worsening picture.

Despite initiation of all possible means of care, he developed multiple complications in the form of

ventilator associated pneumonia, acute kidney injury requiring dialysis and finally succumbed to this illness within 2 months.

Discussion

West Nile virus encephalitis remains a significant public health challenge due to its unpredictable outbreaks, high morbidity in neuroinvasive cases, and lack of targeted treatment. The clinical spectrum of WNV neuroinvasive disease varies widely, ranging from mild confusion to profound encephalopathy, coma, and death. Several studies have highlighted that advancing age, diabetes mellitus, hypertension, and immunosuppression are key risk factors for severe disease progression [1].

Pathogenesis and Immune Response

The ability of WNV to cross the blood-brain barrier is the most crucial factor in neuroinvasive disease. Viral neuroinvasion is thought to occur through direct infection of endothelial cells, transport within immune cells (monocytes), or via increased BBB permeability due to systemic inflammation [2]. Within the CNS, WNV primarily affects neurons in the cortex, brainstem, and spinal cord, leading to neuroinflammation and excitotoxicity. Elevated levels of inflammatory cytokines, including tumor necrosis factor-alpha (TNF- α) and interferon-gamma (IFN- γ), are associated with neuronal injury and disease severity [2].

Clinical and Diagnostic Considerations

The diagnosis of WNE relies on a combination of exposure history, unique clinical features, and CSF findings. The presence of WNV-specific IgM in CSF is the most reliable diagnostic marker, though it may persist for few months after acute infection [3]. In challenging cases, polymerase chain reaction (PCR) or plaque reduction neutralization tests (PRNT) may provide confirmatory evidence, particularly in immunocompromised individuals where antibody responses may be delayed or absent. Neuroimaging findings, though often nonspecific, may reveal leptomeningeal enhancement or basal ganglia involvement.

Treatment and Prognosis

So far no antiviral therapy is proven to be effective against WNV encephalitis. Management remains supportive, including airway protection, seizure control, and management of cerebral edema. Experimental treatments such as interferon therapy, monoclonal antibodies, and ribavirin have been explored, but none have demonstrated definitive clinical benefit [2,5]. Mortality rates range from 10% to 20% in severe cases, with long-term neurological sequelae such as cognitive impairment, muscle weakness, and movement disorders persisting in survivors [6].

Prevention and Future Directions

No vaccines have been approved so far, hence WNV prevention strategies focus on mosquito control and public health interventions. Strategies adopted include reducing stagnant water sources, using insect repellents, and implementing targeted vector surveillance programs. Several vaccine trials including DNA-based and live-attenuated vaccines, are under investigation and hold promise for the future [7].

In conclusion, West Nile virus encephalitis remains a serious neurotropic infection with significant morbidity and mortality. While supportive care is the mainstay of treatment, further research into antiviral therapies and vaccine development is essential to mitigate the impact of this emerging

infectious disease.

Conclusion

In cases of rapidly progressive brainstem encephalitis, less common viral infections like West Nile, Japanese encephalitis and Nipah should be suspected. Although treatment options are limited, it is worth while to detect these etiological agents at the earliest, prognosticate cases and initiate best treatment options available as well as to report/notify such cases and make necessary precautions to prevent an epidemic.

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