Psychosis and Catatonia Associated With West Nile Virus

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West Nile virus (WNV) is a single-stranded RNA flavivirus that is commonly transmitted by mosquitoes. While psychosis has been linked to other flavivirus infections, WNV-associated psychosis is rare and has only been reported a few times in the literature. We present a unique case of WNV-associated psychosis complicated by catatonia.

Case Report

A 60-year-old man presented to the emergency department after a 4-day period of altered mental status, fever with chills, and headache. His white blood cell (WBC) count was 12.7×10^9/L, and other laboratory results were unremarkable. Empiric antibiotics were started for suspected meningitis. After 2 days, he was afebrile with a WBC count within normal limits, and antibiotics were discontinued. Head magnetic resonance imaging was nonspecific. The neurology department performed a lumbar puncture, with a WBC count of 74×10^9/L and polymorphonuclear leukocytes of 14%, lymphocytes of 56%, monocytes of 30%, protein of 47 g/dL, and glucose of 65 mg/dL, which was consistent with viral meningitis. A meningitis panel was negative for cryptococcal antigen, HIV, and Lyme disease, but the cerebrospinal fluid serology was positive for WNV.

Months later, he presented with paranoia and persecutory delusions of police pursuing him and cars spying on his house. He had no prior psychiatric history. He was resistant to redirection, stared out the window, abstained from activities, and became nonverbal. He had no exposure to toxins, substances, or other environmental exposure factors. He believed there was flesh, blood, and babies in his food. He was admitted to the inpatient psychiatry floor and treated with olanzapine 10 mg. His symptoms started to improve. However, he later developed catatonic symptoms including mutism, echolalia, and withdrawal.

He was transferred to the medical floor. His Bush-Francis Catatonia Rating Scale[1] score was 11. After starting lorazepam 2 mg intravenous 3 times/d, the score decreased to 1. Olanzapine was discontinued to avoid worsening of catatonia. After 2 days of lorazepam, he improved and could engage in conversation. Lorazepam was tapered over 3 weeks. His catatonia resolved, and he was restarted on olanzapine and discharged home in stable condition.

Discussion

WNV is the most common mosquito-borne illness in the continental US.[2] Most WNV infections are asymptomatic. Symptomatic patients typically present with self-limiting fever, myalgias, and malaise. Approximately 1 in 150 people develop neuroinvasive disease, such as meningitis, encephalitis, or flaccid paralysis.[3,4]

Along with WNV, the Flaviviridae family also includes Zika, Powassan, dengue, and yellow fever viruses. Zika,[5] Powassan,[6] and dengue[7] viruses have been associated with psychosis. There are 2 reported cases, to our knowledge, of WNV-associated psychosis involving (1) a renal transplant patient with meningitis and psychosis[8] and (2) a patient with psychosis, ataxia, and facial dyskinesia.[9] Neither involved catatonia.

There are links between flaviviral pathophysiology, immunology, and psychosis. Schizophrenia geographic distribution has been linked to tick-born encephalitis, including tick-associated flaviviruses.[10] A murine study[11] found that genome components of flaviviruses, including the Kunjin WNV subtype,[12] had similarities to abnormal protein-encoding sequences in schizophrenia. RNA virus encephalitis was also linked to cerebellar neuroinflammation,[13] which is associated with psychosis in clinical and laboratory research.[14]

Akinetic catatonia, as seen in our patient, has complications including malnutrition, dehydration, pressure ulcers, and venous thromboembolism.[15,16] Catatonia is primarily seen in psychiatric conditions, but it is also seen in a variety of medical conditions such as infections.[17] A systematic review found that 20% of catatonia has a medical cause, and central nervous system inflammation represents 29% of these cases.[18] Catatonia is not yet reported with WNV, to our knowledge, but a report exists of catatonia associated with dengue fever.[19] Catatonia has been associated with autoimmune conditions, such as *N*-methyl-D-aspartate receptor encephalitis,[20] and elevated acute phase reactants, such as C-reactive protein.[21] Regardless of cause, response rates to treatment with benzodiazepines and electroconvulsive therapy are high.[22,23] This suggests

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a common pathophysiology behind catatonia, such as immune system modulation.

In conclusion, we present a patient with no primary psychiatric diagnosis who developed psychosis and catatonia months following WNV encephalitis. This case, along with other similar flaviviral reports, builds the connection between neuropsychiatric symptoms and the immunologic basis of infectious diseases. Further research into the cognitive and functional outcomes of patients recovering from WNV illness will be important in understanding the overall illness burden and long-term psychiatric prognosis.

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