West Nile Encephalitis in the Emergency Department: Prevalence and Recognition

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Contribution to Emergency Nursing Practice

- This article explores the prevalence and significance of West Nile virus encephalitis across the United States.
- The signs and symptoms of West Nile virus neuroinvasive infection in the emergency department are described.
- The primary prevention of West Nile Virus infections among patients seeking care in the emergency department is detailed.

Case Study

A 40-year-old female nurse, in Orange County, California, was brought to the emergency department by her father after she collapsed at home and found herself weak and unable to walk.

The patient reported that her symptoms began approximately 10 days before this episode and included weakness with intermittent periods of blurry vision, headaches, and vertigo-like dizziness. Two days before she collapsed, she awoke with a fever of 104.0° F, extreme fatigue and weakness, repeated vomiting, severe headache, persistent dizziness, and confusion. The fever was not responsive to acetaminophen or ibuprofen.

In the emergency department, the doctor noted that the patient had an altered level of consciousness and was very confused. He suspected West Nile virus (WNV) infection after discovering a large mosquito bite on the back of her calf. Because there is no specific treatment for WNV, the patient was discharged with supportive care instructions to rest, hydrate, and manage her fever.

Two days later, the patient was brought back to the emergency department by her husband because of increased confusion, refractory fever, weakness, and decreased urine output. At this time, she was admitted to the hospital for dehydration and electrolyte imbalance and treated with IV fluids, antipyretics, and antiemetics. Shortly after admission, the diagnosis of WNV encephalitis was definitively confirmed.

The patient stated that she suffered from residual weakness, fatigue, and dizziness for almost 8 months after the acute phase of this infection. In addition, the patient reported having periods of amnesia; for example, she admitted to having no memory of her initial emergency department visit. She stated that she could not have maintained her family and professional life during this period without the extensive assistance of her husband and parents. Although she could not return to work for several months after her hospitalization, she did eventually fully recover and resume her career as a nurse.

Epidemiology and Etiology

West Nile virus is a member of the genus Flavavirus and was first isolated in a patient from the West Nile province of Uganda in 1937.[1] The virus became relevant in the United States in 1999, when it caused 62 cases of encephalitis and 7 deaths in New York City.[2] Since this initial outbreak in 1999, 2 nationwide epidemics have occurred in the United States: in 2003 and 2012.[3] West Nile virus is now endemic in North America[4] and has spread throughout the entire continental United States,[5] in addition to international distribution throughout Africa, the Middle East, parts of Europe, South Asia, and Australia.[6] West Nile virus is now recognized as the most widely distributed arbovirus in the world.[3] Outbreaks of WNV disease since the 1990s have resulted in mortality rates near 10% among patients with encephalitis.[5] Serologic surveys have indicated that approximately 1 in 250 WNV infections results in neuroinvasive disease (meningitis or encephalitis).[7] From 1999 to 2014, the Centers for Disease Control and Prevention (CDC) reported 41,762 cases of WNV disease in the United States, including 18,810 cases of neuroinvasive disease.[5] West Nile virus encephalitis is now the most commonly diagnosed arboviral infection of the CNS[8] and the most common cause of viral encephalitis in the United States.[9]

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Human cases prevail in late summer and early fall,[5] a result of mosquito emergence patterns related to climate and viral amplification in the bird–mosquito–bird cycle. Birds are the amplifying hosts of the WNV, and human infections derive from mosquito vectors.[10] Mosquitos that transmit WNV are most often of the Culex species and vary with geography. Sixty-five mosquito species infected with WNV have been discovered in North America.[3] American crows and other corvids, such as ravens and jays, are common avian hosts,[11] and higher incidence of human West Nile infection has been observed in high crow-mortality areas.[12] In addition to mosquito bites, transmission can occur from mother to child in utero[13] or through breast milk,[14] or from an infected donor to a blood or organ transplant recipient.[15] Since 2003, the United States has screened all blood supplies for WNV by nucleic acid testing (NAT), which has essentially eliminated the risk of transfusion-related WNV infection.[16] However, transmission through donated organs is more difficult to detect, as several case studies have shown that donors who tested negative for WNV RNA resulted in West Nile-infected recipients, suggesting that the virus can sequester in organs without the presence of detectable viremia in the serum.[17] In the United States, organ and tissue donors are not routinely screened for WNV infection.[17]

Close to Home

The CDC estimates that 70% to 80% of human WNV infections are subclinical and therefore not reported.[18] Preliminary data available from the CDC for 2016 reveal 1,002 cases of neuroinvasive WNV, 54% of all reported WNV cases for the year. California had 202 cases of reported WNV neuroinvasive disease.[19] The only state with a larger reported number of neuroinvasive WNV cases is Texas, with 225 cases.[19] California had the largest known number of viremic blood donors in the country and the largest number of WNV-related deaths: 18 reported in 2016.[19] Los Angeles and Orange counties have two of the highest rates of reported human WNV cases in California and, moreover, are among the counties with the highest incident rates across the country.[20]

Pathophysiology

The pathogenesis of severe WNV infection is not completely understood. What is known is that when a human is bitten by a WNV-infected mosquito, the mosquito injects virus-containing saliva into the human host. The incubation period normally ranges from 2 to 6 days, with longer incubation periods up to 14 days seen in immunocompromised hosts.[21] The virus disseminates in 3 phases: first by replicating in the keratinocytes and dendritic cells of the skin, next by replicating in the lymph nodes and spreading to visceral organs, and finally by invading the central nervous system (CNS).[22] The method by which viral invasion of the CNS in achieved is not clearly known, but possibilities include direct viral crossing of the blood–brain barrier (BBB) via cytokine-mediated increased vascular permeability,[23] passive transport through endothelium or choroid plexus epithelial cells,[23] movement of infected macrophages across the BBB,[23] or direct axonal retrograde transport from infected olfactory or peripheral neurons.[24] Neurologic manifestations can occur approximately 1 week after infection.[25] Patients who died from WNV neuroinvasive disease have been found to have inflammation of the spinal cord along with glial nodules, loss of neurons, and perivascular cuffing by mononuclear cells in the gray matter.[26] After patients recover from WNV infection, they are thought to obtain life-long immunity, as reinfection rates are extremely low.[21]

Signs and Symptoms

West Nile virus can cause a range of symptoms in humans, from asymptomatic disease to severe neuroinvasive disease. Only 20% to 40% of patients with WNV are symptomatic.[21] Cases of WNV are considerably underreported, as most infected persons are asymptomatic, do not seek medical care for mild symptoms, or are not tested for WNV.[27] Because WNV is endemic in North America, emergency nurses, in particular, should be familiar with the clinical manifestations, diagnostics, and treatment modalities available for the infection. It is important to note that the clinical signs and symptoms of the WNV infection are generally nonspecific and similar to several other disease processes and are therefore easily missed or misdiagnosed. Some of the most familiar infections that present comparably to neuroinvasive WNV infection are Dengue fever, herpes simplex encephalitis, St. Louis encephalitis, and Japanese encephalitis, as well as other spirochete and tick-borne illnesses. Risk factors for WNV infection include female gender, advanced age, immunocompromised state, and prolonged outdoor exposure.[21]

Initial WNV infection is termed West Nile fever (WNF), which presents suddenly with general signs and symptoms of meningeal irritation such as nonspecific fever, headache, nausea and vomiting, malaise, back pain, anorexia, and—more specifically—include photophobia and nuchal rigidity.[28] A distinguishing maculopapular rash over the chest, back, and arms can be found in 25% to 50% of patients with WNF.[28] The rash may or may not be pruritic, does not