Rare ocular manifestation in a case of West Nile virus meningoencephalitis

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Abstract

West Nile Virus (WNV) is an arthropod-borne flavivirus, which causes flu-like illness and is sporadically associated with encephalitis. Transmission to humans occurs following a bite from an infected mosquito, which acquires the virus after feeding on dead birds. WNV meningoencephalitis is a rare infection with a neuroinvasive disease occurring in less than 1% of the cases, with varied presentations including aseptic meningitis, meningoencephalitis, and acute flaccid paralysis. Chorioretinitis is the most common eye finding in this infection, while other ocular manifestations have been rarely reported in the literature. We present the first case report of WNV meningoencephalitis, with rare ocular manifestations of acute hemorrhagic conjunctivitis, bilateral subconjunctival hemorrhages, and nystagmus. The rare ocular findings of acute hemorrhagic conjunctivitis, bilateral subconjunctival hemorrhages, and nystagmus diagnosed in our case can guide clinicians toward early diagnosis of WNV meningoencephalitis, while serologic testing is still pending.

Key Words

Acute hemorrhagic conjunctivitis, meningoencephalitis, subconjunctival hemorrhages, West Nile Virus

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Case Report

A 40-year-old male presented with confusion, short-term memory loss, and unsteady gait for one month. He also complained of intermittent right facial numbness, diplopia, bilateral lower extremity numbness and weakness, and severe choking spells while coughing. The patient mentioned that when he had visited Ohio four weeks ago, a dead bird was found with West Nile virus infection. On examination, he was drowsy, but oriented to time, place, and person with mild maculopapular rash on his upper back, chest, and face. Cranial nerve examination revealed a subtle downbeating nystagmus. His motor strength, sensory exam, and deep tendon reflexes were normal. The cerebrospinal fluid (CSF) showed lymphocytic pleocytosis, while the remaining results were normal. The patient was started on empirical antibiotics, while the culture results were awaited. Magnetic resonance imaging (MRI) of the brain and complete spine were unremarkable. The patient had pronounced bilateral subconjunctival hemorrhage with progressive drowsiness the next day. West Nile IgM in the CSF was noted to be positive, while his mental status deteriorated and he required intubation within the next 24 hours. Electroencephalography (EEG) showed mild bicentral slowing, with no evidence of epileptiform discharges. An ophthalmology examination showed acute hemorrhagic conjunctivitis, conjunctival chemosis, and periorbital edema, with no chorioretinitis on fundoscopy (Figure 1). Supportive management was continued with gradual improvement of his mental status.

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Discussion

West Nile Virus infection is an arthropod-borne disease, with the highest incidence during the summer season.[1] It is one of the most common causes for epidemic meningoencephalitis in northern parts of America.[2] Transmission to humans occurs following a bite from an infected mosquito, which acquires the virus after feeding on dead birds.[3] Approximately 80% of WNV infections are asymptomatic, while the remaining 20% develop a nonspecific viral syndrome.[4] Diabetics and age > 50 years have been seen to have increased association, with serious systemic and ocular manifestations.[5]

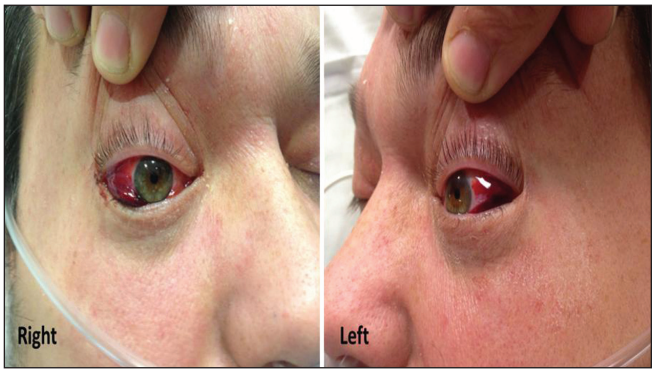

Figure 1: Acute hemorrhagic conjunctivitis with bilateral subconjunctival hemorrhages

Multifocal chorioretinitis involving both eyes, with a self-limited clinical course is the most common ocular manifestation. Rare ocular manifestations of WNV infection can include anterior uveitis, retinal vasculitis, optic neuritis, subconjunctival hemorrhage, sixth nerve palsy, nystagmus, and chorioretinal scarring.[6,7] Detailed ocular examination should be included as a part of the routine evaluation of patients with clinically suspected WNV infection. Central nervous system (CNS) involvement in WNV infection is uncommon and can be categorized into three broad clinical syndromes: Encephalitis, meningitis, and acute flaccid paralysis.[8] Males over 50 years of age have been seen to have increased risk of developing meningoencephalitis.[9] Neuroinvasive disease occurs following penetration of WNV into the blood–brain barrier, leading to direct invasion of neurons in the brainstem, deep nuclei, and anterior horn of the spinal cord. Acute flaccid paralysis or weakness is usually an isolated entity, which has been postulated to be due to an immunological reaction triggered by WNV or through direct invasion of the anterior horn cells of the spinal cord.[10] As seen in poliomyelitis, this denervation causes loss of motor function at that level, with minimal or no sensory involvement. Autonomic involvement manifested as bradycardia, blood pressure changes or bowel and bladder dysfunction may also be present. Prominent bulbar signs, especially dysphagia, can gradually progress and may lead to respiratory failure.

Cerebrospinal fluid studies in neuroinvasive WNV infection present with pleocytosis and elevated protein levels, although the CSF glucose levels are usually normal. Brain MRI findings may be entirely normal or may show hyperintense T2 and fluid-attenuated inversion recovery (FLAIR) signals, with accompanying diffusion abnormalities or gadolinium enhancement. Serum or CSF collected after the onset of illness is diagnostic of WNV infection. With large size of the IgM antibodies, making them unable to cross the blood–brain barrier, detection of IgM antibodies to WNV in the CSF strongly suggests acute WNV CNS infection. Extensive serological cross-reactivity exists among various members of the Flaviviridae family, including, WNV, St. Louis encephalitis virus, dengue virus, tick-borne encephalitis virus, yellow fever virus, hepatitis C virus, and Japanese encephalitis virus, which can give false-positive results. At present, there is no specific therapy with proven efficacy for the treatment of WNV infection. Current treatment of WNV disease is largely supportive, including pain control for headaches, anti-emetics, rehydration for associated nausea and vomiting, clinical monitoring for development of elevated intracranial pressure or autonomic dysfunction, and seizure control if needed.

Our case was unique with a rare clinical presentation of WNV meningoencephalitis, with acute hemorrhagic conjunctivitis and bilateral subconjunctival hemorrhages. Asymptomatic and self-limited chorioretinal involvement is the most common ocular manifestation in patients with WNV infection associated with neurological disease. We present a case of serologically proven, rapidly progressing WNV encephalitis, associated with interesting ocular manifestations of nystagmus and bilateral subconjunctival hemorrhages. In our case, the eye findings preceded the development of severe autonomic instability. The unique ocular findings of acute hemorrhagic conjunctivitis with bilateral subconjunctival hemorrhages and nystagmus, in a patient presenting with meningoencephalitis, may guide clinicians toward early diagnosis even when the serologic tests are pending.

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