West Nile virus (WNV) is an arthropod-borne virus transmitted to humans by infected mosquitoes. Birds are thought to be the natural reservoir (amplifying host) of this virus with humans merely being incidental hosts. It usually presents with no symptoms but may manifest as a febrile illness or involvement of the brain including encephalitis, and meningitis or polyradiculitis. West Nile virus is known to cause severe illness in people with advanced age, underlying malignancy or who have undergone solid organ transplantation.

Case report

A 40-yr-old woman with poorly controlled asthma, hepatitis C virus and polysubstance abuse was brought in from a drug detox centre to the emergency room of a tertiary care centre of St. Luke’s Roosevelt, Hospital, New York, U.S.A. with complaints of high-grade fever, vomiting and poor oral intake for one day. On presentation, she was febrile to 102°C. Physical examination result was grossly benign. The laboratory investigation results were significant for WBC (6.9/μl), lactate (2.5 mmol/l), lipase (571 U/L) and elevated transaminases. Urinalysis showed marked pyuria, positive leukocyte esterase and many bacteria. Empiric ceftriaxone and aggressive IV hydration was initiated for suspected pyelonephritis and she was admitted to the general medicine service. After 12 h into the hospital stay, the patient developed noticeable disorientation with unintelligible speech but an otherwise non-focal neurological examination. Head CT was significant for enlarged ventricular and sulcal spaces, consistent with cerebral edema. A spot EEG was suggestive of a left frontal epileptic focus. CSF analysis revealed WBC (450/μl) and protein (403 mg/dl), prompting empiric vancomycin, ampicillin, ceftriaxone and dexamethasone for suspected bacterial meningitis. After an overnight transfer to the medical ICU, the patient developed respiratory distress requiring mechanical ventilation. Over the next 48 h, the patient experienced elevated but variable temperature and blood pressure readings, associated with waxing and waning mental status. MRI showed abnormal signal enhancement in the basal ganglia and thalamus bilaterally, compatible with acute encephalitis. A repeat lumbar puncture demonstrated WBC (7), and Protein (168), with lymphocytic predominance. The patient gradually improved, ultimately leading to extubation and discharge from ICU to general medicine on Day 6 of admission. About 72 h later, the patient again developed respiratory distress and was re-intubated. At this time, pending CSF serologies returned positive for West Nile IgM. The kit used to detect WNV was an IgM capture ELISA that utilizes recombinant WNV antigens to detect WNV IgM. On Day 11, she was extubated and maintained on the medicine service until her discharge to a subacute rehabilitation facility. Before discharge a repeat MRI was done. The patient

Fig. 1: MRI of brain showing abnormal signal enhancement, affecting the basal ganglia and thalamus compatible with a toxic/metabolic process, accounts for the patient’s clinical diagnosis of encephalitis.

Fig. 2: Repeat MRI of brain showing interval decrease in abnormal signal affecting the basal ganglia and thalamus (in comparison to previous MRI). Findings are consistent with interval improvement of patient’s toxic/metabolic process.
lives in Jersey City, U.S.A. and has no history of recent travel or visit to endemic areas and was up to date on immunizations.

DISCUSSION

We report a unique case of WNV disease with cerebral involvement and neurological manifestations. The recommended modality for diagnosing WNV meningitis and encephalitis is the detection of IgM antibodies to WNV in the serum and CSF. Most people infected with WNV are asymptomatic, while > 20% develop a heterogeneous febrile syndrome. Potentially catastrophic and sometimes fatal cerebral involvement is demonstrated in < 1% of infected patients. It is thought that the severity of neuroinvasion and rate of death due to this increases with age. Those with encephalitis often demonstrate residual neurological deficits and a markedly increased risk of morbidity and mortality. Treatment is thought to be mainly supportive but some advocate early administration of IFN-α/β, which has been reported to protect against long-term neurological sequelae, delay neuronal death and promote survival. The most recent epidemic in the United States was reported in 2012 after the epidemic of 2003, with higher evidence of neuroinvasive disease. Since, there is no vaccine available for this virus and its sequelae can impact quality of life, its early diagnosis and prevention are important. Prevention depends on household and individual efforts to decrease exposure to mosquitoes and screening of blood donations.

REFERENCES