**INTRODUCTION**

WN Neuroinvasive diseases include encephalitis, meningitis, acute flaccid paralysis, among others. However, CSF findings are usually described as an initial polymorphonuclear pleocytosis followed by lymphocytic pleocytosis. We report a case of a 68-year-old female with encephalitis and flaccid quadriparesis with a monocytic pleocytosis.

**Case presentation**

A 68-year-old female patient with a past medical history of hypothyroidism presented to the ER for 2 days of fever and confusion. Glasgow coma eye subscore 3, verbal subscore 2 and motor subscore 4. Neurological exam was remarkable for lethargy, flaccid quadriparesis and was intubated for management of airway and respiratory failure (hypercapnic). Laboratory studies revealed WBC 6.9 × 10^3/uL (85% of neutrophils, 11% of lymphocytes, 3% monocytes, no eosinophils, and 1% immature neutrophils), hemoglobin 14.4 g/dL and platelets 133 × 10^3/uL.

Magnetic Resonance Imaging (MRI) of the brain showed a small focus of right frontal T2 signal hyperintensity with T2 shine through and no basal ganglia involvement. MRI cervical, thoracic and lumbar showed contrast enhancement of the nerve roots, particularly in the ventral root of the lower thoracic and lumbar levels. Nerve conduction studies showed an active motor neuronopathy. The Electroencephalogram (EEG) showed non-specific generalized slowing. At the same time, the Cerebrospinal fluid (CSF) showed a clear colorless fluid with glucose 55 mg/dl and protein 128 mg/dL, monocytic pleocytosis (98 WBC with 62% monocytes, 25% lymphocytes and 13% polymorphonuclear). Pathology review of CSF showed monocytes, small lymphocytes, and occasional neutrophils. Infectious etiologies (Gram stain, aerobic and fungal cultures, cryptococcal antigen, herpes simplex virus, varicella-zoster virus, cytomegalovirus, enterovirus, syphilis, HIV, and tuberculosis) returned negative. She was started on plasma exchange with the suspicion of a variant of Guillain-Barré syndrome. However, after 2 days, the WNV studies in CSF resulted positive for virus-specific IgM and negative for IgG. Thus, plasma exchange was stopped. With only supportive treatment, she regained consciousness and her strength mildly improved, but a tracheostomy and a PEG were warranted. At discharge, she was fully dependent on ventilator by tracheostomy and fully fed by PEG, awake, alert and followed simple commands and with quadriparesis.

**DISCUSSION**

Even though WNV encephalitis and acute flaccid paralysis were initially suspected in this case, CSF findings were atypical from previous descriptions. The most commonly reported features of CSF in patients with WNV encephalitis is early neutrophilic pleocytosis; that is followed by lymphocytic pleocytosis.[1,2] Other CSF findings include elevated protein concentration.[2] Reported findings also included the presence of atypical lymphocytes and Mollaret’s cells in the CSF of patients with WNV meningitis or meningoencephalitis.[1,2] Nonetheless, monocytic pleocytosis without Mollaret’s cells is scarcely described in these patients. Upon starting plasma exchange with the suspicion of a variant of Guillain-Barré syndrome. However, after 2 days, the WNV studies in CSF resulted positive for virus-specific IgM and negative for IgG. Thus, plasma exchange was stopped. With only supportive treatment, she regained consciousness and her strength mildly improved, but a tracheostomy and a PEG were warranted. At discharge, she was fully dependent on ventilator by tracheostomy and fully fed by PEG, awake, alert and followed simple commands and with quadriparesis.
completion of a comprehensive literature review, we could not find any article that addressed monocytic pleocytosis. However, we found 2 references that mentioned this feature in the CSF. One was part of a case series of 7 in 2004 where the patient had monocytic pleocytosis (51%) and no further descriptions of the case. The second one was a case of a Brazilian ranch worker with acute encephalitis and flaccid paralysis, showing 14 leukocytes/mm^3 with 85% monocytic predominance, 274 mg/dL proteinorrachia and a glucose of 59 mg/dL. The clinical presentation of the second case is similar to our case description (encephalitis and acute flaccid paralysis). We could hypothesize that monocytic pleocytosis is underreported and WNV with monocytic pleocytosis is under-recognized.

There is a relationship between WNV and monocytes that relates to the involvement of the central nervous system (CNS). Initially, WNV infects keratinocytes and Langerhans cells, before migrating towards the lymph nodes and beginning its initial replication phase. After spreading to other organs, a second replication occurs in the epithelial cells and macrophages. The affected macrophages cross the blood brain barrier into the CNS. Monocytes play a crucial role in this last step by recognizing WNV by toll-like receptor 3, leading to the production of metalloproteinase MMP-9 and TNF alpha. This in turn leads to a loss of tight junctions and passage of immune cells and WNV to the brain. Other studies have shown that the absence of monocytes resulted in the death of experimental mice that were infected with neuroinvasive WNV.

In summary, we suggest consideration of neuroinvasive WNV despite a monocytic pleocytosis in the setting of encephalitis with flaccid paralysis. Recognition of this atypical finding may lead to an earlier diagnosis of neuroinvasive West Nile Virus and prevent unnecessary additional testing.

**Declaration of patient consent**
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**
There are no conflicts of interest.

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