

Letter to the Editor

West Nile encephalitis presenting with severe persistent chills mimicking malaria

Dear Editor,

The neurological spectrum of West Nile virus (WNV) varies from aseptic meningitis to encephalitis ± flaccid paralysis. In endemic areas in summer, WNE should be suspected in patients presenting with mental confusion ± flaccid paralysis. WNV may also present with rash and less commonly, diarrhea. Like other viral illnesses, WNV may be accompanied by fever, chills, headache, and myalgias. Except for influenza, severe chills are not usually the dominant component of the clinical presentation with systemic viral infections^{1–4}.

We present a case of a male recently returned from a malarious area with continuous high fevers and persistent shaking chills. He did not take malaria prophylaxis but the diagnosis of malaria was suggested by low/normal WBC count, thrombocytopenia, elevated serum transaminases, and an elevated LDH. Being from Long Island, the differential diagnosis included babesiosis and ehrlichiosis as well as bacterial sepsis. During his hospital stay, his violent shaking chills continued for over a week.

A 76-yr old male presented with fever/chills for 5 days. He recently returned from a cruise to Belize/Honduras. Two weeks after he returned, he began experiencing chills, fever, and was confused. The most prominent aspect of his presentation was uncontrollable continuous chills. He developed respiratory distress and was transferred to the intensive care unit (ICU) and was intubated.

Physical examination included a temperature of 103.5°F and pulse of 92/min. He continues to have persistent severe chills, but otherwise his physical examination was unremarkable.

Pertinent laboratory data included a white blood cell (WBC) count of 4.6 K/mm³ (neutrophils= 54%, lymphocytes = 35%, monocytes= 10%), hemoglobin of 14.3 g/dl and a platelet count of 92 K/mm³. The ESR was 31mm/h, and CRP was 1.06 mg/l. His ALT was 39 IU/l (n = 4–36 IU/l), AST was 43 IU/l (n = 13–39 IU/l), and alkaline phosphatase was 67 IU/l (n = 25–100 IU/l). Serum LDH was 315 IU/l (n = 100–250 IU/l), and ferritin was 580 ng/ml (n = 14–235 ng/ml). He was empirically treated with meropenem for possible sepsis, and doxycycline for the empiric treatment of ehrlichiosis.

Because of mental confusion and progressive unre-

sponsiveness, a lumbar puncture was performed. CSF analysis showed 52 WBC/hpf (PMNs = 20%, lymphocytes = 59%, monocytes = 21%), and 25 RBCs/hpf. CSF protein was 136 mg/dl (n = 15–40 mg/dl), and CSF lactic acid level was 3.5 mmol (n < 2.2 mmol). CSF gram stain/culture were negative. EKG and CXR were unremarkable. EEG showed generalized background slowing. Blood and urine cultures were negative. Multiple malaria/babesia smears were negative. Titers (IgM/IgG) for RMSF, parvovirus B₁₉ and *Ehrlichia chaffeensis* were negative. CSF-PCR for enteroviruses, HSV, VZV, and HHV-6 were negative. CSF-PCR and serum IgM were also later reported positive for WNV. Empiric antimicrobial therapy was discontinued.

With systemic viral infections, chills often accompany fever, headache, arthralgia/myalgia, but few systemic viral infectious diseases have chills as a prominent clinical manifestation, e.g. influenza. In this case, the differential diagnosis included malaria, babesiosis, ehrlichiosis, and sepsis of unknown source. Because WNV is endemic on Long Island, diagnostic tests for WNV were sent after other tests were reported negative. His mental status continued to deteriorate and he remained unresponsive. WNE may be mimicking by many infectious and non-infectious disorders, but to the best of our knowledge, this is the first case of WNE mimicking malaria^{1,5}. Against the diagnosis of malaria was the absence of headache and a normal hemoglobin and the absence of atypical lymphocytes^{6–8}. Findings suggesting malaria included WBC count, thrombocytopenia, increased LDH, and highly elevated serum ferritin level⁹. In retrospect, a clue to the diagnosis was an elevated serum ferritin which has diagnostic and prognostic significance in WNE¹⁰. In WNV endemic areas, clinicians should include WNE in the differential diagnosis of severe prolonged chills.

REFERENCES

1. Cunha BA. Differential diagnosis of West Nile encephalitis. *Curr Opin Infect Dis* 2004; 17: 413–20.
2. Emig M, Apple DJ. Severe West Nile virus disease in healthy adults. *Clin Infect Dis* 2004; 38: 289–92.
3. Chowders MY, Lang R, Nassar F, Ben-David D, Giladi M,

- Rubinshtein E, *et al.* Clinical characteristics of the West Nile fever outbreak, Israel, 2000. *Emerg Infect Dis* 2001; 7: 675–8.
4. Cunha BA. West Nile virus encephalitis: clinical diagnostic and prognostic indicators in compromised host. *Clin Infect Dis* 2006; 43: 117.
 5. Cunha BA. West Nile viral encephalitis mimicking hepatic encephalopathy. *Heart Lung* 2005; 34: 72–5.
 6. Pherez FM, Cunha BA. Diagnostic importance of headache as a key symptom in suspecting malaria in adult returning travelers. *Travel Med Infect Dis* 2009; 7: 383–4.
 7. Cunha BA. Triad of non-specific laboratory tests in malaria. *Scand J Infect Dis* 2008; 40: 350–1.
 8. Cunha BA. Atypical lymphocytes in acute malaria. *Arch Intern Med* 1997; 157: 1140–1.
 9. Cunha BA. The diagnosis of imported malaria. *Arch Intern Med* 2001; 161: 1926–8.
 10. Cunha BA. Serum ferritin levels in West Nile encephalitis. *Clin Microbiol Infect* 2004; 10: 184–6.

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