A 51-Year-Old Man With Seizures and Progressive Behavioral Changes

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A 51-year-old man was admitted for evaluation of new-onset generalized seizures in the context of progressive and significant behavioral change. His medical history was only notable for previous outbreaks of genital herpes. He took no medications. He had occasional social alcohol use and no illicit drug use but was a 35-pack-year current smoker. The patient had no relevant occupational exposure history but had recently traveled to Panama. Initially, the patient's significant other noticed a progressive flattening of his affect. The patient then started to experience episodes of "passing out" that led to injuries prompting ED visits. He was prescribed antiseizure medications and scheduled for an outpatient workup. However, with progressive gait instability, lethargy, and an increase in frequency of generalized seizures, the patient was admitted for treatment of suspected viral encephalitis. Despite initiation of antimicrobial and antiviral therapy, the patient's level of alertness continued to decline, ultimately leading to intubation for airway protection. CHEST 2015; 147(3):e86-e89

Physical Examination Findings

On admission, the patient was normotensive and afebrile with sinus tachycardia at 114 beats/min. He was well developed, awake, but not oriented. He was unable to follow commands, although he was moving all extremities spontaneously. He had no nuchal rigidity. The remainder of the physical and neurologic examination was unremarkable. As the patient's level of alertness declined to stupor, no other new neurologic signs were appreciated.

Diagnostic Studies

The patient's CBC and electrolyte levels were normal. Serum alcohol and urine toxicology screen tests were negative. MRI of the brain showed a focal intracortical T2 hyperintense signal abnormality in the right-side cerebellar hemisphere that contained a punctate focus of enhancement (Fig 1A). The patient continued to have encephalopathy and seizures; a second MRI showed a

Manuscript received June 29, 2014; revision accepted July 19, 2014. **AFFILIATIONS:** From the Department of Internal Medicine (Dr Cable), Department of Neurology (Drs Freeman and Rubin), Department of Neurosurgery (Dr Freeman), Department of Critical Care (Dr Freeman), and Department of Pathology (Dr Khoor), Mayo Clinic, Jacksonville, FL; and Departments of Pulmonary and Critical Care Medicine (Dr Karnatovskaia), Mayo Clinic, Rochester, MN. small focus of enhancement in the right-side superior frontal gyrus (Fig 2A). An EEG captured left-sided frontotemporal rhythmic discharges leading up to a clinical seizure.

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Results of cerebrospinal fluid (CSF) analysis were as follows: WBC count, $36/\mu$ L with lymphocytic predominance (93%); RBC count, $19/\mu$ L; glucose level, 75 mg/dL (normal); and protein level, 29.8 mg/dL (normal). CSF Gram stain and blood cultures were negative for bacteria, fungus, and mycobacteria. Additional serum and CSF serologies were negative for cysticercosis, Lyme disease, syphilis, legionella, *Bartonella, Brucella*, Q fever, *Ehrlichia*, and Rocky Mountain spotted fever. Viral serologies were negative for herpes simplex, cytomegalovirus, John Cunningham virus, Epstein-Barr virus, varicella-zoster virus, West Nile virus, HIV, and Saint Louis and eastern and western equine encephalitis. Histoplasma urine antigen test results were normal.

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Figure 1 – A, Initial MRI fluidattenuated inversion recovery sequence showed a focal intracortical T2 hyperintense signal abnormality in the right cerebellar hemisphere, which contained a punctate focus of enhancement (arrow). B, Follow-up sequence after treatment, with the resolution of the right cerebellar lesion.



Figure 2 – A, Initial MRI fluidattenuated inversion recovery sequence showed a small focus of enhancement in the right superior frontal gyrus indicating an inflammatory lesion (arrow). B, Follow-up sequence after treatment, with resolution of the lesion.

What is the diagnosis?

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