St. Louis Encephalitis with Focal Neurological Signs

St. Louis encephalitis (SLE) is a form of epidemic encephalitis named for a 1933 outbreak in that Missouri city when >1000 cases occurred; the fatality rate during this epidemic was ~20% [1]. Although it is the most common type of viral encephalitis, <1% of patients present for medical attention [2]. Symptoms are usually nonspecific and consistent with those of viral meningitis. The virus causing SLE is distributed throughout the United States, and similar viruses exist worldwide. The disease is spread by Culex species mosquitoes, hence the primary risk factor is mosquito exposure. Focal neurological signs are extraordinary: to our knowledge, there are no previous reports of SLE with focal neurological signs in the medical literature. Here we report the case of a young man who presented with delirium and focal neurological signs attributable to SLE.

A 33-year-old man was admitted to the hospital with weakness and delirium. Two days before admission, the patient complained only of dizziness. On the day before admission, he had a right footdrop. He was seen at an emergency department and was discharged. He woke up the next morning with right lower extremity paralysis and was admitted to another institution.

The patient had no significant medical history and had not traveled for several months from northern Louisiana. He regularly abused alcohol and occasionally abused illicit (not intravenous) drugs.

His temperature was 38.4°C. He was anxious and agitated but cooperative. The right lower extremity strength was graded 1/5 with areflexia; the left lower extremity strength was graded 2/5 with hyporeflexia. The remainder of the physical examination was unremarkable. Pertinent findings included a negative urine toxicology screen, and the serum chemistry analysis was normal. Lumbar puncture revealed CSF with a WBC count of 484/mm³ (75% lymphocytes, 23% polymorphonuclear cells, and 2% monocytes), an RBC count of 21/mm³, a glucose level of 48 mg/dL, and a protein level of 87 mg/dL. Repeated cultures and polymerase chain reaction analysis of CSF for herpesviruses were negative.

Electroencephalography revealed widespread axonal degeneration and demyelination without demyelination. A diagnosis was made 3 days later when serum titers of IgG and IgM antibodies to the virus causing SLE were positive at dilutions of 1 : 1024 and 1 : 512, respectively (titers were determined at the State of Louisiana Reference Laboratory, New Orleans). He received supportive treatment and did well, with gradual clearing of his mental status and rehabilitation. When interviewed 6 months after discharge, he still complained of right lower extremity weakness and required the use of a walker, but according to his mother, his mental status was back to baseline.

This young man presented with many features typical of viral encephalitis: sudden onset, alteration in consciousness, and lymphocytic pleocytosis. In addition, he had very high acute-phase serum titers of immunoglobulins to the virus causing SLE.

Many features of SLE are atypical, however. Despite its prevalence, SLE is rarely diagnosed. Focal weakness and pleocytosis (cell count >100/mm³) are rare [3], demyelination and axonal degeneration rarer still. Neurological sequelae are unlikely since the only demonstrated risk factor is advanced age [4]. What caused neurological damage in this case is unclear. An immunologic reaction to the virus is possible although unlikely. Immunologic reactions to viruses are well characterized in Guillain-Barre syndrome and acute demyelinating encephalomyelitis, and neither fits this case. Typical examples of viral neuropathies, including those due to cytomegalovirus and HIV, present as chronic distal symmetrical pain and do not impair consciousness.

This case emphasizes that a broad differential diagnosis must be kept in mind even when it seems obvious to ascribe delirium to alcohol or toxins. Clinicians should be aware of the possibility of viral encephalitis during the summer months. Early reporting is key to initiating mosquito control programs and preventing the spread of infection.

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References
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Spontaneously Resolving Pulmonary Mucormycosis

The term *mucormycosis* is generally applied to infections caused by a variety of fungal organisms, including *Rhizopus*, *Rhizomucor*, and *Mucor* [1, 2]. Although treatment with systemic antifungal agents followed by surgical excision of the affected area [3] is recommended, the mortality rate remains high [3]. We describe a diabetic patient who was admitted to the hospital with ketoacidosis and pulmonary infection with *Rhizopus* species. His infection resolved without any specific therapy except to correct the diabetic metabolic disorder.

A 49-year-old man negative for human immunodeficiency virus (HIV) who had no significant medical or surgical history was admitted to the hospital because of progressive polyuria, polydipsia, weakness, and weight loss. One month before admission, he developed a temporary flu-like illness, continued to feel ill, and developed a productive cough. He was found to have diabetic ketoacidosis. A chest x-ray and CT scan revealed a cavitary mass (4 cm in diameter) in the anterior segment of the right upper lobe (figure 1). The patient underwent CT-guided fine needle aspiration (FNA) and fiberoptic bronchoscopy. Silver staining of FNA and bronchial brush specimens revealed the presence of nonseptate, branched (at 90° angles) fungal hyphae suggestive of mucormycosis (figure 1). Transbronchial biopsy showed invasive fungal hyphae. Culture of the FNA sample eventually yielded a *Rhizopus* species.

The patient recovered from ketoacidosis and started treatment with an insulin regimen. He was urged to receive therapy for the fungal infection. The patient refused and left the hospital against medical advice; medication at that time was insulin alone. Three months later, when he was asymptomatic, the patient sought a second opinion for treatment of the lung lesion. He had returned to work 1 week after discharge, and his diabetes was under control. The cough had disappeared within 4 weeks. A follow-up chest x-ray and CT scan demonstrated scarring resolution of the cavitary lesions in the right upper lobe (figure 1). He had not received antifungal treatment.

Diabetic ketoacidosis and hematologic malignancies predispose for pulmonary mucormycosis, which is associated with a high mortality rate, even with medical and surgical treatment [1–3]. Diagnosis relies on the presence of branching hyphae (at

Figure 1. A. Chest CT scan at the time of admission of a patient with spontaneously resolving pulmonary mucormycosis that shows a large cavitating mass in the right upper lobe. B. Chest CT scan 3 months later that reveals scarring in the right upper lobe. C. Silver stain of a fine needle aspirate from the right upper lobe that demonstrates many fungal elements branching at 90° angles (arrow) (original magnification, ×40).