Benjamin J. Sandefur, MD, Leslie W. Milne, MD

From the Harvard Affiliated Emergency Medicine Residency, Boston, MA (Sandefur); and the Department of Emergency Medicine, Massachusetts General Hospital, Boston, MA (Milne).

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Figure 1. Computed tomography of the brain demonstrating bilateral parieto-occipital hypodensity.

Figure 2. Computed tomography of the brain demonstrating bilateral parieto-occipital hypodensity (arrows).

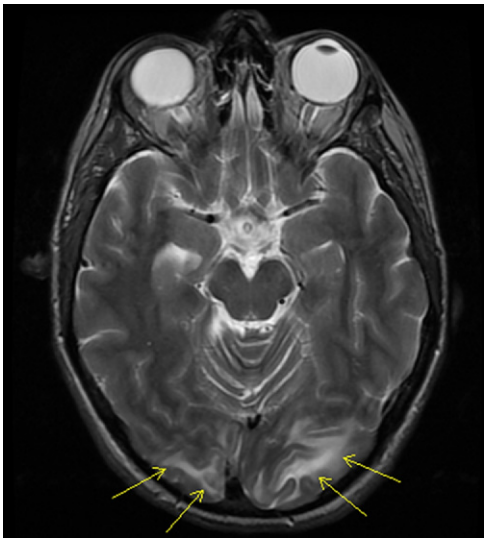

Figure 3. MRI of the brain, T2-weighted, demonstrating bilateral parieto-occipital hyperintensity (arrows). Used with permission of Benjamin J. Sandefur, MD, Harvard Affiliated Emergency Medicine Residency, Boston, MA.

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A 37-year-old woman presented to an emergency department (ED) with seizure. According to emergency medical services (EMS), she was a healthy gravida 2 para 2 2 weeks postpartum after uncomplicated cesarean section delivery at 38 weeks gestational age. She had experienced daily headaches for 1 week, and on awakening this day, she observed difficulty seeing. She subsequently experienced shaking in her left arm, which progressed to rhythmic jerking of all extremities for 2 minutes. EMS found her unresponsive, with snoring respirations and frothing from the mouth. The vital signs were notable for a blood pressure of 220/110 mmHg and a pulse rate of 116 beats/min. The patient was tracheally intubated on ED arrival for airway protection. She was taken urgently for computed tomography (CT) to rule out cerebral hemorrhage.

For the diagnosis and teaching points, see page 142.
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DIAGNOSIS:

Posterior reversible leukoencephalopathy syndrome in a postpartum eclamptic patient. Posterior reversible leukoencephalopathy syndrome is characterized clinically by headache, altered consciousness, cortical vision changes, and seizures and neuroradiographically by bilateral parieto-occipital white matter lesions.[1] Although the syndrome is associated with acute hypertension, less common causes include cytotoxic medications, sepsis, preeclampsia, and eclampsia.[1,2] The pathophysiology is poorly understood, though an accepted theory is that posterior reversible leukoencephalopathy syndrome is mediated by vasogenic edema resulting from loss of autoregulation or endothelial damage. Neuroimaging reveals white matter edema, characterized by hypodensity on CT and hyperintensity on T2-weighted MRI (Figures 1 and 2), commonly involving the parieto-occipital regions in symmetric fashion (arrows) (Figure 3).[1-3] Posterior reversible leukoencephalopathy syndrome is treated by aggressively controlling hypertension, typically with calcium antagonists or labetalol. Appropriate and timely treatment is critical because clinical and radiographic findings are reversible; however, permanent brain damage has been reported.[2,3]

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