



# Images In Medicine

## Eastern Equine Encephalitis: MRI Findings In Two Patients

*Matthew Ethier, MD, and Jeffrey Rogg, MD*

**A PREVIOUSLY HEALTHY 20 YEAR-OLD MALE PRESENTED TO THE** Rhode Island Hospital Emergency Department with 24 hours of worsening abdominal pain, vomiting, and fevers. On physical examination he appeared diaphoretic and febrile, with an oral temperature of 104.3°. The patient was noted to have tenderness to palpation in the epigastric region. Initial laboratory evaluation revealed a white blood cell count of 23,000/ $\mu$ l. A CT scan of the abdomen and pelvis was normal, with no evidence of appendicitis as was suspected clinically. The patient was started empirically on IV antibiotics and admitted for observation. On the first night of admission, the patient developed a severe headache, with subsequent rapid deterioration of his mental status. Status epilepticus soon followed. He was intubated for airway protection and transferred to the Medical Intensive Care Unit.

A non-contrast CT scan of the head showed diffuse cerebral edema and effacement of the basilar cisterns. Brain MRI performed shortly thereafter revealed abnormal T2 and FLAIR signal hyperintensity involving the bilateral thalami in a symmetric manner, as well as the left external capsule and tegmentum pons (Figures 1, 2, and 3; white arrows). Prominent perivascular congestion was also noted (Figure 4; white arrows).

No associated restricted diffusion or post-contrast enhancement was seen in these areas.

Due to diffuse cerebral edema and cisternal effacement a lumbar puncture could not be safely performed. A small amount of CSF obtained from placement of a ventriculostomy shunt yielded negative Herpes Simplex Virus (HSV) 1 and 2 PCR analysis. The patient expired 3 days after admission.

One month later, a 21 year-old male was transferred to the Neurology Intensive Care Unit at RIH from an outside hospital after being intubated for status epilepticus. The patient initially presented with 24 hours of headache and vomiting, and was febrile to 103.8°. His course was rapidly progressive, with mental status deterioration to the point of obtundation and onset of seizures beginning less than 12 hours after admission to the outside facility. His history noted exposure to mosquitoes while golfing in southeastern Massachusetts seven days prior to admission.

An initial brain MRI at the time of admission revealed abnormal T2/FLAIR signal hyperintensity in the mesial temporal lobes bilaterally in a pattern suggestive of viral encephalitis, possibly HSV based on these imaging findings. However, CSF analysis revealed negative HSV 1 and 2 PCR, in addition to negative gram stain and culture. The patient showed only

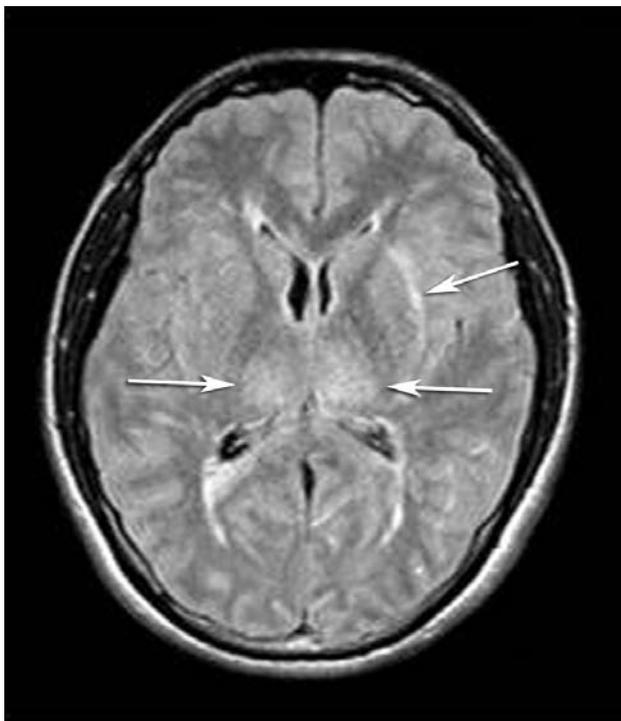


Figure 1.

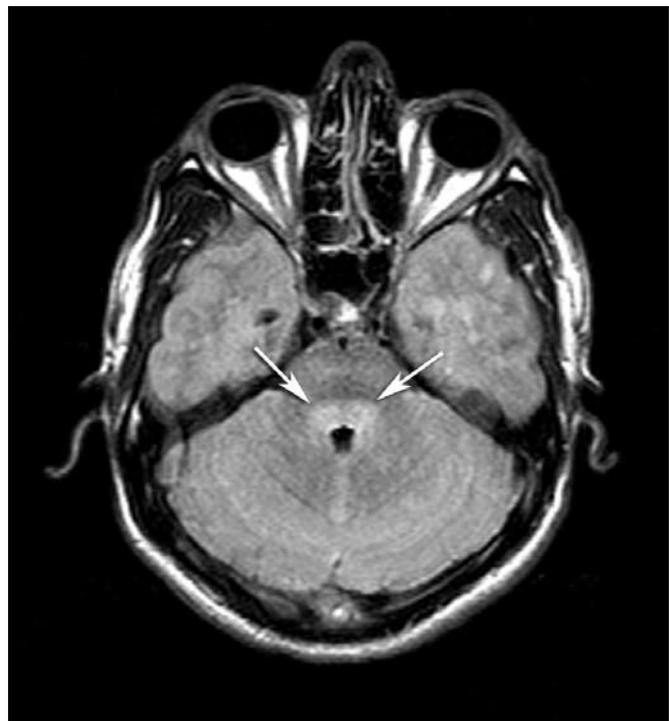


Figure 2.

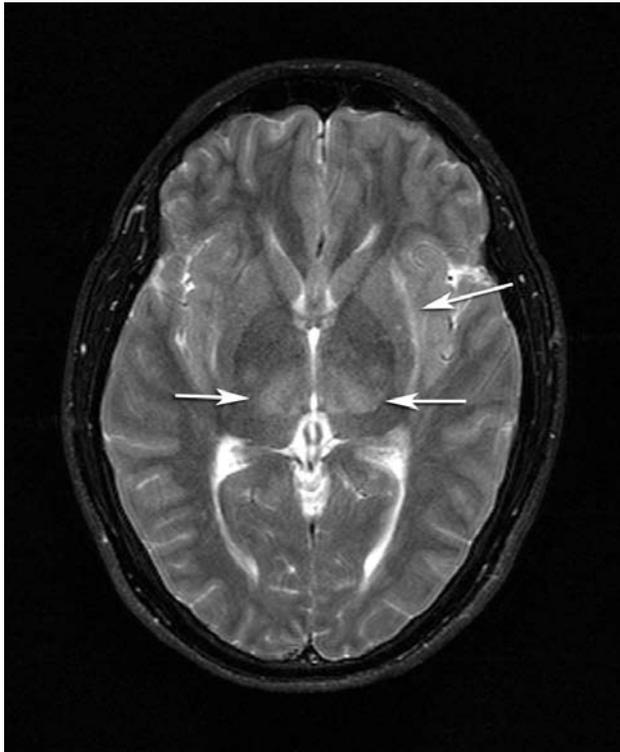


Figure 3.

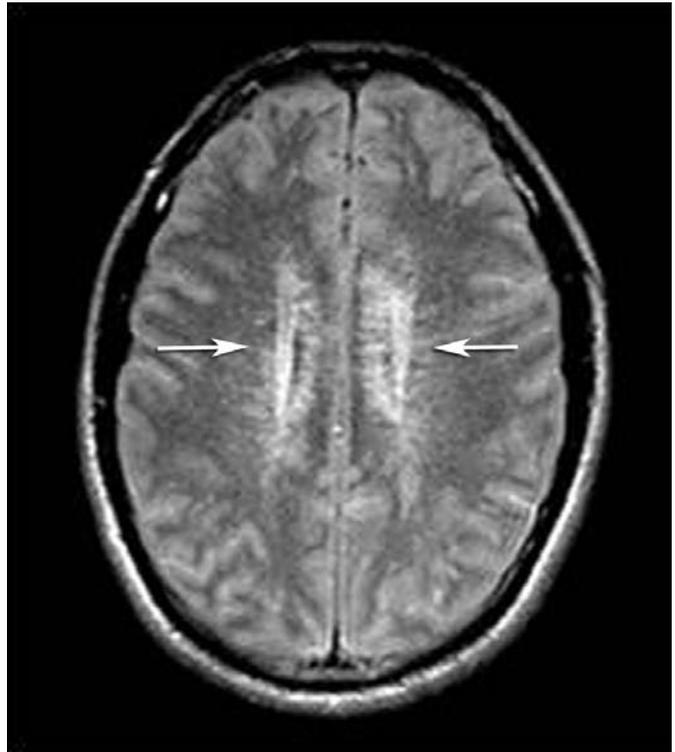


Figure 4.

minimal improvement clinically while on anti-viral therapy, so a subsequent brain MRI was performed 10 days later. This showed abnormal T2 and FLAIR signal hyperintensity within the pulvinar nuclei of the bilateral thalami, the medial aspects of the thalami, the bilateral insular cortex, bilateral putamina, and inferomedial frontal cortex bilaterally (see Figures 5 and 6;

white arrows). Repeat CSF analysis performed within 24 hours of the second brain MRI showed negative gram stain, culture, and negative HSV 1 and 2 PCR analysis, but anti-EEE IgM anti-body were positive. Anti-EEE IgG anti-body was negative. In addition to anti-viral therapy and steroid administration, this patient also received IVIg therapy. His neurologic status

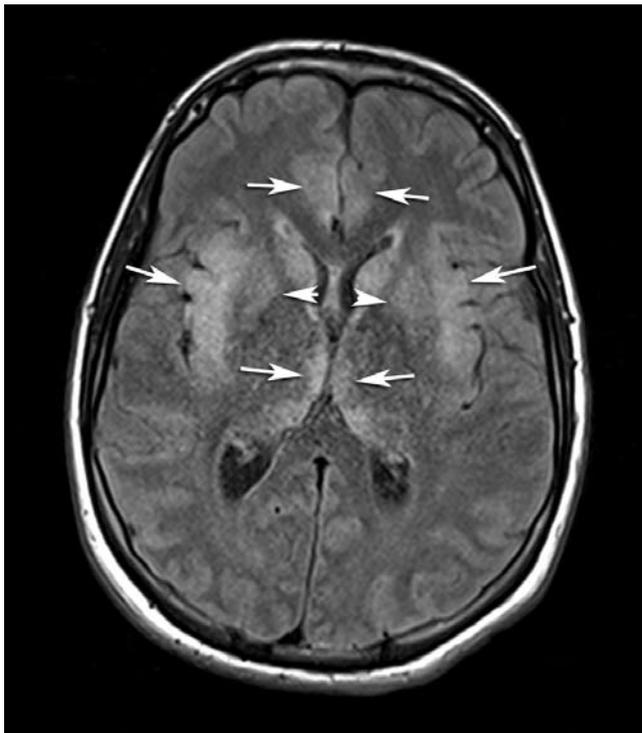


Figure 5.

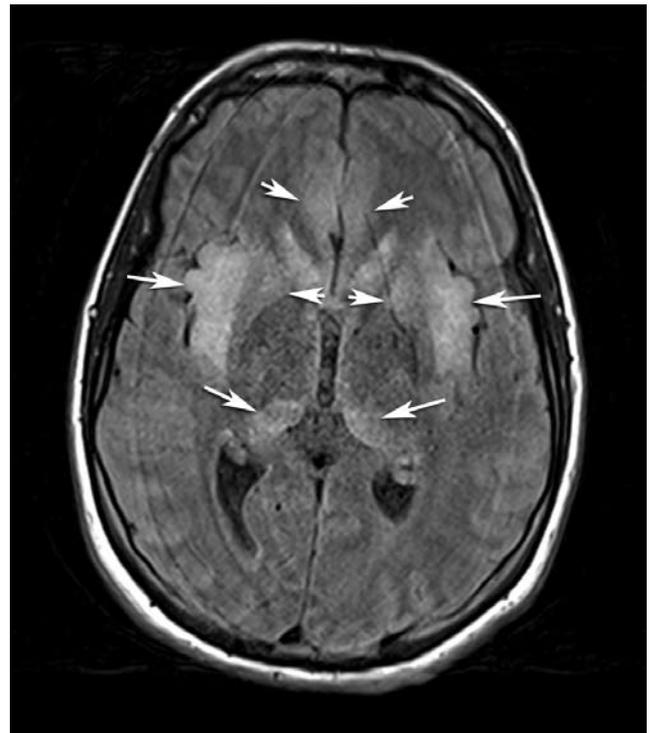
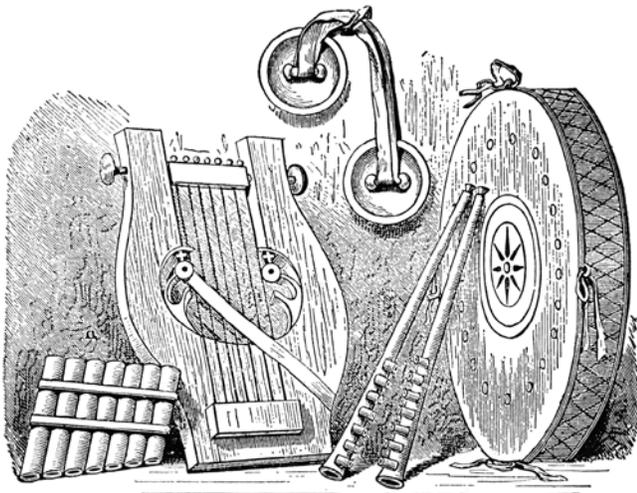


Figure 6.

improved gradually over the course of the admission and he was eventually discharged to a rehabilitation facility.

Eastern equine encephalitis (EEE) is an illness caused by the mosquito-borne arbovirus *Alphavirus togaviridae* which occurs predominantly along the East and Gulf coasts of the United States. Although only about 5% of infections lead to EEE, morbidity and mortality are high with 33-36% of affected patients dying from the illness.<sup>1</sup> In a study of the clinical and imaging manifestations of EEE performed by Deresiewicz et al in 1997, only 1 of the 36 patients with EEE made a full recovery.<sup>2</sup> The early presentation of EEE can be similar to any common viral illness, with symptoms of fever, headache and abdominal pain. Rapid deterioration of neurologic status and seizure activity then occurs due to severe encephalitis, often despite anti-viral and steroid therapy. There is limited evidence suggesting that IVIg may be of some therapeutic value.<sup>3</sup> Definitive diagnosis is made by CSF or serum analysis for anti-EEE IgM and IgG antibodies. While EEE titers were positive in the second case, there was an insufficient quantity of CSF to send for EEE titers in the first case, though this entity became the leading diagnostic consideration based on the imaging findings and the patient's rapidly progressive course. While encephalitis from herpes simplex virus can have a similar clinical course to EEE, the MR imaging findings can aid in distinguishing this entity from EEE. The basal ganglia, brainstem and bilateral thalami demonstrate abnormal T2 signal on MRI early in the course of EEE. These areas are often spared completely in HSV encephalitis or become involved only later in its course. In both of these cases, the imaging patterns were most consistent with EEE rather than HSV infections.



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*Matthew Ethier, MD, is a Diagnostic Radiology resident, at the Warren Alpert Medical School of Brown University.*

*Jeffrey Rogg, MD, is an Associate Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University, and is Director of Neuroradiology at Rhode Island Hospital.*

## Disclosure of Financial Interest

The authors have no financial interests to disclose.

## CORRESPONDENCE

Matthew Ethier, MD  
Department of Diagnostic Imaging  
Rhode Island Hospital  
593 Eddy Street  
Providence, RI 02903  
e-mail: methier@lifespan.org

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