Transient Neuroimaging Abnormalities of the Posterior Thalamus in Status Epilepticus

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**Purpose**
Reversible CT and MR imaging abnormalities ascribed to cerebral edema in focal status epilepticus may mimic the appearance of ischemia. Posterior thalamic involvement may help to differentiate these entities when cortical abnormalities are present in the distribution of the anterior circulation. Transient MR imaging abnormalities of the posterior thalamus have been described in only four previous cases of status epilepticus. We report transient signal alterations of the posterior thalamus due to focal status epilepticus as demonstrated by CT and MR imaging, likely secondary to excessive cortical stimulation.

**Materials & Methods**
A 44-year-old woman with a history of seizures since childhood presented with a left homonymous hemianopsia, hemiparesis, and hemisensory loss with recurrent left-sided focal motor seizures, 1 week after being emergently evaluated for persistent headaches. Laboratory investigation included extensive evaluation of serum and CSF. Video/EEG monitoring was performed. Neuroradiologic procedures included head CT and MR imaging with diffusion-weighted imaging, MRA and MRV. Transthoracic echocardiography and carotid ultrasound also were obtained.

**Results**
CT revealed isolated hypodensity of the right posterior thalamus with extensive hypodensity of the right temporal, frontal, and parietal lobes that was not present on CT performed 1 week earlier. MR imaging demonstrated corresponding signal abnormalities with unremarkable MRA and MRV. Prominent T2-weighted, FLAIR and diffusion-weighted imaging hyperintensity was noted with associated enhancement following gadolinium administration. EEG revealed periodic lateralized epileptiform discharges over the right temporo-parietal region with electrographic partial seizures and focal slowing. Seizure activity abated with phenytoin and phenobarbital. Examination of serum and CSF was unremarkable and evaluation of a possible ischemic etiology including transthoracic echocardiography and carotid ultrasound was unrevealing. Her focal neurologic deficits gradually resolved over a period of several days. Serial MR imaging demonstrated progressive resolution, although normalization of signal abnormalities within the right posterior thalamus was delayed considerably.

**Conclusion**
The posterior thalamus may exhibit transient signal abnormalities in the setting of status epilepticus associated with excessive cortical stimulation. Contemporaneous cortical and focal thalamic lesions defying rational vascular localization may be explained by seizure-related phenomena.
References