
Seizure-Induced Transient Hippocampal Abnormalities on MR: Correlation with Positron Emission Tomography and Electroencephalography

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Summary: We report transient focal abnormalities on MR in a patient having frequent electrographic seizures that were not obvious clinically. Marked mass effect (confirmed with volumetric studies) and abnormal T2 signal intensity in the right hippocampal region correlated with electroencephalographic ictal activity and with increased positron-emitting radiotracer uptake in the medial temporal lobe. The follow-up MR 2 months later, after electroencephalography findings normalized, revealed no hippocampal abnormalities.

Index terms: Seizures; Hippocampus; Brain, magnetic resonance

Transient focal abnormalities on magnetic resonance (MR) and computed tomography imaging of seizure patients have previously been described (1–7). Resolution of zones of radiolucency suggesting transient cerebral edema have been reported on computed tomography. Reversible regions of signal abnormality on T2-weighted MR images have been observed in patients with status epilepticus. However, in all the reports referred to above, the imaging abnormalities did not involve the hippocampus or medial temporal lobe. Two recent reports have described transient signal changes on T2-weighted images in the hippocampal region without associated mass effect (8–9). We report a case in which MR imaging of a patient with seizure activity on electroencephalography (EEG) showed abnormal hyperintensity and marked mass effect in the hippocampus that mimicked a neoplasm. However, a follow-up MR 2 months later was normal. Volumetric hippocampal measurements and positron emission tomography (PET) findings also are reported.

Case Report

A 69-year-old white man was transferred to our hospital approximately 3 weeks after a tonicoclonic seizure with loss of consciousness. The patient had no previous history of a seizure. Since the seizure, the patient had remained in a relatively persistent state of confusion, with disorientation and a fluctuating level of consciousness. Cerebrospinal fluid analysis at the other institution 2 weeks before admission to us showed mild leukocytosis (27 cells per cubic millimeter) primarily attributable to lymphocytes. All cultures of the cerebrospinal fluid, blood, urine, and stool at that time were negative, and viral antibody tests did not indicate the presence of an acute viral infection. The patient was afebrile and cerebrospinal fluid cell counts; protein; and all viral, bacterial, and tuberculosis cultures were negative on admission to our hospital as well.

Because of the suspicion of nonconvulsive seizure activity, the patient was transferred to the epilepsy monitoring unit after being placed on phenytoin and phenobarbital therapy. During the 5-day monitoring period, the patient did not have any obvious seizures. However, EEG monitoring revealed that the patient was having frequent epileptiform discharges, all lasting less than 1 minute. The predominant ictal activity was confined to the right temporal region and consisted of a mixed delta and theta semi-rhythmic activity of 3 to 7 Hz. An MR study without and with gadopentetate dimeglumine administration on the third day of his epilepsy monitoring unit stay revealed enlargement of the right hippocampus, increased signal intensity on T2-weighted images (3000/90/0.75 [repetition time/echo time/excitations]) and mild contrast enhancement (Fig 1 A–C). This mass lesion was interpreted as most likely representing a glioma. Several days after the patient left the epilepsy monitoring unit, a fludeoxyglucose F 18 PET study of the brain was performed (Fig 1D), which showed a focus of increased uptake in the same location as the lesion demonstrated on the MR image. Again, the findings were interpreted as being consistent with a neo-

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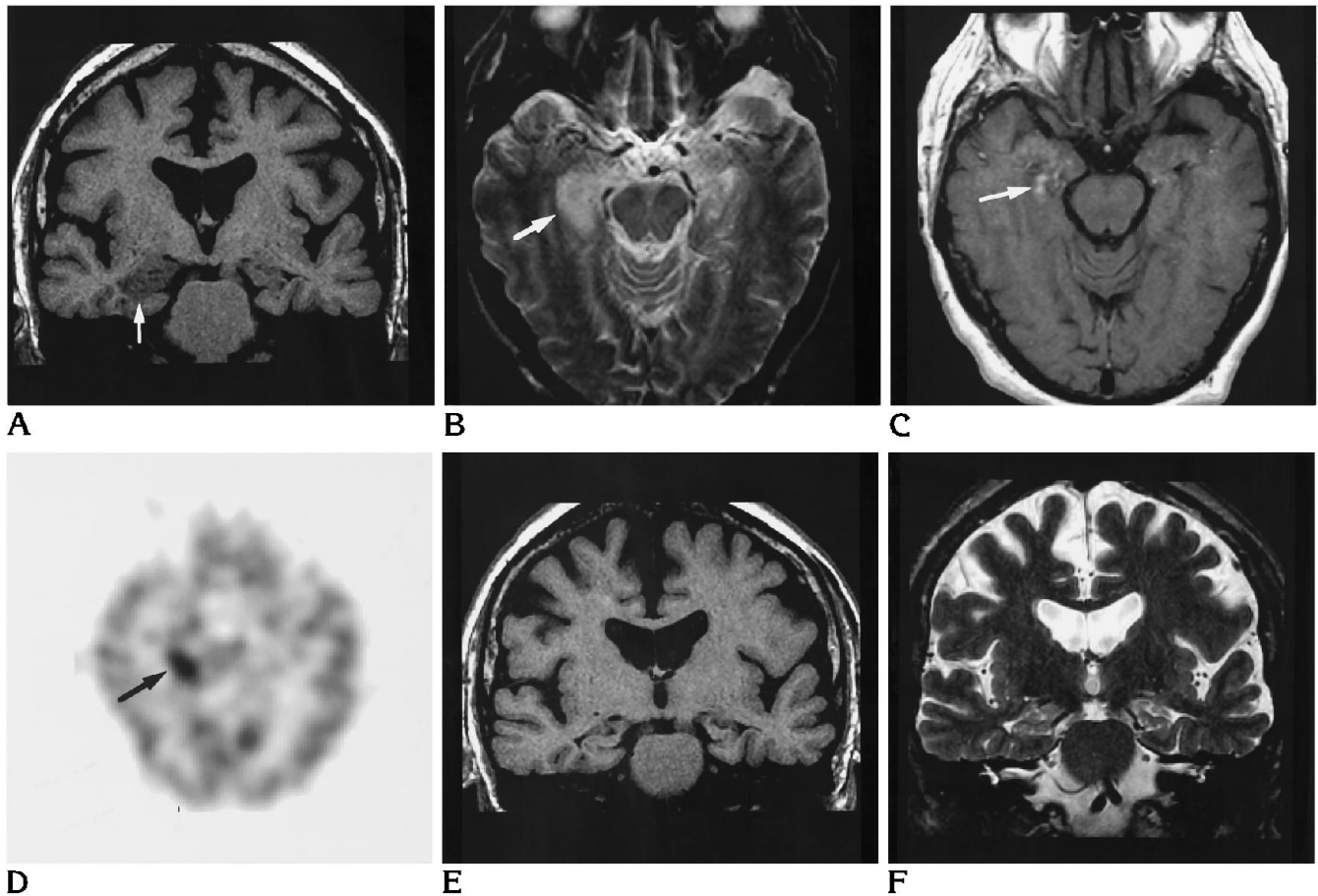


Fig 1. MR images performed while the patient was having temporal lobe seizure activity. A, Coronal T1-weighted image (500/17/2) shows an enlarged, hypointense right hippocampal head (*arrow*). B, Axial T2-weighted image (3000/90/0.75) shows abnormal hyperintensity and enlargement of the right hippocampal body (*arrow*). C, Axial T1-weighted image after gadopentetate dimeglumine administration demonstrates mild contrast enhancement (*arrow*) in the anterior hippocampus. D, Axial image from a fludeoxyglucose F 18 PET study at the same level as B demonstrates a focus of hypermetabolism (*arrow*) in the medial aspect of the right temporal lobe. Coronal MR studies acquired 8 weeks after the initial study show complete resolution of the hippocampal enlargement and signal abnormality on T1-weighted (E) and T2-weighted (F) images.

plasm. Over the next several weeks, the patient's confusion and disorientation began to improve gradually, and a follow-up awake and drowsy EEG recording was normal, with no paroxysmal abnormalities. Eight weeks after the original MR study, follow-up MR imaging was normal (Fig 1E, F). Hippocampal volumetrics were performed retrospectively on both MR studies, using previously described methods (10, 11). On the initial study, the right hippocampus measured 4.4 mL and the left measured 1.5 mL, whereas on the follow-up examination, the right measured 1.8 mL and the left was unchanged.

At a 6-month follow-up evaluation, the patient's mental status had returned to near baseline on carbamazepine therapy. He did not have any evidence of malignancy. His chest x-ray was normal, and there were no abdominal masses or complaints.

Discussion

MR imaging is the radiologic study of choice in the evaluation of epilepsy. To date, the significant MR imaging findings in patients with seizures of temporal lobe origin have included mass lesions (12, 13) and findings suggesting mesial temporal sclerosis (asymmetrically small or atrophic hippocampus with increased signal intensity on T2-weighted images ipsilateral to the seizure focus). It has been postulated that the signal abnormalities are attributable to gliosis, edema, or both (14).

In our case, the patient was imaged at the time he was having ictal activity in the right

temporal lobe. The right hippocampal swelling and increased signal intensity on T2-weighted images presumably was secondary to vasogenic and cytotoxic edema at the seizure focus (1, 2, 6, 9). Although the patient was not having clinical seizures or EEG monitoring at the time of the PET scan, the hypermetabolic zone in the right temporal region strongly suggests that this was an ictal scan.

The cause of the patient's seizures was never established. Encephalitides, especially herpes encephalitis, may involve medial temporal lobe structures but would be unlikely to involve only the hippocampus. In addition, laboratory tests did not clearly indicate the presence of an acute viral infection. Nevertheless, viral encephalitis as a cause of his seizures cannot be completely excluded. The resolution of the hippocampal swelling on the follow-up MR study essentially rules out the diagnosis of a low-grade astrocytoma. The hypermetabolism identified in the right temporal region and the absence of other evidence of posterior cerebral artery ischemic change makes hippocampal ischemia an unlikely cause of these imaging findings. Limbic encephalitis, a rare paraneoplastic disorder, may present with hippocampal enlargement and signal intensity changes, but this is usually a bilateral process. In addition, these patients usually have a known primary cancer, and typically cerebrospinal fluid protein is elevated (15).

In conclusion, reversible unilateral hippocampal enlargement and abnormal hyperintensity on T2-weighted MR imaging in a patient having temporal lobe ictal activity on EEG are consistent with previously reported findings of seizure-induced transient vasogenic and cytotoxic edema. This entity can be easily confused with a neoplasm on both MR and PET imaging. It is possible that hippocampal enlargement with abnormal signal intensity represents the first step in the pathophysiology of mesial temporal sclerosis.

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