

TRANSIENT CORTICAL ABNORMALITIES ON MAGNETIC RESONANCE IMAGING AFTER STATUS EPILEPTICUS: CASE REPORT

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INTRODUCTION

Transient neuroimaging findings associated with seizure activity have received relatively little attention in the neurosurgical literature. These abnormalities may mimic neoplastic or ischemic changes on magnetic resonance imaging (MRI), possibly leading to additional studies and surgical treatment.

CASE DESCRIPTION

A 17-year-old right-handed male was transferred to emergency room in status epilepticus. A MRI obtained 5 months before admission was negative. On the day of admission, he had multiple intermittent upper-left extremity partial seizures and prolonged secondary generalized seizures. An electroencephalogram (EEG) showed frequent epileptiform discharges over the right hemisphere posteriorly. A MRI study performed 2 days after admission revealed non-hemorrhagic abnormalities involving the right occipital region that were hyperintense on fluid-attenuated inversion recovery (FLAIR) and T2 weighted sequences. The apparent diffusion coefficient map was unremarkable. Follow-up MRIs, 3 and 11 months after admission, showed complete resolution of these lesions.

CONCLUSION

Imaging findings after status epilepticus may raise suspicion of ischemic or neoplastic lesions. These findings may be reversible. Further follow-up imaging may prevent unnecessary intervention. © 2004 Elsevier Inc. All rights reserved.

KEY WORDS

Epilepsy, status epilepticus, magnetic resonance imaging, transient, cortical abnormality.

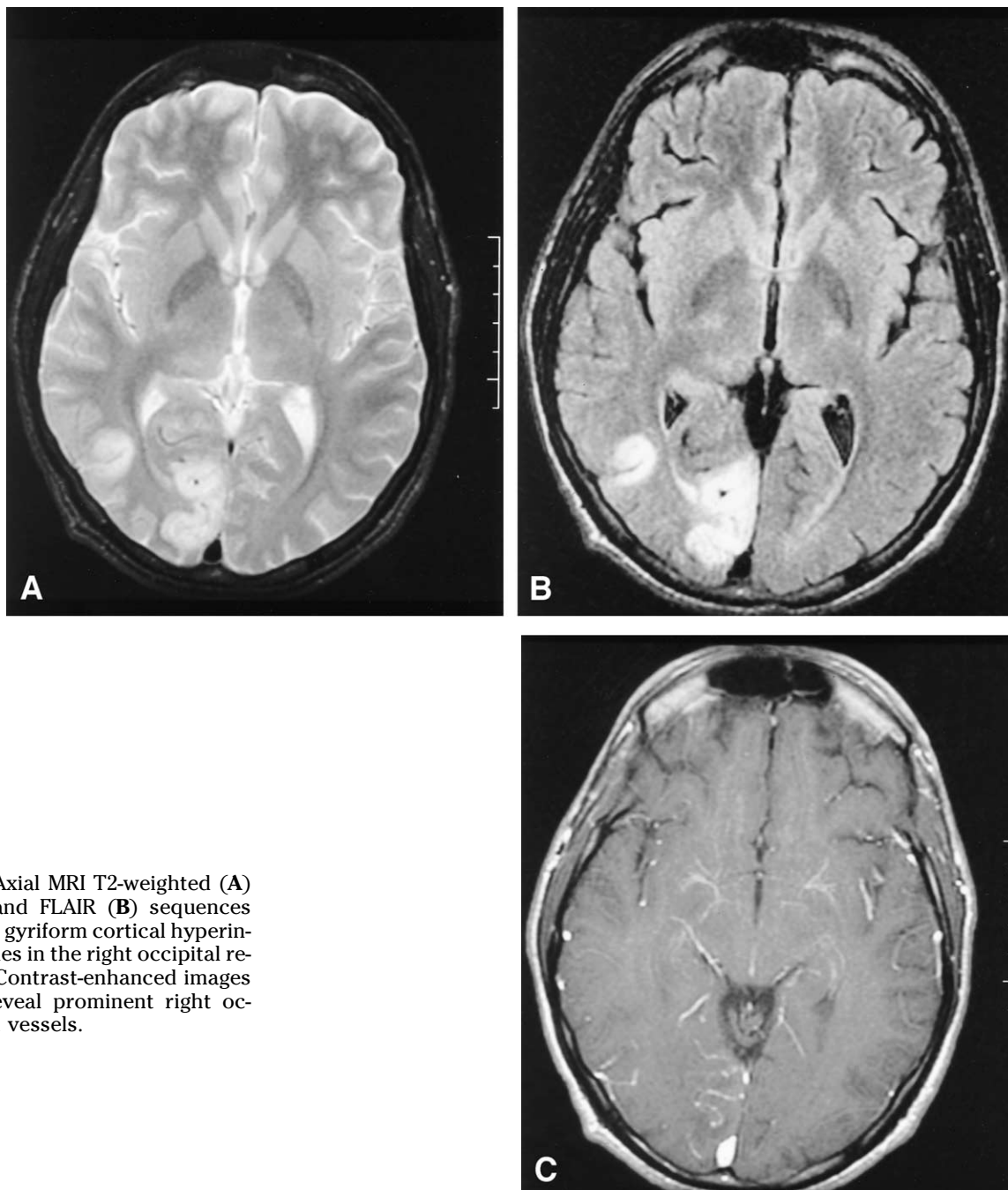
Transient computerized tomography (CT) and magnetic resonance imaging (MRI) abnormalities may occur after a seizure, most often following status epilepticus [2,4,9,10]. It is important to recognize that seizures can produce transient MRI abnormalities to avoid unnecessary neurosurgical procedures, for example, brain biopsy or invasive cerebral vascular studies. Complementary non-invasive neuroimaging studies may help elucidate the nature of these abnormalities.

CASE REPORT

A 17-year-old right-handed male was transferred to the emergency room in status epilepticus. He had a history of febrile seizures and a partial seizure disorder since age 10. He had no history of developmental delay, meningitis, encephalitis, or significant head injury. A MRI obtained 5 months before admission was negative. His typical seizures involved turning of the head to the left, left arm extension, and secondary generalized seizures. He was previously treated successfully with carbamazepine (500 mg twice a day), but was unable to take it the day before admission because of a flu-like illness. On the day of admission, he had multiple intermittent upper-left extremity partial seizures and prolonged secondary generalized seizures. He was admitted, intubated, and was started on lorazepam, fosphenytoin, phenobarbital, levetiracetam, and continued on carbamazepine. Upon discontinuation of his sedation, his neurologic examination was significant for left-sided apraxia and a left homonymous hemianopia. He continued to have simple partial motor seizures involving the distal left upper extremity.

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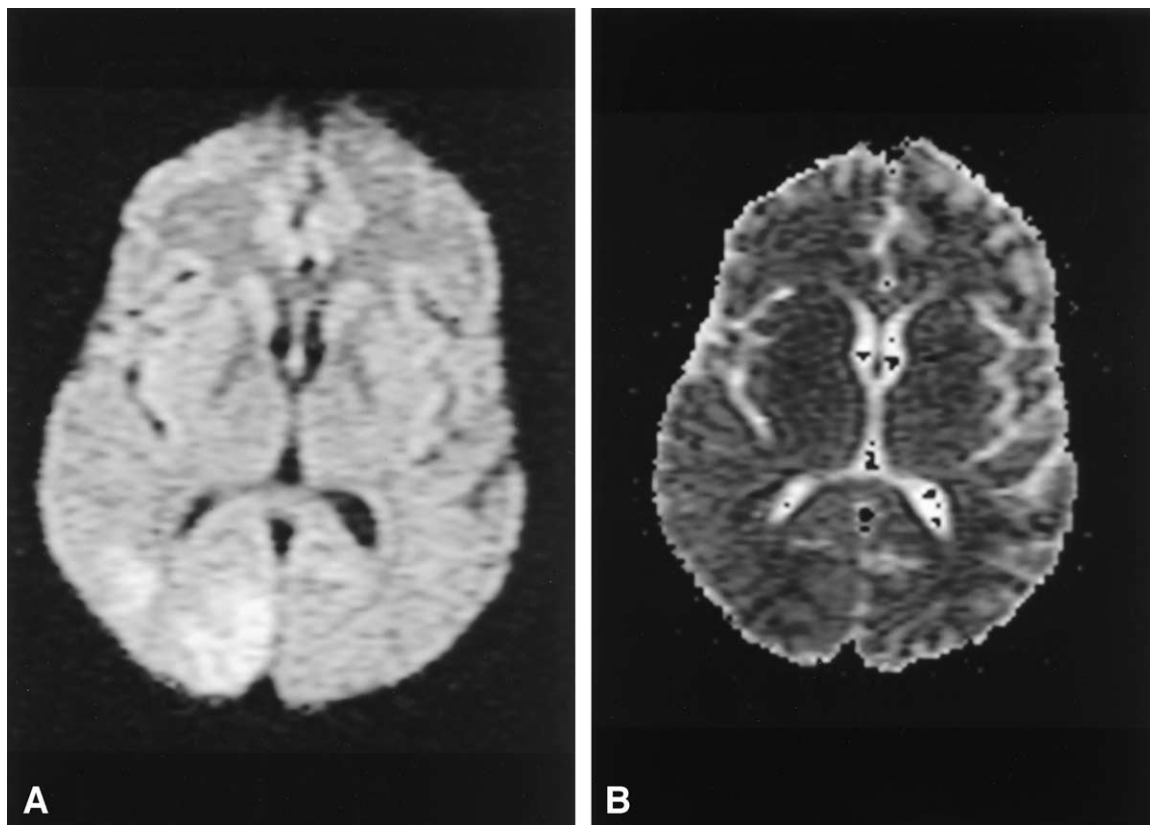
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1 Axial MRI T2-weighted (**A**) and FLAIR (**B**) sequences reveal gyriform cortical hyperintensities in the right occipital region. Contrast-enhanced images (**C**) reveal prominent right occipital vessels.

An electroencephalogram (EEG) showed frequent epileptiform discharges over the right hemisphere posteriorly. A MRI study performed 2 days after admission revealed non-hemorrhagic abnormalities involving the right occipital region that were hyperintense on fluid-attenuated inversion recovery (FLAIR) and T2 weighted sequences (Figure 1 **A, B**). Contrast-enhanced images revealed prominent right occipital vessels (Figure 1**C**). Diffusion

weighted images showed hyperintensity in the similar region (Figure 2 **A**), however; the apparent diffusion coefficient (ADC) map was unremarkable (Figure 2**B**). Follow-up MRIs, 3 and 11 months (Figure 3) after admission, showed complete resolution of these lesions. He continued having intermittent visual auras in the left visual field followed by left upper extremity myoclonic jerks. His left homonymous visual field deficit remained unchanged.



2 A diffusion-weighted axial MRI image (A) reveals hyperintensities in the corresponding regions. The apparent diffusion coefficient map (B) is unremarkable. These features argue against acute cerebral ischemia.

DISCUSSION

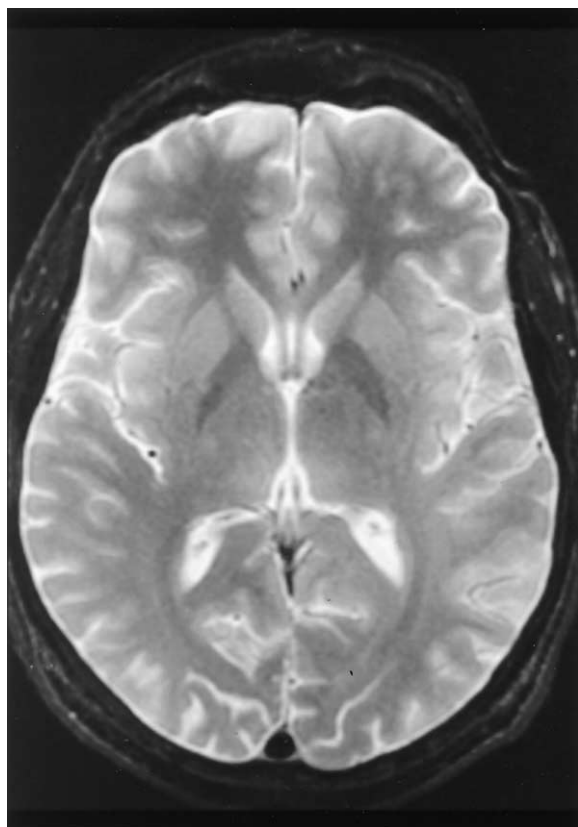
The exact pathogenesis of these transient lesions remains uncertain. Transient focal edema related to breakdown of the blood-brain barrier during a seizure has been postulated to be involved in the development of transient peri-ictal MRI abnormalities [7]. It is important to recognize the radiologic features of transient postictal abnormalities to differentiate them from ischemic, neoplastic, or infectious processes. Contrast enhancement of various intensity may be observed and may be related to alteration of blood–brain barrier [4]. Angiographic findings in the peri-ictal period suggest abnormal vascular permeability and “luxury perfusion” leading to focal edema [6]. Diffuse gyral and focal nodular enhancement and white matter hypo- or hyperdensity with and without mass effect have been described for these reversible lesions in the peri-ictal period [1,3,10].

Diffusion-weighted MRI (DWI) has been proven useful for evaluation of acute ischemia [11]. In the ischemic region, DWI intensity is increased and the apparent diffusion coefficient map (ADC), a measure of water diffusion, is decreased. In our case, high

DWI intensity suggests vasogenic edema; however, a lack of ADC intensity changes argues against ischemia. These findings are limited to the cortex and do not respect vascular territories. Neoplasm may be excluded based on the reversible nature of these lesions. Despite the resolution of these abnormalities, regional brain atrophy suggesting permanent brain injury has been reported in the literature [5]. The persistent left-sided visual field deficit in our patient most likely is another evidence for neuronal injury associated with these lesions.

CONCLUSION

Lack of significant abnormalities on follow-up imaging may support “benign” nature of these reversible peri-ictal changes. Patients with transient peri-ictal neuroimaging findings have undergone angiography or biopsy, with normal or nonspecific findings, and therefore, no change in therapy [8,12]. Recognition of these entities can encourage follow-up imaging to establish their transient nature and prevent unwarranted intervention.



3 An axial T2-weighted MRI obtained 11 months later shows resolution of the right occipital abnormalities.

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COMMENTARY

This interesting case report documents a slightly different angle on a phenomenon that has been known for years: that cortex-supporting ictal activity has complex metabolic changes associated with it that may result in transient abnormalities visible by structural imaging. In this case, the structural image was produced by magnetic resonance.

In the early 1980s, shortly after CT scanning became common, it was noted anecdotally that enhanced scans taken immediately after severe seizures or status epilepticus might show contrast enhancement in the region of the suspected epileptogenic focus. This was so commonly believed that several of us attempted this strategy as a way of helping to identify foci not associated with obvious structural lesions. It was never validated and, in my own personal experience, was capricious at best. Therefore, the strategy died. MRI, however, is much more sensitive and can show abnormalities never seen with CT scans.

So, though the authors' "take-home" message is to be aware of this potential phenomenon so as not to unnecessarily biopsy one of these lesions (which may be difficult to resist if the patient has never been scanned before), another message is that such transient lesions may provide clues to the location of epileptogenic foci that are not otherwise associated with obvious structural abnormalities.

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