Hyperdensity on CT After Seizure: A Pitfall

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Abstract

A 12-year-old boy had a hyperdense area corresponding to a gyral pattern on an enhanced CT brain scan within 12 hours of his last seizure. The hyperdense area disappeared on a subsequent enhanced CT scan after he was seizure free for about 48 hours. The hyperdense area was in a location (mesial frontal lobe) predicted by the interictal physical exam findings and the seizure type recorded on video–EEG monitoring. We postulate that the CT abnormality was due to transitory increase of regional cerebral blood flow and vascular permeability. (*J Child Neurol* 1987;2:276–278).

Angiographic and radionuclide brain scan abnormalities, in the form of blush or increased isotope uptake respectively, during or after seizures, are well described in the literature. Now that computed tomographic (CT) scans have largely supplanted these invasive investigations as a principal tool in the evaluation of epilepsy[1] one might also have expected reports of changes in CT scans in the ictal and postictal periods. However, such reports are scant in the literature, and since the CT scan changes could suggest a mass lesion, we report a patient who has partial elementary seizures with secondary generalization and who had transient changes on the enhanced cranial CT scan in the immediate postictal period.

Case Report

This 12-year-old white male had a 6-year history of solely nocturnal seizures, beginning from 30 minutes after going to sleep to the early hours of the morning. As witnessed by his mother, his seizures always start with awakening with a cry, after which he assumes a flexed position, has generalized clonic movements or sometimes just tonic postures. Each spell lasts for a minute or so and is followed by 1 or 2 minutes of postictal exhaustion, after which he goes back to sleep. He has had one or more such spells every night since they started. He developed significant side effects while on phenytoin and carbamazepine separately, necessitating discontinuation. A limited trial of valproic acid was of no benefit.

He was admitted to the hospital for video–electroencephalographic (EEG) monitoring at which time the general physical and initial neurological examinations were normal. While in the hospital, he continued to have nocturnal spells. Only on one occasion, on routine exam by a neurologist, his right plantar response was upgoing. This sign could not be reproduced later by the same examiner. On further questioning, his mother revealed that the patient sometimes complained of right leg weakness in the morning, which spontaneously resolved, and that some of his seizures started as jerking of the right upper extremity.

He had no significant problems in the neonatal period and reached his developmental milestones appropriately. There was no history of meningitis or head trauma and no family history of a seizure disorder or a neurodegenerative disease.

Investigation

Three seizures were recorded on cable telemetry EEG and audio-video monitoring. The first two seizures started with stiffening of both lower extremities and fencing posture with extension of the right upper extremity. All three seizures later became secondarily generalized. On EEG, all seizures started with appearance of fast activity in the beta range in both temporal electrode chains, followed a few seconds later by rhythmic delta activity in the parasagittal electrodes. On the basis of clinical history and monitoring, he was felt to have partial motor seizures with secondary generalizaton.

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While still having nocturnal seizures, an unenhanced cranial CT scan was normal, but a CT scan after contrast infusion showed focal enhancement in the left hemisphere cortex near the falx in the area of leg representation (see Figure 1). The enhancing area followed the normal gyral pattern. He was started on carbamazepine. After he was seizure free for about 48 hours, the CT scan was repeated with and without contrast. The area of enhancement seen on the previous study had disappeared (See Figure 2). While the patient was seizure free, magnetic resonance imaging (MRI) of the brain also did not confirm the CT scan abnormality seen on the first CT scan.

Discussion

Prensky et al[2] reported four children with repeated focal motor seizures who had positive radionuclide (technetium) brain scans performed within 72 hours of the last seizure. The scans performed from 72 hours to 15 months after the last seizure in all four patients were normal. These patients subsequently had normal development and no focal neurologic signs for the duration of follow-up. Positive brain scans are uncommon in idiopathic seizure disorders, and Prensky et al postulated that the isotope scans in their four patients were positive for two reasons: (1) because the scans were performed within 72 hours of the last seizure, and (2) because the patients had repeated focal motor seizures. In the 13th hour of partial motor status epilepticus, Farrell et al[3] reported abnormalities on carotid angiogram, including vasodilation, abnormal venous flow pattern, and parietal gyral stain. A repeat angiogram 8 days later was normal, and the patient was neurologically normal on follow-up. Regional hyperfusion was also shown by Holmquist et al[4] using cerebral scintiangiography in two patients with partial motor status epilepticus. Hougaard et al[5] came to the same conclusions (ie, ictal blood flow increases markedly in areas presumed to participate in seizure activity) using intra-arterial Xenon-133. Interictally, Oakley et al[6] found a relative increase in CT numbers after enhancement lateralized to the side of an epileptic focus compared with the homologous opposite brain.

To help localize the sites of lesions in partial seizures, elegant studies using positron emission

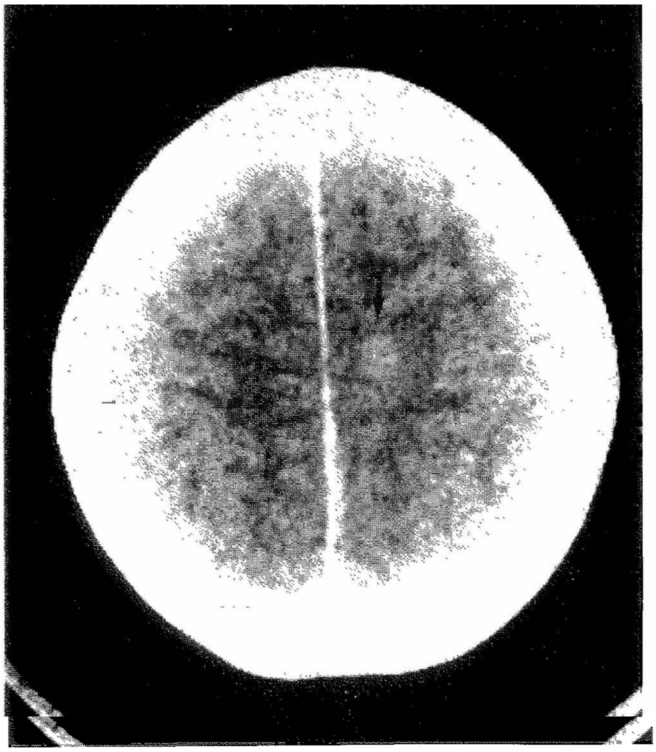

FIGURE 1
Contrast-enhanced CT scan within 12 hours of seizure. Note left parasagittal hyperdensity (arrow).

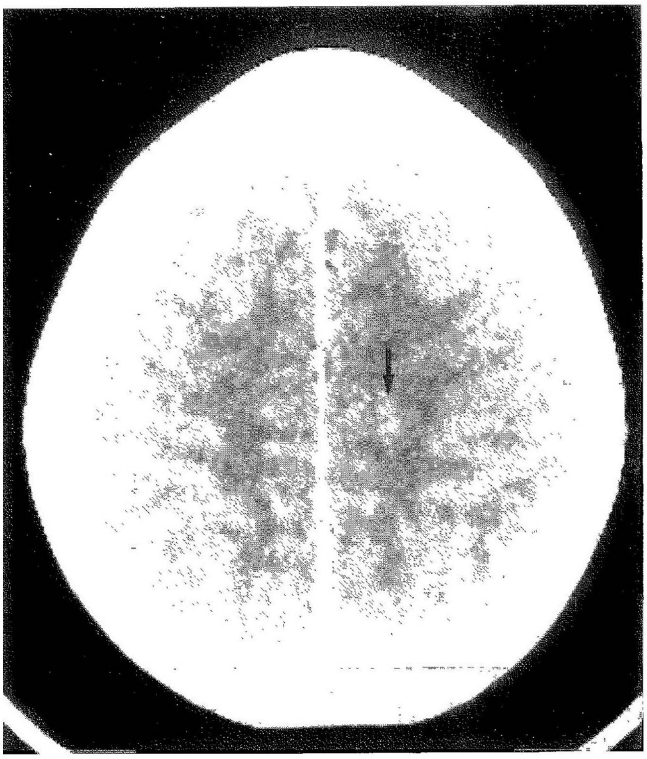

FIGURE 2
Contrast-enhanced CT scan 48 hours after last seizure. Hyperdense area in the left parasagittal area has disappeared (arrow).

computed tomographic (PECT) scans were done by Kuhl et al.[7] In the interictal state, the ^{18}F-fluorodeoxyglucose and ^{13}N-ammonia PECT scans respectively demonstrated broad regions of cortical hypometabolism and hypoperfusion corresponding to the EEG localizations and lateralizations. This is just the opposite of observations made by Oakley et al on enhanced CT scans. However, as in the other studies, Kuhl et al found that metabolism and perfusion increased to twice normal in the cortical epileptic focus during active seizures.

There is ample evidence both from animal experiments and human studies that during seizures, cerebral metabolism increases and the need for substrates rises. This need is met by increased cerebral blood flow.[8,9] That regional cerebral blood flow increases during partial seizures is adequately shown by studies mentioned above. There is also increased vascular permeability during seizures, as shown in animal experiments by Lorenzo et al.[10]

Although there are two other reports of postictal CT scan abnormalities,[11,12] it is not clear why the observation of ictal or immediate postictal changes has not been made more often either on isotope or CT scans. One of us (R.H.) with long-standing experience in neuroradiology has seen only four cases (including our patient) showing this pattern in the last 10 years (Heinz et al, in preparation). Rumack et al[11] reported a case of transient postictal enhancing focal CT scan abnormality in a 2-year-old child. Sammaritano and his colleagues[12] recently reported focal CT scan changes in three patients following partial status epilepticus. These authors do not comment on the appearance of hyperdensity on the postictal CT scan seen in their patients. We observed that the hyperdensity on the CT scan in the immediate postictal period followed the gyral pattern. This observation was also made in the other three patients seen by one of us (Heinz et al, in preparation). We believe that this transient focal enhancement which follows a gyral pattern may help differentiate an ictal–postictal abnormality from brain tumor or infarction. We agree with the suggestion by Prensky et al[2] that detection may depend on the time interval between the scan and the last seizure, as well as the severity of the seizures. Only more experience will clarify this point.

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