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Acute postictal cerebral imaging.

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Acute Postictal Cerebral Imaging

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BACKGROUND AND PURPOSE: Imaging of postictal patients is performed to investigate causes of seizure, such as space-occupying lesions or other “structural” processes; however, abnormalities may be found that reflect physiological or pathologic alterations due to seizure activity. The purpose of this study was to determine the brain imaging findings in patients in the immediate postictal period who presented with altered mental status or weakness.

METHODS: Ten patients who were examined for postictal neurologic derangement were studied (nine by CT and one by MR imaging) within 12 hours of ictus. Four of the CT studies and the one MR study included administration of contrast material. Follow-up examinations were performed 1 day to 11 months later. These studies were reviewed retrospectively.

RESULTS: CT findings included focal gyral swelling (10/10), effacement of adjacent cortical sulci (2/10), decreased gyral attenuation by CT (8/9), and mild to moderate gyral enhancement after injection of contrast material (5/5). MR imaging findings included gyral swelling, increased signal intensity on T2-weighted images, and enhancement after injection of contrast agent. The abnormalities were located in the frontal lobes (9/10, with bilateral involvement in 6/10), the parietal lobes (4/10), the temporal lobes (2/10), and the occipital lobe (1/10). Follow-up studies revealed complete or subtotal reversal of these abnormalities.

CONCLUSION: Although there are numerous causes of gyral swelling and enhancement, such as infarction and neoplasm, if these conditions are reversible and correspond to clinical findings, then the differential diagnosis is narrowed to postictal change, reversible ischemia, complicated migraine, or resolved inflammation/infection.

Most seizure activity resolves in a few minutes without persistent neurologic deficits, and if this activity is chronic in nature and unchanged in pattern, it does not require imaging evaluation. Transient hemiparesis associated with seizures and lasting for hours or days has been described as Todd's paralysis. Other transient postictal events, such as sensory loss, persistent altered mental status, or aphasia, have been called Todd's phenomena. When patients present with such postictal neurologic deficits, the differential diagnosis includes several entities that secondarily cause seizures, such as cerebrovascular accident, transient ischemic attack, hemorrhage, neoplasm, and encephalitis. In addition, at imaging, brain swelling may be encountered (1–4) in association with edema and abnormal enhancement and later found to be transient in nature, leading to a diagnosis of seizure-induced brain swelling and dysfunction affiliated with

the neurologic deficits. Such findings have been reported in the literature, but a characteristic pattern of swelling has not been specifically described.

Methods

Ten patients with acute seizure activity had persistent neurologic deficits or altered mental status and were examined by CT or MR imaging within 12 hours of ictus to ascertain the presence of a primary seizure-generating lesion, such as neoplasm or hemorrhage. The group included three men and seven women, 32 to 81 years old (mean age, 57 years). The time from ictus to the first imaging study ranged from 1 to 12 hours (mean, 4 hours). Nine patients had CT studies and one had an MR study. Four of the CT studies and the MR imaging study included IV administration of contrast material. The time from the first to the second imaging study ranged from 1 day to 11 months (mean, 62 days). Four of these were CT studies and two were MR studies (both with IV contrast). Imaging studies were evaluated for the presence of space-occupying lesions such as hemorrhage, neoplasm, and abscess. Once these were excluded, they were further examined for gyral swelling, sulcal effacement, attenuation or signal alteration, and contrast enhancement.

A review of the medical records revealed that all 10 patients exhibited seizure activity associated with neurologic abnormalities, and that five patients had EEG studies supporting the presence of seizure foci. EEG activity generally corresponded to imaging findings in four of these patients. A second neuro-radiologist evaluated these images while blinded to the clinical findings.

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FIG 1. 77-year-old man with acute postictal left hemiparesis and decreased level of consciousness.

A, Initial axial noncontrast CT scan shows right frontoparietal swelling with decreased attenuation (*arrow*). Note 3.5 × 2.5-cm old infarction anteriorly in right frontal lobe (*arrowhead*).

B, Axial noncontrast CT scan obtained 8 days later shows resolution of cerebral swelling. (Note: A and B were obtained with slightly different CT gantry angles.)

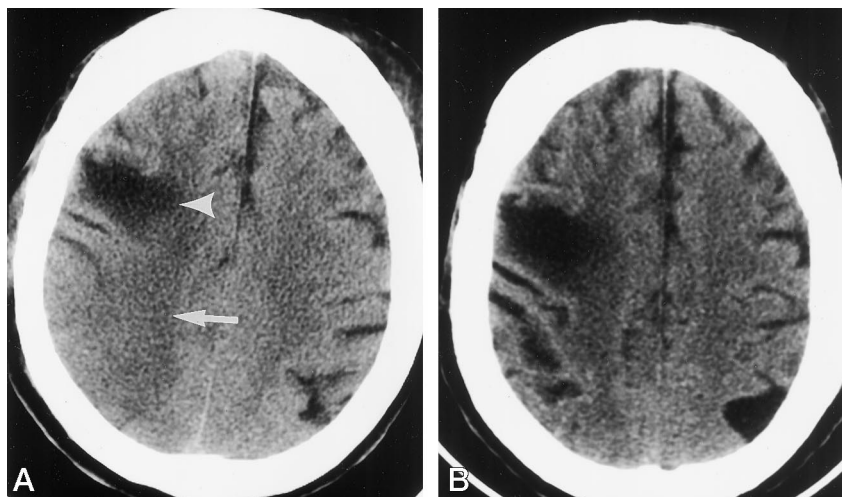
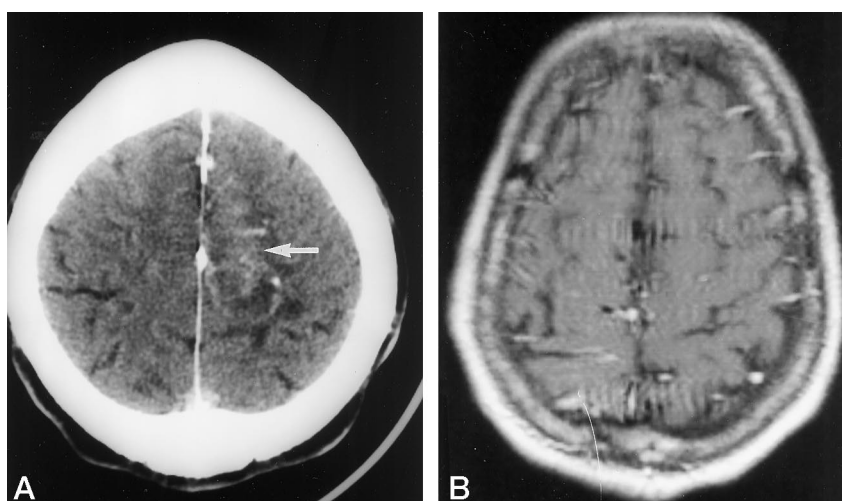


FIG 2. 49-year-old man with acute postictal right arm paresthesia and lethargy resolving by 6 to 7 days.

A, Initial axial contrast-enhanced CT scan shows left frontal swelling and irregular enhancement (*arrow*).

B, Axial contrast-enhanced T1-weighted MR image (540/15/2 [TR/TE/excitations]) obtained 10 weeks later shows resolution of cerebral swelling and enhancement. (Note: A and B were obtained with slightly different scan angles.)



Results

Initial findings included Todd's paralysis with altered mental status (4/10) and persistent altered mental status only (6/10). Medical history included end-stage renal disease treated with chronic hemodialysis (5/10), chronic renal insufficiency without dialysis (3/10), sickle cell anemia (1/10), and mesial temporal sclerosis (1/10). Seizures were chronic in five patients and new in onset in five. Seven patients had generalized tonic-clonic seizures, two had partial seizures with secondary generalization, and one had complex partial seizures. The time to resolve clinical findings was divided into less than 24 hours (7/10) and from 1 to 7 days (3/10).

Initial imaging findings included gyral swelling (10/10), sulcal effacement (2/10), decreased attenuation by CT (8/9), abnormal enhancement (5/5), decreased T1 signal intensity by MR imaging (1/1), and increased T2 signal intensity (1/1) (Figs 1–3). Distribution of these findings was as follows: frontal lobes (9/10, with bilateral involvement in six and unilateral involvement in three), the parietal lobes (4/10), the temporal lobes (2/10), and the occipital lobe (1/10). Lesions conformed to arterial distribution territories

(3/10), with one definite middle cerebral artery (MCA) territory, one possible MCA location, and one possible lesion in the anterior cerebral artery territory. No lesions were solely in arterial watershed territories, but overlap of arterial distribution and watershed territories was the most common pattern (7/10).

Findings on the second imaging studies included complete (5/6) or subtotal (1/6) resolution of initial findings. Underlying lesions, such as hemorrhage or neoplasm, were not discovered in any patient.

Discussion

Seizures have been defined as “a sudden alteration of the CNS resulting from a paroxysmal high frequency or synchronous low frequency, high voltage electrical discharge” (5) and are most commonly due to metabolic derangements that affect neuronal electrical activity, such as hypoglycemia, hyponatremia, uremia, withdrawal from ethanol (in alcoholics), and hypoxic encephalopathy (6). However, space-occupying lesions and other acute or chronic processes can produce seizure activity. The current standard of care in the United States involves imaging of patients with new onset of seizures or a change in previous seizure

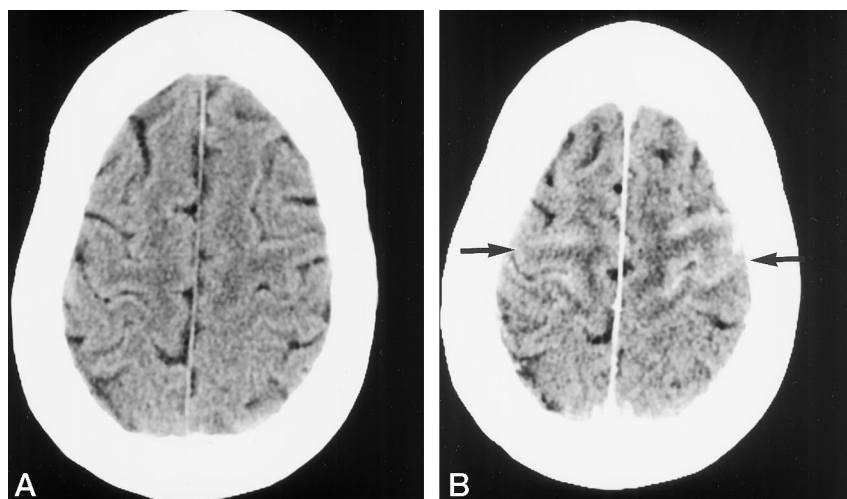


FIG 3. 58-year-old woman with profound acute postictal lethargy and bilateral weakness.

A, Axial noncontrast CT scan shows subtle decreased attenuation of superior frontal and precentral gyri bilaterally.

B, Axial contrast-enhanced CT scan obtained at same time reveals symmetric gyriiform enhancement of the pre- and postcentral gyri and superior frontal gyri (arrows).

pattern. In addition, patients with neurologic deficits or altered mental status suggesting a cerebrovascular accident or transient ischemic attack are generally imaged regardless of the presence of seizure activity. Generalized seizures and partial seizures with secondary generalization entail alterations of consciousness. The classic form of generalized seizure is the tonic-clonic variety, which consists of an aura; a tonic phase lasting 10 to 20 seconds during which there is muscle spasm, glottic closure, and pupillary dilatation; a clonic phase lasting 1 minute or less, including rhythmic salvos of muscle contraction and apnea; and finally a postictal phase of 5 minutes or more that consists of quiet breathing, disorientation, and somnolence. When this postictal altered mental state is significantly prolonged (ie, lasting hours or days) or is accompanied by prolonged focal deficits, such as hemiparesis, hemiplegia, aphasia, or hemisensory deficits, then imaging is clearly indicated.

During generalized seizure activity, the affected regions of the cerebral cortex undergo an increase in metabolic rate and oxygen consumption, while respirations decrease. This leads to decreased PaO_2 and PvO_2 , increased PCO_2 , and increased brain lactate. As a result, cerebral vessels dilate and systolic blood pressure increases to increase cerebral blood flow (CBF) in an attempt to maintain nominal oxygenation (7). Although it is unclear why most patients have a brief postictal period while others suffer from prolonged altered mental status or focal deficit, it may be that these outcomes are dependent on the body's ability to compensate for the decrease in blood oxygen levels with such hemodynamic maneuvers. Posner et al (8) reported that during seizure activity, autoregulation stops in the affected areas of the cortex and resumes in minutes, during which time CBF passively follows systemic blood pressure. Cutler et al (9) and later Lorenzo et al (10) further reported a transient breakdown in the blood-brain barrier during induced seizures; however, it is not clear whether this is part of the overall primary seizure pathophysiological process or a cerebrovascular reactive process. Functional evaluation using single-photon emission

CT (SPECT) in subjects with chronic seizures has shown hypometabolism of seizure foci in the interictal period (11, 12). However, SPECT studies during ictus have shown hyperperfusion with an abrupt "switch" to hypoperfusion within 60 to 90 seconds, with a gradual return to baseline by approximately 10 minutes, in patients without persistent deficits or altered mental status (13–15). It is not clear, though, whether patients with persistent postictal neurologic abnormalities endure a longer duration or greater magnitude of this switch to or from hypoperfusion.

Seizure activity with transient flaccid paralysis was first described by Bravais in 1827 (16) and later by Todd in 1854 (17); in 1890, Jackson added reversible postictal aphasia, stupor, and sensory loss. Transient blindness, psychosis (18), and encephalopathy lasting several days (19) have since been described. In a series of 229 patients with generalized tonic-clonic seizures, Rolak et al in 1992 found 14 patients (6%) with reversible deficits ("Todd's paralysis") (20). In 1975, Yarnell (21) proposed that arteriovenous shunting of blood occurs in response to foci of metabolic acidosis, resulting in further depletion of essential metabolites and accumulation of toxic metabolites. This was thought to be a possible explanation for Todd's paralysis. However, it is not known whether the arteriovenous shunting occurred as a secondary phenomenon immediately after seizures began, as Yarnell indicates, or hours later, or even as a preceding event to seizures. Cerebral arteriovenous shunting has been implicated as a cause of arterial steal in regions of seizure activity, leading to ischemia, acidosis, and blood-brain barrier breakdown (22). Furthermore, Soffer et al (23) reported a detailed pathologic study of a patient who had endured several days of status epilepticus, culminating in death. At autopsy, the cerebral cortex showed massive swelling and characteristic ischemic alterations, including laminar necrosis on the involved side. These authors concluded that seizures can indeed induce brain damage, most likely on an ischemic basis (23).

Seizures have long been known to occur as secondary events in the presence of other lesions, whether

acute or chronic. In 1990, Kilpatrick et al (24) reported a series of 1000 patients with acute strokes, and found 4.4% to have had seizures by 48 hours of their initial events. After dividing these patients by stroke type, these investigators found seizures in 6.5% of 370 patients with ischemic cortical infarctions and in 2% of 189 patients with transient ischemic attacks. In 1992, Bogousslavsky et al (25) described 1280 patients with prior cerebral infarctions who were followed up for as long as 6 years. They found that beyond 2 weeks after infarction, 4% suffered from seizure activity that emanated from the region of the infarction focus.

Reports in the literature have shown reversible cerebral abnormalities acutely after seizure activity. Rumack et al (1) in 1980 described a patient who had decreased attenuation with parenchymal enhancement on CT scans obtained 2 hours after ictus, with normal findings on a follow-up scan. The lesion was located in both the MCA and PCA territories. In 1982, Dillon et al (2) reported two patients with similar findings, also with locations that covered two arterial territories. In 1985, Sammaritano et al (26) described three patients with transient cerebral edema in whom cerebral angiography showed a capillary phase blush with the presence of early-appearing veins. These lesions again overlapped more than one arterial territory. In 1987, Kramer et al (27) described a case of reversible cortical edema with gyral enhancement by CT and edema by MR imaging in the posterior MCA and PCA territories.

Our imaging studies showed evidence of localized acute postictal cortical edema and swelling, abnormality of the blood-brain barrier indicated by abnormal enhancement, reversible clinical and imaging findings, and predominant frontal involvement. Furthermore, these lesions were usually bilateral and frequently did not conform to a watershed or arterial distribution pattern; rather, they overlapped such patterns, as might be expected from seizure-induced edema due to a metabolic cause rather than to an arterial embolus or other cause of early ischemia or infarction.

The differential diagnosis for an initial study that shows acute seizure activity and brain swelling is considerable, and includes infarction, ischemia, venous thrombosis, vasculitis, infection, neoplasm, complicated migraine, and metabolic encephalopathy. However, if the cerebral swelling is bilateral and overlaps arterial distribution patterns while sparing basal ganglia and other deep structures, then arterial thromboembolism and neoplasm become less likely. If the imaging findings resolve on follow-up studies (without specific treatment aside from supportive seizure therapy), then infarction, venous thrombosis, vasculitis, infection, and neoplasm become less likely. Seizure-induced transient cerebral swelling with transient blood-brain barrier breakdown, however, would be a viable consideration, along with transient ischemia and complicated migraine.

Conclusion

Postictal imaging findings, including cerebral swelling, that are transient and correspond to reversible

clinical findings may be directly related to metabolic alterations caused by seizure phenomena.

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