AJNR

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AJNR Am J Neuroradiol 1984, 5 (1) 107-109
http://www.ajnr.org/content/5/1/107.citation

Transient Computed Tomographic Abnormalities after Focal Seizures

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Adult patients with new onset of seizures are often studied by computed tomography (CT) to exclude infarct, neoplasm, vascular malformations, infection, or other pathology as a cause of their convulsion. Previous reports of cerebral angiography and radioisotope scanning in postictal patients with no obvious underlying pathology have demonstrated transient hyperperfusion or blood-brain barrier defects near the site of electroencephalographic (EEG) focus [1–6], especially in patients with epileptogenic foci. Transient CT abnormalities in such patients might also be expected, but have been identified only rarely [7]. We encountered two patients with striking transient CT abnormalities in whom CT scans were obtained shortly after seizures. Both patients were initially believed to have a structural abnormality on the basis of the CT findings and required further invasive diagnostic procedures. We report these potentially misleading CT findings and discuss the possible pathophysiology.

Case Reports

Case 1

A 36-year-old diabetic woman had focal motor seizures involving her right face, arm, and leg that became generalized in the emergency room. She had a long history of intravenous narcotic addiction but no prior seizure or other brain disorder. On initial examination, she was unresponsive with no obvious focal postictal motor deficit. Her vital signs were normal. Initial laboratory studies included a serum sodium of 124 mEq/L and a glucose of 1425 mg/dl. CT (G.E. 8800, 10 mm contiguous scans before and after 42 g of intravenous iodinated contrast material) 1 hr after her last seizure revealed marked gyral enhancement in the left parietooccipital cortex (figs. 1A and 1B). A lumbar puncture was normal. An EEG the same day as CT depicted an epileptogenic focus in the left parietooccipital region. A ^{99m}Tc brain scan the next day demonstrated increased left hemispheric flow and static uptake in the left parietooccipital cortex. A preliminary diagnosis of left posterior cerebral infarction was made on the basis of abnormal CT and isotope brain scans.

Gradual correction of her hyperosmolar state was accompanied by neurologic improvement. Initially, postictal right homonymous hemianopsia, aphasia, dyspraxia, and perseveration were noted. All abnormalities resolved by day 4 with the exception of the persisting visual field deficit. CT (fig. 1C) and isotope brain scans were normal at discharge on day 5.

Case 2

A 20-year-old man was brought to the emergency room after an hour of repetitive focal motor seizures involving his right arm and face. These became generalized. He had no prior convulsive disorder, however, he had a long history of left retroorbital headaches associated with right arm numbness believed to be migrainous in origin. He had complained of headaches before the onset of his seizures. There was no other pertinent history. Postictally, the patient was comatose with normal vital signs. Within several hours he was able to respond to verbal commands. Initial spontaneous ocular nystagmus with deviation of eyes to the right, slight right hyperreflexia, and a right Babinski response also resolved several hours postictus. Mild dysphasia gradually cleared over several days. A lumbar puncture on admission was unrevealing.

An enhanced CT scan (Siemens Somatom II, 10 mm contiguous sections, 42 g iodinated contrast material) within 1 hr of the last seizure showed striking engorgement of the vasculature in the left cerebral hemisphere (fig. 2A). This finding led to a cerebral arteriogram on the same day, which was normal. An EEG that day showed lateralized epileptiform discharges over the left hemisphere. The patient had improved markedly by day 3; however, repeat EEG and CT were essentially unchanged. On day 7, the patient developed a nonspecific viral syndrome associated with small pleural and pericardial effusions. These resolved over the next week. All viral cultures and titres (blood and cerebrospinal fluid samples) were negative, and a repeat lumbar puncture was normal. The final enhanced CT scan on day 14 was normal, 10 days after the last seizure activity (fig. 2B). The patient recovered without sequelae.

Discussion

CT scans are usually unremarkable in the acute evaluation of grand mal and primary generalized seizures unassociated with gross structural lesions [8, 9]. However, it is generally accepted that increased cerebral blood flow occurs with seizures and occasionally may manifest itself as focal abnor-

Received September 7, 1982; accepted after revision December 1, 1982.

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AJNR 5:107–109, January/February 1984 0195–6108/84/0501–0107 $00.00

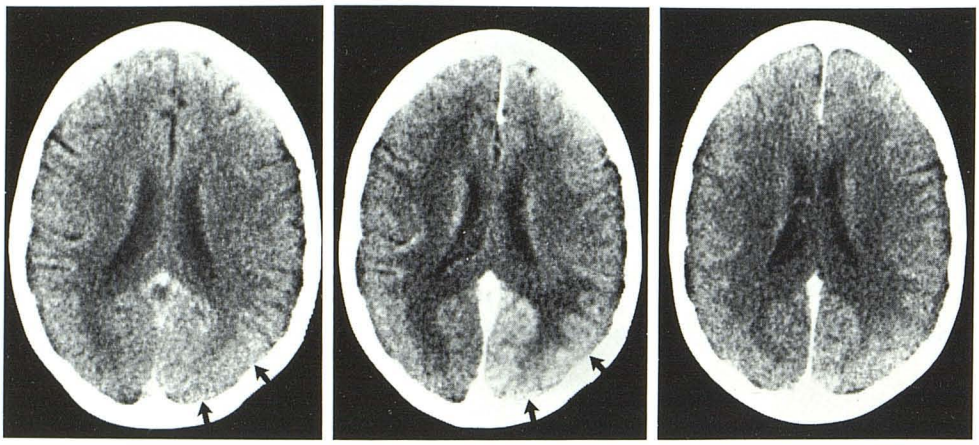

Fig. 1.—Case 1, postictal gyral enhancement. **A**, Unenhanced CT scan 1 hr after last seizure demonstrates equivocally increased attenuation in left parietooccipital cortex (*arrows*). **B**, Marked left parietooccipital gyral enhancement after intravenous contrast administration (*arrows*). Note lack of edema or mass effect surrounding abnormality. **C**, Contrast-enhanced scan 5 days later shows return to normal. There was a residual mild right homonymous hemianopsia.

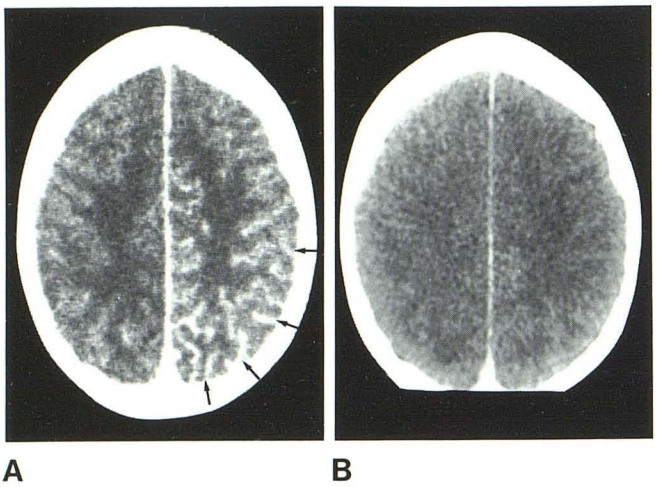

Fig. 2.—Case 2. **A**, Enhanced CT scan 1 hr after last focal left hemispheric seizure demonstrates prominent, asymmetric vasculature suggesting hyperperfusion in left hemispheric sulci (*arrows*). **B**, Enhanced scan 2 weeks later shows return to normal.

malities on angiography [1–6, 10]. Local breakdown of the blood-brain barrier has also been demonstrated after the induction of seizures in an experimental model [11]. Both mechanisms may explain the abnormalities on isotope brain scans in some affected patients. Focal isotope defects are usually transient, appear within 72 hr of the seizure, and occur with an estimated frequency of 11%, especially in patients with repetitious focal seizures [2].

The exact pathophysiology of increased cerebral blood flow after seizure is not known; however, several contributing factors have been proposed. Local vasodilation and increased cerebral blood flow may occur after seizures as a direct result of relatively increased local oxygen consumption and excess lactic acid production by the focus of epileptogenic cells [12–14]. Lassen [15] suggested that local metabolic acidosis around hypoxic or injured neurons may lead to such regional vasodilation and cause the observed "luxury perfusion" angiographic pattern. Such a pattern has been reported occasionally after multiple seizures [4].

With the superior contrast sensitivity of CT in comparison with conventional film-screen angiography, the paucity of reports of postictal CT abnormalities is at first surprising. However, in our relatively large experience of studying patients with CT shortly after focal seizures, the abnormalities reported in the two cases here are unique. Of interest, the CT abnormalities differ in our two patients. The marked gyral enhancement present in case 1 suggests blood-brain barrier breakdown and is not seen in case 2, which depicts asymmetric enhancement of the vasculature. One may speculate, since a mild postictal fixed neurologic deficit resulted in case 1, that some degree of cellular necrosis is reflected by the gyral enhancement. Although we cannot exclude that the primary epileptogenic event in this patient was infarction, we believe this is unlikely in view of the subsequent normal CT examination. It is known that prolonged transient as well as permanent focal postictal neurologic defects may occur, probably on the basis of cellular hypoxia and necrosis [6]. Perhaps gyral enhancement in patients with prolonged seizures may indicate a more serious insult and predict permanent neurologic deficits.

In contrast to the more focal abnormality of case 1, case 2 suggests a regional increase in cerebral blood flow similar to that seen angiographically in some postictal patients. In this case, the patient's entire left hemisphere was hyperemic, unlike the more focal defect previously reported on a contrast-enhanced CT scan [7]. The abnormal postictal enhancing CT pattern in our patient persisted for 3 days, possibly reflecting ongoing subclinical seizure activity as documented by EEG.

These unusual transient postictal CT abnormalities could simulate more serious structural lesions such as vascular malformations, infarction, or local cerebritis. Both our patients were initially suspected of harboring structural lesions on the basis of CT and were subjected to further invasive diagnostic studies. In view of our (admittedly limited) experience, a repeat enhanced CT scan may be warranted before more invasive studies in those patients whose CT scans show acute postictal abnormality.

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