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The reversible posterior cerebral edema syndrome.

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The Reversible Posterior Cerebral Edema Syndrome

In this issue of the *AJNR*, Ito et al report a case in which hypertension, convulsions, visual disturbances, and unconsciousness developed while the patient underwent therapy with cisplatin. T2-weighted MR images showed bilateral increased signal intensity within the white matter of the frontoparietal lobes, which reversed after antiepileptic therapy and control of hypertension. The MR findings of postictal edema are usually, but not always, reversible, and are most frequently related to an antecedent seizure, hypertensive crisis, or both. The findings have been described both on CT and MR (1, 2), and usually occur in ill patients in a variety of clinical settings including certain drug therapies, as well as in transplant recipients undergoing antirejection therapy with cyclosporin. While typically involving the parietal and occipital lobes, an atypical distribution is sometimes seen within the basal ganglia, cerebellum, brain stem, and anterior frontal lobes. Contrast enhancement is not a typical feature; however, some patients have areas of transient enhancement of the overlying cortex and leptomeninges. Recent evidence with diffusion-weighted imaging has shown that these abnormal MR findings do not demonstrate restricted diffusion, and therefore most likely represent vasogenic edema.

In a recent article, Hinchey et al (2) report 15 patients with similar MR findings of cerebral edema and term these changes the "reversible posterior leukoencephalopathy syndrome." Twelve of these 15 acutely ill patients had experienced abrupt increases in blood pressure and 11 had seizures preceding their MR scans; all 15 had imaging findings that were reversible within 2 weeks. Unfortunately, the term "leukoencephalopathy" may be a bit confusing to many neuroradiologists, because there is usually not an accompanying destructive process of the white matter. Perhaps a better term might be "reversible posterior cerebral edema syndrome." Nevertheless, the common denominator of the various cases described in the literature is a recent history of acute seizure or severe uncontrolled hypertension, or both.

Rather than focusing on specific pharmacological agents that may be associated with the hypertensive or epileptic event, it seems more appropriate to focus on the pathophysiology of these interesting, and potentially misleading, MR findings. It has long been recognized that seizures can result in elevations of blood pressure, focal areas of arteriovenous shunting, and loss of autoregulatory capacity of the cerebral vessels. Vasodilatation of cerebral arterioles resulting in regional brain hyperperfusion may result in hyper-

perfusion breakdown of the blood-brain barrier and vasogenic edema may be the sequel (8). Postictal vasogenic edema is best appreciated on T2-weighted FLAIR sequences of the brain, and may also be associated with regions of contrast enhancement reflecting transient defects in the blood-brain barrier (8). While the reversibility of ictus-related vasogenic edema is most characteristic, it should be noted that prolonged seizures, hypertension, or both may result in permanent neurologic deficits and cerebral infarction. The predilection for the more posterior white matter of the parietooccipital lobes is as yet unexplained, but may relate to the observation that the sympathetic innervation of the cerebral vessels, which helps autoregulate cerebral vessels during acute elevations of blood pressure, is relatively reduced in the posterior cerebral arterial circulation (3).

If the history of an acute seizure or uncontrolled hypertension is not obtained, or, as we have seen several times, is an underemphasized aspect of the clinical presentation and not mentioned to the radiologist, an incorrect diagnosis such as gliomatosis cerebri, progressive multifocal leukoencephalopathy, dysmyelinating diseases, or infarction may be advanced on the basis of the MR findings. This may result in invasive biopsies or therapies. In our institution, we have become so familiar with the typical MR pattern that when high signal intensity in the parietal white matter is seen, the first question to the clinician is now "Is there a history of a seizure or hypertension?" In these patients, a follow-up scan in a period of 1 to 2 weeks will most often document the reversibility of the vasogenic edema, and avoid expensive or potentially invasive work-ups for other primary cerebral diseases.

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