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FIGURE 1



A 45-year-old man with renal failure and severe hypertension presents to the ED with visual disturbances. On physical examination, no focal neurologic deficits are identified. CT examination of the head is ordered. Figure 1 is an axial image from that study.

What is your diagnosis?

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CONTINUED

ANSWER

FIGURE 2



Figure 2 demonstrates focal hypoattenuation within the posterior cerebral hemispheres, specifically in the parietal white matter (white arrows). Note the normal white matter (red arrows) on the same image. White matter hypoattenuation is a nonspecific finding indicating either edema or demyelination. There is a wide differential diagnosis for the etiology of this finding, including neoplasm-induced edema, traumatic edema, chronic vascular disease, metabolic disease, and diseases of myelination (eg, multiple sclerosis). However, in a patient with acute hypertension, the diagnosis of posterior reversible encephalopathy syndrome (PRES) should be considered.

PRES, also known as *reversible posterior leukoencephalopathy syndrome* (RPLS), is a clinical/radiographic syndrome that has been associated with a wide variety of medical conditions, including hypertension, preeclampsia/eclampsia, infection, sepsis, shock, autoimmune disease, chemotherapy, and transplantation.^{1,2} Although there have been numerous case reports

FIGURE 3



and series, the incidence and etiology of this condition are unknown. A common theory points to hyperperfusion due to intracranial hypertension in the setting of failed autoregulation. In addition, compromise of the blood-brain barrier resulting from endothelial dysfunction or injury, vasoconstriction, and hypoperfusion has been considered.²

Patients with PRES may present with a variety of neurologic symptoms, including headache, altered mental status, visual disturbances, and seizures. These symptoms are not specific and may also occur with ischemic stroke, intracranial hemorrhage, cerebral venous sinus thrombosis, epilepsy, infection, and toxic or metabolic encephalopathies. PRES is often fully reversible with correction of the underlying abnormality (eg, hypertension), but cases resulting in permanent neurologic deficits and even death have been reported.³

CT is the initial imaging examination of choice in patients with symptoms related to PRES. CT is widely available, takes a short time to perform, and can be used

to rule out intracranial hemorrhage and exclude mass effect. Imaging findings associated with PRES on CT include focal hypoattenuation typically involving the bilateral parietal and occipital lobes. However, lesions may occur elsewhere as well.^{2,4} Hemorrhage has also been reported in a small number of cases.⁵

MRI is useful in documenting the extent of white matter edema and in excluding acute infarct. While both PRES and infarct result in increased signal intensity on T₂-weighted imaging—as seen on this patient's axial FLAIR (fluid-attenuated inversion recovery) image (white arrows, Figure 3)—findings on diffusion-weighted imaging are typically normal in patients with PRES and abnormal in patients with infarct.

The patient in this case was admitted and treated for his hypertension, and his symptoms resolved. Follow-up imaging was normal. **EM**

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