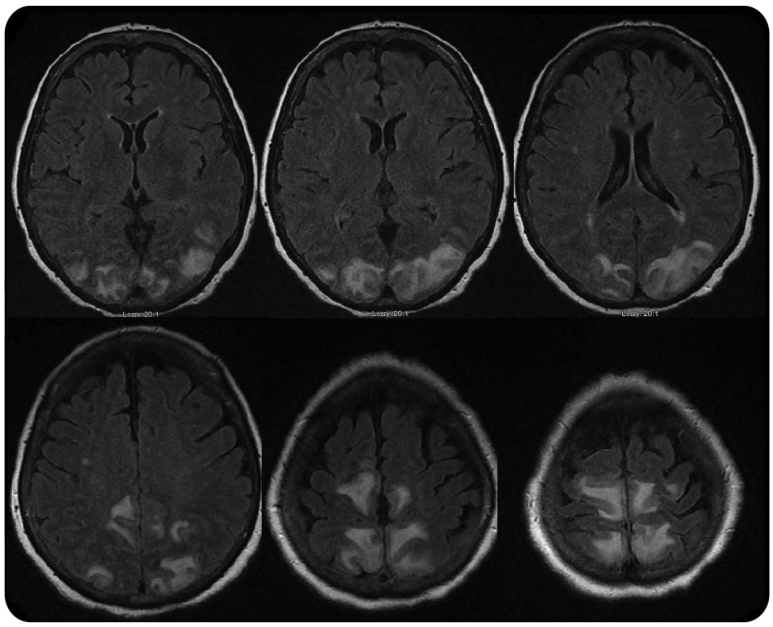
Amaurotic and ophthalmoplegic presentation of Balint syndrome

Figure Posterior predominant white matter abnormalities on brain MRI



Axial fluid-attenuated inversion recovery brain MRI demonstrates patchy hyperintensities in the parietal and occipital subcortical regions bilaterally, typical of the vasogenic edema associated with the posterior reversible encephalopathy syndrome.

A 54-year-old woman developed acute hypertensive encephalopathy associated with acetaminophen-induced liver failure. Examination showed blindness with absence of horizontal and vertical volitional and reflex saccades (video on the *Neurology*® Web site at Neurology.org, first segment). MRI showed biparieto-occipital signal abnormalities consistent with the posterior reversible encephalopathy syndrome (PRES) (figure). Within 24 hours, visual acuity and eye movements improved, but the patient developed ocular apraxia (increased saccadic latency), optic ataxia (impaired visual navigation), and simultanagnosia (inability to recognize more than a single object): the Balint syndrome (video, second segment). This illustrates that a severe expression of oculomotor apraxia can mimic complete ophthalmoplegia[1] and that Balint syndrome may occur at the onset[2] and during recovery from PRES.

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Supplemental data at Neurology.org

Disorders and on the editorial board of *The European Neurological Journal*. S. Allen reports no disclosures relevant to the manuscript. He is a PGY2 Neurology resident at the University of Cincinnati. Go to Neurology.org for full disclosures.

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