Amaurotic and ophthalmoplegic presentation of Balint 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 ophthalmoplegia1 and that Balint syndrome may occur at the onset2 and during recovery from PRES.

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Author contributions: Alberto J. Espay: drafting/revising the manuscript, study concept or design, analysis or interpretation of data, accepts responsibility for conduct of research and final approval, acquisition of data. Scott R. Allen: drafting/revising the manuscript, study concept or design, analysis or interpretation of data, accepts responsibility for conduct of research and final approval, acquisition of data.

Study funding: No targeted funding reported.

Disclosure: A. Espay is supported by the K23 career development award (NIMH, 1K23MH092735); has received grant support from CleveMed/Great Lakes Neurotechnologies, Davis Phinney Foundation, and Michael J. Fox Foundation; has received personal compensation as a consultant/scientific advisory board member for Solvay (now Abbvie), Chelsea Therapeutics, TEVA, Impax, Merz, Solstice Neurosciences, Eli Lilly, and USWorldMeds; and has received honoraria from Novartis, UCB, TEVA, the American Academy of Neurology, and the Movement Disorders Society. He serves as Associate Editor of Movement Disorders and Frontiers in Movement Disorders. Scott R. Allen: has received research funding from the National Institutes of Health (grants K23 MH092735 and RO1 NS076944); has received honoraria from UCB, Teva, and Solstice Neurosciences; has served as consultant to UCB, Teva, and Medtronic; and has served as scientific advisory board member for Solvay (now Abbvie), Chelsea Therapeutics, and Intercranial Therapeutics. He is a co-founder of BioReliance, a company that provides clinical trial services. He is founder and CEO of Neurophye, a public company that develops and markets wearable rehabilitation and monitoring devices. The other authors report no competing financial interests.

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.

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Neurology 2014;82;1844-1845
DOI 10.1212/WNL.0000000000000441

This information is current as of May 19, 2014

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