RESIDENT & FELLOW SECTION

Teaching Video NeuroImages: Posterior Cortical Atrophy Presenting With Balint Syndrome

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Abstract

We present the case of a 68-year-old woman who developed progressive visuospatial deficits in a period of 18 months, leading to the loss of her independence for activities of daily living. After examination, she showed signs of Balint syndrome with optic ataxia, oculomotor apraxia, and simultanagnosia without visual acuity impairment. After brain imaging showing severe bilateral parieto-occipital association cortex atrophy, a diagnosis of posterior cortical atrophy was made according to the 2017 International Consortium’s criteria.

Figure Structural MRI and PET/MRI

(A) Structural MRI T1-3D sequence, showing bilateral parieto-occipital and temporal cortical atrophy (arrows) with enlargement of lateral ventricle's posterior horns (*). (B) 18-FDG PET/MRI showed profoundly impaired posterior cortical metabolism, especially in the parietal and parieto-occipital association cortices (arrows), with relatively preserved metabolism in the primary visual cortex (*).

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A previously healthy 68-year-old woman developed visuospatial deficits, inability to read, and motor dyspraxia for 18 months, leading to the loss of her independence despite spared memory and speech. Several ophthalmology consultations showed no findings. Neurologic examination evidenced oculomotor apraxia, optic ataxia, and simultanagnosia (video 1). Neuroimaging showed cortical atrophy with impaired metabolism on both occipito-parietal association cortices but preserved the primary visual cortex (figure).

Posterior cortical atrophy is a rare variant of Alzheimer disease but can also occur in other neurodegenerative disorders such as corticobasal degeneration. Other causes for Balint syndrome such as PRES or watershed infarcts should be ruled out.

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Disclosure
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Appendix  Authors

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References
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