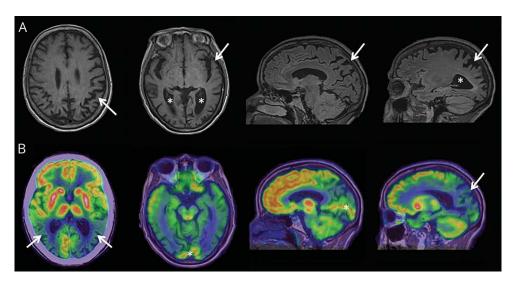
Teaching Video NeuroImages: Posterior Cortical Atrophy Presenting With Balint Syndrome

Roberto Rodríguez-Rivas, MD, Mariana Marcín-Sierra, MD, and Carlos Cardeña-Arredondo, MD Neurology® 2021;96:e1389-e1390. doi:10.1212/WNL.000000000010849

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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 cortexes (arrows), with relatively preserved metabolism in the primary visual cortex (*).

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.

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Go to Neurology.org/N for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

A previously healthy 68-year-old woman developed visuo-spatial 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 cortexes 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.¹ Other causes for Balint syndrome such as PRES or watershed infarcts should be ruled out.²

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Disclosure

The authors report no disclosures relevant to the manuscript. Go to Neurology.org/N for full disclosures.

Appendix Authors

Name	Location	Contribution
Roberto Rodríguez- Rivas, MD	Instituto Nacional de Neurología y Neurocirugía CMDX, Mexico	Designed and conceptualized the study; drafted the manuscript for intellectual content; and edited videos, images, and redacted video vignettes
Mariana Marcin- Sierra, MD	Instituto Nacional de Neurología y Neurocirugía CMDX, Mexico	Data and video collection and analysis
Carlos Cardeña- Arredondo, MD	Instituto Nacional de Neurología y Neurocirugía CMDX, Mexico	Coordinated imaging and molecular analysis

References

- Schott JM, Crutch SJ. Posterior cortical atrophy. Continuum (Minneap Minn) 2019; 25:52–75.
- Galton CJ, Patterson K, Xuereb JH, Hodges JR. Atypical and typical presentations of Alzheimer's disease: a clinical, neuropsychological, neuroimaging and pathological study of 13 cases. Brain 2000;123:484

 –498.



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