Teaching Video NeuroImages: Paroxysmal Dysarthria-Ataxia in Multiple Sclerosis

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Neurology® 2021;96:e2245-e2246. doi:10.1212/WNL.00000000000011498

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Figure MRI of Brain and Spine

T2-Hyperintense right superior cerebellar peduncle lesion (A.a, arrow) with ring enhancement (A.b, arrow). MRI 2 years later reveals new brain (B.a, arrow) and spinal (B.b, arrow) demyelinating lesions.

A 20-year-old man presented with vertigo followed by hourly episodes of dysarthria and incoordination lasting 5–20 seconds (video 1). Neurologic examination was normal between episodes. The only brain/spine MRI lesion was in the right superior cerebellar peduncle (figure, A). CSF analysis revealed elevated white blood cells (6/ μ L [normal, 0–5]) and oligoclonal

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bands (≥ 4). Paroxysmal dysarthria–ataxia associated with CNS demyelination was diagnosed and episodes resolved with oral acetazolamide 250 mg twice daily. Subsequent MRI of the brain and spine (figure, B) confirmed multiple sclerosis. Paroxysmal dysarthria–ataxia arises from ephaptic transmission within midbrain or cerebellar multiple sclerosis lesions and may respond to carbamazepine or acetazolamide.¹ It should be distinguished from genetic or contactin-associated protein-like-2 (CASPR2) immunoglobulin G–associated episodic ataxia.²

Study Funding

No targeted funding reported.

Disclosure

The authors report no disclosures relevant to the manuscript. Go to [Neurology.org/N](https://www.neurology.org/N) for full disclosures.

Appendix Authors

Name	Location	Contribution
Shailee Shah, MD	Mayo Clinic, Rochester, MN	Design and conceptualization of study
Bryan T. Klassen, MD	Mayo Clinic, Rochester, MN	Design and conceptualization of study
Eoin P. Flanagan, MD	Mayo Clinic, Rochester, MN	Design and conceptualization of study, study supervision

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Neurology 2021;96:e2245-e2246 Published Online before print January 5, 2021

DOI 10.1212/WNL.0000000000011498

This information is current as of January 5, 2021

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