Ten days after respiratory infection, a 32-year-old woman presented with headaches, ataxia, and diplopia without encephalopathy. Brain MRI revealed extensive white matter, brainstem, and cerebellar vasogenic edema, without gadolinium enhancement, partially regressive during follow-up (figure, A). CSF revealed transient elevated protein level (1.01 g/L) and hypercellularity (123 neutrophils/mm³). Negative anti-GQ1b but positive anti-GD1a immunoglobulin G led to the diagnosis of Bickerstaff brainstem encephalitis (BBE). Symptoms resolved within 10 days without treatment. ¹⁸FDG-PET showed bilateral temporo-parieto-occipital and cerebellar hypometabolism (figure, B). Neurologists should be aware that diffuse brain hypometabolism or vasogenic edema can be associated with BBE.¹,²

AUTHOR CONTRIBUTIONS
E. Nerrant, C. Fourcade, S. Coulette, C. Lechiche, and E. Thouvenot participated in drafting/revising the manuscript.

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

REFERENCES

From the Department of Neurology (E.N., S.C., E.T.) and the Infectious and Tropical Diseases Unit (C.F., C.L.), Nîmes University Hospital, France.
Figure Brain MRI and PET findings

(A) Initial MRI (T0) reveals fluid-attenuated inversion recovery (FLAIR) hyperintensities of the deep white matter, brainstem, and cerebellum, with increased diffusivity on apparent diffusion coefficient (ADC), progressively regressive at 10 days (D10) and 1 year (M12). (B) Brain 18F-DG-PET performed at 1 month (M1) shows focal temporo-parieto-occipital hypometabolism (arrows) and global cerebellar hypometabolism.
Teaching NeuroImages: Extensive vasogenic edema in Bickerstaff brainstem encephalitis
Elodie Nerrant, Camille Fourcade, Sarah Coulette, et al.
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