The Canadian Journal of Neurological Sciences (2025), **52**, 1016–1017
doi:10.1017/cjn.2025.17

Canadian Journal of Neurological Sciences
Journal Canadien des Sciences Neurologiques

Neuroimaging Highlight

Brainstem and Central Variant PRES in Hypertensive Encephalopathy

Kevin Perera[1,3], Fazeela Mulji[2,3], Rakhbeer Boparai[2,3], Ryan Koo[2,3] and Carlos R. Camara-Lemarroy[1,3]

[1]Department of Clinical Neurosciences, University of Calgary, Calgary, Alberta, Canada, [2]Department of Internal Medicine, University of Calgary, Calgary, Alberta, Canada and [3]Cumming School of Medicine, University of Calgary, Calgary, Alberta, Canada

Keywords: ataxia; brain injury; cerebellum; hypertension

(Received 3 January 2025; date of acceptance 11 January 2025; First Published online 6 February 2025)

A 62-year-old man with no relevant history presented to the emergency department with an occipital headache, and his blood pressure was 258/138. He had evidence of acute kidney injury and elevated troponin. On neurological examination, he was alert, diffusely hyperreflexic, and he had mild bilateral dysmetria and tremor. He had mild gait ataxia but could ambulate independently. MRI of the brain revealed bilateral white matter hyperintensities, characterized by vasogenic edema, involving the brainstem, cerebellum, basal ganglia and bilateral centrum semiovale, suggestive of central variant posterior reversible encephalopathy syndrome (PRES) (Figure 1). Susceptibility weighted imaging (SWI) also revealed multiple microbleeds, mostly affecting the brainstem and cerebellum. There was clear clinic radiological dissociation, with severe abnormalities on imaging but a mild brainstem–cerebellar syndrome. Blood pressure was aggressively controlled, and his symptoms resolved within a few days. Testing revealed fibromuscular dysplasia as a cause for his severe hypertension. He declined further workup or management and asked for a discharge 7 days after admission.

PRES is a Clinico-radiological syndrome characterized by acute onset of neurological symptoms associated with classic MRI findings of white matter vasogenic edema (T1, T2 hyperintense) that most frequently affects brain regions irrigated by the posterior vasculature.[1] Hypertension, uremia, systemic inflammation and chemotherapeutic drugs are common causes. Reversibility is common but not universal, and hemorrhagic lesions can be seen in

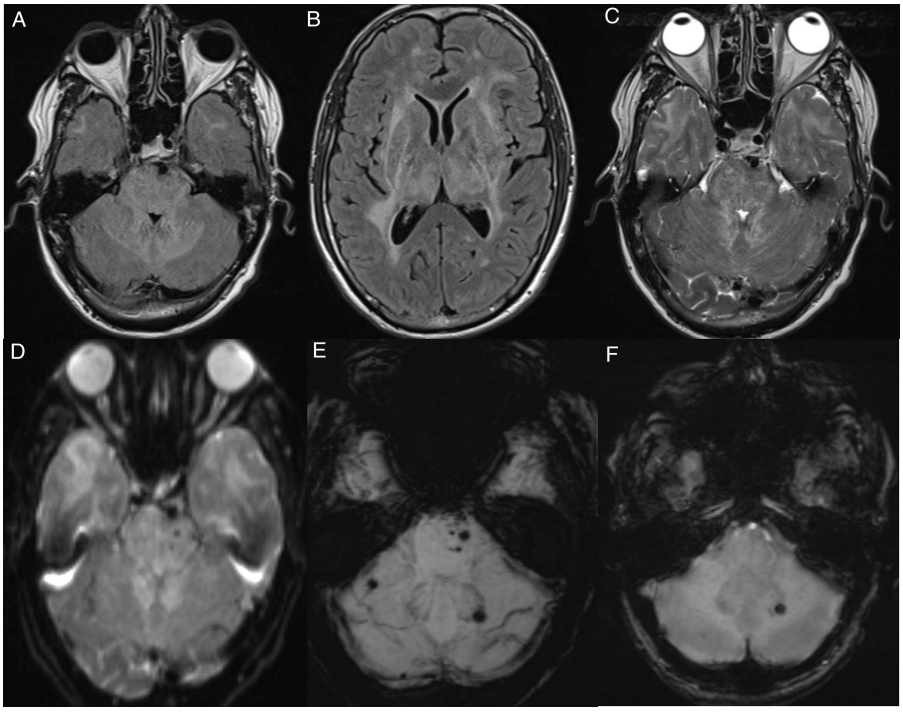


Figure 1. T2 FLAIR (A, B) and T2 (C) images revealed vasogenic edema involving the brainstem, cerebellum and basal ganglia. Apparent Diffusion Coefficient (ADC) (D) revealed shine through hyperintensity, not associated with Diffusion-weighted imaging (DWI) signal change (not shown). Several microbleeds were apparent on susceptibility weighted imaging, particularly in the brainstem and cerebellum (E, F).

Corresponding author: Carlos R. Camara-Lemarroy; Email: Carlos.camara-lemarroy@ahs.ca

Cite this article: Perera K, Mulji F, Boparai R, Koo R, and Camara-Lemarroy CR. (2025) Brainstem and Central Variant PRES in Hypertensive Encephalopathy. *The Canadian Journal of Neurological Sciences* **52**: 1016–1017, https://doi.org/10.1017/cjn.2025.17

© The Author(s), 2025. Published by Cambridge University Press on behalf of Canadian Neurological Sciences Federation.

SWI.[2] The mechanisms are only partially understood, and it is thought that there is cerebrovascular dysregulation in the context of a "two-hit" phenomenon (i.e., hypertension plus inflammation).[1,2] The prognosis is variable, and the mainstay of treatment is the management of the precipitant causes.

In PRES, isolated brainstem involvement or a "central variant," affecting deep gray matter symmetrically, is rare.[3,4] Moreover, there is literature describing hypertensive brainstem encephalopathy (HBE) as a distinct entity.[5,6] However, there are several shared features between brainstem PRES and HBE, and they may simply represent a spectrum of disease. On the other hand, there may be factors that may be used to distinguish a phenotypic variant. First, microbleeds are relatively rare in HBE, while they are frequent in PRES.[7,8] Second, clinico-radiological dissociation seems to be frequently seen in hypertensive encephalopathy and rare in PRES.[5,7] Finally, precipitating factors in PRES are much more varied, and usually, there is more than one factor identified, whereas HBE typically presents with isolated, but severe, elevations in blood pressure.[5,9] As a "clinico-radiological" syndrome, probably representing a spectrum of disease (which may include eclampsia and reversible vasoconstriction syndrome[2,10]), these considerations may not be simply semantic, particularly since there are no specific disease-modifying therapies. More data will be needed to settle these debates.

Author contributions. The manuscript was drafted by Dr Camara-Lemarroy and Dr Perera. Dr Koo, Dr Mulji and Dr Boparai reviewed and suggested revisions for the manuscript and its contents and helped obtain patient consent. All physicians were involved in the patient's care during his stay.

Funding statement. None.

Competing interests. Dr Camara-Lemarroy reports personal fees from EMD SERONO, NOVARTIS and AMGEN, outside the submitted work.

Other authors have no other conflicts to disclose.

References

1. Camara-Lemarroy CR, Gonzalez-Moreno EI, Ortiz-Corona Jde J, et al. Posterior reversible encephalopathy syndrome due to malignant hypercalcemia: physiopathological considerations. *J Clin Endocrinol Metab*. 2014;99:1112–1116.
2. Battal B, Castillo M. Imaging of reversible cerebral vasoconstriction syndrome and posterior reversible encephalopathy syndrome. *Neuroimaging Clin N Am*. 2024;34:129–147.
3. Chen TY, Wu TC, Ko CC, et al. Quantitative magnetic resonance diffusion-weighted imaging evaluation of the supratentorial brain regions in patients diagnosed with brainstem variant of posterior reversible encephalopathy syndrome: a preliminary study. *J Stroke Cerebrovasc Dis*. 2017;26:1560–1568.
4. McKinney AM, Jagadeesan BD, Truwit CL. Central-variant posterior reversible encephalopathy syndrome: brainstem or basal ganglia involvement lacking cortical or subcortical cerebral edema. *AJR Am J Roentgenol*. 2013;201:631–638.
5. Karakis I, Macdonald JA, Stefanidou M, Kase CS. Clinical and radiological features of brainstem variant of hypertensive encephalopathy. *J Vasc Interv Neurol*. 2009;2(2):172–176.
6. Thambisetty M, Biousse V, Newman NJ. Hypertensive brainstem encephalopathy: clinical and radiographic features. *J Neurol Sci*. 2003;208:93–99.
7. McKinney AM, Sarikaya B, Gustafson C, Truwit CL. Detection of microhemorrhage in posterior reversible encephalopathy syndrome using susceptibility-weighted imaging. *AJNR Am J Neuroradiol*. 2012;33:896–903.
8. Yakushiji Y. Cerebral microbleeds: detection, associations and clinical implications. *Front Neurol Neurosci*. 2015;37:78–92.
9. Pascarella A, Quattrone A, Sabatini U, Gambardella A, Labate A. Brainstem posterior reversible encephalopathy syndrome in an asymptomatic patient. *Can J Neurol Sci*. 2020;47:267–269.
10. Camara-Lemarroy CR, Escobedo-Zúñiga N, Villarreal-Garza E, García-Valadez E, Góngora-Rivera F, Villarreal-Velázquez HJ. Posterior reversible leukoencephalopathy syndrome (PRES) associated with severe eclampsia: clinical and biochemical features. *Pregnancy Hypertens*. 2017;7:44–49.

https://doi.org/10.1017/cjn.2025.17 Published online by Cambridge University Press