A 70-year-old woman presented with a 3-week history of progressive left hemichorea-hemiballism. Based on elevated serum glucose (384 mg/dL) and HbA1c (13.2%) plus neuroimaging findings (figure, A and B), she was diagnosed with hyperglycemia-induced hemichorea-hemiballism.¹ She was treated with insulin and risperidone with near resolution after 4 days. Additional neuroimaging (figure, C and D) revealed a developmental venous anomaly (DVA) adjacent to the affected putamen. The DVA was associated with increased cerebral blood flow (E) and increased cerebral blood volume (F) in the affected putamen (arrows).

The right putamen is hyperdense on CT head (A) and hyperintense on T1-weighted MRI brain (B) which is classic for hyperglycemia-induced hemichorea-hemiballism. The characteristic caput medusa of a developmental venous anomaly (arrowheads) is seen on CT head with contrast (C) and gadolinium-enhanced 3T MRI brain (D). CT perfusion demonstrates increased cerebral blood flow (E) and increased cerebral blood volume (F) in the affected putamen (arrows).

A 70-year-old woman presented with a 3-week history of progressive left hemichorea-hemiballism. Based on elevated serum glucose (384 mg/dL) and HbA1c (13.2%) plus neuroimaging findings (figure, A and B), she was diagnosed with hyperglycemia-induced hemichorea-hemiballism.¹ She was treated with insulin and risperidone with near resolution after 4 days. Additional neuroimaging (figure, C and D) revealed a developmental venous anomaly (DVA) adjacent to the affected putamen. The DVA was associated with increased cerebral blood flow (E) and increased cerebral blood volume (F) in the affected putamen (arrows).
that altered hemodynamics within the basal ganglia together with this patient’s metabolic disturbance resulted in the movement disorder: a 2-hit hypothesis.

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