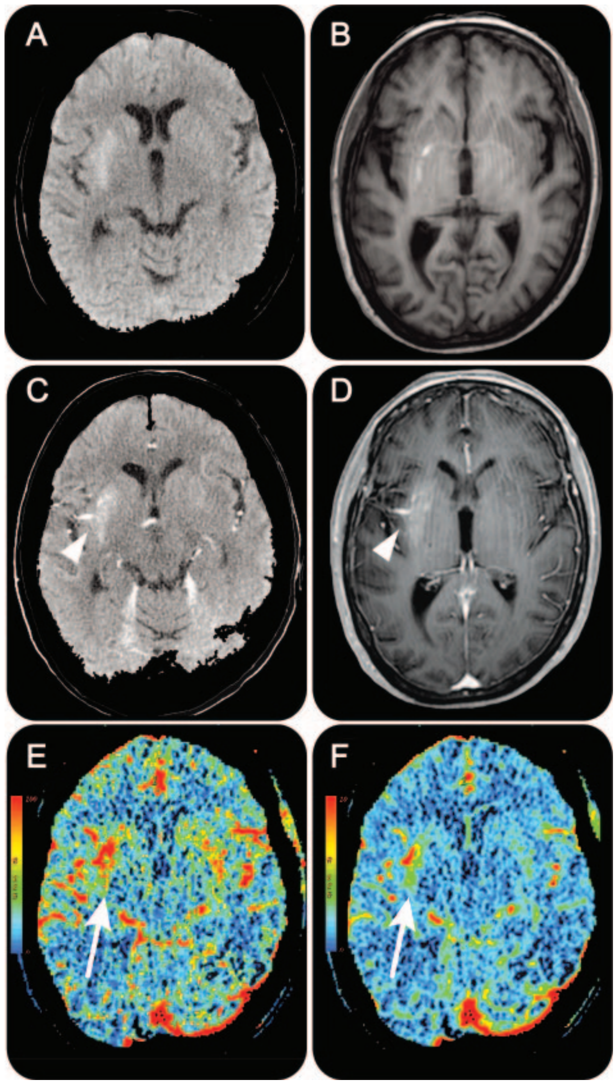
Hemichorea-hemiballism associated with hyperglycemia and a developmental venous anomaly

Figure Neuroimaging



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 and volume[2] (figure, E and F). DVAs are typically asymptomatic. However, we propose 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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