

Editors' Note: In response to "Hemichorea-hemiballism associated with hyperglycemia and a developmental venous anomaly" by Kalia et al., Drs. Civardi and Collini describe their own case of putaminal CT hyperperfusion without the developmental venous anomaly (DVA), resulting in unilateral myoclonus. The authors describe possible explanations for the abnormal CT perfusion (CTP) findings in their own case study.

Megan Alcauskas, MD, and Robert C. Griggs, MD

HEMICHOREA-HEMIBALLISM ASSOCIATED WITH HYPERGLYCEMIA AND A DEVELOPMENTAL VENOUS ANOMALY

Carlo Civardi, Alessandra Collini, Novara, Italy:

Kalia et al.¹ described a case of a hyperglycemia-induced hemichorea-hemiballism with the typical high signal on T1-weighted images on MRI² in the putamen contralateral to the movement. Additional neuroimaging revealed a DVA adjacent to the affected putamen associated with increased cerebral blood flow and volume on CTP.

The authors proposed that altered hemodynamics within the basal ganglia along with a metabolic disturbance resulted in movement disorder. We reported a man with repetitive myoclonus of the left side of the body and contralateral putaminal CT hyperperfusion. Carbamazepine completely controlled myoclonus and, concurrently, CT showed normal perfusion.³ Our patient did not have a DVA and there was no metabolic defect.

As in other hyperkinetic disorders, CT hyperperfusion as observed in these patients^{1,3} may indicate failed autoregulatory mechanisms in the basal ganglia vessels.⁴ These 2 reports showed the same putaminal hyperperfusion, albeit with a different etiology. The perfusional pattern may be related to hyperkinetic status and not to the etiology of the movement disorder. Moreover, this recent report confirms the utility of CTP in the assessment of hyperkinetic disorders.

Author Response: Lorraine V. Kalia, Anthony E. Lang, Richard I. Aviv, Mario Masellis, Toronto: We thank Drs. Civardi and Collini for their thoughtful comments. We agree that there are several potential explanations for the abnormal CTP findings in our

case.¹ As they suggested, one possibility is that the observed increases in cerebral blood flow and cerebral blood volume are a consequence and not a cause of the patient's hemiballism-hemichorea. Another possibility is that this CTP pattern within the putamen was a result of the adjacent DVA. A similar pattern has been previously described with MRI perfusion (MRP) in 4 patients with a DVA.⁵ Follow-up imaging in our patient after complete resolution of her hemiballism-hemichorea may have been useful in differentiating between these 2 possibilities (i.e., resolution of CTP abnormalities with the first possibility vs persistence with the second possibility). However, based on similarities between the MRP and CTP techniques, we anticipate that the latter would be more likely. While the CTP findings in our case¹ and those described by Civardi et al.³ are interesting, further characterization of this type of imaging in hyperkinetic movement disorders is required before we can establish its utility in the assessment of patients with movement disorders.

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DECREASED IRON LEVELS IN THE TEMPORAL CORTEX IN POSTMORTEM HUMAN BRAINS WITH PARKINSON DISEASE

Osamu Kano, Ken Ikeda, Yasuo Iwasaki, Tokyo:

Yu et al.¹ reported that iron levels in the temporal cortex were reduced in patients with Parkinson disease (PD) compared with age-matched controls. They also determined that patients with Alzheimer disease (AD) had no change in iron levels in the temporal cortex. We

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Hemichorea-hemiballism associated with hyperglycemia and a developmental venous anomaly

Carlo Civardi, Lorraine V. Kalia, Alessandra Collini, et al.

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