Subthalamic Lesion on MR Imaging in a Patient with Nonketotic Hyperglycemia-Induced Hemiballism


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SUMMARY: Hemiballism with corresponding striatal T1 hyperintensity on MR imaging has occasionally been reported in patients with nonketotic hyperglycemia. However, the subthalamic nucleus lesion, which is believed to be pathogenetically related to hemiballism, is rarely documented in a living patient with nonketotic hyperglycemia. We describe a patient with nonketotic hyperglycemia-induced hemiballism, whose responsible lesion (ie, the subthalamus) was demonstrated by MR imaging.

Case Report

A 53-year-old woman was admitted for sudden onset of abnormal movement on the left side. This jerky irregular involuntary movement was first noted on the left leg 3 days before admission. Her symptoms worsened during the last 3 days. She had no history of hypertension, diabetes, headache, parkinsonism, or other neurologic diseases. At admission, her initial blood glucose level was 242 mg/dL and her hemoglobin A1c concentration was 11.7% (normal values are 70–110 mg/dL and 4.7–6.4%, respectively). The urine specimen was negative for ketones, indicating nonketotic hyperglycemia. A neurologic examination showed her sensorium to be normal. Deep tendon reflexes were regular, wide-amplitude, and poorly patterned movements.6 Increased viscosity, the presence of myelin-breakdown products, the presence of blood products, or deposition of calcium and other minerals have been suggested as possible explanations as well.7-9

As to the MR signal intensities alone, our imaging findings confirmed results from previous reports. In this case, however, MR imaging revealed the sole involvement of the contralateral subthalamic nucleus (STN) without a putaminal lesion. Although subthalamic involvement has never been reported in hyperglycemia-induced hemiballism, acute hemiballism was reported in a small cavernous angioma or infarction confined to the STN.10,11

The STN serves as the main excitatory input to the medial part of the globus pallidus, which inhibits, by using gamma amino butyric acid, the activity of the ventromedial thalamus. The destruction of the STN causes a decrease of this inhibitory function and increases thalamic excitation of the motor and premotor cortex, resulting in involuntary movement.12,13 This mechanism is supported by findings during deep brain stimulation of the subthalamus in Parkinson disease.13

Possible differential diagnosis in this case includes acute infarction by vascular insult. Unlike T1 hyperintensity due to
nonketotic hyperglycemia, acute infarction of 3 days duration shows bright signal intensity on DWIs and low signal intensity on T1-weighted images. When T1 hyperintensity is noted in acute infarction at 3 days, it should be hemorrhagic transformation and should, therefore, show dark signal intensity on T2*-weighted images. Acute hemorrhage is also unlikely because there is no evidence of magnetic susceptibility artifacts on T2*-weighted images as well as on DWIs with a b-value of 0 s/mm².

In conclusion, unilateral subthalamic involvement can be the sole imaging finding in nonketotic hyperglycemia-induced hemiballism. This case suggests a pathophysiologic explanation for hemiballism with its strategic localization.

References

Fig 1. The T1-weighted axial image (A) shows increased signal intensity (broken arrow), whereas the T2-weighted coronal image clearly demonstrates a high-signal-intensity lesion (arrow) in the right STN.