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


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**CASE REPORT**

H.-J. Kim  
W.J. Moon  
J. Oh  
I.K. Lee  
H.Y. Kim  
S.-H. Han

**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.

**Discussion**

Hyperglycemia-associated chorea and ballismus have been reported predominantly in older patients with diabetes and nonketotic hyperglycemia. The T1-weighted MR images have shown unilateral hyperintensity in the putamen and caudate, but the findings on the T2-weighted MR images have varied. Although a significant association between hyperglycemia and hyperintensity on T1-weighted MR images has been reported, the mechanism underlying the imaging findings has been unknown. Protein dissociation in wallerian degeneration secondary to acute putaminal dysfunction has been proposed as one of the plausible mechanisms behind the imaging findings. 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.

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 STN without a putaminal lesion. Although subthalamic involvement has never been reported in hyperglycemia-induced hemiballism, acute hemiballism was reported in a small caverno us angioma or infarction confined to the STN.

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. This mechanism is supported by findings during deep brain stimulation of the subthalamus in Parkinson disease.

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.