

## Cranial Magnetic Resonance Imaging and Angiography Findings in a Patient With Hyperglycemic Hemichorea-Hemiballism

Junko Nagai, Tsutomu Yamada, Xia Cao, Ayako Fukui, Machiko Tajitsu, Fumiko Yamakawa, Yuko Yamabe, Takashi Murase, Masashi Watanabe, and Fumio Shimada

Departments of Endocrinology and Diabetes (J.N., T.Y., X.C., A.F., M.T., F.Y., Y.Y., T.M.), and Neurology (M.W., F.S.), National Hospital Organization, Nagoya Medical Center, 4-1-1 Sannomaru, Naka-ku, Nagoya, Japan

**A**lthough there are reports of hyperintense lesions in hyperglycemic hemichorea-hemiballism by brain magnetic resonance imaging (MRI) (1,2), such findings by magnetic resonance angiography (MRA) are rare.

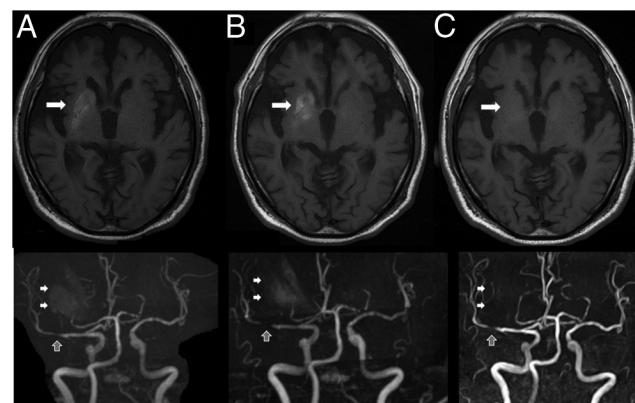
An 82-year-old man presented with a 1-month history of left-sided involuntary movements of his face and limbs. Laboratory data revealed a blood glucose level of 20.8 mmol/L, glycosylated hemoglobin of 11.5%, and serum C-peptide of 2.20 ng/mL. There were no urinary ketones or metabolic acidosis. T1-weighted brain MRI showed a hyperintense lesion in the putamen and caudate nucleus, whereas cranial MRA showed an irregular perivascular hyperintense lesion and an arteriostenosis in the middle cerebral artery (MCA) (Figure 1A, arrows). After a diagnosis of hyperglycemic hemichorea-hemiballism, he was treated with insulin. Upon correction of blood glucose levels, the ballistic movements improved. At the 1-month follow-up visit, the abnormal hyperintense areas had improved slightly (Figure 1B), and they had disappeared completely at the 6-month follow-up visit (Figure 1C). His glycosylated hemoglobin improved to 7.6%.

Hyperglycemic hemichorea-hemiballism is more common among patients with type 2 diabetes, Asians, and the elderly. Possible underlying mechanisms include microhemorrhages due to blood-brain-barrier failure (3), reactive astrocytes associated with ischemia (4), and decreased  $\gamma$ -aminobutyric acid production after dehydration (5), and they present as a high-intensity signal on T1-weighted MRI. We observed high perivascular signal intensity on

MRA, along with stenosis of the MCA at the bifurcation of the lenticulostriate arteries, which supply the putamen and caudate nucleus. This suggests that these lesions have vascular fragility and they are vulnerable to ischemia and metabolic disorders associated with hyperglycemia.

### Acknowledgments

Address all correspondence and requests for reprints to: Tsutomu Yamada, MD, PhD, Department of Endocrinology and Diabetes, National Hospital Organization, Nagoya Medical



**Figure 1.** T1-weighted brain MRI and cranial MRA at presentation (A), 1-month follow-up (B), and 6-month follow-up (C). Abnormal hyperintense areas gradually improved and almost disappeared (white arrows). A stenosis in the right MCA was observed throughout the course (gray arrows).

Center, 4-1-1 Sannomaru, Naka-ku, Nagoya, Japan. E-mail: yamadat@nnh.hosp.go.jp.

Disclosure Summary: The authors have nothing to disclose.

## References

1. Duker AP, Espay AJ. Images in clinical medicine. Hemichorea-hemiballism after diabetic ketoacidosis. *N Engl J Med.* 2010;363:e27.
2. Oh SH, Lee KY, Im JH, Lee MS. Chorea associated with non-ketotic hyperglycemia and hyperintensity basal ganglia lesion on T1-weighted brain MRI study: a meta-analysis of 53 cases including four present cases. *J Neurol Sci.* 2002;200:57–62.
3. Mestre TA, Ferreira JJ, Pimentel J. Putaminal petechial haemorrhage as the cause of non-ketotic hyperglycaemic chorea: a neuro-pathological case correlated with MRI findings. *J Neurol Neurosurg Psychiatry.* 2007;78:549–550.
4. Fujioka M, Taoka T, Matsuo Y, et al. Magnetic resonance imaging shows delayed ischemic striatal neurodegeneration. *Ann Neurol.* 2003;54:732–747.
5. Hashimoto K, Ito Y, Tanahashi H, Hayashi M, Yamakita N, Yasuda K. Hyperglycemic chorea-ballism or acute exacerbation of Huntington's chorea? Huntington's disease unmasks by diabetic ketoacidosis in type 1 diabetes mellitus. *J Clin Endocrinol Metab.* 2012;97:3016–3020.