Hemichorea in a woman with diabetes mellitus

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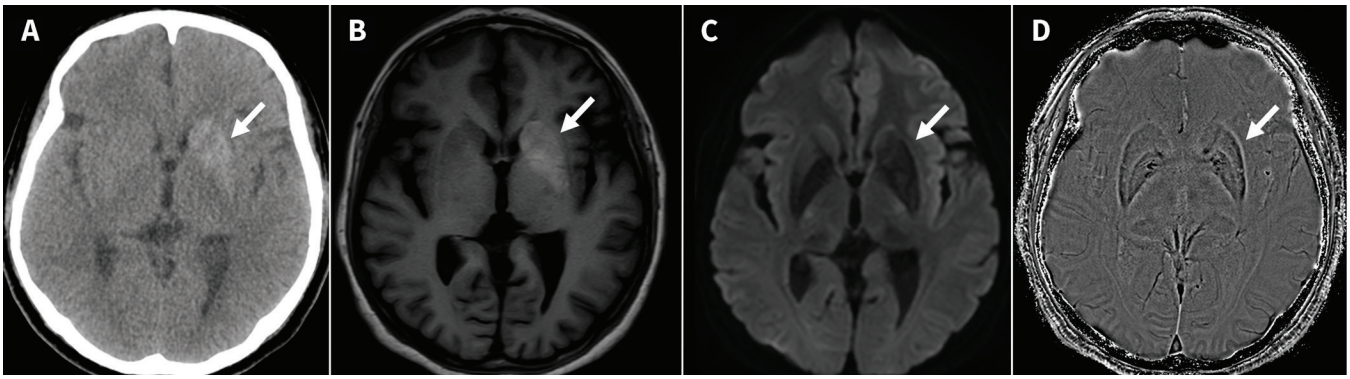

Figure 1: Brain imaging of a 45-year-old woman with right hemichorea. (A) Plain computed tomography showed asymmetric hyperdensity in the left striatum (arrow). (B) T_1-weighted magnetic resonance imaging showed high signalling in the corresponding region (arrow), without restricted diffusion. Diffusion-weighted imaging (C) and susceptibility-weighted imaging (D) confirmed no restricted diffusion (i.e. acute infarct) and no hemorrhage in left striatal lesion (arrow).

A 45-year-old woman with a 10-year history of poorly controlled type 2 diabetes mellitus presented to the emergency department with intermittent involuntary movements of her right face and right limbs for 1 day. She had no family history of movement disorders. Physical examination showed right hemichorea, with a blood pressure of 162/86 mm Hg. She had a high fasting blood glucose level (286 [reference range, 70–110] mg/dL), and hemoglobin A_{1c} level (14.9% [reference range, 4.8%–6.0%]), but normal serum osmolality and no ketosis.

Computed tomography (CT) scans showed hyperdensity in the left striatum (Figure 1A). Magnetic resonance imaging (MRI) showed a T_1-weighted hyperintensity in the corresponding region, without restricted diffusion or hemorrhage (Figures 1B, 1C, 1D). Based on the clinical and radiological findings, we diagnosed diabetic striatopathy. We carefully controlled the patient's blood glucose with insulin therapy and her chorea resolved completely after 3 weeks.

Diabetic striatopathy, also called hyperglycemic nonketotic hemichorea-hemiballism, is seen predominantly in patients with type 2 diabetes that is not well controlled; the prevalence is about 1 in 100 000 people.[1] Blood glucose control is the mainstay of treatment. Diabetic striatopathy is characterized by 1-sided chorea with neuroimaging abnormalities of the contralateral striatum.[1,2] Pathognomonic neuroradiological findings include hyperattenuation in the striatum on CT scan and T_1 hyperintensity on MRI. Four potential pathophysiological mechanisms have been proposed: infarction with astrocytosis, microhemorrhage, mineral deposition and myelin destruction.[3] In our patient, MRI showed no evidence of restricted diffusion and pathological mineral deposition, which suggested that the first 3 theories were unlikely. Protein desiccation from Wallerian degeneration may explain the radiological findings. Diabetic striatopathy may be misdiagnosed as hypertensive hemorrhage and treated inappropriately. The key to differentiating between the 2 conditions is the absence of mass effect and sparing of the internal capsule in diabetic striatopathy.[1,3] Susceptibility weighted imaging with MRI is a useful test to exclude hemorrhage.

References

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