

# Diabetic striatopathy

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## DESCRIPTION

A 70-year-old man presented with involuntary movements of the left lower leg. The patient had recent admission 4 weeks ago with increased drowsiness and raised capillary blood glucose reading of 935 mg/dL with elevated serum osmolality level at 321 mosmol/kg and no ketoacidosis (pH 7.404), resulting in the diagnosis of hyperosmolar hyperglycaemic syndrome (HHS). Intravenous fluid and insulin improved his consciousness. With lower serum C-peptide level and high haemoglobin A1c level (17.6%), the patient was diagnosed with latent autoimmune diabetes in adults with a positive anti-glutamic acid decarboxylase antibody level at high tit  $\geq 2000$  U/mL. Regular insulin was initiated.

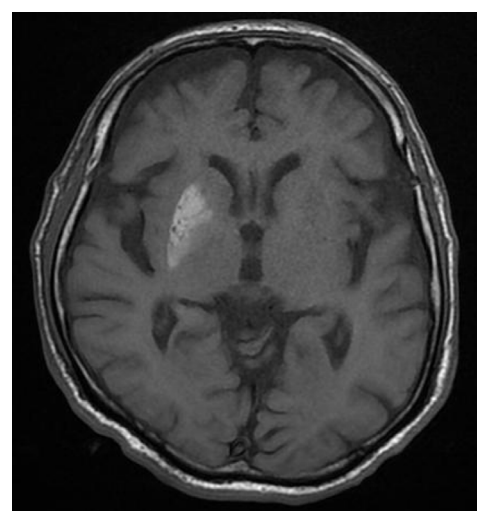
Three days beforehand, involuntary movements of the left lower leg occurred at rest. Other history was unremarkable. He did not take any medications except for insulin. He used tobacco daily for 20 years but quit 30 years before. He did not consume alcohol or use illicit drugs.

On examination, vital signs were stable. His body mass index was 20.8. He was alert and oriented. Neurological examination revealed involuntary movement of the left upper arm and lower limb ([video 1](#)). Strength was 5/5 throughout. Sensation to light touch and pinprick was intact. Reflexes were symmetrical. Cranial nerves were intact. Cerebellar signs were absent. Romberg sign was not performed. Other system examination results were normal.

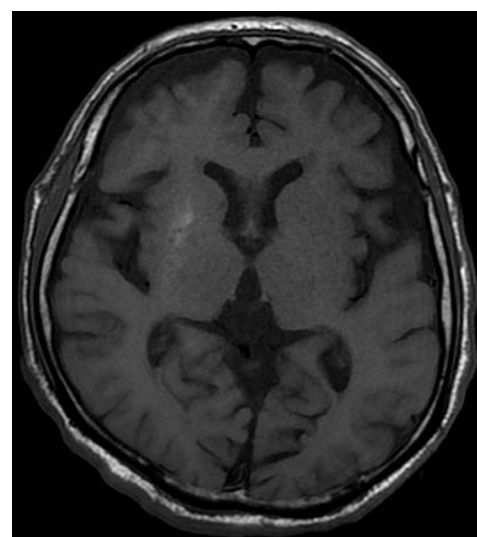
Laboratory studies revealed normal complete blood count, liver and renal function tests, and electrolytes. There was no acidemia (pH 7.36,  $\text{HCO}_3^-$



**Video 1** Involuntary movement of the left upper arm and lower limb was notable.



**Figure 1** A brain MRI scan showed right putamen T1 hyperintensity.



**Figure 2** The abnormal signals of the right putamen on MRI images had almost disappeared.



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27.3 mmol/L). A chest radiograph, ECG and CT of the head were unremarkable. A brain MRI scan showed right putamen T1 hyperintensity (figure 1) with normal T2 intensity and diffusion weighting. Ballism secondary to diabetic striatopathy (DS) was diagnosed. Insulin and sulpiride were commenced and glucose normalised, along with improvement of involuntary movements. Six months later, the abnormal signals on MRI images had almost disappeared (figure 2).

Chorea consists of involuntary, rhythmic, purposeless, jerky movements involving distal limbs. Proximal muscle involvement with larger amplitude is commonly seen in ballism.<sup>1</sup> Basal ganglia and subthalamus dysfunction causes chorea or ballism. Aetiologies of chorea or ballism include cerebrovascular, metabolic, structural, inflammatory, infectious, autoimmune and iatrogenic diseases.<sup>1</sup> DS is characterised by the sudden onset of hemichorea or hemiballism associated with hyperglycaemic and striatal abnormality either by hyperdensity on CT or hyperintensity on T1-weighted MRI.<sup>2</sup> DS usually develops in the elderly population with a female predominance and long-standing, poorly controlled type 2 diabetes mellitus (DM) with an average haemoglobin A1c level of 13.1%. DS infrequently occurs in type 1 DM<sup>3</sup> and even after correction of hyperglycaemic.<sup>4</sup> However, it can be the first manifestation of DM in 17% of patients.<sup>1</sup> The precise pathogenic mechanism of DS is unclear in non-ketotic hyperglycaemic status.<sup>5</sup> However, it is hypothesised that the depletion of gamma-aminobutyric acid (GABA) due to the upregulation of an alternative anaerobic pathway in the Krebs cycle leads to the disinhibition of the subthalamus and basal ganglia associated with hyperkinetic movement.<sup>1</sup>

CT or MRI can detect the striatal abnormality of DS: T1-weighted MRI demonstrates hyperintensity in the putamen,

caudate nucleus and globus pallidus,<sup>6–8</sup> which presumably result from petechial haemorrhage, mineral deposition, myelin destruction or infarction with astrocytosis.<sup>1</sup>

Management of DS includes correction of hyperglycaemic and symptom control. Chorea may improve by glucose control in 25% of patients, while the majority may need treatment including GABA-receptor agonists, selective serotonin reuptake inhibitors and dopamine-depleting agents.<sup>9</sup>

New onset of ballism or chorea in a diabetic patient following HHS should alert the clinician about DS, which can be confirmed by MRI.

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#### Learning points

- Diabetic striatopathy is characterised by the sudden onset of hemichorea or hemiballism associated with hyperglycaemic and striatal abnormality either by hyperdensity on CT or hyperintensity on T1-weighted MRI.
- Chorea may improve by glucose control in 25% of patients, while the majority may need treatment including GABA-receptor agonists, selective serotonin reuptake inhibitors and dopamine-depleting agents.

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