CASE REPORT

45. Diabetic Striatopathy: A Forgotten Diagnosis in Clinical Neurology

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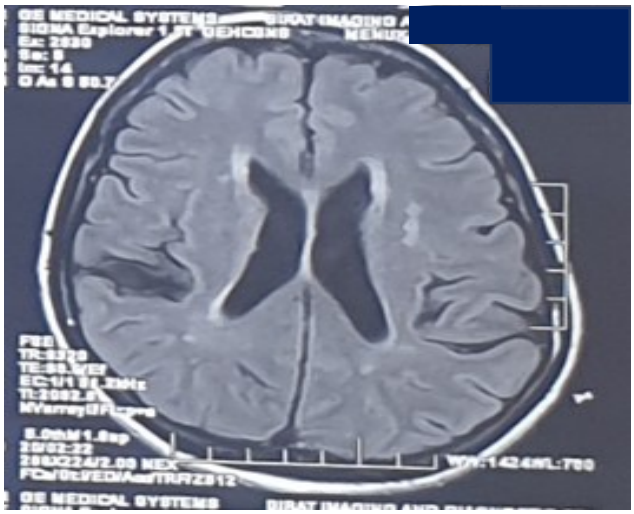
https://www.youtube.com/watch?v=4rJ3DHWeKRs&list=PLhqNq3xJClbafO0Y5bvBcgMmXpgzJxd44&index=6&t=4713s

Background: Diabetic striatopathy primarily affects individuals with type 2 DM, often controlled, and is more common in females from Asian regions; cases have been reported across diverse populations.[1,2] The incidence is approximately 1/100,000.[3] Biopsy of the striatum shows vasculopathy with reactive gliosis. Diabetic striatopathy, a rare complication of Diabetes Mellitus (DM), presents a clinical challenge due to its variable manifestations. It is primarily characterized by abnormal involuntary movements, such as chorea or ballismus, which may affect limbs and, as in our case, the oromandibular region.[4] This atypical presentation highlights the need to include diabetic striatopathy in the differential diagnosis of unexplained movements in patients with diabetes.

The Case A 73-year-old man presented to the emergency department with a 15-day history of involuntary movements involving his left leg and oromandibular region. The symptoms began with mild pain, followed by irregular jaw movements and later choreiform movements of the left leg. He had recently been diagnosed with type 2 diabetes and was on metformin, glyburide, and sitagliptin, with no family history of movement disorders, prior head or neck injuries, or fever. On examination, he was alert, oriented, and hemodynamically stable, with no focal neurological deficits. Laboratory evaluation revealed fasting glucose of 132 mg%, postprandial 148 mg%, HbA1c 7.3%, mild anemia, and elevated thyroid function (0.3 µIU/mL); renal and liver function tests were normal. Initial suspicion of right basal ganglia or subthalamic stroke was revised after MRI revealed right putaminal hyperintensity with preservation of the internal capsule (Figure 1). Endocrinology consultation and anti-chorea therapy with tetrabenazine and clonazepam led to complete resolution, and the patient remained well on follow-up while continuing oral hypoglycemic therapy.

Conclusions: The pathophysiology remains incompletely understood. Evidence suggests a link between hyperglycemia and vascular changes in the striatum, with biopsy showing vasculopathy and reactive gliosis.[1,5] These hyperglycemia-induced microangiopathic changes parallel those seen in diabetic retinopathy.[6,7] Further research is required to elucidate the molecular mechanisms connecting striatal vasculopathy to abnormal movements. Diagnosis relies on hyperglycemia history, chorea or ballismus, and characteristic imaging findings, such as striatal hyperdensity on CT or T1 hyperintensity on MRI, with preservation of the internal capsule distinguishing it from stroke.[8] Management focuses on glycemic control and symptomatic treatment with anti-chorea agents like tetrabenazine and clonazepam, which resulted in resolution in our patient. Prognosis is generally favorable, with clinical and radiologic improvement after controlling hyperglycemia.[9] Despite increasing recognition, research gaps persist regarding pathophysiology, clinical variability, and optimal management strategies.[10] Documenting and sharing cases, particularly from resource-limited regions, will enhance understanding and guide clinical care.

Figure 1. Axial Brain MRI (FLAIR Sequence): Periventricular White Matter Lesions.



Legend: MRI imaging findings showing hyperintensities on right putamen with characteristic sparing of the internal capsule.

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