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DIABETIC STRIATOPATHY PRESENTING AS LEFT HEMICHOREA IN A WOMAN WITH POORLY CONTROLLED TYPE 2 DIABETES MELLITUS: A CASE REPORT

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ABSTRACT:
Diabetic striatopathy (DS) is an uncommon neurological complication of diabetes mellitus characterized by acute or subacute involuntary movements, most commonly hemichorea or hemiballismus, associated with hyperglycemia and characteristic abnormalities involving the striatum on neuroimaging. The putamen and caudate nucleus are frequently affected. Although the condition is classically associated with non-ketotic hyperglycemia, its clinical and radiological presentations are variable, and diagnosis requires correlation between clinical findings, biochemical investigations, and neuroimaging.

INTRODUCTION

Characteristic radiological findings include striatal hyperdensity on computed tomography and T1-weighted hyperintensity on magnetic resonance imaging (MRI). However, imaging appearances vary, and the absence of a typical finding on an individual sequence does not necessarily exclude the diagnosis. Proposed pathophysiological mechanisms include metabolic neuronal dysfunction, altered cerebral perfusion, microvascular injury, and disruption of neurotransmitter pathways within the basal ganglia.

Early recognition is important because appropriate glycemic management, together with symptomatic treatment when indicated, is frequently associated with clinical improvement. We report a case of a 38-year-old woman with poorly controlled type 2 diabetes mellitus who presented with left-sided involuntary movements and characteristic right striatal MRI abnormalities, followed by complete clinical resolution after treatment.

Patient Information

A 38-year-old woman with a history of type 2 diabetes mellitus for approximately 10 years presented with involuntary movements involving the left upper and lower limbs for approximately three weeks. She had discontinued her oral hypoglycemic medications for approximately six months before presentation.

The movements occurred throughout wakefulness, including at rest, and were absent during sleep. The clinical records described the presentation as left hemichorea.

The history of longstanding diabetes mellitus, prolonged interruption of antidiabetic treatment, and subsequent evidence of poor glycemic control raised suspicion of a hyperglycemia-associated movement disorder.

Other relevant medical history, family history, medication exposure apart from the documented diabetes treatment, and details of associated neurological symptoms were not available in the supplied records.

Clinical Findings

The patient presented with involuntary movements involving the left upper and lower limbs. These movements were present during wakefulness, including at rest, and absent during sleep.

Neurological examination revealed normal cranial nerve function. Motor examination demonstrated normal muscle tone and muscle power of 5/5 in all four limbs. Deep tendon reflexes were normal, and bilateral plantar responses were flexor. Sensory examination was normal in all modalities.

The principal neurological abnormality was the involuntary movement disorder involving the left upper and lower limbs, documented clinically as hemichorea.

The patient had a 10-year history of type 2 diabetes mellitus and had discontinued oral hypoglycemic treatment for approximately six months. Laboratory investigations demonstrated markedly elevated glycated hemoglobin, indicating chronic poor glycemic control.

Diagnostic Assessment

Laboratory Investigations

The relevant available laboratory findings are summarized below.

Investigation	Result
Glycated hemoglobin (HbA1c)	11.66%
Postprandial blood glucose (PPBS)	251 mg/dL
Fasting blood glucose (FBS)	93 mg/dL
Urine glucose	3+
Urine ketones	Negative
Serum sodium	141.8 mmol/L
Serum potassium	3.81 mmol/L
Serum calcium	9.6 mg/dL
Serum magnesium	2.1 mg/dL
Serum creatinine	0.79 mg/dL
Hemoglobin	12.3 g/dL
Total leukocyte count	10.91 × 10^9/L
Platelet count	379 × 10^9/L

The HbA1c of 11.66% indicated substantially elevated average glycemia over the preceding months. The PPBS of 251 mg/dL and glycosuria of 3+ further supported inadequate glycemic control.

Urine ketones were negative. Arterial blood gas analysis demonstrated a pH of 7.34, pCO_2 of 25 mmHg, and bicarbonate concentration of 13.5 mmol/L, suggesting metabolic acidosis with possible concurrent respiratory alkalosis. The blood gas analyzer also reported sodium of 105 mmol/L, potassium of 3.0 mmol/L, and ionized calcium of 0.46 mmol/L. However, repeat renal function tests with serum electrolytes were reported to be normal. The cause of the discrepancy between the blood gas analyzer measurements and repeat laboratory results could not be established from the available records. Serum ketones, blood β-hydroxybutyrate, and serum osmolality were not available for further metabolic characterization.

The documented FBS of 93 mg/dL represents a single measurement and does not negate the evidence of chronic poor glycemic control provided by the elevated HbA1c and other elevated glucose readings.

Neuroimaging

MRI of the brain performed on 5 September 2026, during the same clinical episode as the involuntary movements, demonstrated T1 and T2-FLAIR hyperintensities involving the right caudate nucleus and lentiform nucleus. The radiological impression was suggestive of asymmetric non-ketotic hyperglycemic hemichorea.

A focus of diffusion-weighted imaging (DWI) hyperintensity was noted in the right basal ganglia without a corresponding apparent diffusion coefficient (ADC) abnormality. This finding requires interpretation alongside the complete MRI sequences and clinical context rather than being considered definitive evidence of acute infarction.

Additional findings included a chronic infarct with gliosis involving the right parietal cortical, subcortical, and deep white matter, extending into the parasagittal right centrum semiovale. Multiple punctate T2-FLAIR hyperintensities were present in the bilateral frontoparietal periventricular and subcortical white matter, consistent with mild chronic small-vessel ischemic changes, graded Fazekas I. A small left parietal subcortical GRE/SWAN hypointensity was reported, suggesting a microbleed or calcification.

The right-sided striatal abnormalities were anatomically concordant with the left-sided involuntary movements and supported the diagnosis of diabetic striatopathy. The chronic right parietal infarct was an additional cerebrovascular finding that informed the decision to prescribe aspirin for secondary stroke prevention.

Final Clinical Diagnosis

Diabetic striatopathy presenting as left hemichorea in the setting of poorly controlled type 2 diabetes mellitus, with right caudate and lentiform nucleus signal abnormalities on MRI, and an additional chronic right parietal infarct.

Diagnostic Reasoning

The diagnosis was based on the combination of a characteristic movement disorder, evidence of chronic poor glycemic control, and striatal MRI abnormalities corresponding to the side opposite the involuntary movements.

The patient had a 10-year history of type 2 diabetes mellitus and had discontinued oral hypoglycemic medication for approximately six months. Her HbA1c was 11.66%, and PPBS was 251 mg/dL. The presence of left-sided involuntary movements, together with right caudate and lentiform nucleus signal abnormalities, was strongly suggestive of diabetic striatopathy.

The principal differential diagnoses included the following.

1. Ischemic or hemorrhagic basal ganglia lesions: Vascular lesions involving the basal ganglia can produce contralateral involuntary movements. The MRI demonstrated DWI hyperintensity without a corresponding ADC abnormality, as well as a separate chronic right parietal infarct. The overall clinical-radiological pattern favored diabetic striatopathy.

However, review of the complete MRI study and formal radiological report is important to exclude a concurrent vascular lesion.
2. Other metabolic or toxic movement disorders: Electrolyte abnormalities and medication-related causes may produce abnormal movements. The documented sodium, potassium, calcium, magnesium, and creatinine values did not demonstrate a major abnormality sufficient to explain the presentation. A complete medication review and additional metabolic investigations were not available.
3. Other structural basal ganglia disorders: Structural lesions can produce hemichorea. However, the association with prolonged poor glycemic control and the characteristic striatal MRI abnormalities favored diabetic striatopathy.
4. Primary movement disorders: Primary choreiform disorders are less likely when marked chronic hyperglycemia and anatomically corresponding striatal imaging abnormalities are present. Nevertheless, a complete neurological history and examination are essential when evaluating new-onset chorea.
Overall, the clinical presentation, biochemical evidence of poor glycemic control, and neuroimaging findings supported the diagnosis of diabetic striatopathy.

Therapeutic Intervention

Treatment consisted of glycemic management, symptomatic therapy for the involuntary movements, and antiplatelet therapy for secondary stroke prevention in view of the chronic infarct identified on MRI.
The documented treatment regimen was as follows:

Medication	Dose and frequency	Duration or indication
Haloperidol	5 mg orally, 1-0-0	3 days
Clonazepam	0.5 mg orally, 1-0-1	3 days
Tetrabenazine	12.5 mg orally, 1-0-1	3 days
Aspirin	75 mg orally, 0-1-0	Secondary stroke prevention;
Metformin	500 mg orally, 1-0-1	Glycemic control;
Basugine insulin	14 units at night, 0-0-14	Glycemic control;

Haloperidol, clonazepam, and tetrabenazine were administered for short-term symptomatic management of the involuntary movements. Aspirin 75 mg once daily was prescribed for secondary stroke prevention in view of the chronic right parietal infarct identified on MRI. Metformin and nighttime insulin were used to improve glycemic control.
The patient's fasting, postprandial, and capillary blood glucose values subsequently came under control. The involuntary movements resolved completely within three to four days of treatment.
Because glycemic management and symptomatic medications were administered during the same period, the relative contribution of each intervention to clinical improvement cannot be determined from this case alone.

Follow-up and Outcome

The patient demonstrated complete cessation of the involuntary movements within three to four days after initiation of treatment and improvement in glycemic control.
At the two-week follow-up, she had no residual movement disorder and reported no recurrence of the involuntary movements. The neurological outcome at this early follow-up was favorable.
Repeat MRI, longer-term neurological follow-up, and longer-term glycemic outcome data were not available. Therefore, the persistence or resolution of the striatal imaging abnormalities and the risk of later recurrence could not be assessed.

DISCUSSION

Diabetic striatopathy is an uncommon but important cause of acute or subacute choreiform movements in individuals with diabetes mellitus. It is generally associated with severe or prolonged hyperglycemia and characteristic abnormalities of the basal ganglia on neuroimaging. The term encompasses a clinical-radiological syndrome in which hemichorea or hemiballismus occurs alongside striatal abnormalities, most frequently involving the putamen and caudate nucleus.

Pathophysiology

The precise pathophysiological mechanisms underlying diabetic striatopathy remain incompletely understood. Proposed mechanisms include metabolic neuronal dysfunction, altered cerebral blood flow, ischemic injury, blood-brain barrier disruption, and changes in neurotransmitter pathways within the basal ganglia. Microvascular disease and abnormalities in regional glucose metabolism may also contribute.
The variability of imaging findings suggests that diabetic striatopathy may represent a spectrum of metabolic and structural changes rather than a single uniform pathological process. Histopathological descriptions in the literature include reactive astrocytosis, neuronal injury, and changes resembling ischemic or hemorrhagic injury, although the underlying pathology is not identical in every patient.

Clinical Presentation

Hemichorea and hemiballismus are the most commonly reported manifestations. Chorea consists of involuntary, irregular, non-rhythmic movements that may flow unpredictably between body regions, whereas ballismus refers to larger-amplitude, flinging movements, usually involving proximal limbs. The two may coexist and are sometimes described together as hemichorea-hemiballismus.

In the present case, the involuntary movements involved the left upper and lower limbs and were documented as hemichorea. They occurred during wakefulness, including at rest, and were absent during sleep. The symptom duration of approximately three weeks before presentation is compatible with a subacute movement disorder.

The history of discontinuation of oral hypoglycemic medication for six months and an HbA1c of 11.66% support prolonged inadequate glycemic control. A single fasting glucose measurement may be normal or near-normal despite markedly elevated HbA1c and intermittent hyperglycemia. Accordingly, evaluation should incorporate the complete glycemic history rather than relying on one glucose measurement.

Neurological Examination

The neurological examination demonstrated normal cranial nerve function, normal muscle tone, full power of 5/5 in all four limbs, normal deep tendon reflexes, bilateral flexor plantar responses, and normal sensation in all modalities. The principal neurological finding was the unilateral involuntary movement disorder.

These findings document preserved motor power, tone, reflexes, cranial nerve function, and sensation despite the presence of choreiform movements. A normal examination in these domains does not independently exclude a basal ganglia disorder or a concurrent vascular lesion.

Neuroimaging Features

The characteristic radiological findings of diabetic striatopathy include striatal hyperdensity on non-contrast computed tomography and T1-weighted hyperintensity on MRI. The putamen is commonly involved, with variable extension into the caudate nucleus and globus pallidus. Imaging abnormalities are frequently contralateral to the affected limbs.

In this patient, MRI demonstrated right caudate and lentiform nucleus hyperintensities, corresponding to left-sided involuntary movements. This anatomical concordance, together with chronic poor glycemic control, strongly supported diabetic striatopathy.

The reported DWI hyperintensity without corresponding ADC abnormality requires cautious interpretation. Diffusion-weighted signal alone is insufficient to establish acute ischemia; the ADC map, other MRI sequences, clinical presentation, and radiological interpretation must be considered together. The chronic right parietal infarct and mild small-vessel ischemic changes represent additional cerebrovascular findings but do not, by themselves, explain the complete clinical-radiological pattern as well as diabetic striatopathy does.

Differential Diagnosis

The differential diagnosis of acute or subacute hemichorea includes ischemic stroke, intracranial hemorrhage, metabolic disturbances, medication-induced dyskinesia, autoimmune or infectious disorders, neoplastic lesions, and primary movement disorders. In patients with diabetes, new neurological symptoms should not automatically be attributed to hyperglycemia, particularly when focal imaging abnormalities or vascular risk factors are present.

In this case, the laterality of the striatal findings, the absence of a major documented electrolyte disturbance, the elevated HbA1c, and the clinical improvement after treatment supported the diagnosis. Nevertheless, the chronic infarct and DWI finding emphasize the importance of careful review of the complete neuroimaging study.

Management and Prognosis

The cornerstone of treatment is correction of hyperglycemia and restoration of appropriate diabetes management. Symptomatic medications may be considered when the movements are severe, disabling, or persistent. Medications used in published cases include dopamine receptor antagonists and dopamine-depleting agents; selection depends on symptom severity, comorbidities, adverse-effect risk, and clinical assessment.

In this case, metformin and nighttime insulin were used for glycemic management, while haloperidol, tetrabenazine, and clonazepam were administered for short-term symptomatic control. Aspirin was prescribed for secondary stroke prevention in view of the chronic right parietal infarct. The movements resolved within three to four days, and no recurrence was reported at two weeks.

This outcome is consistent with the favorable short-term response described in many published cases, although recovery time varies and some patients experience persistent or recurrent symptoms. The contributions of glycemic correction and symptomatic treatment cannot be separated in this case. Furthermore, no follow-up MRI was available to establish whether the radiological abnormalities resolved in parallel with the clinical symptoms.

Clinical Significance

This case highlights the importance of considering diabetic striatopathy in patients with diabetes who present with new-onset unilateral involuntary movements. Recognition of the characteristic clinical and MRI pattern can guide appropriate investigations and treatment. It also underscores the importance of medication adherence, sustained glycemic management, and appropriate evaluation of additional cerebrovascular findings.

Strengths and Limitations

Strengths

This case illustrates a clinically and radiologically concordant presentation of diabetic striatopathy. The patient had a documented history of prolonged interruption of diabetes treatment, an elevated HbA1c, contralateral striatal MRI abnormalities, and complete early resolution of the involuntary movements. The neurological examination was documented, showing normal cranial nerve function, normal tone and power, normal reflexes, bilateral flexor plantar responses, and intact sensation. The two-week follow-up provides additional evidence of a favorable short-term clinical outcome.

LIMITATIONS

This report has certain limitations. Detailed characterization of the movement phenomenology beyond the clinical documentation of hemichorea was unavailable. Serum osmolality and blood β-hydroxybutyrate measurements were not available for comprehensive metabolic characterization. Although the patient received both glycemic management and symptomatic treatment, their individual contributions to clinical recovery could not be determined. Repeat MRI and longer-term follow-up were unavailable to assess radiological resolution and the possibility of recurrence. Finally, as a single case report, these findings cannot establish causal relationships or be generalized to all patients with diabetic striatopathy.

CONCLUSION

Diabetic striatopathy should be considered in patients with diabetes mellitus who present with acute or subacute hemichorea, particularly when glycemic control has been poor and MRI demonstrates contralateral striatal abnormalities. In this 38-year-old woman, left-sided involuntary movements, an HbA1c of 11.66%, and right caudate and lentiform nucleus signal abnormalities supported the diagnosis. The neurological examination demonstrated preserved cranial nerve function, normal tone and power, normal reflexes, flexor plantar responses, and intact sensation. Glycemic management with short-term symptomatic therapy was followed by complete cessation of movements within three to four days, with no recurrence at two weeks. Aspirin was prescribed for secondary stroke prevention in view of the chronic infarct on MRI. Early recognition and appropriate management may lead to favorable clinical recovery.

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Conflict of Interest

The authors declare no conflict of interest.

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