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**Case Report**

# Nonketotic hyperglycaemia associated hemichorea-hemiballismus in an elderly woman with type 2 diabetes mellitus

**S. Saurabh Biswal*, Ashik Mohammed, K.C. Misra, Guruprasad H.**

Department of Critical Care Medicine, CARE Hospital, Banjara Hills, Hyderabad, Telangana, India

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***Correspondence:**
Dr. S. Saurabh Biswal,
E-mail: biswal.saurabh@gmail.com



## ABSTRACT

Hyperglycaemia-induced chorea, also referred to as Diabetic Striatopathy (DS), is a rare but clinically significant neurological complication of uncontrolled diabetes mellitus. It is most frequently associated with non-ketotic hyperglycaemia and manifests as hemichorea or hemiballismus, occasionally progressing to generalised choreiform movements. Although uncommon, DS is increasingly recognized as the second most frequent cause of hemichorea-hemiballismus after basal ganglia cerebrovascular events and the most common metabolic etiology of this syndrome. The condition is typically characterized by severe hyperglycaemia in the absence of ketosis accompanied by either choreiform movements or radiological abnormalities of the basal ganglia on CT or MRI. However, normal neuroimaging does not exclude the diagnosis and clinical suspicion remains paramount. Misdiagnosis is common, particularly when basal ganglia hyper density on CT mimics intracerebral haemorrhage leading to unnecessary interventions and delayed treatment. We present the case of a 71-year-old woman with long-standing type 2 diabetes mellitus and chronic kidney disease who developed acute onset hemichorea in the setting of profound hyperglycaemia. Despite unremarkable neuroimaging, the diagnosis of DS was established based on clinical features and metabolic derangements. The patient demonstrated partial improvement with strict glycaemic control and symptomatic therapy, though abnormal movements persisted beyond three months. This case underscores the importance of recognizing DS even in the absence of classical imaging findings. Early diagnosis and prompt initiation of glycaemic control, supplemented by symptomatic pharmacotherapy, when necessary, can significantly improve outcomes. Greater awareness among clinicians is essential to prevent misdiagnosis and ensure timely management of this rare but reversible complication of diabetes mellitus.

**Keywords:** Hyperglycaemia, Diabetic striatopathy, Hemichorea, Hemiballismus, Non-ketotic hyperglycaemia, Basal ganglia dysfunction

## INTRODUCTION

Diabetes mellitus is a global health challenge, with its complications extending far beyond the metabolic sphere. While vascular, renal and ophthalmic complications are well recognized, neurological manifestations of diabetes are less frequently encountered and often underappreciated. Among these, hyperglycaemia-induced chorea or Diabetic Striatopathy (DS), represents a rare but striking clinical entity. DS is defined by the triad of non-ketotic hyperglycaemia, choreiform movements, and characteristic basal ganglia abnormalities on neuroimaging. First described several decades ago, DS has since been reported across diverse populations, though its prevalence remains exceedingly low. Epidemiological studies estimate an incidence of approximately 1 in 100,000, with higher rates observed among elderly women with poorly controlled type 2 diabetes mellitus. In

hospitalized cohorts, prevalence has been reported at 0.58% of all admissions with uncontrolled diabetes, rising to 1.2% among those presenting with neurological symptoms.[1] Despite its rarity, DS is clinically important because it is potentially reversible with appropriate treatment.

The condition is currently regarded as the second most common cause of hemichorea-hemiballismus after basal ganglia strokes and the leading metabolic cause of hemichorea- hemiballismus.[2] Yet, DS remains underdiagnosed and frequently misinterpreted. Physicians often mistake basal ganglia hyper density on CT for intracerebral haemorrhage, leading to unnecessary investigations or inappropriate management.

The clinical presentation of DS is variable. Most patients develop hemichorea or hemiballismus typically unilateral, though generalized chorea has been described. Movements are irregular, non-rhythmic and involuntary, often impairing daily activities. Neuroimaging findings while supportive, are not universal. CT scans may reveal hyper density of the striatum, while MRI often demonstrates T1 hyperintensity of the basal ganglia. However, up to one-third of patients may have normal imaging, emphasizing the need for clinical vigilance.[2]

The pathophysiology of DS remains incompletely understood Proposed mechanisms include depletion of gamma-aminobutyric acid (GABA) due to altered glucose metabolism, ischemic changes within the basal ganglia, metabolic derangements and microvascular injury. The metabolic hypothesis suggests that in non-ketotic hyperglycaemia, impaired glucose utilization forces the brain to metabolize GABA, leading to neurotransmitter depletion and basal ganglia dysfunction. This imbalance between inhibitory and excitatory pathways manifests clinically as chorea.

Treatment of DS centres on strict glycaemic control, achieved through hydration and insulin therapy. While some patients experience rapid resolution of symptoms within days, others require additional pharmacological agents such as antipsychotics, benzodiazepines or dopamine-depleting drugs. In refractory cases, invasive interventions including deep brain stimulation or pallidotomy have been attempted with variable success.[13] Prognosis is generally favourable, though recurrence rates up to 20% have been reported, necessitating long-term follow-up.

In this report, we describe the case of a 71-year-old woman with poorly controlled type 2 diabetes mellitus who developed hemichorea in the setting of severe hyperglycaemia. Despite normal neuroimaging, the diagnosis of DS was established based on clinical features and metabolic abnormalities. This case highlights the importance of recognizing DS even in atypical presentations and underscores the need for heightened awareness among clinicians.

## CASE REPORT

A 71-year-old woman was admitted with complaints of giddiness, generalized weakness and difficulty walking for 2-3 days. She also reported excessive thirst and paraesthesia in the form of tingling sensation and pain involving the right upper and lower limbs, more pronounced in the right upper limb. She was a known case of hypertension, type 2 diabetes mellitus, coronary artery disease with mild left ventricular dysfunction and chronic kidney disease with a baseline serum creatinine of approximately 2 mg/dl. Two days prior to admission, she was independently ambulatory; however, she required support to sit and walk over the preceding 48 hours.

On arrival, her vital signs were stable: pulse rate 82/min, blood pressure 130/80 mm Hg, respiratory rate 20/min and $SpO_2$ 99% on room air. Random blood glucose was markedly elevated at 549 mg/dl with blood ketones of 1.0 mmol/l. HbA1c was >12% indicating poor long-term glycaemic control. Thyroid profile, parathyroid hormone, vitamin B12, serum ammonia, calcium, magnesium and phosphorus levels were within normal limits. Viral markers and ASO titres were negative. She appeared dehydrated with a dry tongue.

During hospitalization, the patient developed sudden onset irregular, non-rhythmic involuntary movements predominantly involving the right upper limb consistent with choreiform movements. Neurological examination revealed normal higher mental functions, intact motor power (5/5 in all limbs), normal deep tendon reflexes, bilateral plantar flexor responses and no cerebellar signs. Cardiovascular and respiratory examinations were unremarkable. Ultrasound abdomen revealed minimal gallbladder sludge with bilateral grade Il renal Parenchymal disease. Urine culture grew Escherichia coli. MRI brain showed no diffusion restriction or focal lesions.

In view of severe hyperglycaemia and dehydration, the patient was managed with intravenous fluid resuscitation and a human insulin infusion. Over the next 24 hours, blood glucose levels improved to ~172 mg/dl. Neurology consultation confirmed the diagnosis of hyperglycaemia-induced chorea based on clinical features and metabolic abnormalities.

Symptomatic therapy was initiated with tetrabenazine 12.5 mg twice daily and haloperidol as required. resulting in partial reduction of abnormal movements. Due to persistence of involuntary movements, tetrabenazine was discontinued and clonazepam was introduced (0.25 mg in the morning and 0.5 mg at night), along with strict glycaemic control (<180 mg/dl). This led to transient cessation of abnormal movements, but choreiform activity recurred on the same side and persisted despite medications. The patient was readmitted twice over the last 3 months for dysglycemia. The choreiform movements have been persisting since their first onset 3 months back.

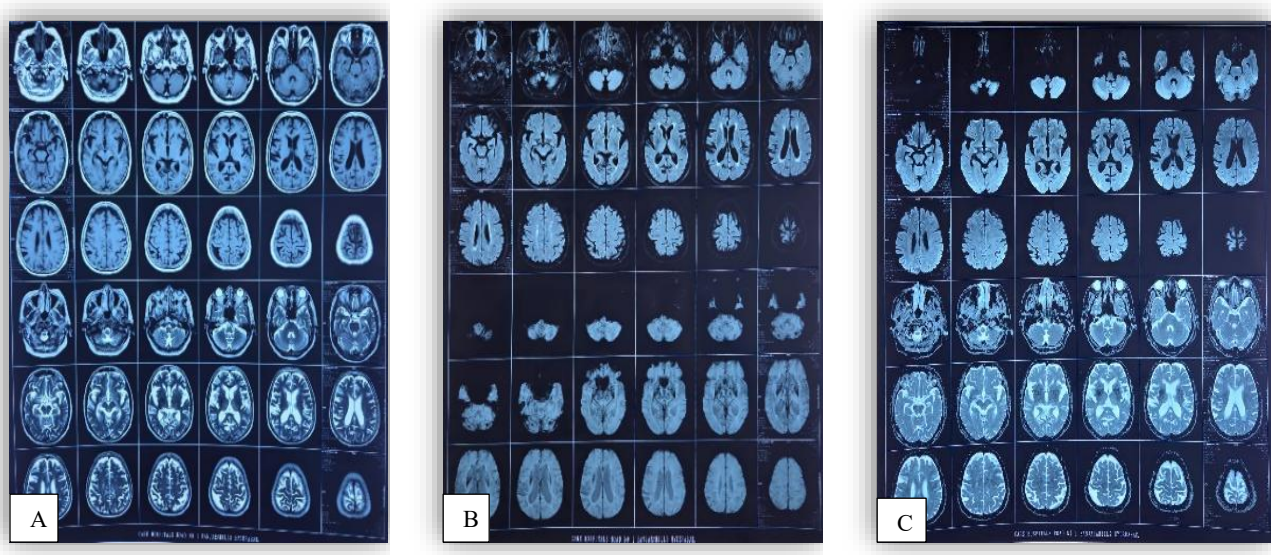


**Figure 1 (A, B and C): MRI brain showed no diffusion restriction or focal lesions.**

## DISCUSSION

Hyperglycemia-induced chorea is a rarely encountered but a known complication of uncontrolled diabetes mellitus which is predominantly associated with non-ketotic hyperglycemia. The exact pathophysiology remains unclear but the proposed underlying pathways include depletion or exhaustion of GABA, ischemic changes in the basal ganglia, metabolic derangements and petechial hemorrhage.

According to the metabolic theory, non-ketotic hyperglycemia is thought to play an important role in contributing to the development of hyperglycemia-induced chorea. During hyperglycemia, the brain shifts from its normal aerobic glucose metabolism to an anaerobic pathway leading to inactivation of the Krebs cycle. When glucose utilization is impaired, the brain uses GABA as an alternative energy source converting it into succinic acid. However, this pathway supplies only 10-40% of the energy required by the basal ganglia resulting in energy deficiency and metabolic acidosis. As GABA and acetate are rapidly consumed, acetylcholine synthesis decreases. The combined depletion of GABA and acetylcholine (imbalance in neurotransmitters) along with metabolic acidosis and reduced energy availability leads to basal ganglia dysfunction producing the characteristic clinical manifestations.[2]

On the one hand, loss of inhibition of the subthalamic nucleus leading to excessive activation of the motor cortex via thalamic projections thereby producing choreiform movements.[3,4] In ketotic hyperglycemia, ketone bodies serve as an alternative substrate for the brain allow re-synthesis of GABA which may protect from developing chorea and explain its low incidence in these patients.

Nevertheless, this metabolic hypothesis does not fully explain several observed clinical features. Despite hyperglycaemia being systemic metabolic disturbance, clinical manifestations are typically unilateral suggesting a focal vulnerability of the basal ganglia rather than a generalised metabolic effect. Chorea may persist despite normalization of blood glucose levels suggestive of ongoing neuronal dysfunction beyond metabolic correction.[5] Rapid correction of hyperglycaemia may precipitate chorea by unmasking basal ganglia dysfunction i.e. chorea develops following rapid correction of hyperglycaemia implying that abrupt metabolic shifts may unmask or worsen basal ganglia dysfunction.[6-9]

A proposed explanation for delayed-onset chorea is that elevated ketone levels transiently compensate for GABA depletion during the hyperglycaemic state with symptoms emerging once ketone availability decreases after glycaemic correction. However, this remains speculative.[8] Furthermore, not all patients exhibit ketoacidosis and among those with ketotic hyperglycaemia, delayed hemichorea after glucose normalization has not been consistently documented suggesting that additional mechanisms beyond GABA metabolism are likely involved.[6-10] Neuroimaging often demonstrates hyper density of the striatum on CT or T1-weighted hyperintensity on MRI. However, as highlighted in the above case, imaging may be normal and diagnosis should be based on clinical suspicion and metabolic evaluation.

Reducing blood glucose levels achieved by adequate hydration and administration of insulin is the cornerstone of DS treatment.[11] The time taken for involuntary movements to resolve after correction of hyperglycaemia is highly variable. In some patients, symptoms improve within a few days where-in some others recovery may take up to ten months with an average duration of about six

months for complete resolution. Some patients experience only partial improvement even after prolonged follow-up ranging from three months to over five years. Importantly, glycaemic control alone is sufficient for complete symptom resolution in only about one-quarter of patients. The majority require additional medications to achieve meaningful clinical improvement.

Most patients require additional medications to achieve meaningful clinical improvement. These drugs involve five classes: benzodiazepines, antipsychotics, dopamine-depleting agents, anticonvulsants and selective serotonin reuptake inhibitors.[2] Benzodiazepines like diazepam and clonazepam enhance inhibitory neurotransmission through GABA receptors. Among the typical antipsychotics, haloperidol is the most commonly used followed by chlorpromazine, sulpiride, pimozide and tiapride. Atypical antipsychotics like risperidone and quetiapine can also be used. However, the use of antipsychotic agents must be carefully weighed due to the risk of tardive dyskinesia.

Dopamine-depleting agents including tetrabenazine and reserpine act by blocking the presynaptic monoamine transporter. Anticonvulsants consisting of sodium valproate and topiramate as well as selective serotonin reuptake inhibitors like escitalopram have also been reported to be beneficial. In some cases, combination therapy has been used. Interestingly, the average time to symptom resolution is significantly shorter in patients treated with glycaemic control alone (approximately 2 days) compared with those requiring additional anti-choreic medications (around 14 days) which is suggesting that patients responding to glycaemic control alone likely have a less severe form of the disease.

For patients with symptoms refractory to medical therapy, invasive approaches have been attempted with moderate success which includes pallidotomy, ventrolateral thalamotomy, transcranial magnetic stimulation, internal globus pallidus deep brain stimulation.[13]

Some patients relapsed after stopping anti-chorea drugs over two months and two years after the first episode of chorea. In most patients, the chorea recurred on the same side previously affected but some patients who initially had a unilateral onset developed bilateral chorea. A relatively high recurrence rate of about 20% was observed even after the resolution of the neuroimaging abnormalities, thus pointing to the need for regular follow-up independent of neuroimaging results.[12]

Differential diagnoses include acute ischemic stroke, Fahr syndrome, autoimmune chorea, drug-induced movement disorders, Wilson's disease and infectious causes. Fahr syndrome commonly presents with a combination of movement disorders including parkinsonism, chorea and dystonia along with progressive cognitive decline, psychiatric manifestations, seizures and frequently associated with metabolic abnormalities consisting of hypoparathyroidism or pseudohypoparathyroidism resulting in disturbances of calcium–phosphate metabolism. Neuroimaging usually demonstrates extensive bilateral calcifications. Prompt recognition is essential as symptoms are typically reversible with correction of hyperglycaemia. Dopamine-depleting agents and benzodiazepines may be used for symptomatic control when movements are severe in nature

## CONCLUSION

Hyperglycaemia-induced chorea is a rare, reversible neurological complication of poorly controlled diabetes mellitus. Clinicians should maintain a high index of suspicion in elderly diabetic patients presenting with acute onset involuntary movements even in the absence of classical imaging findings. Early diagnosis and strict glycaemic control lead to excellent clinical outcomes and prevent unnecessary investigations.

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