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ORIGINAL ARTICLE

Diabetic striatopathy in Hispanic patients: recognizing a variable yet treatable movement disorder

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Abstract

Objective: *To describe clinical features, diagnostic challenges, and therapeutic response of diabetic striatopathy in Latin American patients, an underrepresented population in current literature, and to highlight the importance of early recognition and interdisciplinary collaboration in improving diagnosis and management.* **Methods:** *Case series of two Latin American women with poorly controlled type 2 diabetes mellitus who developed hyperkinetic movement disorders. Clinical presentation, neuroimaging findings, diagnostic approach, and clinical outcomes following metabolic and symptomatic treatment were analyzed.* **Results:** *One patient presented with acute-onset chorea accompanied by pain and hypoesthesia, with striatal hyperdensity on computed tomography and T1 hyperintensity on magnetic resonance imaging, achieving complete symptom resolution within 48 hours after intensive insulin therapy. The second patient developed progressive choreiform movements, initially misdiagnosed as epilepsy, without response to antiepileptic drugs. Neuroimaging confirmed diabetic striatopathy; insulin therapy produced partial improvement, and adjunctive haloperidol reduced movement severity, with residual symptoms at discharge. Both cases demonstrated characteristic imaging findings despite distinct clinical phenotypes.* **Conclusions:** *Diabetic striatopathy shows heterogeneous clinical presentations and may mimic other neurological disorders, leading to diagnostic delays. Early recognition and prompt glycemic control are essential, although treatment response varies and may require individualized management strategies.*

Keywords: *Diabetes. Hyperglycemia. Chorea. Basal ganglia. Case report.*

Estriatopatía diabética en pacientes hispanos: reconociendo un trastorno del movimiento variable pero tratable

Resumen

Objetivo: *Describir las características clínicas, los desafíos diagnósticos y la respuesta terapéutica de la estriatopatía diabética en pacientes latinoamericanos, una población escasamente representada en la literatura, y resaltar la necesidad de reconocimiento clínico temprano y colaboración interdisciplinaria.* **Métodos:** *Serie de casos de dos mujeres latinoamericanas con diabetes mellitus tipo 2 mal controlada que desarrollaron trastornos del movimiento hipercinéticos. Se analizaron las manifestaciones clínicas, hallazgos de neuroimagen, abordaje diagnóstico y evolución clínica tras tratamiento metabólico y sintomático.* **Resultados:** *Una paciente presentó corea de inicio agudo asociada a dolor e hipoestesia, con hiperdensidad estriatal en tomografía e hiperintensidad en T1 en resonancia magnética, logrando resolución completa tras control glucé-*

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mico intensivo con insulina en 48 horas. La segunda paciente desarrolló movimientos coreiformes progresivos, inicialmente diagnosticados como epilepsia, sin respuesta a antiepilépticos. La neuroimagen confirmó estriatopatía diabética; el tratamiento con insulina produjo mejoría parcial y la adición de haloperidol redujo la severidad de los movimientos, persistiendo síntomas residuales. Ambos casos mostraron hallazgos radiológicos característicos pese a fenotipos clínicos distintos. **Conclusiones:** *La estriatopatía diabética presenta manifestaciones clínicas heterogéneas y puede simular otras enfermedades neurológicas, favoreciendo retrasos diagnósticos. El reconocimiento temprano y el control glucémico oportuno son fundamentales, aunque la respuesta terapéutica puede variar y requerir manejo individualizado.*

Palabras clave: *Diabetes. Hiperglucemia. Corea. Ganglios basales. Reporte de caso.*

Introduction

Diabetic striatopathy (DS) is a rare neurological complication associated with poorly controlled Type 2 diabetes (T2D). It is characterized by non-ketotic hyperglycemia, hyperdensity of the basal ganglia on computed tomography (CT), and contralateral choreiform movements. While predominantly reported in elderly Asian females, cases in Latin American populations remain underrepresented, limiting understanding of potential ethnic variations in presentation and management[1-3].

The term DS is relatively recent and corresponds to previously used diagnostic labels such as hyperglycemia-associated chorea, chorea hyperglycemia basal ganglia syndrome (CHBG), and non-ketotic hyperglycemic hemichorea. Including these synonymous terms helps prevent diagnostic and terminological confusion in the literature. In this manuscript, the term Latin American is used instead of Hispanic to more precisely describe individuals originating from Spanish-speaking countries in Latin America. Unlike the broader term Hispanic, which may include people from Spain or Spanish-speaking populations in the United States, Latin American specifically refers to individuals from Mexico, Central America, South America, and the Spanish-speaking Caribbean, which aligns with the origin of the patients presented in this case series.

A previous prospective series by Cervantes-Arriaga et al., reported 10 Mexican patients with hyperglycemia-associated movement disorders and described similar clinical and neuroimaging features[4]. However, that study predated the widespread adoption of the term “diabetic striatopathy” and used earlier nomenclature. Our report adds to this limited but growing body of literature by applying current diagnostic terminology and focusing specifically on clinical variability, diagnostic challenges, and treatment response in Latin American patients.

This case report was prepared in accordance with the CARE (CAse REport) guidelines to ensure completeness, transparency, and accuracy in reporting.

Clinically, DS presents as an acute or subacute hyperkinetic movement disorder, frequently misdiagnosed as stroke, epilepsy, or structural lesions of the basal ganglia. Involuntary movements, predominantly chorea or ballism, typically affect one side of the body. Choreic movements are irregular, non-repetitive, and random, while ballistic movements are more forceful and wide-amplitude. These abnormal movements are often exacerbated by voluntary activity and tend to resolve during sleep[5-9].

Several pathophysiological mechanisms have been proposed to explain the clinical manifestations. One prevailing theory suggests that non-ketotic hyperglycemia depletes gamma-aminobutyric acid (GABA) levels, leading to reduced acetylcholine and resulting in basal ganglia dysfunction and chorea. Another theory implicates ischemic injury to the basal ganglia in the context of uncontrolled diabetes as the underlying cause. In addition, neurodegeneration due to hyperosmolarity has been proposed as a plausible mechanism. The presence of gemistocytes-swollen reactive astrocytes observed following ischemic injury, may contribute to the T1 hyperintensity on MRI, potentially integrating elements from the aforementioned theories[2,3,10,11].

Average blood glucose and glycated hemoglobin (HbA1c) levels in DS patients tend to be markedly elevated (414-481.5 mg/dL and 13.1-14.4%, respectively), underscoring the association with hyperglycemia. While DS is predominantly linked to non-ketotic hyperglycemia, 81.7% of patients in one study exhibited no evidence of ketosis, it has also been reported in patients with diabetic ketoacidosis and other ketotic states[3,12-16].

Neuroimaging plays a critical role in diagnosis. CT scans often reveal unilateral hyperdensity in the striatum contralateral to the side affected by involuntary movements. Magnetic resonance imaging (MRI), particularly T1-weighted sequences, is more sensitive and

typically shows characteristic striatal hyperintensity without mass effect and with preservation of the internal capsule[17-20].

Prompt glycemic control with insulin remains the cornerstone of treatment. However, in some cases, adjunctive therapy with antichoreic agents may be required. Clinical resolution can occur within 2-14 days with glycemic correction and symptomatic treatment. Symptom resolution through glycemic control alone occurs in only 25% of patients, while the addition of antichoreic agents increases the response rate to approximately 76%[3,9,12,21].

Resolution of radiological abnormalities often lags behind clinical improvement, with a median duration of 60 days for CT findings and up to 180 days for MRI. Approximately 20% of patients experience recurrence of abnormal movements even after initial radiological resolution, emphasizing the importance of ongoing clinical and imaging follow-up[1,3,12].

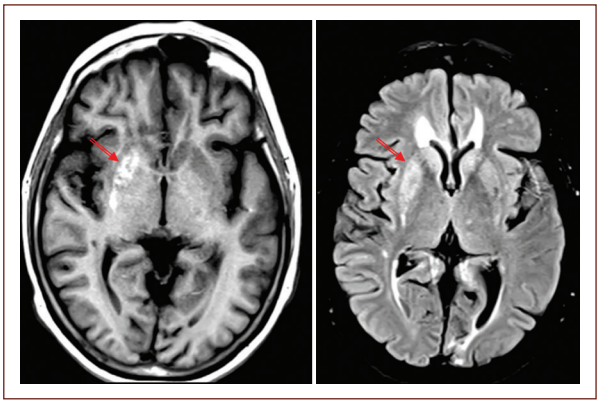

Figure 1. Axial T1-weighted and fluid-attenuated inversion recovery magnetic resonance imaging of case 1 demonstrating a hyperintense lesion in the right striatum (arrow), consistent with diabetic striatopathy.

Case presentation

Case 1

A 62-year-old Latin American woman with a history of long-standing T2D and hypertension presented with an acute-onset hyperkinetic movement disorder affecting her left upper limb. The involuntary movements, described as continuous, non-rhythmic, and multidirectional, began insidiously during the night and progressively worsened over 4 days, prompting medical evaluation. The patient also reported pain and hypoesthesia in the affected limb and ipsilateral facial region.

She denied any prior history of movement disorders, epilepsy, or stroke, as well as any family history of neurological conditions. Her glycemic control had been consistently poor, with blood glucose levels persistently exceeding 300 mg/dL. Her current pharmacological treatment included Losartan for hypertension, with no recent modifications in medication use or evidence of intercurrent infections.

Neurological examination revealed choreiform movements predominantly affecting the left hand, with mild involvement of the proximal upper limb and ipsilateral face. The movements were involuntary, continuous, and exacerbated by voluntary action and distraction maneuvers, while subsiding at rest. The remainder of the neurological and systemic examination was unremarkable, with no cognitive impairment, motor weakness, or pyramidal signs.

On admission, her blood glucose level was 331 mg/dL (reference range 7-100 mg/dL), and laboratory workup, including venous blood gas, renal function panel, complete blood count, and serum electrolytes, was within normal limits. Non-contrast brain CT revealed a subtle hyperdensity in the right striatum, without associated mass effect or hemorrhagic transformation. MRI demonstrated a T1-weighted hyperintense lesion in the right striatum, with corresponding hypointensity on T2 and fluid-attenuated inversion recovery sequences (Fig. 1), consistent with DS. No additional structural, metabolic, or vascular abnormalities were identified.

A diagnosis of DS was established. Intensive glycemic control with insulin therapy was initiated, achieving a reduction in blood glucose levels to < 100 mg/dL within 48 h. The choreiform movements resolved completely, along with the associated pain and sensory disturbances. The patient was discharged 48 h after admission, entirely asymptomatic, with no residual neurological deficits.

Case 2

A 70-year-old Latin American woman with a history of poorly controlled T2D presented to the emergency department with recurrent, episodic involuntary movements affecting her right hemibody. She reported a 6-month history of progressive hyperkinetic episodes, which had initially been intermittent and brief (lasting seconds to minutes) but had become continuous over time. A previous misdiagnosis of epilepsy had led to treatment with phenytoin, with no clinical benefit.

She denied a history of stroke, traumatic brain injury, or neurodegenerative disorders, and there was no

family history of movement disorders. Her diabetes had been poorly controlled, with erratic glucose monitoring and lack of adherence to prescribed treatment.

On neurological examination, the patient exhibited non-rhythmic, dance-like choreiform movements, predominantly affecting the proximal right upper and lower limbs. The movements were present at rest, exacerbated by voluntary action, and significantly interfered with daily activities, including walking and feeding. Importantly, they did not occur during sleep, and no myoclonus, dystonia, or pyramidal signs were noted. The rest of the neurological and systemic examination was unremarkable.

Initial laboratory investigations revealed severe hyperglycemia of 469 mg/dL (reference range 70-100mg/dL), with HbA1c levels consistent with chronic poor glycemic control. Complete blood count, renal function panel, serum electrolytes, venous blood gas analysis, and ketone bodies were within normal limits. Brain CT demonstrated a hyperdense lesion in the left basal ganglia, with no associated hemorrhage or ischemic infarct (Fig. 2). Based on these findings, DS was diagnosed.

Intravenous insulin therapy was promptly initiated, leading to partial glycemic control. Given the persistence of disabling choreiform movements, haloperidol (5 mg twice daily) was introduced as adjunctive therapy. After 3 days of treatment, the severity and frequency of the involuntary movements improved partially, but mild residual symptoms persisted at discharge.

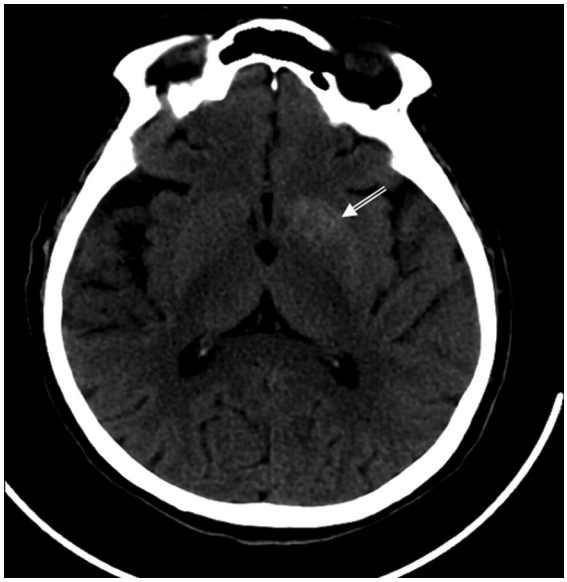

Figure 2. Non-contrast computed tomography of case 2 showing a hyperdense lesion in the left basal ganglia (arrow), a characteristic finding in diabetic striatopathy.

Discussion

This case series highlights the heterogeneous clinical presentation of DS and underscores the importance of early recognition and accurate diagnosis, particularly in underrepresented populations. A prior case series conducted in Mexico by Cervantes-Arriaga et al., described 10 patients with hyperglycemia-associated movement disorders exhibiting similar neuroimaging and clinical features, although the term “diabetic striatopathy” was not yet in use. This supports the presence of this clinical entity in Latin American populations and reinforces the importance of recognizing it under its current diagnostic framework[1,2,4].

DS presents with variable phenotypes, ranging from acute-onset continuous movements with sensory symptoms (Case 1) to progressive episodic choreiform movements (Case 2). The diagnostic delay in Case 2, initially labeled as epilepsy, underscores a recurrent issue in DS: its mimicry of other neurological conditions, particularly stroke, seizure disorders, or functional movement disorders. These diagnostic pitfalls can lead to ineffective or inappropriate treatment, delaying targeted interventions such as glycemic correction or dopamine antagonism[3,5,11,12]. Our findings reinforce the need for heightened clinical suspicion in patients with poorly controlled diabetes and movement disorders[6].

Neuroimaging continues to be pivotal in diagnosis, as both cases showed striatal hyperdensity on CT and characteristic T1 hyperintensity on MRI. These findings support the role of neuroimaging not only in excluding structural lesions but also in confirming DS in the appropriate clinical context. Interestingly, the radiological features in our patients were subtle, highlighting the importance of clinician awareness in interpreting these findings when DS is suspected[6,12,19,20].

Pathophysiologically, both cases reinforce the notion that DS may not be exclusively related to non-ketotic hyperglycemia. One patient exhibited a clinical profile consistent with prolonged metabolic dysregulation and required antichoreic therapy, suggesting a possible cumulative or neurotoxic mechanism. The variation in treatment response aligns with the theory that striatal injury in DS may result from a combination of factors, including ischemia, astrocytic swelling (gemistocyte

Table 1. Comparative summary of diabetic striatopathy case series reported in the literature

Authors	Year	Country	Sample size	Mean age (years)	F:M	Glc mg/dL	Choreic/ballistic (%)
Oh et al.[6]	1990-2001	South Korea	53	71	1:0.57	481.5	90.57
Chua et al.[8]	1992-2018	China	176	67.6	1:1.7	414	88
Aras et al.[7]	2018-2021	Turkey	19	72.2	1:0.19	649	68.4
Dubey et al.[16]	2014-2021	India	59	55.4	1:1.1	419	69.5
Cervantes et al.[5]	2011	Mexico	10	67.7	7:3	359.7	100
Ours	2023-2024	México	2	66	-	400	100

Data include study authors, publication period, country, sample size, mean age, female-to-male ratio (F:M), mean plasma glucose levels (mg/dL), and the percentage of patients presenting with choreic or ballistic movements.
Glc: glucose; F:M: female-to-male ratio. Data were extracted from the cited references.

accumulation), and neurotransmitter depletion (e.g., GABA and acetylcholine), rather than a single metabolic insult[2,3,11,12].

A comparison of DS cases across different populations (Table 1) reveals key epidemiological and clinical differences. The mean age of presentation in our patients (66 years) is comparable to cohorts from South Korea (71 years), China (67.6 years), and Turkey (72.2 years) but is notably higher than in India (55.4 years)[6,17]. This may reflect regional or genetic factors influencing disease onset. Glucose levels (400 mg/dL) in our cases were within the reported range but lower than in Turkey (649 mg/dL), suggesting that hyperglycemia alone may not fully account for DS pathogenesis[3].

Interestingly, our patients exhibited 100% choreiform/ballistic movements, consistent with findings from another Latin American series by Cervantes-Arriaga et al., in which all 10 patients also presented hyperkinetic motor phenotypes. This contrasts with the 68.4-90.57% choreiform presentation rates reported in non-Latin American cohorts[6-8,17]. This observation raises the possibility that ethnic, genetic, or metabolic factors may influence the motor phenotype of DS. In addition, while a female predominance is well-documented in DS, the small sample size in our cohort precluded sex-based comparisons[3,18].

Case 1 responded rapidly to insulin alone, while Case 2 required adjunctive haloperidol, aligning with reports that glycemic control alone resolves symptoms in ~25% of cases, whereas dopamine receptor antagonists increase resolution rates to ~76%[12]. The differing responses suggest variability in underlying mechanisms, ranging from acute metabolic dysfunction to chronic neurotoxicity or vascular injury[2,12].

Our findings highlight the need for expanded research in Latin American populations to assess whether these age, metabolic, and clinical differences hold statistical and clinical significance[3,9]. Future studies should focus on long-term outcomes, genetic predisposition, and neuroimaging evolution to refine diagnostic and therapeutic approaches for DS[2,9].

Several limitations must be acknowledged. First, this is a small case series, limiting the generalizability of findings to the broader Latin American population. Second, the lack of long-term follow-up prevents assessment of potential symptom recurrence or neuroimaging evolution over time. Finally, while neuroimaging was essential for diagnosis, more detailed pathophysiological analyses (e.g., metabolic imaging or histopathological correlation) were not performed[5].

Conclusion

DS should be a key differential diagnosis in patients presenting with acute hyperkinetic movement disorders and poorly controlled diabetes, particularly in populations where it remains underrecognized. Early diagnosis and glycemic management are essential for optimal outcomes, but therapeutic response varies, warranting further investigation into predictors of treatment success. Expanding research in Latin American populations will provide crucial insights into the epidemiology, pathogenesis, and tailored management of this rare but clinically significant diabetic complication.

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The authors declare that this work was carried out with the authors' own resources.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical considerations

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. The authors have obtained approval from the Ethics Committee for the analysis of routinely collected and anonymized clinical data; therefore, individual informed consent was not required. Relevant ethical recommendations have been followed.

Declaration on the use of artificial intelligence (AI). The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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