The Case | The sweet twirl—chorea as a diabetic complication: an elderly patient with CKD, hyperglycemia, and chorea

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An 81-year-old patient with chronic kidney disease presented to the emergency department with disorientation, somnolence, and marked hyperglycemia (36 mmol/l). Laboratory tests revealed chronic kidney disease stage G3bA1 (creatinine 1.97 mg/dl, estimated glomerular filtration rate 31 ml/min per 1.73 m^2, and urinary albumin-to-creatinine ratio 4 mg/g), hyponatremia (129 mmol/l), and an A1C of 18.1%, indicating poorly controlled diabetes.

Renal ultrasound demonstrated bilaterally small kidneys (93–94 ml) relative to the patient's body weight (97 kg), consistent with chronic parenchymal disease. Noncontrast cranial computed tomography excluded acute hemorrhage or infarction but revealed hyperdense lesions in the putamen and caudate nucleus (Figure 1a).

After 48 hours of insulin treatment with a reduction of glucose to <11 mmol/l and partial correction of hyponatremia to 134 mmol/l, the patient developed sudden right-sided hemichorea, resulting in skin abrasions due to involuntary movements (Figure 1b). Magnetic resonance imaging confirmed T1-hyperintense signals in the left lentiform nucleus (Figure 1c).

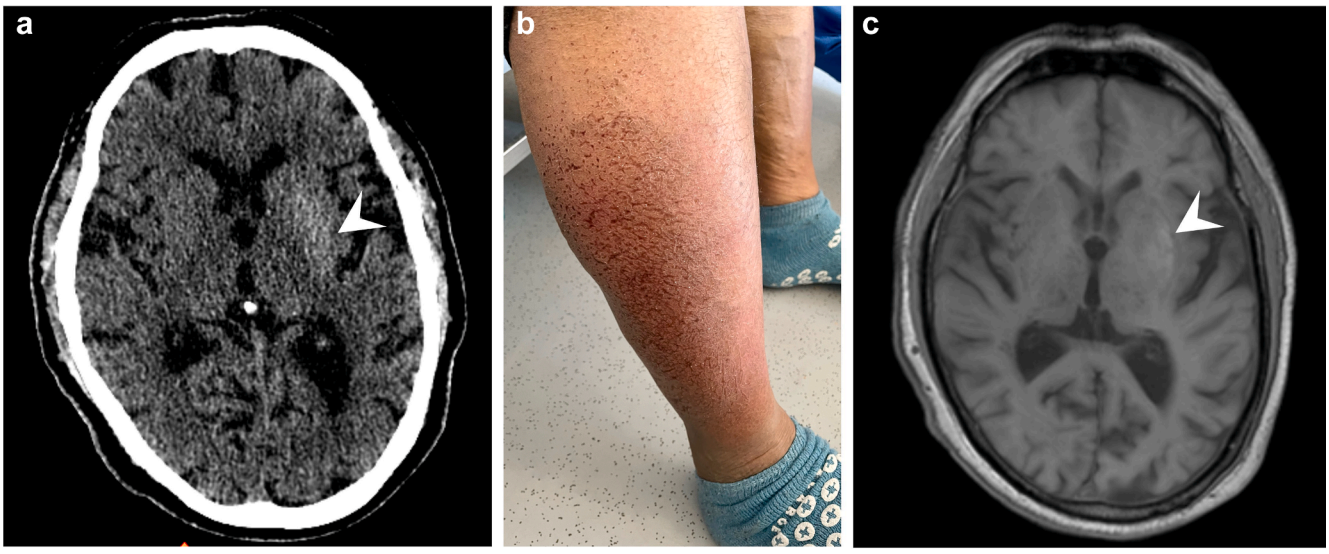

Figure 1 | (a) Noncontrast cranial computed tomography with hyperdense lesions in the putamen and caudate nucleus (arrowhead). (**b**) Skin abrasions due to involuntary movements. (**c**) Magnetic resonance imaging with T1-hyperintense signals in the left lentiform nucleus (arrowhead).

What is your diagnosis?

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The Diagnosis | **Chorea-hyperglycemia-basal ganglia syndrome**

Chorea-hyperglycemia-basal ganglia (C-H-BG) syndrome, also known as nonketotic hyperglycemic hemichorea, is a rare neurologic complication of poorly controlled type 2 diabetes.[1] It classically affects elderly Asian women and typically presents during episodes of severe hyperglycemia, with symptom resolution after glycemic correction.

Thus, the demographic characteristics of our patient were atypical, and he developed hemichorea after glucose correction, suggesting a delayed metabolic or structural response in the basal ganglia.

Neuroimaging is key to diagnosis: computed tomography reveals hyperdensity, and magnetic resonance imaging shows T1 hyperintensity in the contralateral basal ganglia, typically without infarction or hemorrhage.

The exact pathophysiology remains unclear; proposed mechanisms include hyperviscosity-induced ischemia, petechial hemorrhage, and astrocytic dysfunction.[2]

Management focuses on achieving and maintaining euglycemia, typically through insulin therapy and close metabolic monitoring. If hemichorea persists, pharmacologic dopamine receptor antagonists or benzodiazepines may be required to manage symptoms.[2] In our case, risperidone and tetrazepam proved effective.

Generally, the prognosis of C-H-BG is favorable, though the symptoms may persist for weeks before full resolution. In this case, symptoms improved significantly within a week of maintaining euglycemia, though residual chorea persisted when the patient was transferred for rehabilitative care.

Associations between chronic kidney disease and C-H-BG have so far not been reported. However, recent evidence suggests that uremic toxins and chronic renal dysfunction may contribute to basal ganglia vulnerability and modify clinical manifestations of metabolic encephalopathies such as C-H-BG.[3]

In addition to glycemic correction, simultaneous hyponatremia normalization may have contributed to neurologic symptoms. Rapid plasma osmolality shifts during concurrent correction of hyperglycemia and hyponatremia can stress central nervous system structures, including the basal ganglia. Although osmotic demyelination is considered the main complication of rapid sodium correction, movement disorders such as chorea have also been reported with abrupt electrolyte and glucose shifts. In our case, combined partial correction of hyperglycemia and hyponatremia may have triggered a delayed basal ganglia response, underscoring the need for cautious, gradual correction.

In summary, this case illustrates an atypical form of C-H-BG syndrome, with chorea emerging *after* glucose normalization and preceded by altered mental status. Chronic kidney disease may be a modifying factor in this presentation.

KEYWORDS

basal ganglia; chorea; chronic kidney disease; hyperglycemia; hyponatremia;

DISCLOSURE

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PATIENT CONSENT

Informed consent was obtained from the patient for publication.

AUTHOR CONTRIBUTIONS

MSB wrote the original draft. APS, L-TL, K-UE, MSB reviewed and edited the writing.

REFERENCES

1. Dong M, E J-Y, Zhang L, et al. Non-ketotic hyperglycemia chorea-ballismus and intracerebral hemorrhage: a case report and literature review. *Front Neurosci*. 2021;15:690761.
2. Chua C-B, Sun C-K, Hsu C-W, et al. "Diabetic striatopathy": clinical presentations, controversy, pathogenesis, treatments, and outcomes. *Sci Rep*. 2020;10:1594.
3. Kim YJ, Kim SJ, Kim J, et al. Chorea due to diabetic hyperglycemia and uremia: distinct clinical and imaging features. *Mov Disord*. 2015;30:419–422.