Um Caso de Convulsões Induzidas por Hiperglicemia

A Case of Hyperglycemia-Induced Seizures

Mafalda S.B. Sequeira, Bruno Sousa, Francisca Abecasis, Henrique Santos

Resumo:

Os distúrbios metabólicos como a hiperglicemia, apesar de raramente, podem ser responsáveis por alterações neurológicas focais, e devem sempre ser considerados como diagnóstico diferencial de crises convulsivas. Tipicamente cursam com hipointensidade T2 subcortical e em sequência de recuperação de inversão atenuada por fluidos (FLAIR), bem como difusão restrita em difusão nos mapas de coeficientes (ADC) na ressonância magnética cerebral (RM-CE). Os autores relatam o caso de um homem de 49 anos, com antecedentes pessoais de diabetes com mau controlo metabólico, internado com quadro clínico de convulsões e défices neurológicos focais causados, após extensa investigação, por hiperglicemia. Este caso merece atenção pela sua raridade na prática clínica e sugere a necessidade de considerar etiologias menos frequentes para as crises.

Palavras-chave: Convulsões/etiologia; Hiperglicemia/complicações.

Abstract:

Metabolic disorders, like hyperglycaemia, may be responsible for focal neurological alterations and should always be included in the differential diagnosis of seizures with subcortical T2 hypointensity and in fluid attenuated inversion recovery sequence (FLAIR), as well as restricted diffusion in apparent diffusion coefficient maps (ADC) on brain magnetic resonance imaging (MRI). The authors report a case of a 49-year-old man, with personal history of diabetes with poor metabolic control, admitted to hospital with seizures and focal neurological deficits caused, after extensive investigation, by hyperglycaemia. This case highlights the rarity of hyperglicemic-induced seizures in clinical practice and the need to consider less frequent aetiologies for seizures.

Keywords: *Hyperglycemia/complications; Seizures/etiology.*

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Introduction

The pathophysiology of hyperglycaemia-induced seizures is unclear; however, the most reliable evidence states that transient accumulation of free radicals and iron may lead to secondary cortical ischemia.[1]

The authors report a rare case of seizures and focal neurological deficits caused by hyperglycaemia. Moreover, it is noteworthy that the recognition of this association enables early diagnosis and prescription of appropriate treatment, thus preventing progression to more severe clinical pictures like seizures, hemiplegia, and chorea-like movements.

Case Report

A 49-year-old Caucasian man, with personal history of diabetes with poor metabolic control diagnosed 5 years ago (having discontinued the therapy 1 year ago), dyslipidemia, and obesity, presents to the hospital with behavioral and language alterations for the past five days. Two weeks before hospital admission, the patient reported frontal headache and photosensitivity, without other symptoms. There was no relevant family or social history.

Physical examination revealed tympanic temperature of 38ºC, tachycardia, normal blood pressure and signs of dehydration. Neurological exam was remarkable for disorientation and puerile behaviour and Wernicke's aphasia (fluent speech, does not name, could not follow simple orders or repeat), without other deficits. Meningeal signs including Kernig's and Brudzinski's, were not present.

After admission, three episodes of self-limited generalized tonic-clonic seizures were observed.

Evaluation on admission showed a blood glucose above 500 mg/dL, a ketonemia of 1.2 mg/dL, serum sodium of 130 mg/dL (glucose corrected) and glycosylated hemoglobin of 17.1% (target value < 7%); plasma osmolality was normal and urine ketone was negative. Blood gas analysis: pH 7.483, pCO2 40 mmHg, pO2 89.9 mmHg; Na^{+}127 mmol/L; K^{+} 4.11 mmol/L; Ca^{2+} 1.130 mmol/L; Lactat 1.4 mmol/L; HCO_3 26.4 mEq/L; AG 9.8 mEq/L; renal function was preserved, with serum creatinine of 0.9 mg/dL (normal range: 0.5-1.1 mg/dL). Hepatitis B, C and syphilis were negative. Inflammatory parameters (leukocytosis and C-reactive protein) were negative, chest teleradiography showed no evidence of pleuroparenchymal alterations.

Computed cranial tomography (CT- scan) was promptly performed and dismissed acute vascular event but highlighted

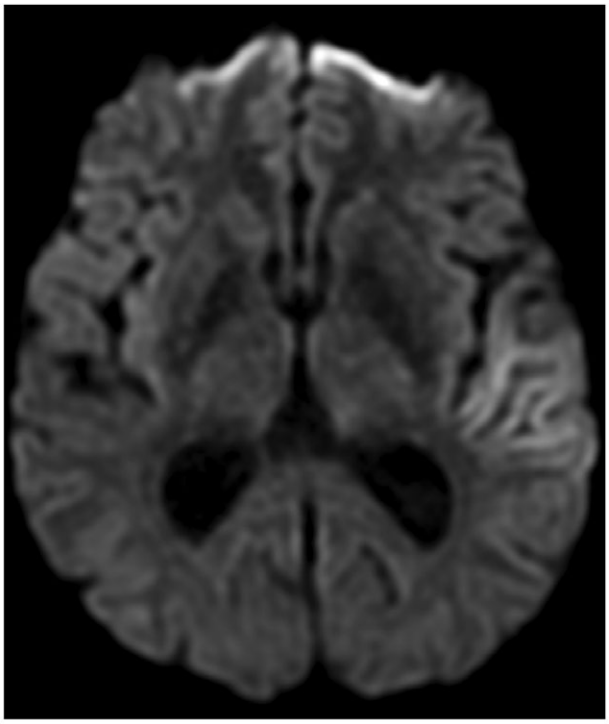

Figure 1: Axial DWI in post ictal state showing increased cortical sign in the upper left temporal lobe confirmed on the ADC map (not shown) to be diffusion restriction.

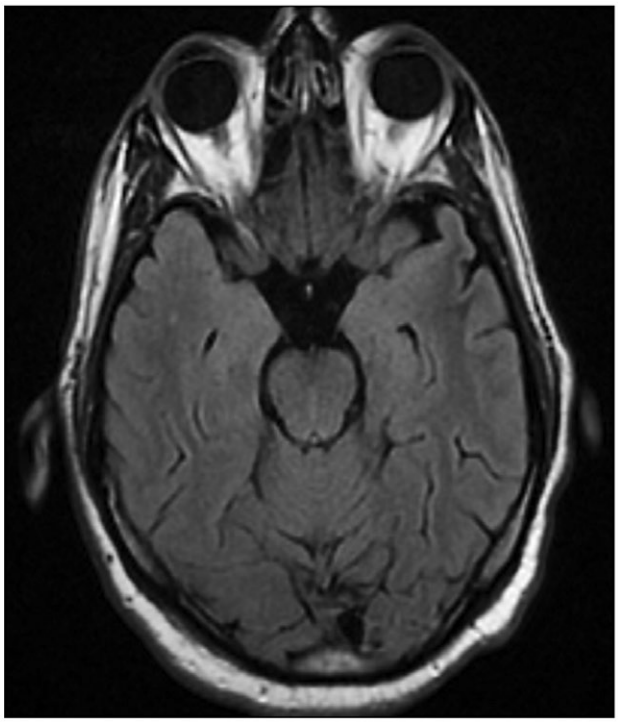

Figure 2: Axial T2 FLAIR in hyperglycemic hyperosmolar state shows hypointensity in the subcortex of the upper left temporal lobe..

slight cerebral atrophy in the region responsible for language. Magnetic resonance imaging (MRI) of the brain showed cortical hyperintensities in the upper left temporal cortex and diffusion with restriction in the ADC maps. It also revealed T2 hypointensity and FLAIR (restriction) in the subcortical white matter, which are classical MRI findings seen in hyperglycemia-induced seizures (Figs. 1 and 2). Lumbar puncture was performed to exclude infectious or neoplastic causes. Cerebrospinal fluid (CSF) was clear and colorless, with 39 mg/dL of proteins (normal range: 15-45 mg/dL), glucose of 211 mg/dL (normal range: 30-90 mg/dL) and only two cells in the cell count. For complete clinical evaluation, the patient underwent electroencephalogram (EEG) monitoring that showed slow left temporal activity, without paroxysmal activity.

Based on these results, treatment with intravenous insulin infusion and anti-epileptics was started and marked clinical improvement was seen after glycemic control was achieved. On clinical evaluation, 3 months after hospital discharge, the patient maintained good glycemic control with 9.3% of HbA1c. Neurological exam was unremarkable, and reversal of MRI findings was noted.

Discussion

Hyperglycaemia-induced seizures can be diagnosed when high blood glucose is accompanied by normal plasma osmolality and negative urine ketones. It is more frequently seen in people over 50 years old and is relatively rare in young adults and children.[3] Hyperglycaemia-induced seizures are rare and might occur in patients without prior history of diabetes or diabetic symptoms. Approximately 25% of diabetic patients experience seizures during the disease that are usually explained by other causes, such as ionic changes (mainly sodium and potassium), infectious conditions, fever, or dehydration.[4]

Although several hypotheses attempt to explain the physiological mechanism by which hyperglycaemia causes seizures, the true mechanism is still poorly understood. Studies indicate that the value of blood glucose is not the only factor implicated on the development of seizures and it is believed that factors such as genetic predisposition and co-morbidities, such as obesity and cardiovascular diseases, should also play a role.[5]

Since hyperglycaemia-induced seizures significantly affects neurological outcomes and can be responsible for misdiagnosis, awareness is fundamental. The most common types of seizures observed in this condition are focal seizures, as opposed to the generalized seizures observed in hypoglycaemia-induced seizures.[6]

Early identification of radiological changes typically caused by hyperglycaemia influences patient's short-term prognosis.[3]

Characteristic magnetic resonance imaging (MRI) findings in hyperglycaemia-induced seizures include focal altered signal intensity, subcortical T2 hypointensity with gyral hyperintensity involving the cortex and cortical or leptomeningeal postcontrast enhancement. A good clinical-radiological correlation has an impact on early treatment, prognosis, length of hospital stays and mortality.[3,7]

Brain MRI and EEG are the most valuable diagnostic tools, and it should be performed in all patients with seizures of unknown cause. The role of anti-epileptics is controversial, and studies indicate that treatment should focus on glycaemic control, hydration, and supportive therapy in case of other hemodynamic repercussions.[8] Treatment in these cases should address the cause and not the seizures themselves.

Conclusion

In summary, our case emphasizes the importance of MRI as a complementary diagnostic exam, since hyperglycaemia induced seizures are a rare condition in patients under 50 years old. Also reinforce the importance of good glycemic control of diabetic in patients to avoid the acute and chronic complications of the disease. ■

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