Reversible Visual and Neurological Manifestations of Nonketotic Hyperglycemia: A Case Report

Manifestações Visuais e Neurológicas Reversíveis da Síndrome Hiperosmolar Hiperglicémica: A Propósito de um Caso Clínico

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Abstract:

Nonketotic hyperglycemia (NKH) has been associated with visual field and neurological alterations, typically reversible with glycemic control.

A 54-year-old woman with hypertension and dyslipidemia presented in the emergency department with a 2-week history of headaches, left visual field photopsia and polydipsia and polyuria. Examination revealed homonymous left hemianopia, along with brief episodes of head and eye deviation to the left, consistent with focal epileptic seizures originating in the right hemisphere, later confirmed by electroencephalogram. Ophthalmologic exams ruled out ocular alterations, while lab results showed hyperglycemia with normal pH, excluding infections and autoimmune causes. Magnetic resonance imaging (MRI) revealed right occipital lobe changes consistent with a postictal state. With glycemic normalization and antiepileptic treatment, her visual symptoms and seizures resolved. After two months, she remained asymptomatic, and her MRI was normal.

This case demonstrates that NKH can cause reversible visual and neurological symptoms, including specific MRI findings, underscoring the importance of recognizing these manifestations for timely diagnosis and treatment.

Keywords: Hemianopsia/etiology; Hyperglycemia, Nonketotic/complications; Hyperglycemia, Nonketotic/diagnostic imaging; Seizures/etiology.

Resumo:

A síndrome hiperosmolar hiperglicémica tem sido associada a alterações visuais e neurológicas, tipicamente reversíveis com o controlo glicémico.

Uma mulher de 54 anos apresentou-se no serviço de urgência com duas semanas de evolução de cefaleia, fotópsias no hemicampo visual esquerdo, polidipsia e poliúria. À observação, verificou-se hemianópsia homónima esquerda e episódios breves de desvio cefálico e ocular para a esquerda, compatíveis com crises epilépticas focais de origem no hemisfério direito, posteriormente confirmadas por eletroencefalograma. O exame oftalmológico excluiu alterações oculares e os resultados laboratoriais revelaram hiperglicemia com pH normal, descartando infeções e causas autoimunes. A ressonância magnética revelou alterações occipitais direitas, consistentes com um estado pós-crítico. A clínica reverteu com a normalização da glicemia e antiepiléticos. Após dois meses, a doente manteve-se assintomática e a ressonância estava normal.

Este caso relembra que a síndrome hiperosmolar hiperglicémica pode causar sintomas visuais e neurológicos reversíveis, incluindo alterações na ressonância, sublinhando a importância de reconhecer estas manifestações para um diagnóstico e tratamento atempados.

Palavras-chave: Convulsões/etiologia; Hemianopsia/etiologia; Hiperglicemia Não Cetótica/complicações; Hiperglicemia Não Cetótica/diagnóstico por imagem.

Learning Points

1. NKH can lead to occipital seizures, often accompanied by visual defects like homonymous hemianopia. This underscores the need for clinicians to consider hyperglycemia as a potential cause of new-onset seizures and visual field defects.
2. Many neurological symptoms associated with NKH are reversible upon normalization of blood glucose levels. MRI abnormalities also tend to resolve with effective treatment.
3. MRI findings in NKH-related neurological presentations may include reversible hyperintensities in the occipital region. EEG may reveal epileptiform activity, particularly in the occipital region. These patterns can help differentiate NKH-related conditions from other seizure etiologies.

Introduction

Nonketotic hyperglycemia (NKH) typically develops in patients over the age of 50 who lack proper glycemic control. It is characterized by hyperglycemia, hyperosmolarity, and intracellular dehydration without ketoacidosis.[1,2] Seizures occur in 15%-40% of NKH patients. While motor seizures are

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frequently observed, only a few cases of occipital seizures, with or without hemianopia, have been reported.[3]

Homonymous hemianopia (HH) is more commonly linked to structural etiologies, primarily retrochiasmal lesions, which can arise from various causes such as tumors, head trauma, global hypoxia, migraines, seizures, occipital ischemia, anemia, and others. However, other etiologies can lead to functional HH, often with normal or only minor alterations on neuroimaging studies.[3,4]

Case Report

A 54-year-old woman with a history of controlled hypertension and dyslipidemia presented in the emergency department with a 2-week history of headache and photopsia in the left visual field. She also reported polydipsia and polyuria. The patient had no fever, weight loss, fatigue, other neurological deficits, or additional symptoms.

On examination, homonymous left hemianopia was identified and confirmed by visual field campimetry, along with brief episodes of leftward cephalic and ocular rotation, suggestive of focal epileptic seizures originating from the right cerebral hemisphere.

A cranioencephalic computed tomography (CT) scan was performed, excluding vascular or structural abnormalities. The electroencephalogram (EEG) showed attenuated baseline electrogenesis in the right parieto-occipital region, with slow activity in the ipsilateral temporal region, confirming right-sided epileptic seizures. The patient was examined by an ophthalmologist, who ruled out ocular abnormalities, particularly retinal ones. A computerized static perimetry (CSP) confirmed the homonymous left hemianopia (Fig. 1). Laboratory results revealed hyperglycemia (greater than 500 mg/dL) with normal pH and ruled out infectious or autoimmune causes (HIV, HCV, and HBV serologies were negative, no acute-phase reactants were present, and ANA and ANCA antibodies were negative). Magnetic resonance imaging (MRI) revealed hyperintensity on diffusion-weighted imaging in the right occipital cortico-subcortical region, with restriction on apparent diffusion coefficient (ADC) maps and T2 hypointensity in the right occipital juxtacortical white matter, without evidence of vascular defects, suggesting a postictal etiology (Fig. 2). Abdominal CT was also performed, excluding pancreatic lesions.

A diagnosis of newly onset diabetes mellitus was established, with a glycated hemoglobin (HbA1c) of 14.2%, and no evidence of target organ damage. The patient began treatment with insulin and oral antidiabetic drugs, achieving normalization of blood glucose levels, as well as with antiepileptic drugs. A combination of three drugs, levetiracetam, phenytoin, and lacosamide, was required to stop the visual disturbances and epileptic activity on the electroencephalogram. During hospitalization, the antiepileptic drugs were gradually tapered, and she was discharged on levetiracetam and lacosamide only. Two months later, at a follow-up appointment, the patient was asymptomatic, with good glycemic control, and MRI showed resolution of the previously observed abnormalities.

Discussion

We presented the case of a 54-year-old woman with new-onset diabetes, who presented with homonymous left hemianopia and occipital seizures secondary to NKH. Supporting this association are the minimal changes observed in the MRI, which exclude structural etiologies, and the reversible nature of the visual defect upon glycemic control.

Numerous reports have described NKH associated with visual defects and epileptic episodes that are reversible with glycemic control.[1,5] The biochemical mechanisms underlying

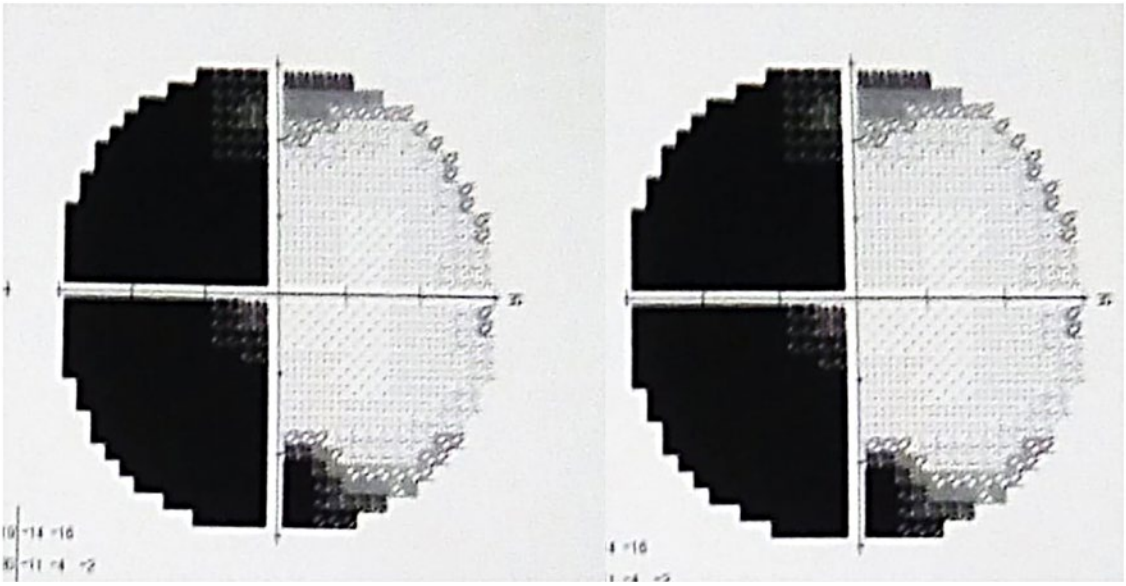

Figure 1: Computerized static perimetry (CSP) showing homonymous left hemianopia.

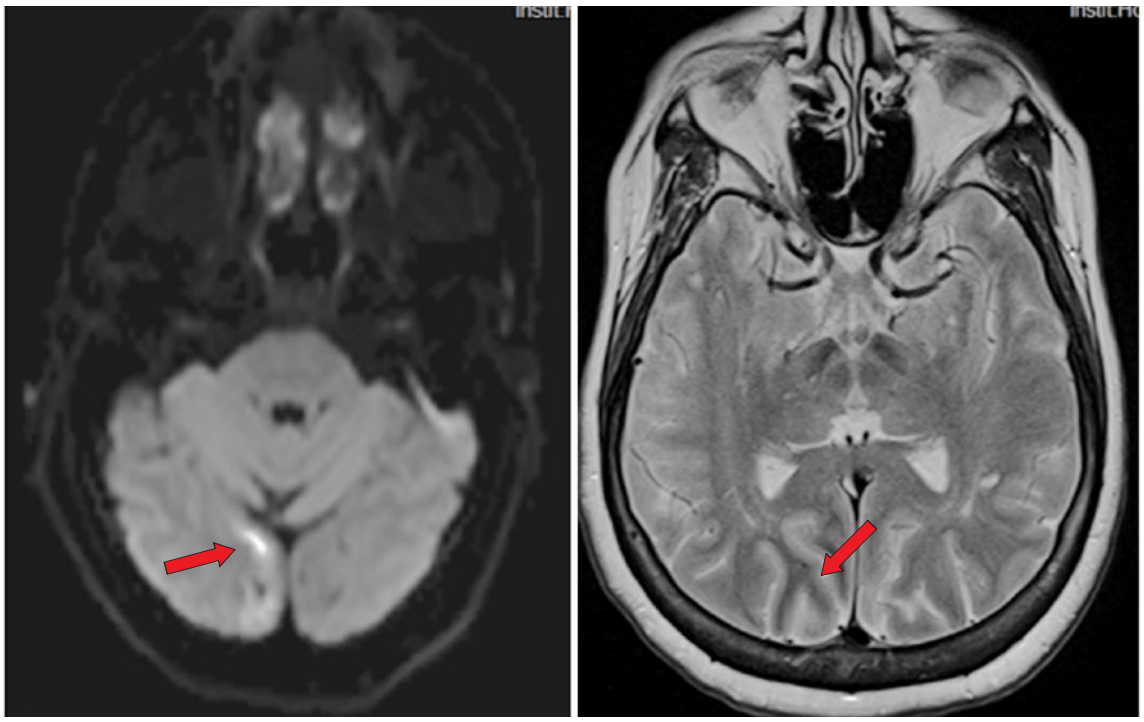

Figure 2: Hyperintensity on diffusion-weighted imaging in the right occipital cortico-subcortical region and T2 hypointensity in the right occipital juxtacortical white matter.

this condition are not fully understood. Potential contributing factors include an increased threshold for neuronal excitability due to elevated glucose levels, as well as an imbalance in intracellular glutamate and gamma-aminobutyric acid (GABA), a stabilizing neurotransmitter that is decreased in NKH patients, promoting a proconvulsive state.[1–3,6]

Seizures associated with hyperglycemia typically present as *epilepsia partialis continua*, a form of motor seizure. Occipital seizures have also been reported in patients with NKH, with glucose levels ranging from 200 mg/dL to over 500 mg/dL. However, these hyperglycemia-induced occipital seizures are mainly described in case reports.[3]

Patients may present with visual defects, most commonly HH (87.8%), which is typically bilateral (84.6%) and accompanied by positive visual symptoms (73.2%). In our patient, the presentation was consistent with this pattern, as she exhibited homonymous left hemianopia with associated photopsia. Seizures can also occur in 15%-40% of patients, primarily in the occipital lobe.[3,5] This aligns with our findings, as the patient experienced focal epileptic seizures with leftward cephalic and ocular movements, confirmed by EEG, which showed right parieto-occipital involvement.

MRI findings may be normal or reveal minor abnormalities, such as low-intensity white-matter signals (due to hyperosmolarity causing fluid loss from cells) or high-intensity gray-matter signals on T2-weighted and FLAIR images. Diffusion-weighted images may show restricted diffusion, predominantly in the posterior hemisphere contralateral to the hemianopia.[1,2,5] In our patient, MRI revealed a hyperintense lesion on diffusion-weighted imaging with restricted diffusion in the right occipital cortico-subcortical region, consistent with a postictal state. These findings are typically reversible, which was confirmed on follow-up imaging after glycemic control was achieved. EEG may detect epileptiform discharges, mainly in the occipital region. In this case, EEG confirmed focal epileptic activity in the right hemisphere, reinforcing the diagnosis of hyperglycemia-induced seizures.[1,5]

The primary goal of treatment is to control blood glucose levels, as most symptoms tend to be resolved following this. However, in some cases, antiepileptic drugs may be necessary to control symptoms and resolve abnormalities on the EEG, as seen in our patient.[1–3] Despite initial resistance to treatment, a combination of levetiracetam, phenytoin, and lacosamide was necessary to control epileptic activity, allowing for the gradual tapering of medication before discharge. Most patients recover completely after achieving glycemic control.[1–3,5]

NKH has been associated with visual field and neurological alterations assumed in the context of metabolic encephalopathy with specific MRI changes, with or without epileptic activity, typically reversible with glycemic control. Early recognition of this condition is essential, as prompt intervention can prevent complications and enhance patient outcomes. This underscores the importance of maintaining vigilant monitoring of glycemic levels, as the neurological prognosis is highly favorable with early diagnosis and appropriate treatment. ■

Awards and Previous Presentations / Prémios e Apresentações Anteriores

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Declaração de Contribuição

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