Case Report
Occipital seizures and persistent homonymous hemianopia with T2 hypointensity on MRI in nonketotic hyperglycemia

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A B S T R A C T

Introduction: Nonketotic hyperglycemia (NKH) is known to cause focal motor or secondarily generalized seizures. Occipital seizures in NKH are seldom reported, especially with visual hallucinations and persistent homonymous hemianopia (HH) with characteristic radiological and electroencephalographic (EEG) findings.

Summary: Our patient was a middle-aged man who presented with a new onset, single episode of generalized tonic–clonic seizure and NKH. He complained of seeing intermittent colorful stripes in his right visual field. Examination revealed persistent complete right HH and he was observed to have complex partial seizures. Magnetic resonance imaging (MRI) showed subcortical T2 hypointensity within the left occipital lobe in T2W and FLAIR images. The EEG showed electrographic seizures originating from the left occipital region. Random blood glucose at presentation was 581 mg/dl with HbA1c of 11.4%; the seizure and visual field deficits were successfully terminated by the introduction of antiseizure medication and glycemic control.

Conclusion: Occipital seizures with visual field deficits can occur in hyperglycemic states. These can be associated with specific MRI brain and EEG changes. The HH is reversible with apt treatment primarily including glycemic control with or without antiseizure medication.

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1. Introduction
Nonketotic hyperglycemia (NKH) signifies presence of high blood sugars in the absence of ketosis or hyperosmolar state. It is known to cause focal motor seizures with or without secondary generalization. Only a few case reports describe occipital seizures in NKH associated with a particular finding on MRI brain and EEG.

1.1. Case report
Our patient was a 53-year-old Indian male, known to have hypertension, type II diabetes mellitus, and chronic kidney disease. On the day of presentation, he had complaints of visual impairment in both eyes, followed a few minutes later by a single brief episode of generalized tonic–clonic seizure. There was no past history of epilepsy. He denied alcohol or drug abuse. However, he had a history of skin abscesses secondary to poor glycemic control.

In the ER, he was seen in the postictal period and was agitated and drowsy suggestive of global brain dysfunction. There were no focal neurological deficits at that time. Laboratory results were significant for hyperglycemia with random plasma glucose level of 581 mg/dl and HbA1c of 11.4%; ketones were negative, findings suggestive of NKH. Serum sodium and creatinine were 136 mg/dl and 2.9 mg/dl respectively. The cerebrospinal fluid analysis was done to rule out the possibility of concomitant inflammatory or infective process, which turned out to be normal. Nonenhanced CT brain was also unremarkable. Intravenous phenytoin was instituted in the ER along with insulin infusion and hydration.

Later, after regaining consciousness, his neurological examination showed a complete right HH. During examination, multiple episodes of complex partial seizures were observed characterized by sudden head and eye turning to the right, right-sided nystagmus, and cessation of speech and awareness. These episodes lasted around 60 s with spontaneous recovery. However, right HH persisted until Day 3. He also reported seeing vertical colorful stripes in his right visual field intermittently.
The MRI brain showed hypointensity on T2WI and FLAIR sequences as shown in Fig. 1. Because of the abnormal renal function tests, MRI brain with contrast was not performed.

During conventional scalp EEG recording, the patient had three seizures, each one lasting about 1 min, associated with high amplitude fast activity over the left occipital region with ipsi- and contralateral spread followed by gradual reducing in frequency and amplitudes in the postictal phase (Fig. 2).

The visual phenomena persisted despite optimal phenytoin doses. Therefore, oxcarbazepine was introduced following which the HH gradually disappeared after 3 days of treatment.

2. Discussion

Hyperglycemia is known to present with diverse neurological phenomena including focal seizures including epilepsia partialis continua [1]. Mostly reported cases are of motor origin, with scanty reports of occipital seizures especially in the setting of NKH. The occipital seizures may have a variety of visual symptoms like visual hallucinations, blurred vision, flickering lights and objects, as well as visual field defects as seen in our patient [1].

We encountered a similar case by Putta et al., with NKH, visual hallucinations, and ictive seizures associated with similar MRI brain and EEG findings as in our patient [2]. However, in our patient, it was interesting to note that there was persistent HH lasting for almost 3 days, recovering only after initiation of a second antiseizure medication along with better glycemic control. This is contrary to the abovementioned report where seizures were controlled promptly with phenytoin and lorazepam within an hour [2]. This prolonged visual deficit has previously been reported in hyperglycemia, possibly a postictal Todd’s phenomena or epilepsy partialis continua. In the absence of motor seizure, the visual phenomenon needs careful interpretation. Possible differential diagnosis includes migraine, stroke/transient ischemic attack, or posterior reversible encephalopathy.

Occipital seizures in hyperglycemic states have been described with blood glucose levels ranging from 200 mg/dl to over 500 mg/dl. Only a few cases have mentioned the HbA1c, usually high, similar to our case, indicating poor long-term control [6]. Hence, it is suggested that occipital seizures may be a result of long-term poor glycemic control rather than an acute hyperglycemic state.

The exact pathophysiology of seizures in hyperglycemia is not known. Recently, KATP channels have been implicated in the pathogenesis of hyperglycemia-related seizures [1].

Variable EEG and visual evoked potential findings have been described including ictal, interictal, and postictal epileptiform discharges [1]. Our patient had 3 seizure episodes during the EEG with progressive electrographic changes of high-amplitude, high-frequency sharp/spike-and-waves activity in the left occipital region with secondary spread to the contralateral side (Fig. 2).

Magnetic resonance imaging brain finding is typically similar to our case, i.e., focal subcortical hypointensity on T2W and FLAIR images [3]. Donat et al. have described diminution of NAA and lipid spikes on spectroscopy [4]. Underlying mechanisms for these changes are not yet known. The postulated mechanisms include a free radical injury with iron deposition due to hypoxic ischemic injury (not supported by an animal model), transient seizure effect (not supported by the typical MRI findings in postictal states i.e., hyper intensity in the affected cortical and subcortical regions on T2WI), and intracellular dehydration due to the seizure.

The mainstay of treatment is good glycemic control, hydration, and use of antiseizure medication although the need for long-term use is not established yet. Notably, phenytoin has been known to worsen hyperglycemia, and in some reported cases, it failed to control the seizures as well, like in our patient. Our patient continued to have recurrent seizures after initiation of phenytoin that later remitted promptly with initiation of oxcarbazepine.

3. Conclusion

Occipital seizures can present in NKH with specific MRI and EEG findings. A persistent visual field defect could represent a postictal Todd’s phenomenon or epilepsy partialis continua. Treatment includes glycemic control and antiseizure medications. The optimal duration of antiseizure medications remains unknown considering the symptomatic nature of these seizures, due to a potentially reversible cause. Prompt recognition is thus crucial in order to begin timely management,
thus, prevent consequences like complex partial status epilepticus and neuronal damage.

**Conflict of interest**

We report no conflict of interest.

**References**

[1] Moien-Afshari F, Ilez-Zenteno JFT. Occipital seizures induced by hyperglycemia: a case report and review of literature. Seizure 2009;18:382–5.

[2] Putta SL, Weisholtz D, Milligan TA. Occipital seizures and subcortical T2 hypointensity in the setting of hyperglycemia. Epilepsy Behav Case Reports 2014;96–9.

[3] Chen CCI, Chai JW, Wu CH, Chen WS, Hung HC, Lee SK. Neuroimaging in seizure patients associated with nonketotic hyperglycemia. Neuroradiol J 2011;24:215–20.

[4] Donat A, Guilloton L, Bonnet C, Depreux C, Lamboley JL, Drouet A. Partial visual seizures induced by non-ketotic hyperglycemia: magnetic resonance imaging and visual evoked potential descriptions. A study of two cases reports with radiologic and electrophysiologic abnormalities. Rev Neurol 2013;169:154–61.