

Hyperglycemia-induced Nonconvulsive Status Epilepticus (NCSE) and Cranial Magnetic Resonance Imaging Results: A Case Presentation



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Hiperglisemi ile İndüklenen Non-Konvülfiz Status Epileptikus ve Kraniyal Manyetik Rezonans Görüntüleme Bulguları: Olgu Sunumu

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Summary

Epileptic seizure occur in up to 25% of cases of, non-ketotic hyperglycemia (NKH). These seizures are the first finding of diabetes mellitus in 50% of the patients. The most common epilepsy is known as epilepsia partialis continua (EPC), Occipital lobe seizures and aphasic seizures may also be seen in these patients. The recovery from seizures can be with the correction of hyperglycemia. The cranial magnetic resonance imaging (MRI) findings of hyperglycemia induced status epilepticus (SE) and/or epileptic seizures have been described; however, their significance and underlying mechanisms have not been understood clearly. Although there is a consensus about the acute treatment of status epilepticus, insufficient information is available about the acute symptomatic SE treatment. Here we described the clinical and the radiological findings of a patient with NKH who was diagnosed with nonconvulsive status epilepticus in the light of the literature.

Keywords: Hyperglycemic hyperosmolar nonketotic coma; magnetic resonance imaging; status epilepticus.

Özet

Non-ketotik hiperglisemide (NKH) %25'e varan sıklıkta nöbetler görülür ve hastaların %50'sinde nöbetler diyabet hastalığının ilk bulgusudur. En sık epilepsiye parsiyalis kontinua (EPK) görülürken, oksipital lob kaynaklı nöbetler ve afazik nöbetler de görülebilir. Nöbetler hipergliseminin düzeltilmesi ile düzelir. Hem status epileptikus (SE) hem de hiperglisemi ile indüklenen nöbetlerle ilişkili kraniyal manyetik rezonans görüntüleme (MRG) bulguları tanımlanmıştır ama bunların önemi ve altında yatan mekanizmalar anlaşılabilmiştir. Status epileptikusun akut tedavisi üzerinde konsensus sağlanmış olsa da özellikle akut semptomlu SE'lerde idame tedavisinin nasıl düzenleneceği konusunda yeterli bilgi bulunmamaktadır. Bu çalışmada NKH'si olan, non-konvülfiz status epileptikus (NKSE) tanısı konan ve kraniyal MRG bulguları olan bir hasta bildirilecek ve literatür eşliğinde tartışılacaktır.

Anahtar sözcükler: Hiperglisemik hiperosmolar nonketotik koma; manyetik rezonans görüntüleme; status epileptikus.

Introduction

Hyperglycemia may be associated with several neurological symptoms such as hallucinations, choreoathetosis, hemiballismus, dysphagia, somatosensory symptoms, nausea

and vomiting, headache, and even coma.^[1,2] NKH is a clinical syndrome with severe hyperglycemia, hyperosmolarity, and intracellular dehydration without ketoacidosis.^[1,2] Seizures occur in up to 25% of NKH, and these seizures are the first finding of diabetes mellitus in 50% of patients.^[3] The most

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common type of seizure in NKH is *epilepsia partialis continua* (EPC). Thus, these seizures are predominantly originate from the frontal lobe.^[4] Hyperglycemia-related occipital lobe seizures^[4–8] and aphasic^[9–12] seizures have also been reported. Seizures due to hyperglycemia are usually resistant to antiepileptic drugs and improved with correction of hyperglycemia.^[4]

Typical magnetic resonance imaging (MRI) findings of hyperglycemia-induced seizures are; focal subcortical T2 hypointensity in the posterior cerebral hemisphere, gyral swelling, and contrast retention in the surrounding meninges and diffusion restriction.^[5–7,13,14] Also, MRI changes triggered by status epilepticus (SE) are mostly restricted by focal diffusion and T2 hyperintensity.^[15–18]

SE has high morbidity and mortality rates if untreated. The first step in the treatment is the administration of intravenous benzodiazepine. Irrespective of the response to the treatment, it is usually advised to start a second antiepileptic drug immediately to prevent the early recurrence of SE, which may occur with the wearing off of the benzodiazepine effect.

Phenytoin/Fosphenytoine, valproate, levetiracetam (LEV), lacosamide, or phenobarbital can be chosen as the second drug that can be intravenously administered and rapidly titrated.

Despite no evidence of the superiority of these drugs to each other in terms of efficacy, the selection is made according to the etiology and presence of accompanying comorbid conditions. The first two steps are considered as refractory SE, which does not respond to treatment, and coma induction is performed with midazolam, propofol, or pentobarbital.^[19]

Here we describe a case of NKH, who was diagnosed with focal nonconvulsive status epilepticus (NCSE) with electroencephalogram (EEG) and the MRI changes due to SE.

Case Report

A 65-year-old female patient who had no similar medical history admitted to the the emergency clinic with several complaints such as drowsiness in the last 2–3 days, inability to identify her relatives, and difficulty in understanding. Her blood sugar, sodium level, and serum osmolarity were detected as 359 mg/dL, 131 mm/L and 295 mOsm/L, respectively. She was diagnosed with nonketotic hyperglycemia. Also, a neurology consultation was requested because the patient had mental fog and her nystagmus was recognized by the emergency physician. No additional feature was found in the patient except confusion. The patient had no signs of fever and meningeal irritation. Moreover, she had no leukocytosis and C-reactive protein level is normal Contrast-enhanced cranial MRI revealed T2-weighted signal in

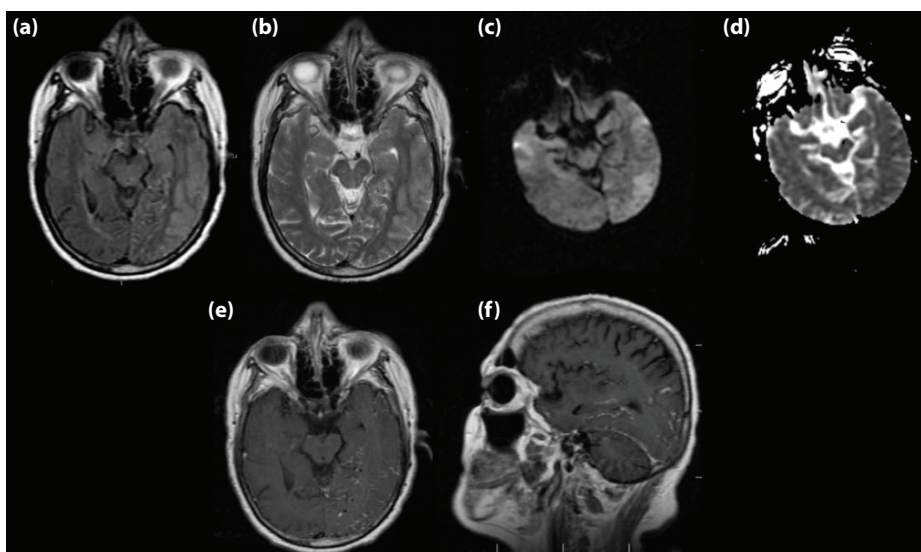


Fig. 1. A glare was observed in the left temporo-occipital region with (a) T2, (b) FLAIR, and (c) DWI in the cranial MRI taken 2–3 days after the onset of complaints. (d) No change in the same region with ADC. (e and f) A gyral-type contrast involvement consistent with leptomeningeal involvement was seen in the same region at T1 contrast examination.

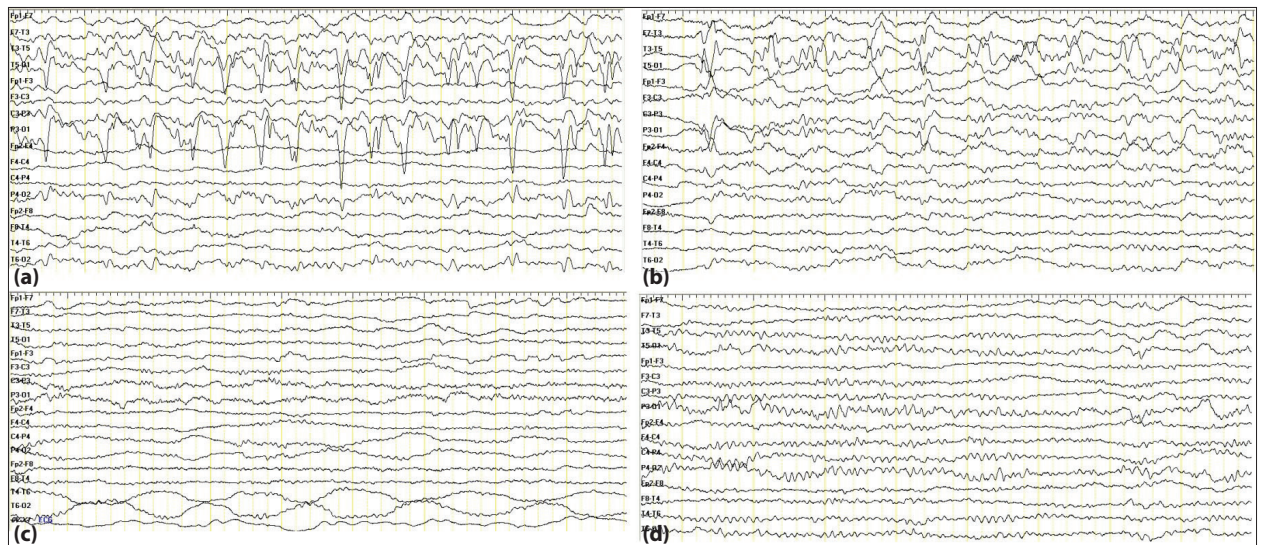


Fig. 2. (a) A continuous spike and slow wave activity were seen in the left temporo-occipital region in the first EEG performed 3–4 days after the onset of complaints. The patient was in a confused state at that time and did not identify her relatives. The patient was diagnosed with focal NCSE. (b) The EEG repeated 4 days after the first round showed marked decrease in spike and slow wave discharges in the left temporo-occipital area. At that time, the patient who was clinically better and started identifying her relatives had visual hallucinations. (c) The results of EEG repeated 7 days after the first round were within normal limits (except that the amplitudes of the alpha waves were low). At that time, the patient completely recovered. (d) The EEG was completely normal after 2.5 months, and the patient had no complaints.

the left temporo-occipital region. Hyperintensity was seen in both fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI). No change was seen in the apparent diffusion coefficient (ADC). Further, leptomeningeal contrast enhancement was seen on T1 contrast examination (Figure 1). A central nervous system (CNS) infection or NCSE was considered based on existing findings. No cells were seen in the lumbar puncture examination. The cerebrospinal fluid (CSF) protein level was 59.9 mg/dL, and

CSF glucose was 168.7 mg/dL. CNS infection was not considered. Insulin therapy was started.

EEG examination revealed a continuous spike and slow wave activity in the left temporo-occipital region (Figure 2a). The hyperglycemia-related focal NCSE was considered, and cranial MRI findings were thought to be related to SE. 2000 mg LEV was administered intravenously following 10 mg intravenous diazepam.

Four days after the first EEG the spike and slow wave discharges disappeared in the left temporo-occipital region (Figure 2b). the patient was clinically better and began to identify her relatives. However, oral haloperidol 2 mg/day was started for the visual hallucinations. The blood sugar levels were maintained between 150 and 200 mg/dL by insulin therapy. The hemoglobin A1C (HbA1C) value was detected as 14%.

The findings of EEG repeated 7 days after the first round were within normal limits; only the amplitudes of the alpha waves in the back hemisphere were low (Figure 2c). Clinically, the patient’s confused state was improved and she was recovered. Haloperidol was discontinued. The patient was discharged with insulin therapy.

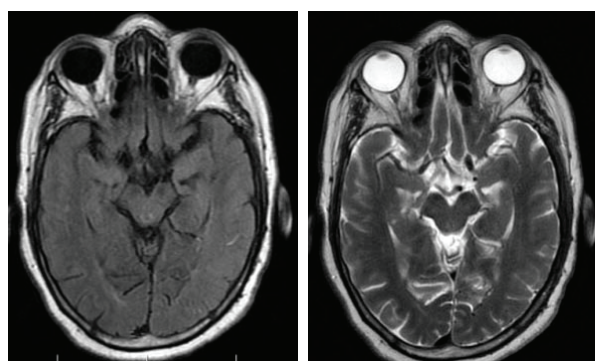


Fig. 3. The T2 and FLAIR cranial MRI (from left to right, respectively), which was repeated 2.5 months after the first round, revealed that hyperintensity in the left temporo-occipital region, which was seen in the previous MRI, completely recovered.

The cranial MRI (Figure 3) and EEG (Figure 2d) examinations repeated 2.5 months after the discharge were completely normal. The patient had no complaints. Insulin therapy was continued.

Discussion

Tiamkao et al. showed that plasma glucose levels were >290 mg/dl and osmolarity was >288 mOsm/L in patients with hyperglycemia-induced seizures. Moreover, plasma osmolarity was lower than 320 mOsm/L in this study, which was the limit for classical NKH. Furthermore, when the seizures were controlled the patients were normoglycemic or hyperglycemic; so the seizures in these patients could not be explained only by hyperglycemia and hyperosmolarity.^[4] A study showed that high plasma glucose levels played a role in uncontrolled seizures in SE.^[20] Another study on adults newly diagnosed with epilepsy showed that seizure clusters in the diabetic group were significantly higher than those in the nondiabetic group and those with worse glycemic control (HbA1C >9%).^[21] Further, prolonged uncontrolled diabetes mellitus was shown to have more dramatic acute effects on the development of seizures than blood sugar peak, and patients with HbA1C >75 mmol/mol had a significantly increased the risk of seizure repeat and clusters.^[13,22] The blood sugar and serum osmolarity of the patient in the present case were 359 mg/dL and 290 mOsm/L, respectively, and they were in accordance with Tiamkao's criteria.^[4] Also, the patient had poorly controlled diabetes (HbA1C 14%).

Focal seizures and EPC have been frequently reported to be associated with NKH.^[4] It has been shown that seizures can be reversed with hyperglycemia treatment, but they can recur with the impairment of glycemic control.^[23] Occipital seizures triggered by hyperglycemia have also been reported in the literature.^[4-8] Photopsia, bright lights, and visual hallucinations were reported in these patients.^[4,8] The prognosis was good. The clinical neuroimaging and EEG findings were usually recovered with the correction of hyperglycemia, and the long-term antiepileptic treatment was not required.^[4-6] Although carbamazepine was started, seizures lasted for 11 days in one patient. Moreover, seizures recurred after 9 months with the deterioration of glycemic control in this patient.^[5] Aphasic seizures and SE were reported in patients with NKH. In these patients, the course of disease was good, and aphasia was resolved in almost all patients; also, long-term antiepileptic treatment was not

needed.^[9-12] In only one patient, seizures were not improved with the correction of hyperglycemia and carbamazepine treatment was needed.^[9] Temporary MRI changes were seen in about half of the patients. The patient in the present case was considered to have focal NCSE originating from the occipital lobe. Nystagmus was noticed by the emergency physician on his first visit. Intravenous diazepam, LEV, and accompanying insulin treatments were given to the patient. The patient gradually recovered within 7 days.

The NCSE-associated morbidity and mortality rates are similar to those associated with convulsive SE, and the etiology is the actual determinant of the outcome for both.^[24] No systematic prospective studies are available including all types of NCSE on different treatment protocols and outcomes, but a treatment algorithm similar to that for convulsive SE is recommended.^[25] The underlying etiology of acute symptomatic NCSE or initial onset complex partial SE are usually metabolic disorders or sepsis. Since it is usually seen in intensive care patients, it is categorized in the "case of NCSE in critically ill" group.^[25,26] The treatment of this NCSE type is difficult, and the morbidity that would be caused by the treatment should also be considered.^[27] The patient in the present case developed hyperglycemia-related acute symptomatic NCSE; however, NCSE of the patient could not be evaluated as "NCSE in critically ill." Considering the morbidity and mortality that NCSE could cause if the early treatment was not initiated, intravenous benzodiazepine with hyperglycemia treatment was immediately given to the patient. Since phenytoin increased hyperglycemia,^[3] we preferred intravenous LEV. The patient's mental fog was not severe, and we did not performed coma induction due to the the potential side effects.

One study concluded that having the first seizure in the form of SE increased the risk of seizure repeat, but acute symptomatic SE did not increase the risk.^[28]

Long-term antiepileptic treatment was not needed in the case of avoiding provoking factors in the first acute symptomatic SE,^[25] but a short-term prophylactic treatment might be given until the clinical status became better.^[29] However, another study reported that SE recurrence risk was 31.7% in the patients who had their first SE and acute symptomatic causes such as metabolic disorders,^[30] suggesting that antiepileptic drugs (AED) treatment might need to be started when the first seizure was SE. SE should

be treated as soon as possible regardless of the cause, but it is not known whether long-term treatment is required especially in acute symptomatic SE. Also, long-term antiepileptic treatment was not required for our patient.

Hallucinations, illusions, and delusions may be associated with simple and complex partial seizures. Especially elemental hallucinations are closely related to primary sensory areas, and hence have highly localized values.^[31] They generally lead to positive symptoms (such as seeing bright lights) and are associated with cortical excitation, but they can also lead to negative symptoms (hemianopia) caused by the loss of direct function or inhibitor nets.^[32–34]

Hallucinations can be short-lasting and self-limiting, but they can also be continuous and associated with EPC^[35] in cases of consciousness and with NCSE in cases of unconsciousness.^[36,37] Antiepileptic drugs can be used for the treatment of ictal hallucinations whereas antipsychotics can be given in addition to antiepileptic drugs in the presence of postictal hallucinations or psychosis.^[32] However, it should not be forgotten that almost all antipsychotics have mild epileptogenic properties.^[38] The onset of visual hallucinations after a clear improvement in the EEG of the patient in the present case suggested that they might be postictal hallucinations. Our patient took 2 mg/day haloperidol during the course of admission. However, it was also possible that visual hallucinations in the patient might be seizures, so antipsychotics should be used carefully in epileptic patients.

Various ideas have been suggested to explain why focal seizures are seen in NKH. It has been reported that acute or chronic focal lesions in the brain^[2,35] and thrombus-related focal flow reduction in arterioles or venules^[2] may predispose to the development of focal seizures in hyperglycemic patients. However, it has also been reported that brain images of patients may be normal^[39] and hyperglycemia may result in focal seizures leading to focal ischemia without permanent damage.^[40] The patient in the present case had focal NCSE. Some SE-related changes were detected in cranial MRI, and these changes were resolved in repeated MRI after 2.5 months. Apart from this, no structural lesion was reported.

It has been suggested that hyperglycemia in NKHD leads to seizures by lowering gamma-aminobutyric acid levels and seizure threshold. This is supported by few seizures in

hyperglycemia accompanying ketosis.^[8,41–43] Moreover, it has been suggested that the ATP-sensitive potassium channels, which close and prevent potassium export and cause membrane depolarization and insulin secretion in response to excess glucose entering the pancreatic cells,^[44] may be present in the brain. Neuronal hyperexcitation may occur when these channels close in hyperglycemia, suggesting that the distribution of these channels may be related to the origin of focal seizures.^[8] The focal subcortical T2 hyperintensity on cranial MRI is common in seizures triggered by hyperglycemia,^[22] but is rarely seen on postictal MRI;^[15] this may be due to the accumulation of free radicals and iron.^[14] If the increase in compensatory cerebral blood flow during SE does not meet the needs of the epileptic tissue, pathophysiological changes may lead to cytotoxic edema,^[45] destruction of blood–brain barrier, and possible cell death due to vasogenic edema.^[46] An increase,^[47] reduction,^[48] or both may be seen in ADC depending on the presence of cytotoxic or vasogenic edema in the epileptogenic area.^[49] Cortical or leptomeningeal contrast involvement is a rare finding associated with seizures^[50] and has also been shown in patients with NKS.^[6,14] Focal cortical perfusion enhancement in the epileptogenic area and disruption of the blood–brain barrier are the possible mechanisms.^[7,23,45,46] The left temporo-occipital region was hyperintense on T2, FLAIR, and DWI in the cranial MRI taken 2–3 days after the onset of symptoms. ADC remained unchanged. These findings suggested to consider vasogenic edema. Moreover, leptomeningeal involvement was observed in T1 contrast examination in the same region, suggesting an increase in focal cortical perfusion in response to hypermetabolism in the epileptogenic center. No T2 hypointensity was seen. The cranial MRI repeated after 2.5 months showed complete recovery of the lesions. These cranial MRI changes were thought to be transient MRI changes associated with SE.

Metabolic disorders are expected to lead to global neurological deficits, but it should be remembered that hyperglycemia may lead to focal NCSE and MRI changes, and also differential diagnosis with CNS infection is necessary, as our patient. It should be kept in mind that the findings of cranial MRI in patients having seizures may be the lesions caused by the seizures. Whatever be the cause, when SE is present, intravenous benzodiazepine and antiepileptic drugs should be administered immediately to prevent morbidity and mortality besides the treatment of the causative agent, and then an intravenous antiepileptic drug loading should

be done to prevent early SE recurrence. However, no agreement exists in the literature on the idiopathic treatment.

Conflict of interest

None declared.

Authorship contributions

Concept: M.Ç.A.; Design: M.Ç.A.; Data collection &/or processing: M.Ç.A., M.M.A.; Analysis and/or interpretation: M.Ç.A., M.M.A.; Literature search: M.Ç.A., M.M.A.; Writing: M.M.A.; Critical review: M.Ç.A., M.M.A.

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