Sweet Seizures – Epilepsia Partialis Continua
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ABSTRACT

Epilepsia partialis continua (EPC) refers to focal and recurrent seizures that happen every few seconds to minutes for extended periods of time. The most common causes of these seizures are stroke, Rasmussen’s encephalitis (in children), and viral encephalitis. Metabolic disorders, like hyperglycemic hyperosmolar state (HHS), infrequently cause EPC. Correction of the HHS stops the EPC and eliminates the need for antiepileptic drugs. Synaptic transmission in the central nervous system requires normal glucose concentrations. Hyperglycemia can lower the seizure threshold, and this possibly explains the development of seizures in patients with HHS.

Key words: Hyperglycemic hyperosmolar state, epilepsy partialis continua, focal seizures, diabetes

INTRODUCTION

Epilepsia partialis continua (EPC) refers to focal and recurrent seizures that happen every few seconds to minutes for extended periods of time. Cerebrovascular stroke, Rasmussen’s encephalitis, and viral encephalitis can cause EPC. Metabolic disorders can rarely cause EPC.

CASE PRESENTATION

A 65-year-old woman presented with recurrent involuntary movements of both upper extremities. This was disregarded as voluntary movements by the emergency room (ER) physician. However, these recurrent focal movements were consistent focal seizures. She was conscious and oriented and was conversing coherently during the episodes. Some of the episodes were followed by transient paralysis affecting the upper limbs. She had no prior history of seizures, and she denied any history of fever, alcohol use, or exposure to toxins or drugs. She had diabetes mellitus type 2 and had a plasma glucose of 814 mg/dl and a negative serum acetone in the ER. The measured serum osmolality was 350 mOsm/L. The involuntary movements stopped when euglycemia was reestablished. She had similar movements two days before this episode, when she was treated in the ER for hyperglycemia (plasma glucose 800 mg/dl), and these movements also stopped after treatment of the hyperglycemia. An EEG (when euglycemic) and brain MRI were unremarkable. These recurrent focal seizures with short interictal periods, associated with Todd’s paralysis, suggest that the patient had EPC precipitated by a hyperglycemic hyperosmolar state (HHS).

DISCUSSION

EPC is considered a rare form of focal status epilepticus. Its characteristic features help differentiate it from other movement disorders and myoclonic
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Hyperglycemia is a rare cause of seizures but is the most common reversible cause. However, this association is easily overlooked, or EPC may not be identified, as occurred in our patient during her initial presentation. EPC can also be the presenting symptom of diabetes. Increased metabolism of inhibitory neurotransmitter gamma amino butyric acid (GABA) leading to lower seizure thresholds has been proposed as one reason for seizures in these patients.

Experimental studies have demonstrated that a threshold glucose concentration is required for synaptic transmission. Both hyperglycemia and hypoglycemia can cause neuronal hyperexcitability and reduce the seizure threshold in experimental studies. Schwechter et al exposed slices of exposed rat endorhinal cortex-hippocampus to various concentrations of extracellular glucose and found enhanced burst amplitudes with an increase in glucose concentrations to 20mM (360 mg/dl). The exact mechanism of neuronal hyperexcitability associated with hyperglycemia is unknown. Ketosis can also lower the seizure threshold. Correction of the underlying metabolic abnormality is the preferred treatment for these patients rather than antiepileptic drugs. This is especially important since phenytoin can worsen glycemic control which could predispose patients with diabetes to HHS and EPC.

In addition to seizures, hyperglycemia/HHS can cause several other neurological abnormalities, including delirium, coma, and dyskinesias, such as hemiballismus and hemichorea. Dyskinesias are more common in Asians (86% of reported cases). Potential causes include ischemia in the contralateral putamen and/or depletion of GABA. The hyperosmolar state could cause mild ischemia of the putamen, and hyperviscosity and hyperosmolality might damage the blood brain barrier leading to small petechial hemorrhages in the putamen. This metabolic derangement also causes calcium deposition in the basal ganglia which could lead to more impairment. Hyperglycemia favors anaerobic metabolism at the cellular level. This depletes the acetoacetate in the cell which is a precursor substrate for the formation of GABA, an inhibitor of the dopaminergic neurons in the nigrostriatal pathway. Therefore, depletion of GABA leads to hyperdopaminergic state which initiates chorea. Post-menopausal women with estrogen deficiency are hypersensitive to dopamine, and this helps explain why the majority of cases occur in women in the ages 50-80 years. These dyskinesias subside slowly with the correction of hyperglycemia, although some persist for months. CT of the head and the T1 weighted image of an MRI may show hypodensity in the contralateral putamen and caudate nucleus.

Our patient had bilateral abnormal movements consistent with seizures (with postictal paralysis) and did not have any imaging abnormalities in the basal ganglia. The movements stopped with the achievement of euglycemia.

**CONCLUSIONS**

EPC is a rare form of focal seizures. Although HHS is an uncommon cause of EPC, it needs to be considered in the differential diagnosis when a patient presents with continuous focal seizures. Treatment aimed at correction of hyperglycemia helps control the seizure activity.

**KEY POINTS**

1. Hyperglycemia is an under recognized cause of seizures.
2. Hyperglycemic hyperosmolar states (HHS) can be associated with epilepsia partialis continua.
3. Correction of HHS usually treats the EPC and avoids the need for antiepileptic drugs.
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