case report

Forced eye closure-induced reflex seizure and non-ketotic hyperglycemia

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We report an uncommon case of a middle-aged diabetic patient presenting with uncontrolled diabetes and partial seizures induced by forced voluntary eye closure.
comas accompanied by severe hyperglycemia, hyperosmolarity and dehydration with minimal or no ketoadidosis. In this condition, focal motor seizures and epilepsy partialis continua (EPC) are the most common type, although generalized tonic-clonic seizures may also occur. Focal seizures have been reported in 19% of patients with NKH and focal seizures induced by eye movements have also been reported with NKH.

The pathogenesis of focal seizures in NKH has not been completely understood. While focal seizures associated with NKH are refractory to antiepileptic drugs, they may be treated with adequate control of blood glucose. However, hyperglycemia alone is not an explanatory cause of seizures. One of the suggested mechanisms is presence of a hyperosmolar gradient due to hyperglycemia between intracellular and extracellular neuronal compartments with subsequent dehydration, which may produce partial seizures. The Krebs cycle is also inhibited in hyperglycemia. This may cause an increase in gamma-aminobutyric acid (GABA) metabolism in the succinil semialdehyde pathway. Depressed brain GABA levels decreases the seizure threshold, decreasing the level of GABA, which is known to be an inhibitory neurotransmitter in the central nervous system. Due to depressed brain GABA levels, the seizure threshold decreases.

Reflex epilepsy refers to seizures that are regularly precipitated by a specific identifiable stimulus. Based on the provocation mechanism, “reflex” seizure can be divided into a simple and a complex group. Triggering factors include visual, auditory, somatosensory, mental, motor and others. In the presence of a cortical lesion, hyperglycemia can facilitate an increase in frequency of focal motor seizures. Localized tissue hypoxia may account for focal seizure activity in diabetics. On the other hand, in cases with NKH, focal motor seizures related to posture have been reported, and it was suggested that proprioceptive-induced reflex seizures were related to unstable levels of blood glucose. In these cases the seizures can result from active or passive movements of a limb, with or without any brain lesion.

Various abnormalities in ictal EEG were reported in seizures with NKH. In some cases ictal EEG demonstrated periodic lateralized epileptiform discharges correlated with clonic contractions. In some, focal seizure discharge was recorded in the frontal, parieto-occipital or temporal region, and in the others no paroxysmal activity was recorded. In our patient, ictal EEG showed discharges of high amplitude spikes and multiple spikes-waves which localized to the right temporoparietal electrodes involving the adjacent area. Photoparoxysmal response was not observed during the photic stimulation.

Eye closure and closed-eyes tend to increase spontaneous EEG paroxysmal abnormalities. Previously, the effect of darkness and proprioceptive impulses on EEG abnormalities, which occur after closing of the eyes and disappear when the eyes are opened, were reported. It was hypothesised that proprioceptive impulses generated by eye closure or the mechanisms associated with moving the eyes may trigger paroxysmal abnormalities. In our case, a partial motor seizure was occurring at the eye circle during forced voluntary eye closure at the base of NKH. It may be related to increased GABA metabolism correlated to hyperglycemia without any ischemia. It was suggested that increased activity in reflex pathways might be secondary to the possibility of decreased GABA utilization in cortical and subcortical levels. Seizures induced by forced eye closure are a rare but well-recognized form of reflex epilepsy. In our patient, these reflex focal seizures might be triggered by proprioceptive stimuli due to the pressure during strong closing of her eyes. Our patient had no photic sensitivity and, visual symptoms were not revealed by the patient during the seizure. Seizure control was achieved after she became normoglycemic and with adequate control of blood glucose, she continued to remain seizure free even by doing the movement, which previously induced the seizure. It is essential to improve metabolic deficiency for seizure control because seizures are generally refractory to anti-epileptic drugs and patients become seizure free with improvement of metabolic deficiency.
REFERENCES