LETTER TO THE EDITOR / EDİTÖRE MEKTUP

Epilepsia Partialis Continua Associated with Non-Ketotic Hyperglycemia: A Case Report

Epilepsi Parsiyalis Kontinua ile İlişkili Non-ketotik Hiperglisemi: Olgu Sunumu

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Dear Editor,

Epilepsia partialis continua (EPC) is a rare neurological condition associated with cortical cerebral lesions (central nervous system [CNS] tumors, trauma, abscess, cortical dysplasia, head trauma, cerebral infarction, intracerebral hemorrhage, cerebral abscess, and vascular malformation) and metabolic disorders (hyperglycemia, hyponatremia, uremic or hepatic encephalopathy).[1,2] The association between non-ketotic hyperglycemia (NKH) and EPC is infrequent.[3] Presently described is case of patient with EPC and non-ketotic hyperosmolar hyperglycemic state (HHS).

A 67-year-old female patient presented at our emergency department with continuous rhythmic clonic jerks (partial motor seizures) of right arm and paresis lasting for 2 days. Patient was a known hypertensive with no diabetes mellitus (DM). Head computed tomography (CT) scans appeared normal, and magnetic resonance imaging (MRI) scan with diffusion-weighted imaging (DWI) showed no acute abnormality but evidence of mild bilateral microangiopathic disease. Laboratory tests were normal apart from elevated serum glucose level of 1000 mg/dL and serum osmolality of 320 mmol/kg. Urine analysis revealed glucosuria (3+) and ketonuria (-). Blood gas parameters were normal (pH 7.42; pCO2 43 mmHg), consistent with HHS. Electroencephalography (EEG) 24 hours after admission showed rhythmic, sharp waves over frontoparietal regions. Administration of diazepam (10 mg intravenous [IV] bolus) and phenytoin (1000 mg, IV infusion, 30 minutes) on admission to medical ward had no effect. Addition of levetirasetam (2000 mg IV infusion, 20 minutes) to phenytoin 3 days later was similarly without benefit. Due to continued seizures, patient's hyperglycemia was managed with insulin and fluid replacement; clonic jerks decreased about 15 days after glucose level returned to normal. Patient remained free of seizures and discontinued taking antiepileptic after 1 month.

Pathogenesis of seizures due to metabolic disorders is not fully known.[1] Hyperglycemia is possible mechanism, precipitating EPC by reducing gamma-aminobutyric acid (GABA) levels, known to be an inhibitory neurotransmitter, and intracellular acidosis presumably decreases seizure threshold due to metabolic disturbance.[2,3] EEG abnormalities of EPC may contain focal spikes and focal slow waves.[4] However, in some cases, EEG report was normal.[3,5]

EPC may be very rare manifestation of DM and response to antiepilepitc drugs (AED) is poor. This condition should be kept in mind for early diagnosis and treatment.

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