Correspondence

Hyperglycemia: Status Epilepticus

Sir,

In type 2 diabetes mellitus, ketoacidosis is rare but hyperosmolar non-ketotic coma is more common. Neurological symptoms have been reported in hyperosmolar non-ketotic coma. However, status epilepticus has not been reported to our knowledge.

A, 60 years old male, hypertensive on regular medication, nonsmoker, not a known diabetic, brought to the hospital in unconscious state at 4.00 A.M., with the alleged history of an episode of tonic-clonic movements followed by unconsciousness during sleep at about 3.30 A.M.. After 5 minutes had another episode of tonic-clonic movements with froth from mouth and passed urine in clothes, and had third similar episode of seizure while in ambulance on the way to hospital. There was no history of any head injury, intake of any intoxicants, drugs, anti-epileptic drugs, alcohol or fever and headache. There was nothing to suggest such episode in the past.

On examination: moderately build male, unconscious, with froth from the mouth and tongue bite. Pulse 80 per minute, B.P. 130/84 mm of Hg, and Respiratory rate was 20 per minute. Pupil bilateral slightly miotic, reacting to light equally and fundus was normal. Bilateral planters were extensor without any focal neurological deficit. Chest was full of crackles. Other systemic examination was non-contributory.

During examination he had an episode of generalized tonic-clonic movements of all the four limbs and neck, managed with i/v diazepam 10 mgm. slowly, apart from general supportive measures like throat suction, O₂ inhalation and i/v line with normal saline. In next 10 minutes had another episode of seizure, again i/v diazepam given followed by phenytoin sodium infusion and systemic antibiotics.

On investigations: haemogram – normal; blood biochemistry revealed blood sugar (random) was 585 mgm%, blood urea 34 mgm% and s. creatinine 1.2 mgm%; S. electrolytes and lipid profile was normal. Urine analysis revealed sugar ++, albumin nil and without any ketone bodies. EKG suggestive of old q wave inferior wall myocardial infarction. CT head was normal.

In addition to the above treatment, he was managed as a case of hyperosmolar non-ketotic diabetic coma i.e. with i/v fluids and human regular insulin. At 6.50 A.M. again he had an episode of seizure, but of less intensity, though phenytoin sodium infusion was continued. At 11A.M. patient regained consciousness, blood sugar was 210 mgm% and patient was change over to oral phenytoin sodium from infusion. On further evaluation for seizures i.e. EEG and MRI brain was normal. Echocardiography revealed akinesia of inferior segments and carotid doppler studies were normal. After a week patient was switched over to oral hypoglycemic agents and anti-epileptic drugs were stopped, and is under regular follow up. Till date i.e. for more than 6 months patient without any AED, is still asymptomatic.

The reported case presented in status epilepticus and found to be in non-ketotic hyperglycemic coma. As the blood sugar approached to normal range, patient regained consciousness and had no seizure. After detailed evaluation not able to find any cause for seizure, moreover, patient had seizure free interval of more than 6 months without any AED, may suggest that hyperglycemia may be the cause of status epilepticus in this patient.

Available literature was reviewed, though spontaneous hypoglycemia is one of the metabolic cause of repeated convulsions, but presentation of non-ketotic hyperglycemic coma i.e. in status epilepticus is not reported so far.

References


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