Case Report

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Internal Medicine Section

A Case of Undiagnosed Diabetes Mellitus with Alcohol Dependence Presenting as Epilepsia Partialis Continua

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ABSTRACT

Non-Ketotic Hyperglycaemia (NKH) or hyperosmolar hyperglycaemic state is a rare cause of Epilepsia Partialis Continua (EPC) or any other types of seizures. However, it poses a diagnostic challenge when a previously undiagnosed diabetic patient presents with partial seizures owing to NKH. We report a 53-year-old male, alcohol dependent patient who was not a known diabetic, presenting with jerky movements of the right arm. He was initially started on phenytoin without any signs of improvement. His blood glucose was 900 mg/dL. He was treated in lines of NKH induced partial seizure with regular insulin infusion and the anti-epileptics were stopped. He showed dramatic improvement with control of blood glucose. We recommend that elderly patients presenting with first episode of partial seizure should be investigated for metabolic disorders along with imaging studies. Also, partial seizures in elderly patients with alcohol dependence should arouse suspicion for metabolic disorders.

Keywords: Hyperosmolar hyperglycaemic state, Mixtard insulin, Non-ketotic hyperglycaemia (NKH), Partial seizures

CASE REPORT

A 53-year-old male presented to the Emergency department with involuntary jerky movements of right upper limb for seven days. History revealed that he was alcohol dependent for many years. His last alcohol intake was seven days back. He was diagnosed to be a case of alcohol dependence syndrome with withdrawal seizure and was referred to Psychiatry department. On further evaluation, it was found that he stopped alcohol only after the onset of these abnormal jerky movements because it was incapacitating. The jerky movements were observed to be localised to the right upper limb. The movements were almost continuous, lasting throughout the entire day with 10 to 20 seconds interval between the bouts of jerky movements. The movements occurred even during sleep and the patient also developed dysarthria. On neurological examination, plantar reflex was indeterminate on the right side and supinator, bicep and triceps jerks were exaggerated on the same side. He had a clear consciousness and was oriented. CT scan brain revealed mild cortical atrophy and Electroencephalogram (EEG) was suggestive of partial seizure. He was diagnosed as a case of EPC and started on phenytoin with a loading dose of 1000 mg in 100 ml normal saline given over 20 minutes followed by another dose of 600 mg one hour after the first dose. The jerky movements continued unabated despite the second dose of phenytoin. However, investigations revealed Random Blood Glucose (RBS) level of 900 mg/dL. HBA1c level was 13.2%. Blood pH was 7.4 and urine and blood tested negative for ketone bodies and also there was no history of pain abdomen or vomiting. Serum sodium level was 133 meq/L, potassium was 3.5 meq/l and blood urea nitrogen level was 64 mg/dL. Calculated plasma osmolality (2{Na+} + {Glucose}/18+{BUN}/2.8) was 339 mOsm/L. A diagnosis of NKH was made and phenytoin was discontinued. Volume correction with normal saline was done and the patient was started on regular insulin infusion with a dose of 6 IU/hr and was titrated upwards. Within four days, as his blood glucose levels came under control, the jerky movements disappeared completely. The patient was discharged on mixtard insulin. On follow up his symtoms had not recurred and his fasting and post prandial blood sugar level was below 130 mg/dL and 180 mg/dL respectively.

DISCUSSION

Epilepsia Partialis Continua (EPC) is a rare epileptic syndrome considered to be the status epilepticus equivalent of simple partial motor seizures [1]. In children, it may present as progressive mental and motor deterioration and is recognised as Rasmussen encephalitis. It is progressive and usually unresponsive to treatment. It may however also present in adults where it is secondary to an underlying cerebrovascular accident, trauma, poisoning, neoplastic conditions and metabolic disturbances [2].

Among metabolic conditions a non-ketotic hyperglycaemic state as a causative factor of partial seizures has been cited in a few case reports [1,3]. A NKH may manifest in a previously diagnosed case of Diabetes Mellitus (DM) with poor glycaemic control or in a previously undiagnosed and concurrently treatment naive DM patient. NKH patients present with high blood glucose levels with no evidence of ketoacidosis. Studies estimate that around 25% of NKH patients may present with focal seizures or EPCs but most textbooks do not mention EPC as a presenting symptom of NKH [3].

Though case reports associating partial seizure and EPCs with NPH are not plenty however, the first case report dates back to 1965 by Macarrio M et al., which has been succeeded by reports by other authors [1,3,4]. Most of the focal seizures are frontal or central in origin and present as motor seizures but, cases of occipital seizures presenting with visual problems have also been reported recently [5]. The authors of these case reports suggest various underlying pathophysiological mechanisms of which most accepted are: (1) A state of cellular dehydration due to serum hyperosmolarity which increases GABA catabolism and decreases the seizure threshold [6]; (2) reversible focal ischaemia due to hyperglycaemia [7]; (3) a reversible disruption of Blood Brain Barrier (BBB) [8].

Our patient was 53-year-old which conforms with the previous finding where most cases are seen beyond the 5th decade of life [9,10]. Our case was however different in that the patient was also a known case of alcohol dependence syndrome. However, his symptoms predated the cessation of alcohol consumption making it unlikely to be a consequence of alcohol withdrawal. Also, the seizure type

was partial in nature which further goes against alcohol withdrawal seizure where seizure is mostly generalized tonic clonic type. This made us suspect some other aetiology. Some case reports suggest alcohol can directly induce a seizure or unveil a seizure potentiated by underlying metabolic or structural brain dysfunction by permanently reducing seizure threshold [11]. The direct seizure inducing ability of alcohol has been attributed to disturbances in microcirculation, hypomagnesia or other electrolyte imbalances [12]. We opine that in our case NKH was the cause of the seizure and alcohol played a contributory role.

Partial seizures in EPCs are unresponsive to antiepileptics as it was in our case and, to make matters worse phenytoin may aggravate hyperglycaemia by inhibiting insulin secretion [13]. Rehydration and glycaemic control with insulin suffices and the outcomes for this condition are usually good which was true in our patient as well.

CONCLUSION

Though NKH is a recognized entity but for it to present as EPC is rare and may pose challenges in its timely recognition and treatment. Co-morbid ethanol abuse may act as a contributing factor. However, if a proper alcohol history is not taken it may be misdiagnosed as alcohol withdrawal and treated erroneously. Also, the prevalence of DM, diagnosed and hidden cases, is precariously high and is on the rise. This means the incidence of NKH will inevitably increase. So, any elderly presenting with first episode of partial seizures needs to be evaluated for the same.

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