Movement Disorder - A Rare Presentation of Diabetic Ketoacidosis

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Abstract

Diabetic ketoacidosis is a common acute complication of Diabetes Mellitus. Diabetic ketoacidosis is known to cause impaired consciousness due to the osmolar and acid base imbalance which in turn cause central nervous system involvement. Here we report a rare presentation of Diabetic ketoacidosis with movement disorder in form of hemichorea and facial tics. The movement disorder improved with treatment of ketosis.

Introduction

Diabetic ketoacidosis is an acute complication of Diabetes Mellitus. It is associated with volume depletion, electrolyte and acid-base abnormalities. Common presenting symptoms are nausea, vomiting, excessive thirst, polyuria, abdominal pain, shortness of breath, etc.

Here we are presenting rare case of diabetic ketoacidosis as movement disorder in form of hemichorea and facial tics in a previously undiagnosed young female of Diabetes mellitus. Nonketotic hyperglycemia is an established cause of chorea and many cases have been reported. On the other hand, Diabetic ketoacidosis has rarely been reported as a cause of chorea.

Case Report

An 18 year old female, married, one parity, presented to us in casualty medicine unit in altered sensorium. As per history given by the mother and brother she had complains of irrelevant talking, abnormal movements of right arm and right side of face since one day. The onsets of these symptoms were described as gradual and then she was in altered sensorium when brought to hospital. She also had complaint of generalized abdominal pain, moderate in intensity, dull in character and without any precipitating/relieving factor since 10 days. She had no history of vomiting or aspiration. She had no significant past medical/surgical history, no history of seizure disorder, and no psychiatric illness and no significant family history.

On examination, the patient was fairly built and nourished, was not fully conscious and oriented, her Glasgow Come scale was E2M4V2.

Her vitals were: pulse rate – 138/min; BP 140/70 mmHg; RR- 34/min; afebrile; oxygen saturation- 98% on room air. Pupils were round, regular and reactive.

Her nervous system examination revealed GCS- 6, no signs of meningeal irritation, generalized increase in tone in all four limbs, with normal deep tendon reflexes and superficial reflexes. Planter reflexes were bilateral mute. She was having choreiform movements on right side of the body along with facial tics on same side.

At the time of admission her random blood sugar was 452 mg% and urine ketones were large. Complete blood count, peripheral blood smear, renal function tests, liver function tests, serum electrolytes, ECG, chest skiagram, and USG abdomen, all were within normal limits.

On the basis of the above evaluation the patient was shifted into ICU and treatment of diabetic ketoacidosis was started. After 2 days when the ketones were nil in the urine, the patient was taken on the basal-bolus insulin regime.

Even after regaining full consciousness, her choreiform movements involving the right arm and tics on right side of face persisted which could not be suppressed with voluntary effort, although marked reduced during sleep.

For further neurological evaluation detail fundoscopy by expert ophthalmologist and EEG were performed and found unremarkable.

MRI brain showed significant subcortical T2 and FLAIR hyper intensity seen predominantly in the left parieto-occipital lobe and part of left temporal lobe with subtle hyperintensity in the overlying cortex, which includes possibilities of either hyperglycemic injury or subacute hypoxic insult (Figures 1, 2, 3).

The frequency and the amplitude of choreiform movements decreased as the serum glucose approached normal levels. She was discharged on insulin basal bolus regimen.

On follow up after one month she was alert and oriented with disappearance of chorieform movements. Only the facial tics persisted which were milder in intensity as compared to when she was admitted

Discussion

Movement disorders e.g. chorea or ballismus have been reported in different states of diabetes mellitus. Despite the large number of cases of chorea being reported in nonketotic hyperglycemia, after excluding other causes diabetic ketoacidosis has been reported rarely as sole trigger for movement disorder, as seen in our case. This was confirmed by the temporal relationship between diabetic ketoacidosis and the abnormal movements.

The pathophysiology of movement disorders in Diabetic ketoacidosis remains speculative. It is probably multifactorial and hyperglycemia is undoubtedly one of the factors.

One hypothesis is related to the lowered threshold for seizure or dysfunction of the basal ganglia due to a deficiency of the inhibitory neurotransmitter gamma amino butyric acid (GABA). During hyperglycemic crisis the activity of tircarboxylic acid cycle (Krebs cycle) and glucose

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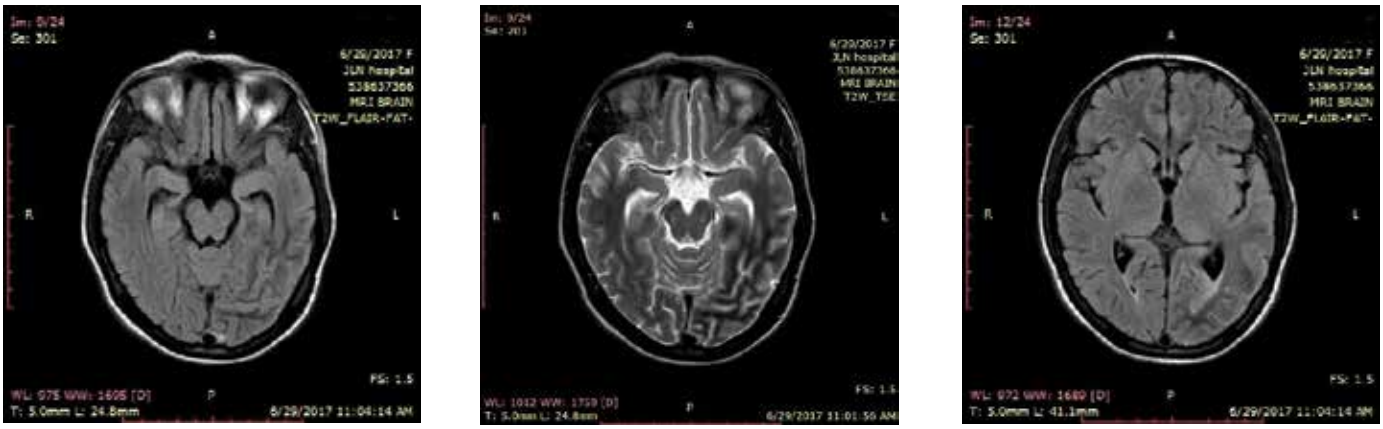

Figs. 1:(a, b, c) MRI brain images showing significant subcortical T2 and FLAIR hyperintensity seen predominantly in the left parieto-occipital lobe and part of left temporal lobe with subtle hyperintensity in the overlying cortex

utilization are depressed in the brain, so the cerebral metabolism shifts to alternative pathways.[1]

Another hypothesis involves transient focal cerebral ischemia caused by hyperglycemia. Cerebral hypoperfusion may result from an increase in cerebrovascular resistance due to the higher brain water content during hyperglycemia or to a loss of flow regulation caused by impaired metabolism.[1]

In patients with Diabetic Ketoacidosis, cerebral oxygen utilization is impaired, and there is hyper viscosity of the blood. A substantial part of the brain's energy source is derived from ketones, which themselves can depress sensorium. Extracellular hyper osomolality is present, which may also contribute to the genesis of coma. In addition, most ketoacidotic patients have associated medical conditions, which may further impair consciousness. Biochemical changes in the brains of animals with DKA include impairment of both phosphofructokinase activity and pyruvate oxidation and accumulation of citrate. The net effect upon sensorium in ketoacidotic patients probably represents the interaction of most of the above factors and differs markedly among individuals.[2]

The hyper intensity on MRI histologically corresponds to selective neuronal death and gliosis with preservation of the macroscopic structure of the brain that appear after brief ischemia. These microscopic lesions may offer an explanation why movement disorder sometimes outlast the period of hyperglycemia, as seen in our patient. The focal neurological deficits are more easily explainable in the cases of non ketotic hyperglycemia.[1]

Patients with non ketotic hyperglycemia manifest not only depression of sensorium, but also focal motor seizures, hemiparesis, and other neurologic changes, such as aphasia, hypereflexia, sensory defects, autonomic changes, and brainstem dysfunction. Most of the aforementioned changes revert to normal after correction of hyper osomolality.[2]

We suggest that multiple mechanisms are involved in pathogenesis of movement disorder in diabetic ketoacidosis. Diabetic vasculopathy with a consequent cerebrovascular insufficiency is another hypothesized mechanism but this may not hold true in our case as she is a case of new onset diabetes.[3]

Neuromuscular irritability in diabetic ketoacidosis is also ascribed to electrolyte imbalance especially hyperkalemia, hyponatremia and hypocalcemia. These may lead to localized involuntary movements at different places. Subtle basal ganglia injury can also occur in context of ketosis and is reported in literature as a complication of ketogenic diet.[4]

Conclusion

The case presenting as the choreiform movements and facial tics as the initial presentation of Diabetic ketoacidosis is a rare entity. The possible hypothesis have been cited. The treatment proven effective was the adequate management of hyperglycemia with adequate insulin regime and dietary advice.

Therefore although DKA has been rarely reported as a trigger for chorea, it should be in the differential diagnosis of a patient presenting with an acute chorea. Given the reversible nature of this disease, early recognition and treatment are imperative.

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