Hemifacial Spasm Complicating Diabetic Ketoacidosis

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Abstract
Chorea, hemichorea, hemiballismus and other parkinsonian movement disorders have been described in type 1 diabetic patient with uncontrolled hyperglycemia. In comparison, abnormal movements in diabetic ketoacidosis are rare though ketosis due to other causes can cause parkinsonism-like movement disorders. We report two cases of diabetic ketoacidosis where hemifacial spasm was the predominant clinical manifestation for which no organic cause could be detected with relevant investigations. The symptoms subsided with conventional therapy for diabetic ketoacidosis and never recurred. ©

INTRODUCTION
Various movement disorders including convulsions are known to occur in hypoglycemic states but abnormal movements in the context of hyperglycemia/ketoacidotic coma is rarely reported in literature. We report two cases of diabetic ketoacidosis where hemifacial spasm was the predominant clinical manifestation.

CASE REPORTS
Case 1
A 17 years male, diagnosed to have type 1 diabetes mellitus because of increasing thirst and polyuria 3 months back, presented with hemifacial spasm involving all the facial muscles of the left half of face and lid spasm. There was no history of fever, head injury, pain behind auricles, loss of consciousness, deafness, alteration of higher mental functions or any other neurodeficit. Patient received subcutaneous insulin for 15 days after diagnosis and stopped on his own. On examination, patient was pale, dehydrated, tachypnoeic without temperature or cyanosis. Capillary blood glucose by glucometer showed a value of 766 mg%. His urine was tested positive for glucose and ketone bodies and arterial blood gas showed metabolic acidosis (pH = 7.28, HCO₃⁻ = 14 mmol/L, lactate = 1.7 mmol/L) with hypocapnia (pCO₂ = 22 mm Hg). A diagnosis of diabetic ketoacidosis with hemifacial spasm was made. Routine hematology and biochemical profile were within normal limits. Treatment started with normal saline, soluble insulin, and later on potassium chloride. Facial spasm became gradually less in intensity with progressive reduction of blood glucose and correction of acidosis and ultimately disappeared.

Case 2
A 29 years lady, diagnosed to have type 1 diabetes mellitus 10 years back and received insulin and oral hypoglycemic agents irregularly (prescribed from outside) presented with dehydration, altered sensorium, and hemifacial spasm involving right half of face. There was neither any rise of temperature, neck rigidity, neurodeficit, nor any history of head injury or intake of oral hypoglycemic drugs in the recent past. Blood and urine examination established a diagnosis of diabetic ketoacidosis. The abnormal movement subsided with control of blood sugar and acidosis and disappeared completely once the blood sugar was brought under 280 mg% and pH normalized to 7.37. No anticonvulsant/anti cholinergic/antiparkinsonian drug was given.

In both of our patients, awake EEG record showed 8 to 10 Hz symmetrical background activity of average amplitude of 40 to 60 mV with out any paroxysmal or focal abnormality recorded. On plain and contrast CT scan, there was no focal area of altered density in supratentorial brain parenchyma. Posterior fossa structures, supratentorial ventricular systems, extra-axial CSF spaces were normal. No abnormal vascular structure was visualized. Multiplanar MR imaging of brain and brainstem using T1- wt. FSE axial and sagittal, T2- wt. FSE axial, T2- wt. FLAIR axial, T2- wt. GRE coronal, T1 -wt post- contrast axial, sagittal and coronal sequences did not show any area of abnormal signal intensity. Normal flow void signals were seen in major cerebral blood vessels in both patients. Serum electrolytes were also normal.

DISCUSSION
Hemifacial spasm is characterized by tonic and clonic contractions of the muscles innervated by the ipsilateral
The paroxysm may be induced or aggravated by voluntary or reflexive movements of the face. It is important to distinguish it from other causes of facial spasms, such as psychogenic facial spasm, facial tic, facial myokymia, blepharospasm, and tardive dyskinesia. Magnetic resonance imaging and angiography studies frequently demonstrate vascular compression of root exit zone of the facial nerve. Importantly, an underlying space occupying lesion needs to be excluded in patients with associated atypical features such as facial numbness and weakness.

Focal motor abnormalities may be the chief initial presentation of diabetes mellitus in the nonketotic hyperglycemic state in 6% of patients. Nonketotic hyperglycemia (NKH), in particular, may manifest any of a wide variety of movement disorders. These have been described as focal seizures, epilepsia partialis continua, myoclonus, and opsoclonia. There are descriptions of movement disorders in hyperglycemia that are similar to the coarse flapping tremor of asterixis, the posturing of paroxysmal kinetogenic choreoathetosis, and of “fencing (stance) seizures.” Disorders of facial motor function including aphasia, facial muscle twitching and jerking, and disorders of muscular tone have been described. Potential pathogenetic mechanisms include relative dopaminergic hypersensitivity, impaired synthesis of acetylcholine or gamma-aminobutyric acid, or an undefined effect of hyperosmolarity, perhaps unmasking a previously subclinical lesion of the basal ganglia. Few cases are due to infarct or hemorrhage in the basal ganglia or adjacent region, and MRI of the brain in patients with hemiballismus often reveals a hyperintensity lesion in the contralateral basal ganglia in T1-weighted images.

Neuromuscular irritability in diabetic ketoacidosis is also ascribed to electrolytic imbalance specially hyperkalemia, hyponatremia and hypocalcemia. This leads to localized involuntary movements at different places. Subtle basal ganglia injury can also occur in the context of ketosis, and is reported in literature as a complication of ketogenic diet.

Spasm in both cases, lasted for few hours, occurred only in the stage of uncontrolled hyperglycemia, and subsided with control of ketosis and hyperglycemia. Both of them did not require any specific drug, and the abnormal movement never reappeared in follow up. As no specific pathologic lesion could be detected in our patients, we conclude that hemifacial spasm in both cases occurred as a complication of hyperglycemic ketoacidotic state though it was not possible to establish whether hyperglycemia and ketosis together or in isolation could contribute to the movement abnormality.

**REFERENCES**


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**Golden Jubilee Year Celebrations of Diabetic Association of India**

The Diabetic Association of India founded in 1955 is celebrating its Golden Jubilee Year and to commemorate the occasion, an International Congress on Diabetes is being organised in the month of November 2005, 25th – 27th, at Hilton Towers (Oberoi Hotel), Nariman Point, Mumbai – 400 021.

International faculty consists of recognized scientists from U.S.A., U.K., France and other countries. National faculty consists of Senior Diabetologists with long years of research background.

This Congress is for the people who are interested in the ‘current trends’ in the management of Type II Diabetes and its Cardio-vascular complications. Interested delegates are requested to contact the Organising Secretary for the registration at the following address:

The Organising Secretary, Diabetic Association of India : Golden Jubilee Year International Congress on Diabetes S.L.Raheja Hospital Road, Mahim, Mumbai 400 016. Tel : 91-022-5652 9999; Fax : 91 022 2444 9418; E-mail : dai_golden@yahoo.com