

Case Report

**NON-KETOTIC HYPERGLYCEMIC HEMICHOREA –
A RARE PRESENTATION**

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ABSTRACT

Diabetes mellitus is a common condition where physicians come across daily. Though the management of diabetes is quite a simple task for physicians, it occasionally manifests in a very uncommon patterns very difficult to correlate them with diabetes, one such being the hemichorea. Hemichorea alone can be the sole manifestation of a variety of non-neurological conditions, such as metabolic derangements. Hemichorea as the first presentation of type 2 diabetes mellitus has been very rarely described. This case depicts the association highlighting that especially in elder patients with newly diagnosed hemichorea, non-ketotic hyperglycemia should promptly be recognized even though CT and MRI scan of brain might be normal.

Keywords: *Diabetes Mellitus, Non-Ketotic, Hemichorea*

INTRODUCTION

Chorea is an irregular, poorly patterned, involuntary movement disorder. Movement disorders like hemichorea are generally caused by genetic disorder, infections, drugs, endocrine disorders, vascular disorders and stroke. Here we report a case of hemichorea presenting as a sole manifestation of type 2 diabetes mellitus. The main purpose to present this case is hyperglycemia should always be kept as a cause for hyperkinetic movements especially in older people even though patient CT/MRI brain is normal. Proper and immediate control of hyperglycemia with or without neuroleptic drugs is the mainstay of the treatment.

CASES

A 55 years old male patient, a known case of systemic hypertension for one year on regular treatment of tab amlodipine 10 mg once daily, not a known case of type 2 diabetes mellitus, coronary artery disease, cerebro-vascular accident has presented to our emergency room with chief complaints of involuntary purposeless movements of right upper limb and lower limb for the past 1 week. The movements were sudden in onset started in right upper limb and progressed to lower limb in a duration of 1 week, relieved during sleep and were progressively worsening. These involuntary movements were not associated with generalization or post ICTAL state and bowel or bladder involvement. No other significant past history of any other diseases or intake of neuroleptic drugs was noted. Family history of huntington's chorea or other neurological disorder was not present for this patient. No history of any specific addictions.

On general examination patient did not show any abnormalities or any neuro-cutaneous markers. Vitals were stable at the time of the admission. Patient was very much oriented to time, place, person. Neurological examination showed higher mental functions to be normal, all twelve pairs of cranial nerves intact, deep tendon reflexes normal and involuntary movements of the right upper limb and the lower limb with decreased tone and normal power and sensory system intact. Examination of cardiovascular system was unremarkable with normal S_1S_2 , no murmur in mitral area or tricuspid area or aortic area or pulmonary area. Examination of respiratory system was also normal with normal vesicular breath sounds in all areas both sides. Per abdomen examination was soft and no organomegaly. Investigations like complete blood picture was normal, urine routine was also normal, serum electrolytes were within normal limits, serum calcium and serum magnesium were normal, blood urea and serum creatinine were normal, random blood sugar was found to be 496 milligram/decilitre and estimated blood osmolality was 307 milliosmoles/litre. No ketones were detected in urinalysis and urine ph was normal. His ECG on

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admission showed normal sinus rhythm, chest X-Ray pa view was normal, computed tomography of brain plain study was normal, MRI brain plain study was normal. Initially patient was diagnosed to have hyper kinetic disorder. The patient was investigated thoroughly for various causes of hyper kinetic disorder. Finally, patient was diagnosed to have non-ketotic hyperglycemic hemichorea and was started on insulin injection and was normal without any movement disorder within two days.

The patient signed an informed consent to allow his data for publication.

Conclusion

Hyperkinesia and hyperglycemia are closely related and has complete recovery if hyperglycemia is treated. Several hypotheses have been put forward to explain this relation, but there is no clear cut explanation. Theories which were thought to be correlated with hyperkinetic movements associated with nonketotic hyperglycemia are relative dopaminergic hypersensitivity, undefined effect of hypersomolality, decrease in aminobutyric acid and hypometabolism of striatal cells due to hypoperfusion (Patil *et al.*, 2013). In a case report recently published has shown that decreased glucose metabolism in basal ganglia as one of the cause for hemichorea in hyperglycemic state (Bizet *et al.*, 2014). In our case patient's complete blood counts, renal function test, serum electrolytes were found to be normal. Both CT and MRI were found to be normal. As the other causes are ruled out in this case and rapid response of hemichorea to correction of hyperglycemia points hyperglycemia causing hyperosmolality perse as pathogenetic factor of hemichorea. Movement disorders like hemichorea and hemiballismus are one of the unusual presentation of nonketotic hyperglycemia and have a very good prognosis with rapid correction of hyperglycemia (Lai *et al.*, 1996).

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