Nonketotic Hyperglycemia Induced Dystonia in Middle Age

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Case Report

ABSTRACT

Movement disorders are clinical syndrome with either excess or paucity of voluntary or involuntary movement which is not related to weakness or spasticity (1). Causes of abnormal movements consist of neurological diseases like Parkinson's disease, progressive supranuclear palsy, drug induced, idiopathic and some metabolic causes like uncontrolled blood sugar level. Non-ketotic hyperglycemia (NKH) has been associated with various neurological disorders, and among these, chorea-ballismus is one of the most frequently observed movement disorder. A patient presented to our hospital with involuntary, jerky, unpredictable, non rhythmic, repetitive movements involving left upper and lower limb. On examination his blood sugar level was high with no focal neurological deficit. He was started on insulin therapy. As his blood sugar level came down to normal limits, his movements started disappearing.

KEY WORDS: dystonia, hyperglycemia, non-ketotic hyperglycemia (NKH),

INTRODUCTION:

Nonketotic hyperglycemia, also known as diabetic striatopathy, is a rare cause of involuntary movements as a primary manifestation of diabetes mellitus (2). it mainly affects elderly individuals, presenting as the triad of hemichorea-hemiballism, hyperglycemia, and a lesion in the basal nuclei showing a hyperintense signal on T1-weighted images. Clinical and imaging findings are typically unilateral, although they can be bilateral in up to 11.4% of cases, being potentially reversible.

CASE REPORTS:

A 55-year-old man, with involuntary, jerky, unpredictable, non rhythmic, repetitive movements involving left upper and lower limb. was admitted for movement disorders involving the left side of the body which had appeared acutely 2 weeks previously. He was conscious and alert. Neurological examination revealed choreiform and proximal athetotic movements involving left upper and lower limb. He didn't have any history of focal neurological deficit or preceding trauma or diabetes mellitus and never took medication for hyperglycemia. On examination his blood sugar level was high with no focal neurological deficit.

Laboratory studies demonstrated high glucose values (540 mg/dl) with no evidence of ketosis with normal arterial blood gas with pH (7.44) & HCO3 (22.4 mmol/l). CT-head study shows hyperdense right basal ganglia without mass effect. Patient was initiated with insulin and olanzapine and as blood sugar level came within normal limits, his abnormal movements start disappearing.

DISCUSSION:

Involuntary movements compose a group of uncontrolled movements that may manifest as tremors, tic, myoclonic jerk, chorea, athetosis, dystonia or hemiballismus. These movements can be divided into two broad categories: (a) Hypokinetic-Parkinson's disease, Hallervorden Spatz disease, progressive supranuclear ophthalmoplegia, striatonigral degeneration; (b) Hyperkinetic- dystonia (drug induced or familial), Rheumatic chorea, Huntington's chorea, Tardive dyskinesia, Tourette's syndrome, Tic disorders, Cerebral palsy, and some rare causes like metabolic causes like nonketotic

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Figure 1:Patient showing abnormal position of hand and lower limb due to \abnormal movements.

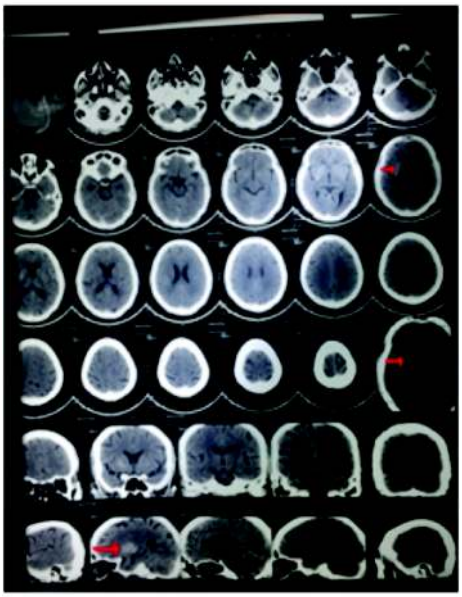

Figure 2:CT head study shows hyperdense right basal ganglia.

hyperglycemia.

Although the pathophysiology of nonketotic hyperglycemia is unknown, some proposed mechanisms include metabolic changes such as the deposition of proteins and degradation products of myelin, blood, calcium, or other minerals, which are likely to decrease as serum glucose level is controlled.

Another accepted theory is that the changes produced by hyperglycemia in perfusion results in reduced Krebs cycle activity, which induces anaerobic metabolism, causing the brain to use alternative sources of energy, and metabolizing the gamma-aminobutyric acid (GABA) inhibitory neurotransmitter. In nonketotic hyperglycemia, GABA and acetate level drops rapidly, leading to decrease in acetylcholine synthesis. It has therefore been speculated that the reduced levels of acetylcholine and GABA in the basal nuclei leads to dysfunction of those nuclei, thus producing involuntary movements such as those seen in chorea-hemiballism(4).

CONCLUSION:

In case of involuntary movements, it becomes necessary to investigate for hyperglycemia for ensuring appropriate and timely treatment. Proper diagnosis of hyperglycemia induced involuntary movements can avoid unnecessary and often ineffective initiation of antiepileptic group of medications or other unwanted medication that can cause economic burden to the patient.

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