



Nonketotic hyperglycemia induced involuntary movement's disorder, An rare presentation of diabetes mellitus

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Abstract

Diabetes mellitus most commonly presents as polyuria, polydipsia, polyphagia, autonomic and peripheral neurological manifestation, but involuntary movement are rarely seen in clinical practice. In this article we describe our diabetic patient who developed involuntary movements due to high blood sugar. The purpose of presenting this case is to bring into notice the fact that movement's disorder may be the clinical presentation of high blood sugar which reverts on its correction.

Keywords: diabetes mellitus, polyuria, polydipsia, polyphagia, autonomic

Introduction

Diabetes mellitus is the most common endocrine disorder which present mainly as polyuria, polydipsia and polyphagia. However, it can present with acute complications like diabetic ketoacidosis and nonketotic hyperosmolar coma. The neurological complications of diabetes includes stroke, altered mental status, neuropathy, seizures, visual disturbances, and movement disorders. Involuntary movement disorders like chorea, athetosis and hemiballismus are rarely seen in uncontrolled diabetes mellitus. Movement's disorder like chorea is usually caused by variety of hereditary neurological diseases, metabolic disorder, post rheumatic fever sequelae and other vascular diseases. We present a case report of 54 year old diabetic female in whom involuntary movements was the presenting symptom associated with high blood sugar.

Case Report

A 54year old female, known case of diabetes for last 13 years presented with sudden onset of involuntary movements of left upper and lower limbs for last 2 days, increased with activity and decreased with sleep. She was on oral hypoglycemic drugs but she stopped medicine by herself about 2 week ago. On examination she was conscious, cooperative, oriented to time, place and person. Vitals were normal. Cardiovascular and respiratory system examination was normal, on neurological examination no focal neurological deficit was found. Initially random blood sugar level was 507 mg/dl, urine ketone negative and arterial blood gas was normal. Patients HbA1c was -14.2%, serum triglyceride 280mg/dl. MRI study of brain revealed small dural base extra axial lesion in posterior fossa, along left petrous bone likely meningioma. Patient was started on IV insulin infusion with hourly blood sugar monitoring for glycemetic control and then switched over to subcutaneous insulin along with oral hypoglycaemic agent. Patients was kept in hospital for 5 days and patient showed

improvement in terms of involuntary movements gradually as her blood sugar improved. Follow up after 10 days of discharge showed no involuntary movement and adequate blood sugar control.

Discussion

Movement disorders related with hyperglycemia may be in the form of chorea and/or ballismus. On cranial MR examination, hyperintense lesions are observed in the putamen and caudate nucleus especially in the T1 sequence. In previously published series of HRBC cases, it was reported that lesions with different intensities could also be observed in the T2 sequence on cranial MR examination might be normal [1 2 3]. Although the pathophysiology of nonketotic hyperglycemia is unknown, potential mechanisms include metabolic changes such as the deposition of proteins and of degradation products of myelin, blood, calcium, or other minerals, which tend to decrease as serum glucose is controlled [4]. Another accepted theory is that a hyperglycemia-induced change in perfusion results in reduced Krebs cycle activity, inducing 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 levels drop rapidly, leading to a 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-hemiballismus [5 6].

Conclusion

Prognosis of hyperglycemia induced involuntary movements are excellent and correction of hyperglycemia causes reversal of involuntary movement. Screening for diabetes should be done for all patient with abnormal involuntary movements

including who have no previous history of diabetes. Our case report increase awareness about rare but treatable cause of involuntary movement and promote treatment of hyperglycemia and prevent fatal outcome.

Reference

1. Oh SH, Lee KY, Im JH, Lee MS. Chorea associated with non-ketotic hyperglycemia and hyperintensity basal ganglia lesion on T1-weighted brain MRI study: a meta-analysis of 53 cases including four present cases. *Journal of the neurological sciences*. 2002; 200(1-2):57-62.
2. Chang KH, Tsou JC, Chen ST, Ro LS, Lyu RK, Chang HS, *et al*. Temporal features of magnetic resonance imaging and spectroscopy in non-ketotic hyperglycemic chorea-ballism patients. *European Journal of Neurology*. 2010; 17(4):589-93.
3. Ahlskog JE, Nishino H, Evidente VG, Tulloch JW, Forbes GS, Caviness JN, *et al*. Persistent chorea triggered by hyperglycemic crisis in diabetics. *Movement disorders*. 2001; 16(5):890-8.
4. Hegde AN, Mohan S, Lath N, Lim CT. Differential diagnosis for bilateral abnormalities of the basal ganglia and thalamus. *Radiographics*. 2011; 31(1):5-30.
5. Aggarwal A, Bansal N, Aggarwal R. Nonketotic hyperglycemia presenting as monoballism. *The Journal of emergency medicine*. 2016; 50(3):e133-4.
6. Hansford BG, Albert D, Yang E. Classic neuroimaging findings of nonketotic hyperglycemia on computed tomography and magnetic resonance imaging with absence of typical movement disorder symptoms (hemichorea-hemiballism). *Journal of radiology case reports*. 2013; 7(8):1.