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CASE REPORT

HYPERGLYCEMIA: REVERSIBLE CAUSE OF INVOLUNTARY MOVEMENT

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ABSTRACT

Diabetes Mellitus commonly presents as polyuria, polydipsia, fatigue and polyphagia, though patients presenting with acute complications at the time of diagnosis are not uncommon. Movement disorders like chorea, hemichorea-hemiballismus, choreoathetosis are rarely associated with metabolic derangements. Hyperglycemia-induced involuntary movements (HIIM) are rarer and less well known. In this article, we describe two diabetic patients who developed abnormal movements, one developed hemichorea-hemiballism (HCHB) and the other onedeveloped nonketotic hyperglycemic (NKH) chorea which are unusual and very rare presentations.

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INTRODUCTION

The diabetes mellitus is the most common disorder of endocrine disorders. It can present with acute manifestations like diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) other than usual presentation of polyuria, polydipsia and polyphagia. The neurologic manifestations of diabetes are varied and include stroke, altered mental status, neuropathy, seizures, visual hallucinations, and movement disorders. Several movement disorders have subsequently found to associated with hyperglycemia like hemichorea-hemiballismus (HCHB) being the most common and NKH chorea. Hemichorea hemiballism (HCHB) is a spectrum of involuntary, continuous non-patterned movement involving one side of the body. Hemiballism often evolves into hemichorea. Chorea is a spontaneous, brief, semi-purposive, jerky, irregular muscle contractions, nonrepetitive or rhythmic, but flows from one muscle to the next.In our study, both patients were known case of diabetic mellitus, and their diabetes was uncontrolled. Both patients presented with different abnormal movements severity. One was having hemi chorea hemiballism and other was having NKH chorea.

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CASE REPORT

Case 1: Hemichorea Hemiballism

A 50-year-old female presented with sudden-onset involuntary movements of the right side, first involving the upper limb and 6 hours later involving the lower limb. There was no history of stroke or preceding trauma. She is a known case of diabetic mellitus from 15 years taken regular oral hypoglycaemic drugs. She was conscious and alert, with no neurological deficits. She had periodic choreiform and ballistic movements of the left upper and lower extremities that could not be suppressed voluntarily but that ceased during sleep. Initial random blood glucose of 480 mg/dL, and negative blood ketones (0.5 mmol/L), with normal arterial blood gas with pH (7.37) & Hco3 (21.5 mmol/l). The patient's HbA1C was 10.3%. Urine routine microscopy show high level of sugar (4+). An MRI study of the brain was performed in which, T1-weighted image showed hyperintensity in the right putamen (Fig 1). No significant signal abnormality was detected on T2-weighted, fluid attenuation and inversion recovery and gradient echo images except that diffusion-weighted images showed slightly elevated signal intensity. Patient was initiated with regular human insulin preparation and with long acting insulin until normal glycemia was restored. The patient's movement disorder decreased in severity and completely disappeared within 10 days after starting of the treatment. Patient was

discharged with the fixed dose of short acting regular insulin with long acting insulin. On follow up, patient's blood sugar was normal and no abnormal movement was reported.

Case 2: NKH chorea

A 70-year-oldmale is a known case unstable angina, diabetic mellitus and hypertension with status post PTCA done before. He presented with complain of flailing-like movements of his right upper extremity gradually progressed to the whole right side of the bodysince one month. The patient denied any loss of consciousness, headache, blurting vision, slurred speech, fever. He also denied taking any neuroleptic drugs. On examination, he showed spontaneous, brief, semi-purposive, jerky, irregular muscle contractions with mild hypotonia in the right upper and lower extremity with no other focal weakness in all extremities.

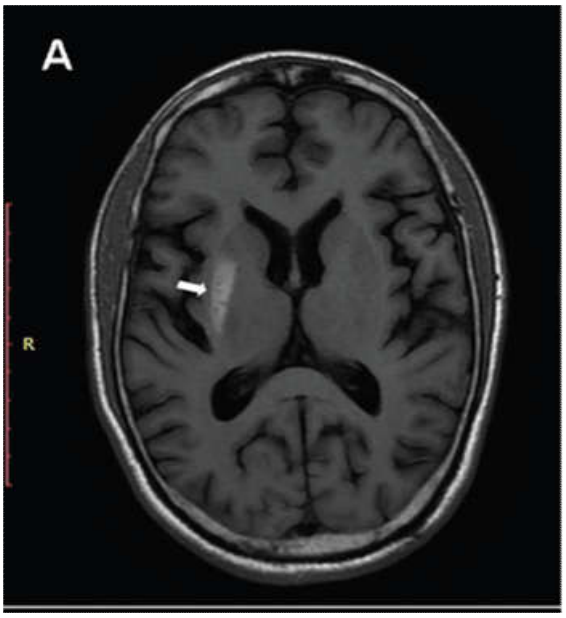

Fig. 1. Brain MRI (sagittal): asymmetric T1 hyperintensity of the left putamen (arrow)

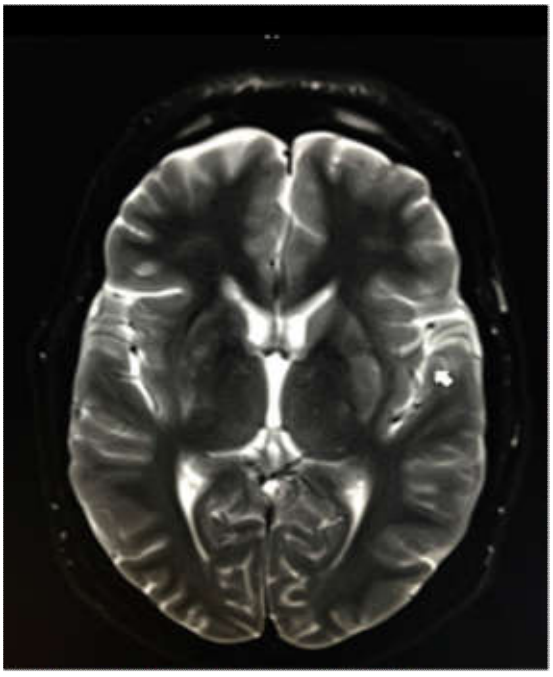

Fig. 2. Shows hyperintensity within the left putamen

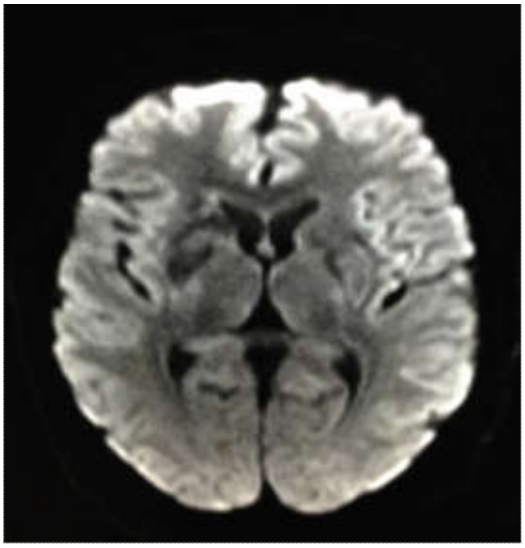

Fig. 3. Image does not show evidence of restricted diffusion

Laboratory investigations showed blood sugar 330mg/dL, with no anion gap. HBA1c was more than 13%. CT scan of the brain did not show any abnormalities. MRI showed highT2W signal in both putamina of the basal ganglia and lowT1 weighted (T1W) signal in the left putamen of the basal ganglion with no restricted diffusion on the axial diffusion weighted imaging (DWI) (Fig 2, 3). Patient was started with regular human insulin preparation and with long acting insulin. Once normoglycemia was achieved in around five days with titrated insulin therapy, abnormal movements in the lower limb disappear and almost 50% improvement was noted in upper limb movements too. Patient was discharged with fixed dose of short acting regular insulin with long acting insulin with antihypertensive and antianginal drugs.

DISCUSSION

Diabetes has wide spectrum of presentation ranging from asymptomatic incidental diagnosis, classical presentation of polyuria, polydipsia, fatigue and polyphagia to acute presentations in form of diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS). The diabetes also has many reported neurological manifestations like stroke, altered mental status, neuropathy, seizures, visual hallucinations, and movement disorders. Involuntary movements associated with hyperglycemia have been well described in the neurology literature for over 50 years. Chorea secondary to hyperglycemia was first reported in 1960 (Bedwell, 1960). Majority of the patients reported with movement disorders were Asians with mostly occurs in females of 50- 80 years of agedue to possible genetic predisposition. Since that time many similar cases reported worldwide; most of them are secondary to NKH in type 2diabetic patients (Lee *et al.,* 2001). The pathogenesis is thought to be related to hyperglycemia-induced perfusion changes in the contralateral striatum. It has been reported that hyperglycemia shifts cerebral metabolism to an anaerobic pathway and that striatal GABAergic neurons are particularly vulnerable to ischemia (Oh *et al.,* 2002). The selective loss of striatal GAB Aergic neurons may be related to disinhibition of the thalamocortical pathway, resulting in the motor cortical hyper excitability (Shan *et al.,* 1998). It was suggested that the presence of acanthocytes in circulating peripheral blood might render people with diabetes prone to develop HCHB (Pisani *et al.,* 2005). The neuroradiological findings of HCHB involve

the putamen in all cases, the head of the caudate nucleus in most cases, and the globus pallidus in a minority of cases. In both the cases, we diagnose the patient son the basis of typical clinical and radiological features. In our first patient which showed the presence of ballistic or choreiform movements, MRI finding were suggestive of hyperintensity in the right putamen. Other patient showed spontaneous, brief, semi-purposive, jerky, irregular muscle contractions with MRI finding were high signal of that both putamina of the basal ganglia and low signal in the left putamen of the basal ganglion in the setting of marked hyperglycemia and no evidence of ketoacidosis. The mainstay of treatment is aggressive control of hyperglycemic. With normalization of glycemic control, usually abnormal movement also subside. In refractory cases, additional treatment with haloperidol, risperidone, or tetrabenazine have been discussed as an option to improve the movement disorder (Battisti *et al.,* 2009). Recognition of this unique clinic-radiological manifestation is important because correction of the underlying hyperglycemia will lead to rapid improvement. The patient having NKH chorea shown improvement in form of mark reduction in abnormal movement as hypergylcemia was corrected within few days. The other patient also had significant decrease in movement after correction of hyperglycemia. Surprisingly both patients had mark improvement in lower limb movement as compare to upper limb. By reporting these cases of HIIM, we wish to highlight rare but importantly reversible etiology for abnormal involuntary movement.

Conclusion

The prognosis for HIIM is excellent and tends to be self-limited, mainly resolves within hours to days of glucose normalization. Control of hyperglycemia and normalization of metabolic status is the most effective treatment. Such case report increases the awareness about uncommon yet treatable causes of involuntary movement. So screening for such causes must be done in all cases of involuntary movement. Our case reports highlights the need for thorough clinical examination to prompt diagnosis and treatment of hyperglycemia leads to complete resolution of the symptoms and signs of both the disease to prevent further fatal complication.

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