Hyperglycemia induced dyskinesia

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ABSTRACT

Diabetes has been documented early as a noteworthy cause of involuntary movements like chorea. We report a case presenting with involuntary movements of right half of body. There was continuous rapid, dance like movements of right sided limbs, with intermittent sudden violent flinging movements of proximal limbs of same side. Lab investigations and non contrast cranial CT scan proved vital in diagnosis of hyperglycemia-induced hemiballism-hemichorea (HB-HC).Patient responded well once normoglycemia was achieved.

***Key words*:** nonketotic hyperglycemia hemiballismus, Hemichorea, Putamen

INTRODUCTION

Diabetes mellitus is common all over the world. After decades of sensitisation to both physicians and patients, it is now diagnosed early but still its complications are on rising trend. Strict glycemic control to avoid its complications is not cared of by the patients. High blood glucose can damage any organ system and most alarming complication is CNS involvement. Diabetes is a known risk factor for stroke and it can present as involuntary movements like chorea. But the fact often missed is that chorea can also be caused by hyperglycemia itself, called hyperglycemia-induced hemiballism-hemichorea. There is paucity of literature available, it is therefore, obligatory to reiterate the existence of this condition. The unique clinical and radiological characteristics of HB-HC could provide an early clue for diagnosis. Prompt treatment can produce full recovery within hours.

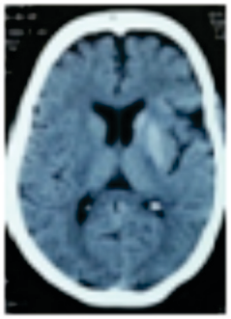
CASE REPORT

A 45 year old woman was admitted to emergency department with complaints of involuntary movements of right side of body since last 3 days, which increased in intensity and became more violent since last 5 hours. She was known case of adult onset diabetes mellitus, since last 10 years, for which she was taking oral hypoglycaemic agents but her glucose level was persistently high. There was no history of any exposure to neuroleptic drugs; hyperthyroidism; autoimmune disorders including SLE, Sjögren's syndrome; infectious disorders including HIV; or trauma. She had no family history of movement disorders. On examination, her vitals were stable. Her blood pressure was 126/74 mm Hg in right arm supine position. It was found that she had continuous rapid, semipurposeful, non patterned dance like movements of right sided limbs both proximally and distally, with superimposed sudden violent, flinging movements of proximal parts, occurring intermittently. Distal tendon reflexes, tone and power of all limbs were normal. Her plantars were flexor on left side while mute on right side. There was no cranial nerve involvement, and no sensory, meningeal or cerebellar involvement. The involuntary movements were suggestive of right sided hemiballism-hemichorea.

Investigations: Investigation showed her blood glucose level: 398 mg/dl and glycosylated hemoglobin A1C was 09.9%. Urine for ketones was negative. Other routine blood tests

including hemogram, kidney and liver function tests and serum electrolytes were within normal range. Brain non-contrast CT scan showed selective hyperintensity of the left putamen and caudate nucleus (Figure-1). The diagnosis of non ketotic hyperglycemia induced hemiballism-hemichorea was made.

Fig.1. Non-contrast CT scan of brain showing selective hyperintensity of the left sided putamen and caudate nucleus



Treatment: The patient responded partially after correction of hyperglycemia by insulin therapy. The amplitude of movements decreased within one day. Patient was also given oral tetrabenazine, 250 mg per day. The movements completely disappeared on 5th day of treatment.

DISCUSSION

Diabetes is common all over the world and India leads the world with largest number of diabetic subjects. It was estimated 31.7 million people were having diabetes in year 2000 which is projected to be 79.4 million by 2030.[1] Both the figures are highest in the world. Diabetes can lead to complications involving any body system. Hyperglycemia in diabetic patients lead to oxidative stress in central nervous system (CNS) which is important factor in the pathogenesis of neuron degeneration in CNS.[2] This could lead to hemiballism-hemichorea (HB-HC) clinically and hyperperfusion in dentate nuclei and striata leading to striatal hyperintensity on CT scan.[3] The condition was first described in humans by Rector et al in 1982.[4] Clinical manifestations can be unilateral or bilateral; if unilateral, the imaging abnormality is typically present at contralateral basal ganglia. Non ketotic hyperglycemia associated HB-HC more commonly occurs in females and most reported cases are of elderly Asians, suggesting genetic influence or inadequate diabetes control in these less developed countries.[5] The mechanism of brain damage in HB-HC associated with hyperglycemia is not completely understood. A meta-analysis on cases of non ketotic hyperglycemia induced chorea suggested synergistic effects of uncontrolled non ketotic hyperglycemia and vascular insufficiencies, which cause an incomplete transient dysfunction of the striatum and chorea.[5] A reduction in both GABA and acetylcholine in the basal ganglia, along with metabolic acidosis and the lack of energy production may then produce basal ganglia dysfunction and subsequent chorea.[5] An ischemic process is another possible mechanism of non ketotic hyperglycemia associated HB-HC.[5] An explanation of the imaging appearance on CT and MRI is still largely under debate. MR examination of previously reported cases revealed that increased density of CT corresponded with an abnormal hyper signal intensity in T1-Weighted and normal or hypo signal intensity on T2-Weighted images.[6] The continuous resolution of high density on CT scans suggest evolution of petechial hemorrhages with hemosiderin deposition as underlying pathophysiology rather than previously postulated calcification, which is unlikely to resolve over time.[6] Hyperviscosity caused by hyperglycemia can result in partial neuronal death and dysfunction of the

vulnerable striatum and may be associated with Wallerian degeneration of the internal white matter of putamen.[7,8] The protein desiccation that occurs during the course of Wallerian degeneration could explain the hyper attenuation in the noncontrast CT brain, and the hyper signalintensity on T1 Weighted and restricted diffusion in the DW images in the early phase.[8] Our patient presented with classic triad of acute onset chorea, non-ketotic hyperglycemia and hyper density on CT scan. The Involuntary movements in our patient recovered completely 5 days after correction of hyperglycemia.

CONCLUSION

Chorea associated with non-ketotic hyperglycemia should be rapidly detected as early correction of hyperglycemia produces full recovery. It may resemble stroke but active neurological intervention is not required. All diabetic patients should be sensitised towards the need for strict glycemic control to avoid these neurological complications.

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