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Case Report

Unilateral Basal Ganglia Hyperdensity in a Previously Undiagnosed Diabetic Patient Presenting with Hemichorea-Hemiballism (Hchb)

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Abstract

Non-ketotic hyperglycemia induced hemichorea-hemiballism (HCHB) is a rare condition with characteristic neuroimaging findings in a diabetic patient. This paper reports a 61-year-old male previously undiagnosed diabetic patient presenting with HCHB as the first presentation of diabetes.

Key Words: *Basal ganglia hyperdensity, Computed tomography (CT) scan, Diabetes mellitus, Hemichorea-hemiballism, Non-ketotic hyperglycemia.*

Introduction

Hemichorea-hemiballism associated with hyperglycemia is a rare and potentially reversible condition in a patient with non-ketotic hyperglycemia [1] and was first described by Bedwell in 1960 [2]. Literature describes this condition occurring most frequently in elderly patients with type II diabetes mellitus and majority are female and Asians [3, 4].

Neuroimaging studies of these patients show characteristic findings of contralateral basal ganglia hyperdensity in non-contrast Computed tomography (CT) scan, corresponding with T1 shortening in contralateral basal ganglia in Magnetic resonance imaging (MRI) [3-5].

However, HCHB as a rare presenting manifestation of type II diabetes mellitus in patients with no previous history of diabetes has been described and should always be thought of in elderly patients presenting with HCHB [5-9].

Case Presentation

A 61-year-old male presented to emergency department of our hospital with

two days history of involuntary movements of right upper and lower limbs. There was no past history of hypertension, diabetes mellitus, trauma, stroke, movement disorders or intake of neuroleptic drugs, with no family history of similar illness or other movement disorders. On examination he was fully conscious and oriented. HCHB of right upper and lower limb was present. Muscle strength and tone were normal. Blood glucose level was 494 mg/dl and HbA1c was 7.8 %. On urinalysis no ketones were detected. Diagnosis of type II diabetes mellitus with non-ketotic hyperglycemia was made and patient was referred for CT scan of brain. Non-contrast CT scan of brain showed hyperdensity of left lentiform and caudate nucleus with sparing of anterior limb of left internal capsule. No surrounding edema or mass effect was seen. There was no change in appearance of lesion on post contrast images. No other significant intracranial abnormality was seen. (Figure 1).

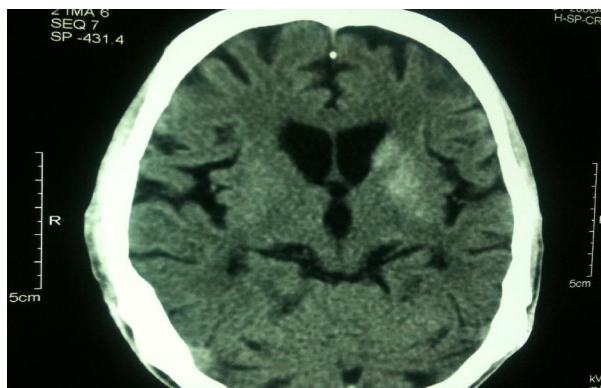


Figure 1: Non-contrast CT scan of brain showing hyperdensity of left lentiform and caudate nucleus.

Patient was treated with insulin and oral hypoglycemic agents for elevated blood sugar level. Haloperidol was given for involuntary movements. With treatment blood sugar level was restored too normal and marked symptomatic improvement was seen. After a week of treatment HCHB completely disappeared.

Discussion

Even though HCHB associated with non-ketotic hyperglycemia is mostly seen in previously diagnosed diabetic patients with poor glycemic control, there are literatures describing HCHB as a rare presenting manifestation of diabetes [5-9], as in this case. In these patients, the characteristic neuroradiological findings are seen in basal ganglia contralateral to the side of the patient's symptoms as seen in present case, with putamen involved in all cases and no isolated involvement of caudate nucleus or globus pallidus [3]. CT scan of brain shows non-enhancing hyperdensity in contralateral basal ganglia. Similarly, MRI scan of brain shows hyperintensity in contralateral basal ganglia in T1 weighted images, whereas T2 weighted / FLAIR images show hypo or isointensity. Although findings are characteristically seen unilaterally, changes in bilateral basal ganglia may be noted [3-5, 10, 11].

The exact underlying pathophysiology for radiological changes seen in non-ketotic

hyperglycemia induced HCHB are not clearly known. There are various theories for this, which includes blood hyperviscosity, depletion of gamma aminobutyric acid (GABA) and acetylcholine, ischemia, petechial haemorrhage, calcium deposition, metabolic acidosis, myelinosis, gliosis with abundant gemistocytes and manganese accumulation on gemistocytes [6, 12, 13]. Various differential diagnosis depending upon clinical and imaging findings of chorea, basal ganglia hyperdensity and T1 basal ganglia hyperintensity are described, that includes intracranial hemorrhages, cerebral ischemia, vasculitis, drugs, central nervous system lupus, multiple sclerosis, basal ganglia calcifications, chronic hepatic encephalopathy, manganese toxicity (following long term parenteral nutrition), hypoglycemic coma, neurofibromatosis, Wilson disease, Fahr disease and carbon monoxide poisoning, which can be excluded depending upon other clinical and imaging findings in these conditions [5, 13, 14].

The clinical improvement and resolution of symptoms in these patients with HCHB occurs once blood glucose level returns to normal. Also the radiological abnormalities in basal ganglia will resolve over a period of time [13].

Conclusion

Non-ketotic hyperglycemia induced HCHB may be the first presenting manifestation of type II diabetes mellitus and should be considered in differential diagnosis of patients, especially elderly presenting with involuntary movements. It has characteristic neuroimaging findings, and together with clinical findings early recognition of this condition is important as it is easily treatable and correction of hyperglycemia leads to spontaneous resolution of symptoms.

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