Brief Report

Diabetic nonketotic hyperosmolar state: Interesting imaging observations in 2 patients with involuntary movements and seizures

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We report two patients of diabetic nonketotic hyperosmolar state presenting acutely with “self-limiting hemichorea - hemiballismus” and “generalized convulsive status epilepticus”. CT scan in both the patients revealed a hyperdense nonenhancing basal ganglia. Magnetic resonance imaging brain of patient 1 showed it to be hyperintense on T1W image and iso-hyper intense on T2W image, minimally enhancing with contrast injection

Key words: CT scan, diabetic nonketotic hyperosmolar, hemichorea - hemiballismus, Magnetic resonance imaging, nonketotic hyperosmolar state, seizures.

Introduction

Diabetic nonketotic hyperosmolar (DNKH) state manifests with diverse neurological manifestations that include clouded sensorium, partial motor seizures, transient hemiplegia, chorea, hemiballismus and hemichorea - hemiballismus.[1,2]

We describe interesting neuroimaging observations (CT/MRI) in two patients of DNKH, one presenting with involuntary movements and the other with status epilepticus.

Case Reports

Case 1

A 55-year-old lady presented with history of eight days duration of sudden onset involuntary movements of right side, first involving the upper limb and six hours later involving the homolateral lower limb. She was on insulin for diabetes for the past seven years. There was no other significant history. She was conscious, alert with normal higher mental functions and cranial nerve examination. She had continuous, violent, flinging involuntary movements of the right upper and lower extremities that persisted but significantly reduced during sleep. Her random blood sugar was 292 mg% and serum osmolality was 317 milliosm/L. Urine analysis did not reveal ketones. Peripheral blood smear for acanthocytes was not performed. A diagnosis of “diabetic nonketotic hyperosmolar state” causing hemichorea and hemiballismus was made. CT brain (plain and contrast) revealed nonenhancing hyperdense caudate and putamen on the left side. On MRI brain, the lesion appeared hyperintense on T1WI and isointense on T2WI, enhancing with contrast injection [Figure 1].

Her blood sugar was controlled with insulin. She required multiple medications for these involuntary movements like sodium valproate, clonazepam and carbamazepine. These movements decreased after a week and completely subsided within a fortnight. After six months, there was no neurological deficit or involuntary movement.

Case 2

She was a 60-year-old lady; a diabetic of two years duration and was on oral hypoglycemic agents. She presented to us with history Sanjib Sinha

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Figure 1: CT scan of brain of patient 1 (a- plain, b - contrast): showing nonenhancing hyperdense left striatum; MRI of brain of same patient (a - T1W, b - T2W, c - FLAIR): showing hyperintense signal change of left striatum
of recurrent episodes of generalized tonic-clonic seizures and altered sensorium for five hours. There was no other relevant history. At presentation, patient was unconscious (post-ictal state) with Glasgow Coma score of 6. She did not have any focal neurological deficit. Her blood sugar was 543 mg/dl but urinary ketone bodies were absent. A diagnosis of “diabetic non-ketotic hyperosmolar state” manifesting as generalized convulsive status epilepticus was considered. The CT of brain (plain and contrast) showed hyperdense nonehancing left caudate and putamen [Figure 2]. MRI could not be performed. She received parenteral sodium valproate and insulin. The seizures stopped immediately and patient recovered completely within three to four days. She was lost to follow-up.

**Discussion**

Hemichorea-hemiballismus (HCHB) is a characteristic relatively benign disorder affecting the elderly and may often be the presenting manifestation of hyperglycemia.

Hyperosmolar state. Hyperosmolar coma can be seen without manifesting as hemichorea-hemiballismus as exemplified by our second patient. She had similar imaging features but presented with status epilepticus and the occurrence of seizures expend the spectrum of the clinical picture, possibly by involving her cortical neurons. However, MRI could not be performed. This raises several questions especially regarding the cause and effect relation of these imaging to involuntary movements and seizures. Whether it means an abnormal underlying dysmetabolic state needs to be considered.

Thus, HCHB is not always a self-limiting disorder and is associated with DNKH state and interesting radiological features. The cause and effect of the radiological observations and clinical manifestation are not known conclusively.

**References**

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Accepted on 22-08-2006

Source of Support: Nil, Conflict of Interest: None declared.