

# Hyperglycemia Induced Reversible Hemiballismus as the Main Presentation of Newly Diagnosed Diabetes Mellitus

Waseem Zaid Alkilani, Hassan Tahir\*, Nathan Gibb, Saad Ullah, Nagadarshini Ramagiri Vinod

Department of Internal Medicine, Temple University/Conemaugh Memorial Hospital, Johnstown, PA 15905, USA

\*Corresponding author: [hassantahir\\_01@hotmail.com](mailto:hassantahir_01@hotmail.com)

**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. Stroke and neuropathies are the most common neurological complications of diabetes. Movement disorder like chorea and hemiballismus are very rarely associated with diabetes mellitus. Primary care physicians should be aware of these rare and complicit presentation of diabetes. We present a case of nonketotic hyperglycemic hemiballismus (NHH) with no acute abnormality seen on MRI brain.

**Keywords:** *hemiballismus, hyperglycemia, nonketotic hemichorea/hemiballismus*

**Cite This Article:** Waseem Zaid Alkilani, Hassan Tahir, Nathan Gibb, Saad Ullah, and Nagadarshini Ramagiri Vinod, "Hyperglycemia Induced Reversible Hemiballismus as the Main Presentation of Newly Diagnosed Diabetes Mellitus." *American Journal of Medical Case Reports*, vol. 4, no. 7 (2016): 245-247. doi: 10.12691/ajmcr-4-7-8.

## 1. Introduction

Diabetes Mellitus is the most common endocrine disorder, which presents mainly as polyuria, polydipsia and polyphagia. However, it can rarely present initially with acute complications like diabetic ketoacidosis and nonketotic hyperosmolar coma. Stroke and neuropathies are one of the most common neurological complications of diabetes, which usually occur after long term uncontrolled diabetes. Movement disorders like chorea, athetosis and hemiballismus have many causes but are rarely seen in uncontrolled diabetes mellitus. We present a case of 59-year-old male who presented with hemiballismus as the chief complaint of new onset diabetes mellitus.

## 2. Presentation

A 59-year-old African American male with the past history of hyperlipidemia and cervical radiculopathy presented to the emergency department (ED) with new onset involuntary left upper extremity movements. He had presented to emergency a couple of days ago for evaluation of neck and left arm pain when he was found to have multilevel cervical degenerative disc and mild cord compression at C4-5 on MRI (Figure 1). He was discharged on painkillers and steroids with the recommendation to follow up with neurosurgery on outpatient basis. He came to ED two days later with worsening involuntary movements of left upper extremity. These episodes of involuntary movements started to happen more frequently and to last for longer periods, approximately 20 minutes each. He had to hold his arm

down so that it would not move erratically. He also reported difficulty ambulating, slurred speech and blurred vision occurring during the episodes.



**Figure 1.** MRI Neck showed mild cord compression at C4-5

He was hemodynamically stable when he came to ED. On neurological exam, he was found to have rapid involuntary wide flailing movements of left arm, which lasted for few minutes and were non suppressible. Pupils were equal and reactive to light; and Cranial nerves 2-12 were intact. Power was 5/5 in all extremities and sensations were intact bilaterally. Cerebellar testing showed marked dysmetria on finger-to-nose and heel-to-shin testing on the left side of the body. He had CT scan of head in ED, which did not show any acute intracranial pathology. Baseline investigations showed glucose level of 598 mg/dl and betahydroxybutyrate level of 2.27. No acidosis was found on ABGs and rests of the labs were

unremarkable. Patient did not have any history of diabetes, though he did have family history of diabetes in mother. He was experiencing polyuria and polydipsia for 2 weeks prior to presentation which had worsened since he had been started on the dexamethasone 2 days ago for his cord compression. He was given IV fluids and started on insulin.

He had extensive work up to find the cause of hemichorea/hemiballismus. He underwent MRI and MRA of brain that were unremarkable (Figure 2). Prolactin, TSH, liver function, urine drug screen and ESR were all normal. EEG was also done to rule out seizure, which came back normal with no epileptical focus. Echocardiography showed LV EF 60-65% with normal systolic function. Carotid Doppler showed mild stenosis

of the right and left internal carotid arteries in the range of 1-40%. For his recent diagnosis of mild cord compression, neurosurgery was consulted that did not feel that his symptoms in the left upper extremity were related to the findings in his cervical MRI. In the mean time, hyperglycemia was treated with insulin and subsequently a level of 200-300 was achieved throughout hospital stay. HBA1c was found to be 15%. Surprisingly his symptoms improved with less frequent episodes in next 24 hours and with complete resolution of symptoms in 2 days. His blurred vision and slurred speech episodes also resolved completely. Repeat neurological examination revealed normal motor/sensory system and cerebellar functions. He was discharged on metformin and insulin. He did not have any more symptoms on follow up in clinic.

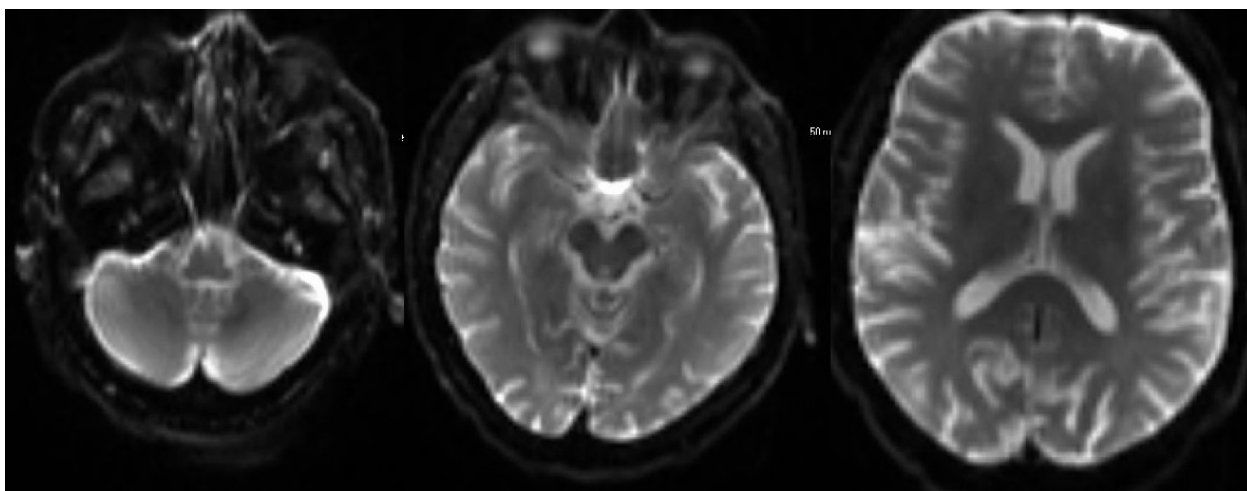


Figure 2. MRI Brain is normal

### 3. Discussion

Hemichorea-hemiballismus (HCHB) is a hyperkinetic disorder characterized by continuous, nonpatterned, proximal, involuntary movements on one side of the body, resulting from involvement of the contralateral basal ganglia and particularly the striatum. HCHB can be manifested as the first presentation or a complication of DM [1]. It is the main presenting symptom in this reported case. HCHB can be classified into primary or acquired; each contains a long list of etiologies. Hypoglycemia, hypernatremia, hyponatremia, hypomagnesemia, and hypocalcemia also represent some of metabolic portion of acquired etiologies [2]. It can also be a manifestation of hypoglycemia [3].

Non-ketotic hyperglycaemia can present with multiple neurological symptoms including hyperosmolar coma, seizures and hemichorea/hemiballismus. Other manifestations can be delirium, aphasia, hemiparesis, hemisensory loss, nystagmus, and hemianopia [4]. Non-ketotic hyperglycaemia is a rare cause of chorea/ballism and bright basal ganglia on MRI that is reported most frequently in elderly Asian females with type II DM, although it occurs in other patient population [5,6]. Underlying pathophysiology is not fully understood yet. Peticheal hemorrhage, microcalcifications, edema, reactive astrocytic, and interneuronal responses have been studied as possible etiologies. Ischemic events, blood brain barrier disruption and neuronal apoptosis all have been also implicated in the pathophysiology of this condition [7]. MRI is the

mainstay to pick putamen and/or caudate changes. Hyperintensity of T1 is the most consistent finding. Other findings include hypointensity on T2/flair and restricted diffusion on DWI. Subtle hyperdensity in striatal region can also be seen on CT scan. These imaging findings resolve but slower than clinical improvement, which can explain some cases of positive imaging findings without clinical manifestations. MRI without acute changes is also reported in non-ketotic hyperglycemic HC-HB [8]. In contrast, acute basal ganglia changes on MRI are noted in some cases of asymptomatic non-ketotic hyperglycemia [9].

Control of patient symptoms can be readily achieved by serum glucose level normalization, although radiographic evidence may take up to 6 months to resolve [10]. In refractory and severe cases, haloperidol, risperidone and possibly topiramate and benzodiazepines can be used among other agents mainly atypical antipsychotics [4,11]. Generally, prognosis is excellent with prompt identification and management.

In our case, symptoms described were typical for hemiballismus/ hemichorea in the setting of nonketotic hyperglycemia secondary to newly diagnosed DM augmented by steroid use. Control of patient symptoms was gradually obtained by controlling hyperglycemia. Extensive work up was done to find the cause of movement disorder. All laboratory and radiological investigation were unremarkable. Spinal cord compression was not a likely cause of our patient symptoms as he was also having slurred speech, blurry vision and signs of cerebellar dysfunction during episodes of abnormal arm movement.

His symptoms in the presence of hyperglycemia that got resolved with insulin treatment led to the diagnosis of hyperglycemia induced reversible hemiballismus. Interestingly, hemiballismus was the main presenting symptom of DM. Although there were no acute changes on radiographs that would suggest NHH but the presentation was typical as hemiballismus resolved almost 24 hours after initiation of insulin therapy.

#### 4. Conclusion

1. Non ketotic hyperglycemic hemiballismus is a rare presentation of diabetes mellitus which should be considered in any patient with sudden onset movement disorder.

2. Severe hyperglycemia can cause a variety of acute neurological complications such as cerebellar dysfunction and movement disorders. Brain imaging may be normal in such cases. These complications are most of the times reversible with correction of hyperglycemia.

3. Primary care physicians should be aware of rare presentations of diabetes.

#### References

- [1] Sayantan Ray et al., "Hemichorea-Hemiballismus as the First Presentation of Type 2 Diabetes," *Clinical Diabetes* 2015 Apr; 33(2): 87-89.
- [2] Bhidayasiri R1, Truong DD, "Chorea and related disorders," *Postgrad Med J* 2004;80:527-534.
- [3] Hefter, H., P. Mayer, and R. Benecke. "Persistent Chorea after Recurrent Hypoglycemia." *European Neurology Eur Neurol* 33.3 (2008): 244-47.
- [4] Mary Kate McCullen, Jeffrey Miller, Serge Jabbour, Kevin Furlong, Monika Shirodakar, Intekhab Ahmed, Steven Mandel. Expert Opinion: Chorea in the .Setting of Hyperglycemia: A case report and review of literature .*Practical Neurology*, April 2010.
- [5] Dewey RB Jr., Jankovic J. Hemiballismus-hemichorea: clinical and pharmacologic findings in 21 patients. *Arch Neurol* 1989; 46: 862-867
- [6] Wilson TJ, Than KD, Stetler WR et-al. Non-ketotic hyperglycemic chorea-hemiballismus mimicking basal ganglia hemorrhage. *J Clin Neurosci.* 2011;18 (11): 1560-1.
- [7] Yeting Zhou et al., "Hemichorea in nonketotic hyperglycemia: Putamenal and cerebellum lesion on MR imaging," *World Journal of Neuroscience*, 2012, 2, 138-140 WJNS.
- [8] D. Branca, O. Gervasio, E. le Piane, C. Russo, and U. Aguglia, "Chorea induced by non-ketotic hyperglycaemia: a case report," *Neurological Sciences*, vol. 26, no. 4, pp. 275-277, 2005.
- [9] Barry G. Hansford, Dara Albert, and Edward Yang, "Classic neuroimaging findings of nonketotic hyperglycemia on computed tomography and magnetic.
- [10] Damani A, Ghoshal A, Salins N, Deodhar J, Muckaden MA. Management of Hemichorea Hemiballismus Syndrome in an Acute Palliative Care Setting. *Indian Journal of Palliative Care.* 2015;21(1):72-75.
- [11] Dewey RB, Jr, Jankovic J. Hemiballismus-hemichorea. Clinical and pharmacologic findings in 21 patients. *Arch Neurol* 1989; 46: 862-867.