# HEMIBALLISMUS-HEMICHOREA IN A 55 YEARS OLD DIABETIC FEMALE PATIENT WITH NON-KETOTIC HYPERGLYCAEMIA

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## Article

**Introduction**

Hemiballismus-hemichorea (HC-BC) are involuntary rapid irregular jerks, invariably unilateral and affect the arms more frequently than legs1. The causative lesion is commonly found in the corpus striatum, but can occur anywhere along the afferent or efferent pathways connecting the striatum to its projection areas. Although the most common cause of HC-BC is a cerebrovascular insult in the region of the striatum and subthalamic nucleus but a variety of pathologies including tumors, neurodegenerative disorders, encephalitis, drugs, systemic lupus erythematosus, and metabolic disorders have been implicated as a cause2. Non-ketotic hyperglycaemia a in newly diagnosed or poorly controlled diabetic has been described as a metabolic case of Hemiballismus-hemichorea particularly in elderly female patients2. More dramatic ballistic movements of limbs usually occurs unilaterally in vascular lesions of subthalamic lesions3 .

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**Case Report**

A 55 years old Diabetic lady presented with a sudden onset involuntry movements on right side of five days duration.These abnormal movements initially involved the upper limb and after three hours also affected homolatreral lower limb. She was on oral hypoglycaemics for the past six months. She was hypertensive for the past six years and her blood pressure was well controlled. There was no family history of movement disorder. She was not on any medication known to cause abnormal movements, was conscious and well oriented in time and space with normal higher mental functions. All the cranial nerves were intact and there was no sensory/motor deficit. She had continuous violent flinging involuntary movements of the right upper and lower limbs used to reduce during sleep. She had no other neurological deficit. Her random blood glucose level was 471 mg/dl, urine for ketones negative , serum osmolality 282 milliosmol/L, HbA1c was 10.1%, serum sodium 138 mmol/L, serum potassium 3.5 mmol/L while Renal and Liver function were normal. Rest of other investigations including serum ceruloplasamin, RA factor, ASO titer and ANA were normal. Kayser- fleischer rings were absent and fundoscopy was normal. CT scan Brain plain and with contrast was also normal. Her blood glucose level was controlled with Insulin and oral hypogycemics. Her Hemiballismus-hemichorea improved after achieving euglycaemia and completely settled within two weeks of hospitalization. She was regularly followed up in outdoor for four months and had no recurrence of involuntry movements.

**Discussion**

Hemiballismus-hemichorea is an acute dramatic movement disorder and its severity ranges from mild chorea to the wild flinging movements of ballismus1. Hemiballismus is usually caused by infarction or hemorrhage in the contralateral subthalamic nucleus4 .

Non-Ketotic hyperglycaemia in poorly controlled new diabetics is an unusual cause of abnormal movement disorder2.

The mean HbA1c in two separate case series were 10.6% and 12.9% and our result (10.1%) is comparable to these results5,6 .Similarly the age of our patient is in line with the published mean age5,6 .

Menopause and acanthosis increase the predisposition of these patients to develop this movement disorder5,7. In menopause there is incresed dopamine receptor sensitivity secondary to oestrogen receptor deficiency5.

The pathphysiological mechanism is not well established and the most favoured mechanism is a deficiency of GABA and acetyltylcholine along with metabolic acidosis2,5. Other proposed mechanism include hyperviscosity and cerebrovascular accident2.
The characteristic imaging features in Hemiballismus-hemichorea are non-enhancing hyperdensity of the striatus8. Focal hypointensity of the left putamen both T-2 weighted and gradient echo MRI sequences implying that petechial hemorrhages and /or cytotoxin oedema may still occur in cases of Hemiballismus-hemichorea secondary to non-ketotic hyperglycaemia, either as a contributing cause of the injury or as a consequence of ischaemic damage but in our case the CT Scan brain (plain/contrast) was normal9.
In our case a combination of reduction in glucose level and the use of neuroleptic drugs resulted in the disappearance of the abnormal movements10. The patient showed improvement with in one week (as her blood glucose was controlled on Insulin therapy) and her symptoms completely disappeared with in two weeks after hospitalization. She was followed for four months in out door. Her blood glucose was well controlled and is symptom free without medication for Hemiballismus-hemichorea.

**Conclusion**

One must consider abnormal blood glucose in any patient presenting with Hemiballismus-hemichorea. Early detection of hyperglycaemia and its treatment can lead to early recovery from this distressing movement disorder.

## Reference


1.Richard K, Disorder of movement and Imbalance. Harrison, Principles of Internal Medicine, Ed 16; Mc Graw-Hill Medical Publication Division 2005;139 .
2.Demssie NY, Kaushal K. Newly diagnosed type-II diabetes presenting with HC-HB.J R Coll Physcian Edinb. 2008; 218-20.
3.Allen CMC, Lueck CJ, Dennis M . Neurological Disease . Davidson,s Principles & practice of medicine ; Ed 20th . Churchill Livingstone 2005 ;1182.
4.Charles RA, Neurological Disease . Kumar and Clark Clinical Medicine : Ed 6th; Elervier Saunders 2005 ;1232.
5.Lee EJ,Choi JY,Lee SH et al. Hemichorea-hemiballism in primary diabetic patients: MR correlation.J Comput Assit Tomogr 2002; 26:905-11.
6.Lee BC, Hwang SH,Chang GY. Hemiballism- hemichorea in older diabetic women: Clinical syndrome with MRI correlation. Neurology 1999;52:646-8.
7.Lin JJ, Lin GY,Shih c. Presentation of striatal huperintensity on T1-weighted MRI in patient with Hemiballism-hemichorea caused by non-ketotic hyperglycaemia: report of seven new cases and a review of literature. J Neurol 2001; 248:750-5
8.Shobha N,Sinha. Diabetic nonketotic hyperosmolar state.Interesting imaging observation in 2 patients with involundary movements and seizurs. Neuro Ind 2006; 54:440-2.
9.James S. MRI changes in a patient with Hemichorea-Hemiballismus and non-ketotic hyperglycaemia. Univ Alb Health Sci Jan 2009; 5:748-50
10.Bouatay S et al. Biballism due to nonketotic hyperglycaemia. Diabetes and Metabolism 2008; 34:617-19.