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Diabetic Striatopathy: Rare manifestation in Diabetics

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ABSTRACT: Diabetic striatopathy also know as hyperglycaemia Non-ketotic hemichorea – hemiballismus or chorea hyperglycemia basal ganglia syndrome (CHBGS) refers to a rare condition characterized by spontaneous, persistent involuntary jerky motions with ballisum typically being more proximal and larger amplitude than chorea. Diabetic striatopathy was defined as hyperglycemic condition associated with both or either one of two following conditions: a) Chorea or Ballism, b) striatal hyperdensity on CT or hyperintensity on T1-weighted MRI. We present 3 cases who presented with acute onset of involuntary movements involving one half of the body and contralateral limb. Diabetic striatopathy is an uncommon presentation associated with hyperglycaemic state. This can be first presenting sign of unknown diabetes mellitus or can occur after months of poor glycemic controls in patients with diabetes mellitus.

KEYWORD: Diabetic Striatopathy; Hyperglycemia; Hemiballism; Hemichorea; Diabetes Mellitus.

I. INTRODUCTION

Diabetic striatopathy (DS) also know as hyperglycaemia Non-ketotic hemichorea – hemiballismus or chorea hyperglycemia basal ganglia syndrome (CHBGS) refers to a rare condition characterized by spontaneous, persistent involuntary jerky motions with ballisum typically being more proximal and larger amplitude than chorea.

Diabetic striatopathy was defined as hyperglycemic condition associated with both or either one of two following conditions: a) Chorea or Ballism, b) striatal hyperdensity on CT or hyperintensity on T1-weighted MRI.(1)

Diabetic striatopathy is a rare manifestation reported to occur with severe hyperglycemia, and is in most cases reversed with glucose control. These patients often exhibit contralateral striatal hyperdensity (caudate/putamen or both) on computed tomography (CT) and/or a striatal hyperintense signal on magnetic resonance imaging (MRI).(2) This syndrome has been reported more frequently in elderly Asian women and symptom resolution is typically associated with serum glucose levels and plasma osmolarity normalization.(3) Very less reported cases with the Indian context, here we present 3 cases presented with Diabetic striatopathy.

Case: All clinical cases presented to emergency department of general medicine of Malla Reddy Medical Sciences, Hyderabad.

	Case 1	**Case 2**	**Case 3**
Age (yrs)	50	65	65
Gender	Female	Female	Female
Duration of DM	Detected on presentation	1 yrs	2 yrs
Duration of symptoms	4 days	7 days	2 days
Side involuntary movements	Right upper limb & lower limb, left lower limb	Left upper limb > left lower limb	Right upper & lower limb
Type of involuntary	Hemiballism +	Choreiform	Hemiballism >

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movement	Hemichorea		Hemichorea
Associated factors	Nil	Lower Respiratory infection	Nil
Response to treatment	Adequate	Poor	Adequate
Urine Ketone bodies	1+	Negative	Negative
Plasma Glucose at time of presentation (mg/dL)	>600	452	521
HbA1c (%)	18.2	13	14.8
CT findings	Left – putamen > caudate; Right – caudate	B/L – caudate + putamen	Left caudate and putamen

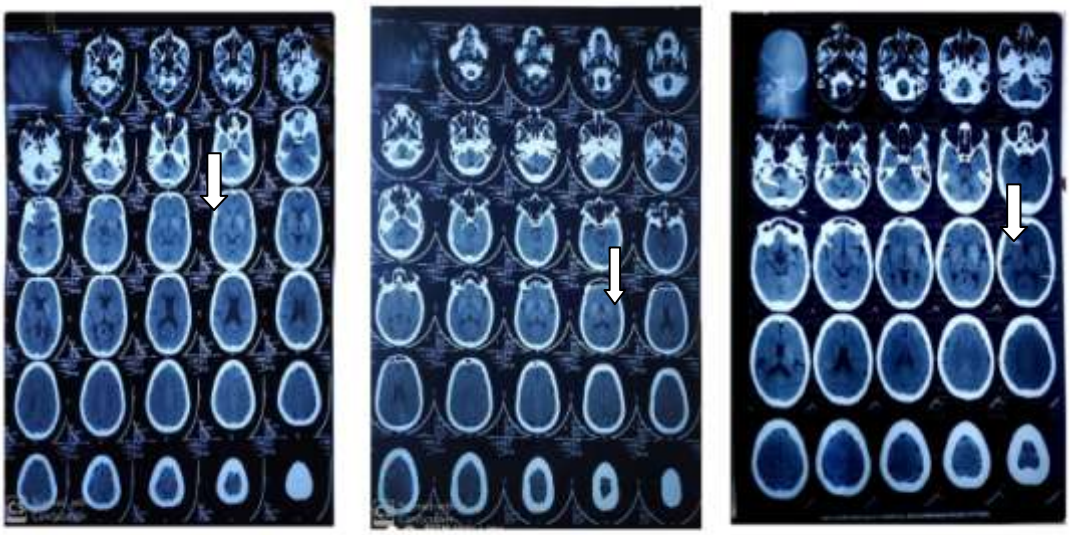

Figure 1: showing the CT findings in case 1 - with left putamen>caudate, right caudate; case 2 - bilateral caudate and putamen; case 3 - left caudate and putamen showing the abnormalities

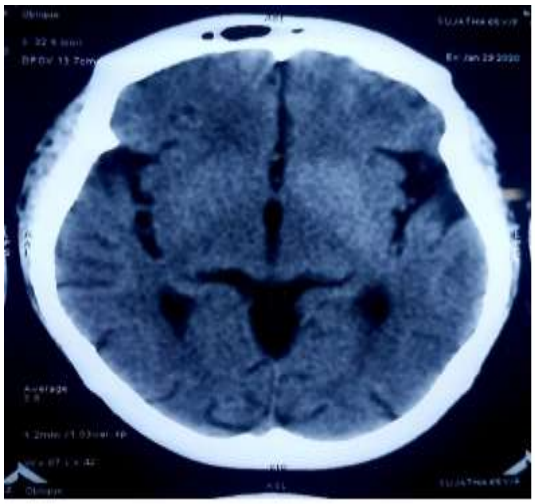

Figure 2: CT Abnormality

II. DISCUSSION

Diabetic striatopathy is a rare hyperkinetic movement disorder characterized by a rare condition characterized by spontaneous, persistent involuntary jerky motions with ballisum typically being more proximal and larger amplitude than chorea.(4,5) The typical triad includes involuntary unilateral (or bilateral) movements, contralateral (or bilateral) striatal neuroimaging abnormalities, and hyperglycemia in patients with known or previously unrecognized diabetes mellitus.(6,7) The involuntary movements start acutely to subacute, often deteriorating over several days. Abnormal involuntary movements can be classified by type and severity from mild chorea to severe ballism.

Diabetic striatopathy exact pathophysiology remains largely unknown; however, focal metabolic distortions secondary to hyperviscosity caused by hyperglycaemia, microvascular ischaemic injury and subsequent hypoxia, changes in dopaminergic activity in predisposed patients, and reactive gliosis were postulated as hypotheses of the acute putaminal dysfunction seen in this syndrome.(6–9) As hyperglycemia slowly converts brain cell metabolism into anaerobic metabolism due to reduced regional cerebral blood flow and failure of the glucose metabolism, the potential mechanisms may be Gamma-aminobutyric acid (GABA) then becoming the main energy source for the brain cells. Acetoacetate can be used to synthesize GABA in ketosis patients. In patients with non-ketotic hyperglycemia, GABA is rapidly depleted due to lack of acetoacetate, thus damaging the normal activity of the basal ganglia.(3,10–12)

By definition, all our subjects had hyperglycemia prior to chorea/ ballism onset. This disorder is associated with type 2 diabetes, and less often type 1 diabetes mellitus. This syndrome has been reported more frequently in elderly Asian women and symptom resolution is typically associated with serum glucose levels and plasma osmolarity normalization.(3) Hemichorea / hemiballism improve slowly in the days following serum glucose correction and by antichorea medications like tetrabenazine and haloperidol and are often aids to speed up symptomatic resolution.

III. CONCLUSION

Hemichorea – hemiballism can be observed during hyperglycaemic crises and clinicians should be able to recognize this rare syndrome and its characteristic findings of neuroimaging and differentiate it from vascular ischemia or hemorrhage. Poorly controlled diabetes is the most common cause of hemiballism and hemichorea from acute dysfunction in the basal ganglia. In most patients, rapid glucose lowering therapy and hydration quickly resolves the symptoms along with antichorea medications.

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