

ORIGINAL RESEARCH PAPER

General Medicine

DIABETIC STRIATOPATHY - A RARE CAUSE OF HEMICHOREA IN A 66 YEAR'S OLD MALE: A CASE REPORT

KEY WORDS:

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INTRODUCTION

Diabetic Striatopathy is defined as hyperglycaemic condition associated with one of the following condition:

- 1. Chorea/Ballism
- Striatal Hyperdensity on CT or Hyperintensity on T1W MRI as previously reported

Diabetic Striatopathy is also a rare complication and also known as hyperglycemic non-ketotic hemichorea/hemiballism with a prevalence of 1 in $100,000^{(1)}$. Present case is a 60 year's old male referred from a tertiary center with Choreaform movements with suspicion of caudate nucleus infarct but MRI brain findings suggest a less known complication known as Diabetic Striatopathy.

Case Report

A 66 year's old right handed man with poorly controlled Type 2 Diabetes Mellitus presented to Emergency Department of Sarojini Naidu Medical College, Agra with 10 months history of involuntary and sudden onset movements involving right side of the body suggestive of choreaform movements of right upper and right lower extremity that were involuntary, couldn't be suppressed in awakening state but resolved during sleep. Movements were not associated with tongue bite, uprolling of eye balls, rotation of the neck, frothing from mouth or incontinence. There was no history of Head trauma, epilepsy, cerebrovascular accident, or any chronic illness.

On CNS examination patient was oriented with time, place, person and intact higher mental function his cranial nerves were normal. No motor or sensory deficits were found. His gait could not be assessed due to choreaform movements. No Cerebellar signs could be demonstrated and had no signs of meningeal irritation. There were no abnormal movements of the mouth or tongue. There was no myoclonus or dystonia.

Initial bio chemistry showed a random serum glucose of 344mg/dL, HbA1c suggestive of poorly controlled diabetes, there was no evidence of persistent microalbuminuria and no evidence of retinopathy or small fibre neuropathy.

MRI brain which was done previously in the tertiary care center showed T1 shortening in left caudate nucleus and left lentiform nucleus, which is showing blooming on GRE imaging and there was no evidence of restricted diffusion suggestive of rare complication like Diabetic Striatopathy as shown in fig. 1 and fig. 2.

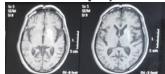


Fig. 1.T1 shortening noted in left side caudate and lentiform nucleus.

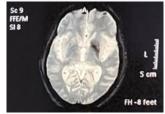


Fig.2.Blooming on GRE image of left side caudate and lentiform nucleus.

Patient was put on Oral Hypoglycemic Agent like Gliptin and Metformin as patient was unwilling to take Insulin, and antichorea treatment Dopamine depleter Tab Tetrabenzine 25mg OD and for hemichorea Tab. Sodium Valproate CR 300mg BD and Tab Clonazepam 0.50mg OD. His blood sugar fasting was found to be 205 mg/dl and Post Prandial was 250 mg/dl after 10 days of treatment further patient was discharged in a satisfactory condition and advised to follow in OPD after 1 months as he was not willing to stay in hospital.

DISCUSSION

The diagnosis for this case of diabetic striatopathy was made with typical clinical as well as radiological manifestations which included choreaform movements together with poorly controlled diabetes in the absence of keto acidosis.

To date, there have been four hypothesis to explain the pathogenesis underlying the observed striatal anomalies on neuroimages, namely petechial hemorrhage (a), mineral deposition (i.e. calcium or magnesium) (a), myelin destruction (a), and infarction with astrocytes (a). Patients with poorly controlled diabetes have certain metabolic disturbances which are associated with hyperglycemia along with vascular insufficiency which together contribute to renal metabolic failure in the brain this in turn produces is chemic/hypoxic damage leading to metabolic derangement causing rapid escalation of swollen astrocytes knwon as gemistocytes (b).

Te mainstay of Diabetic Striatopathy treatment is control of hyperglycemia with proper hydration to correct the underlying metabolic imbalance. Te present study revealed that although chorea could be successfully treated with glucose control only in one-fourth of the patients, the majority needed additional anti-chorea medications for symptom control. There are four main categories of anti-chorea medications, namely antipsychotics, GABA-receptor agonists, selective serotonin reuptake inhibitors and dopamine-depleting agents. Te current study showed that haloperidol was the most common monotherapeutic agent against Diabetic Striatopathy associated chorea, followed by

tetrabenazine, risperidone and clonazepam. Other antichorea medications included tiapride, quatiapine, pimozide, diazepam and vaproate.

CONCLUSION

Diabetic Striatopathy is a poorly understood and lesser known condition associated mainly with elderly female who have poorly controlled diabetes the condition can cause Hemichorea, Hemiballismus which are resistant to treatment with usual therapy fortunately condition can resolve quickly and has a good prognosis in most of the cases.

Screening of Diabetic Retinopathy in patients with Diabetic Striatopathy gives an idea regarding the prognosis of movement disorder associate with the disease.

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