

Case Report

Chorea as a Rare Manifestation of Hyperglycaemia

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Abstract

Introduction : We present a case of chorea presenting as a clinical manifestation of hyperglycaemia. The purpose of presenting this case is to highlight the fact that movement disorder may be the clinical presentation of hyperglycaemia and it reverts on treatment of hyperglycaemia.

Case Presentation : A 66-year-old female known case of type 2 diabetes mellitus and on oral hypoglycaemic drugs presented with abnormal and involuntary movements of the whole body and face since 7 days and high plasma glucose (446 mg/dl) and without ketosis. On controlling the blood sugar, there has been significant decrease in choreiform movements within 48 hrs and complete resolution of involuntary movements found at discharge at 1 week.

Conclusion : Movement disorder like chorea may be the clinical presentation of the hyperglycaemia which could completely recover on rapid detection and correction of hyperglycaemia.

Introduction

Movement disorder like chorea is usually caused by variety of hereditary neurological diseases, metabolic disorders, post rheumatic fever sequel, strokes and other vascular diseases. We present a case of chorea presenting as a clinical manifestation of hyperglycaemia. Such hyperkinesia in the form of chorea, ballismus or choreoathetosis has been reported in patients with nonketotic hyperglycaemia, with complete response to therapy for hyperglycaemia.¹ The purpose of presenting this case is to highlight the fact that movement disorder may be the clinical presentation of hyperglycaemia and it reverts on treatment of hyperglycaemia.

Case Presentation

66 yr old female known case of diabetes mellitus on oral medications, presented to our emergency room with chief complaint of involuntary movement of both sided arms and legs since 7 days. The movements began suddenly, remitted during sleep, but were otherwise constant and progressively worsening. The patient also noted polyuria and polydipsia since last 2-3 weeks. No other significant past history or family history of Huntington's chorea or other neurological disorder present. Neurological examination revealed choreiform movement disorder with decreased tone and normal strength. Deep tendon reflexes and sensation were intact, and mental status was within normal limits.

Kayser Fleischer ring was absent on slit lamp examination. Investigations revealed haemoglobin of 14 g%, total count of 10,200 /mm³, differential count of N 80 E 1 L 19; and normal liver function tests. Serum glucose was elevated to 446 mg/dl. Glycosylated haemoglobin A1C was significantly elevated at 9.7%. Arterial blood gas analysis reveals no acidosis and serum was negative for ketone bodies. Estimated blood osmolality was 297 mosm/L. His BUN and serum creatinine were 24 mg% and 1.0 mg% respectively. He had serum sodium of 132 mEq/L; potassium 4.0 mEq/L; serum calcium and magnesium levels were

normal. Serum antistreptolysin-O titre was < 200 IU, ANA was negative. CT brain and MRI brain revealed no abnormalities.

Patient was started with long acting insulin once daily, with short acting pre-prandial insulin coverage. At the end of 48 hours, his blood sugar was less than 200 mg% and the involuntary movements had significantly decreased. By the time of discharge on hospital day number 7, neurological symptoms had completely resolved. Patient shifted on oral hypoglycaemic drugs without recurrence of choreiform symptoms.

Discussion

Hyperkinesia in the form of chorea, ballismus or choreoathetosis has been reported in patients with nonketotic hyperglycaemia, with complete response to therapy for hyperglycaemia.¹ Several hypotheses put forward to explain the pathogenesis of chorea or ballismus associated with nonketotic hyperglycaemia these movement disorders associated with nonketotic hyperglycaemia include, relative dopaminergic hypersensitivity, undefined effect of hypersomolality, decrease in aminobutyric acid and hypometabolism of striatal cells due to hypoperfusion. In nonketotic hyperglycaemia, the shift to anaerobic metabolism causes brain to utilise aminobutyric acid which is synthesised from acetoacetate. Unlike in ketoacidosis, acetoacetate is rapidly depleted in nonketotic hyperglycaemia causing cellular dysfunction.^{2,3} Chorea had been reported in patients of nonketotic hyperglycaemia with high serum osmolality, which however was normal in our case. Hyperintense lesions in the basal ganglia, on T1WI of MRI have been demonstrated in one study.⁴ Another study revealed hypoperfusion in corresponding areas on SPECT.⁵ Chang et al⁶ compared six nonketotic hyperglycaemic chorea patients with 10 age matched controls, not having any structural lesion by MRI. The SPECT scan showed the presence of hypoperfusion in the striatum contralateral to the symptomatic chorea in five patients and hypoperfusion in both striatum in one. The CT scan and MRI of brain were normal in our case, as in earlier reports.^{3,6} and as SPECT scan could not be performed in the present case, hypoperfusion or structural lesion of basal ganglia could not be ruled out. Bilateral chorea at presentation, hyperglycaemia with normal serum osmolality and rapid response of chorea to correction of hyperglycaemia point towards the direct effect of

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Received: 03.12.2012; Accepted: 06.02.2013

hyperglycaemia as the pathogenesis of chorea in this case.

Conclusion

Movement disorder like chorea may be the clinical presentation of the hyperglycaemia which could completely recover on rapid detection and correction of hyperglycaemia.

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