

A Rare Clinical Presentation of Diabetes Mellitus

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Abstract

This case report highlights a rare presentation of new-onset diabetes. Hemichorea-hemiballismus due to non-ketotic hyperglycemia is a rare disorder, which comprises of unilateral, periodic choreiform movement of the extremities in the setting of hyperglycemia without evidence of ketoacidosis. Radiographically, there is hyperdensity in the basal ganglia but no evidence of bleeding or mass effect on CT scan of the brain. MRI of the brain shows increased T1-signal intensity in the contralateral putamen. The immunopathology is hyperglycemia mediated, which causes ischemic excitation of GABAergic-neurons in the basal ganglia. This leads to excessive inhibition of sub-thalamic nuclei and excitatory cortical input causing involuntary movement unilaterally. The mainstay treatment of this condition is insulin therapy, and the movement disorder disappears once glyceemic control is achieved.

Keywords: Diabetes; Hyperglycemia; Ketoacidosis; Hypertension; Hyperlipidemia; Neuropathy

Case Presentation

A 70-year old male with past medical history of hypertension, hyperlipidemia, and peripheral arterial disease presented with unintentional, uncontrollable movements of his left leg and left arm. His review of systems were positive for polyuria, polydipsia, malaise, dizziness, and fatigue for a month prior to presentation. He denied any chest pain, dyspnea, fever, abdominal pain, weakness, sensory loss, dysphagia, diplopia, or neuropathy. His medications included atorvastatin, aspirin and amlodipine. His family history was significant for hypertension and hyperlipidemia. He had a 40-pack year smoking history, consumed alcohol occasionally, but denied any illicit drug use.

His physical examination was notable for mild sinus tachycardia, with dry mucus membranes. He exhibited akathasia, and periodic choreiform movement of his left leg and left arm. He had 4/5 strength in upper and lower extremities, reduced pinprick sensation in bilateral legs, negative pronator drift, negative Romberg, and negative cerebellar signs. He did not have any cranial nerve deficits.

His laboratory data was significant for hyperglycemia with glucose of 705 mg/dL, trace ketones in urine, and negative serum ketones. His hemoglobin A1c was 14.5%. His thyroid studies, RPR, HIV antibody, anti-nuclear antibody, parathyroid hormone, erythrocyte sedimentation rate, C-reactive protein were normal. CT head showed chronic vessel ischemia. There was no evidence of hemorrhage or mass effect but a hyper-density was present in the right caudate nucleus. MRI brain showed high T1-weighted signal in the right basal ganglia. Treatment with insulin and intravenous fluids was initiated. The patient was also treated with haloperidol and clonazepam. His movement disorder resolved about two weeks after initiation of therapy and hence the dopamine antagonists and benzodiazepines were stopped. He continued to use insulin for diabetes.

Discussion

This case exemplifies a rare manifestation of new onset diabetes, which is called hemichorea-hemiballismus due to non-ketotic hyperglycemia. This disorder is characterized by unilateral, involuntary, poorly patterned movements in conjunction with severe hyperglycemia in the absence of ketoacidosis [1]. Typically, the CT head shows area of hyper-density in the basal ganglia with no mass effect, edema or signs of hemorrhage. T1-weighted MRI shows high signal intensity in the contralateral putamen [2,3].

The pathogenesis is related to hyperglycemia causing ischemic excitation of GABA-ergic neurons. This results in excessive inhibition of sub-thalamic nuclei and excitatory cortical output [1,4].

The main treatment for this disorder is correction of hyperglycemia. Insulin is the drug of choice, and hydration is essential in the acute phase of presentation. Dopamine antagonists, anti-epileptics, and benzodiazepines have been used with varying success, to control the involuntary movement during the acute phase [5]. These patients typically require insulin long-term, since symptoms resolve once glyceemic control is achieved.

The differential diagnosis of acute to sub-acute movement disorders is vast, and hence it is prudent to rule out more common entities such as intracranial bleed, mass, or stroke prior to making the diagnosis of hemichorea-hemiballismus. In the right clinical context of new onset hyperglycemia, it can be the presenting sign of diabetes mellitus, especially in the elder population [6]. Radiographic features help confirm this diagnosis and exclude other ominous entities.

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