Mixed Movement Disorder as the Presenting Manifestation of Non-Ketotic Hyperglycemia

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Abstract
Focal neurological symptoms may provide the first clue to the presence of nonketotic hyperglycemia (nKH). We report a patient with hemichorea-hemiballism (HC – HB) as the first manifestation of nKH. CT Brain revealed hyperdensity in bilateral globus pallidus (GP) and putamen predominantly on right side. Blood sugar was 580 mg/dl and s. osmolality was 316 mosm/l. This condition resolved after correction of hyperglycemia. The possible mechanism by which nKH causes this condition is discussed.

Introduction
In nonketotic hyperglycemia (nKH), hyperglycemia, hyperosmolality and intracellular dehydration occurs without significant accumulation of ketone bodies. It is usually observed in patients over 50 years with type II diabetes mellitus (DM) and resolves after correction of hyperglycemia. Nonketotic hyperglycemia frequently presents with neurological manifestations including partial or generalized seizures, hemiparesis, hemisensory loss. Another well defined but uncommon complication of nKH is chorea-ballismus syndrome.

Case Report
A 70 year old previously neurologically normal male was admitted because of proximal, wide swinging movements, distal semipurposive flowing movements in his left limbs and repetitive, twisting movements in his right upper limb for a week. These movements were exaggerated during emotion and absent during sleep. These involuntary movements were more in left limb than right. He had suffered from type II DM for the past 5 years which had been controlled with oral hypoglycemic agents. He denied history of cerebrovascular disease, hypertension, infection, trauma or history of neuroleptic drug intake. There was no family history of movement disorder.

On admission patient had chorea and hemiballismus involving left face, arm and leg and dystonic posturing of right hand. Other neurological examination was normal. Initial biochemical values included serum glucose 580 mg/dl without
ketonemia, serum sodium 138 meq/l, serum potassium 4 meq/l and blood urea 48 mg/dl. The calculated serum osmolality was 316 mosm/l. CT brain taken 7 days after symptoms onset revealed hyperdensity in bilateral GP and putamen more on right side.

Hyperglycemia was corrected by insulin therapy followed by treatment with oral hypoglycemic agents. The involuntary movements decreased as serum glucose approached normal levels. After a month CT brain revealed resolution of basal ganglia lesion.

Discussion

Focal neurological symptoms may provide the first clinical clue for the presence of non ketotic hyperglycemia. Seizures, chorea and focal neurological deficits have been reported due to NKH. Another well defined and uncommon complication of NKH is chorea – ballismus syndrome. Neurological symptoms improve with correction of hyperglycemia.

The exact mechanism of how NKH causes focal neurological symptoms is unknown. One hypothesis is related to depletion of inhibitory neurotransmitter GABA, which is metabolized in the brain as an energy source in NKH. The deficiency of GABA in basal ganglia may lead to hemichorea – hemiballismus (HC-HB) syndrome. Another hypothesis involves transient focal cerebral ischemia caused by hyperglycemia. Cerebral hypoperfusion may result from an increase in cerebrovascular resistance due to the higher brain water content during hyperglycemia or to a loss of flow regulation caused by impaired metabolism. Hyperdense lesion in putamen on CT Brain is commonly found in HC – HB with NKH. This functional alteration could be associated with some degree of Wallerian degeneration of the internal white matter of the putamen. Protein desiccation occurring in the course of Wallerian degeneration could explain the CT hyperdensities as well as the variable evolution of imaging features with time.1

Conclusion

Focal neurological manifestations may occur with NKH including hemichorea and hemiballismus. Rapid recognition of their association will help prompt diagnosis, correct treatment and resolution of hyperdense lesion in basal ganglia.

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