



Hemichorea-Hemiballismus due to Non-ketotic Hyperglycemia *Non-Ketotik Hiperglisemiye Bağlı Gelişen Hemikore-Hemiballizm*

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Dear Editor,

We have read the clinical report "Hemichorea-Hemiballismus due to Nonketotic Hyperglycemia" with great interest. We wanted to share a similar case to emphasize certain important points related to this topic.

A 41-year-old female patient came to our clinic for involuntary movements starting from her right arm and spreading to her right leg. These complaints had started 3 months ago. She was diagnosed with type-2 diabetes 15 years ago and had been taking insulin aspart 60 IU/day, insulin glargine 30 IU/day, vildagliptin 100 mg/day and metformin 2 gr/day. Except for right hemichorea and hemiballismus, neurological examination did not show significant findings. In her laboratory evaluations, her hemogram, liver, kidney and thyroid function tests, and vitamin B12 and folic acid levels were found to be normal. Pre-meal blood sugar was measured as 365 mg/dl, hemoglobin A1c 14.7%, sodium 135 mEq/L, and serum osmolality 295 mOsm/kg. In his cranial MRI, T1 and T2 weighted sequences showed hyperintensities in left caudate nucleus and putamen (Figures 1 and 2). The patient was diagnosed with hemichorea due to nonketotic hyperglycemia. She was started on 10 mg/day haloperidol and all of her symptoms improved in 1 week.

Hemichorea involves sudden, spasmodic, irregular and transitory finger, hand, arm, face, tongue or head movements that affect one side of the body. The concept of ballismus corresponds to high amplitude, strong flailing or jerking type of these motions (2). Chorea and ballismus develop as a result of basal ganglion damage due to metabolic diseases, infections, drugs or toxic materials (3-5).

Hemichorea due to hyperglycemia is commonly seen in diabetic patients whose condition is not controlled for extended periods of time (6). Our patient fits this description. The ways in which hemichorea and hemiballismus develop due to nonketotic hyperglycemia are unknown. Positron emission tomography and single-photon emission computed tomography studies show decreased blood flow and glucose metabolism in the basal ganglion contralateral to hemichorea (7,8). Certain hypotheses were proposed to explain this phenomenon. Among these hypotheses are the reduction of GABA in basal ganglia, increase of dopaminergic activity in striatum due to hyperglycemia, blood-brain barrier damage due to hyperviscosity, and petechial hemorrhages or infarctions of striatum (4).

Radiologically, it is typical to see striatal hyperintensities in the T1-weighted sequence (1). Restricted diffusion can also be observed in diffusion weighted images (9). We observed the striatal hyperintensity in the T1-weighted MRI. Most of these findings disappear following the successful treatment of hyperglycemia (10).

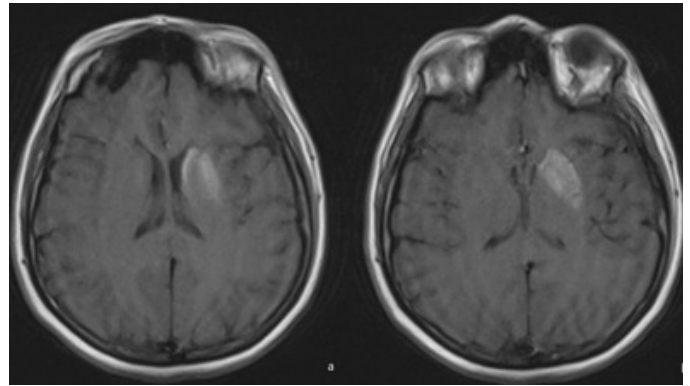


Figure 1A ve 1B. Hyperintensity in caudate nucleus and putamen seen on T1-weighted image.

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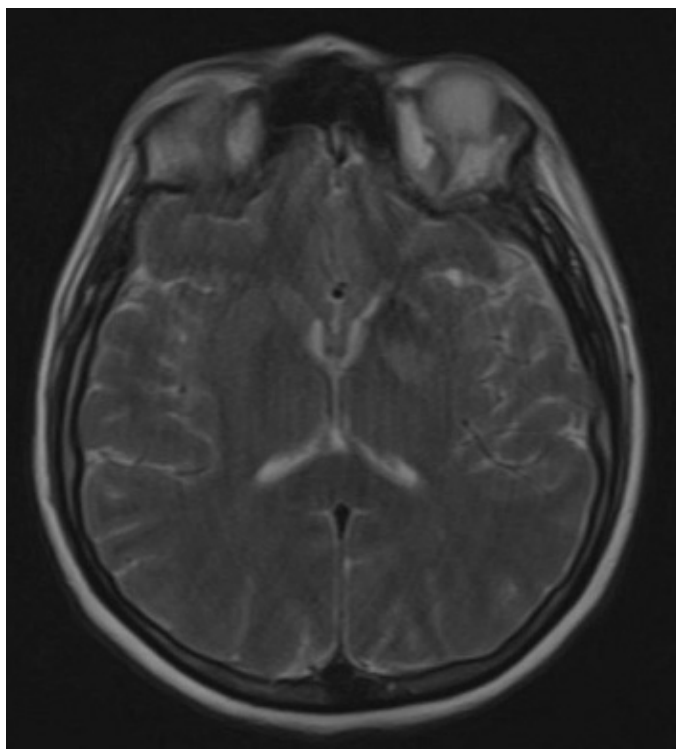


Figure 2. Putamen hyperintensity in T2-weighted image.

Hemichorea and hemiballismus develop due to nonketotic hyperglycemia usually disappears when the blood sugar is brought to the regular levels. In the event that this does not happen, the patient may be given dopamine blockers, tetrabenazine or topiramate (7). Our patient benefited from 10 mg/day haloperidol. In conclusion, the presence of hemichorea hemiballismus in patients with diabetes mellitus should suggest nonketotic hyperglycemia and the treatment should be started swiftly after a definitive diagnosis is made.

Key words: Hemichorea, hemiballismus, nonketotic hyperglycemia

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