CASE REPORT

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Hemichorea-hemiballismus developing due to non-ketotic hyperglycemia: A rare case report

Non-ketotik hiperglisemiye bağlı gelişen hemikore-hemiballismus: Nadir bir olgu sunumu

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ABSTRACT

Hemichorea-hemiballismus can be seen as a rare complication of non-ketotic hyperglycemia in patients with type 2 diabetes mellitus. We detected hemichorea-hemiballismus secondary to hyperglycemia in a patient who presented to the emergency department with speech disorder and involuntary movements in the left arm, as a result of our investigations. We decided to present this case because it is rare in the literature.

Keywords: Athetosis, hemiballismus, hemichorea, hyperglycemia

ÖZ

Hemikore-hemiballismus , Tip 2 diyabetes mellitus hastalarında non-ketotik hipergliseminin nadir ortaya çıkan bir komplikasyonu olarak görülebilir. Acil servise konuşma bozukluğu ve sol kolda istemsiz hareketler nedeniyle başvuran bir hastada yaptığımız tetkikler sonucunda hiperglisemiye sekonder gelişen hemikore-hemiballismus tespit ettik ve literatürde nadir rastlandığı için bu vakayı sunduk.

Anahtar Kelimeler: Ateatoz, hemiballismus, hemikore, hiperglisemi

Highlights

- Hemichorea-hemiballismus can manifest as a rare neurological complication of non-ketotic hyperglycemia, particularly in patients with Type 2 Diabetes Mellitus.
- The case reported describes an 81-year-old female presenting to the emergency department with a speech disorder and involuntary left-sided movements, with a blood glucose level of 726 mg/dl, leading to a diagnosis of hemichorea-hemiballismus secondary to hyperglycemia.
- Contrary to common findings, the patient's Cranial Computed Tomography (CT) and Diffusion Magnetic Resonance Imaging (DWI) did not reveal any acute or pathological findings.
- The management involved immediate blood glucose regulation, complemented by an initial valproic acid (1200 mg) infusion in the emergency department, followed by combination therapy with haloperidol during hospitalization.
- The patient's symptoms completely resolved by the third day of hospitalization following successful blood sugar regulation and combination pharmacological treatment.
- This case underscores the importance for emergency physicians to consider non-ketotic hyperglycemia as the etiology for gait disturbance and involuntary movements, even when initial cranial imaging is unremarkable.

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INTRODUCTION

Chorea; refers to involuntary movements that are irregular, purposeless, brief, sudden, and rapid in nature, which can spread from one part of the body to another, such as the extremities, trunk, neck, and face. Ballismus, on the other hand, refers to higher amplitude, coarser involuntary movements than chorea, involving the proximal joints like the shoulder and hip, causing throwing or violent flailing movements. It is often unilateral and therefore called hemiballismus. Hemichorea can be seen in patients with diabetic nonketotic hyperglycemia. Symptoms usually resolve after blood sugar regulation, but in some cases, long-term medical treatments may be required (1-3). Although T1-weighted cranial magnetic resonance imaging (MRI) in most cases shows hyperintensity in the contralateral putamen and basal ganglia, no pathology was found in the brain computed tomography (CT), brain diffusion, and non-contrast MRI performed in our presented case. In this case, we examined a patient diagnosed with hemichorea-hemiballismus, one of the rare complications of Type 2 diabetes mellitus.

CASE

An 81-year-old female patient was brought to the emergency department due to speech disorder that started in the last 3 days and involuntary movements developing in the left upper and lower extremities. The patient's history included atrial fibrillation, diabetes mellitus type 2, hypertension, and a history of cerebrovascular disease. It was learned that she was irregularly using her medications (metformin, empagliflozin, candesartan, methimazole, insulin aspart, edoxaban). Physical examination revealed the patient was conscious, oriented, and cooperative, with no facial asymmetry, lateralizing motor deficit, or pathological reflexes. Cranial nerve examination was normal. Licking-like movements of the mouth and lips and choreiform movements in the left upper extremity were present. Laboratory tests showed blood sugar 726 mg/dl, serum sodium (Na^+) 129 mmol/l, serum osmolality 317 mOsm/l, venous blood gas pH: 7.327, urine ketone negative, glucose ++++. There were no pathological findings in the patient's other laboratory tests. No urgent, acute pathology was detected on the patient's cranial CT and brain diffusion MRI. (Figure 1, 2) Blood sugar monitoring was performed while the patient was in the emergency department,

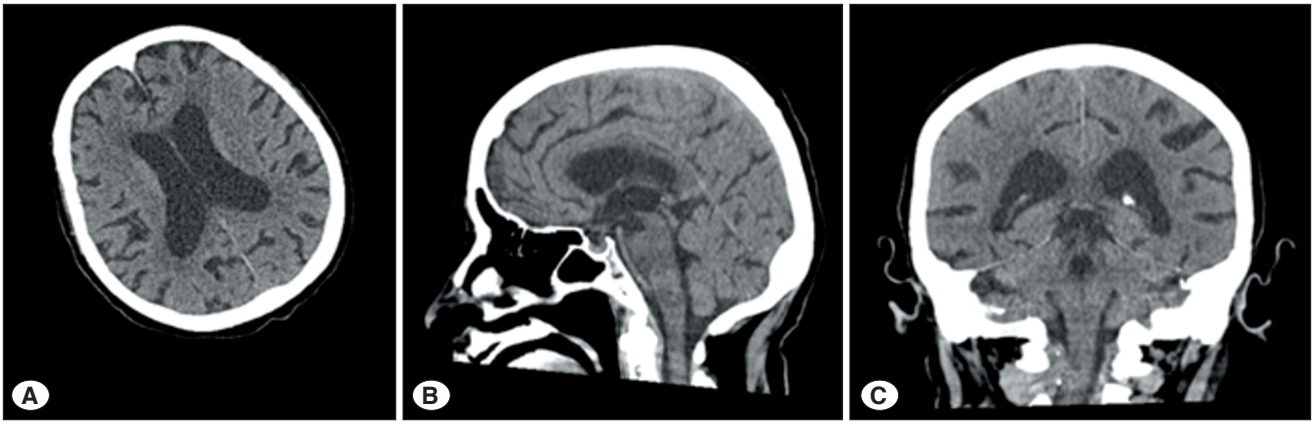

Figure 1: A) Axial view of patient's cranial CT, **B)** A Sagittal view of patient's cranial CT, **C)** Coronal view of patient's cranial CT.

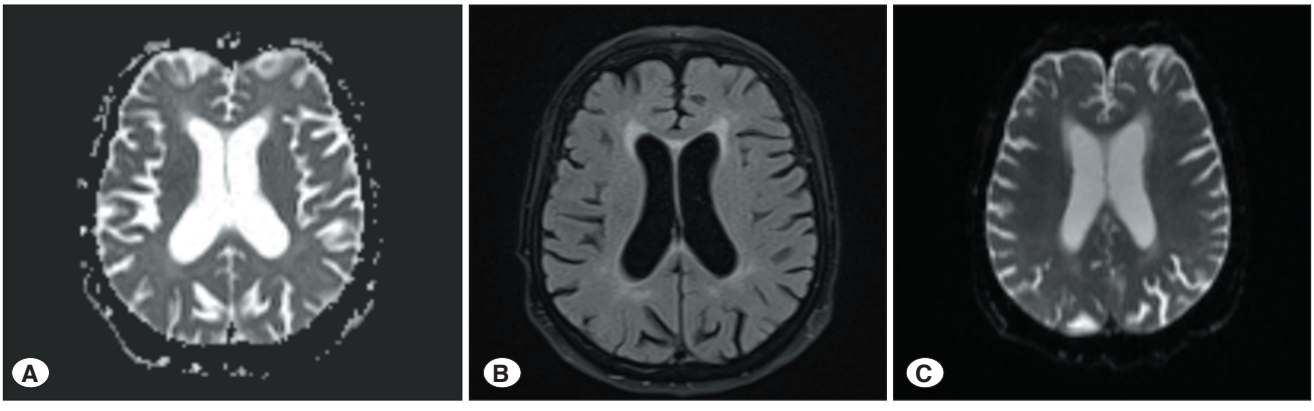

Figure 2: A) Axial DWI of patient's difussion MRI, **B)** T2 Flair of patient's difussion MRI , **C)** T1 images of the patient's difussion MRI

and upon the recommendation of the neurology consultant, valproic acid 1200 mg was administered as a 40-minute infusion. The patient was admitted to the neurology service with a diagnosis of hemichorea-hemiballismus secondary to non-ketotic hyperglycemia. It was learned that the patient's symptoms regressed on the third day of hospitalization in the neurology service with blood sugar regulation and haloperidol treatment, and she was discharged with recovery.

DISCUSSION

Chorea-ballismus can be seen in patients with diabetic non-ketotic hyperglycemia. Hemichorea can occur in elderly female patients, especially those with unregulated blood sugar and irregular medication use. Rarely, cases with bilateral chorea have also been reported. It has been reported that cranial MRI and CT imaging are normal in these patients, and abnormal findings regress with the correction of hyperglycemia (4). In our case, our patient also had Type 2 diabetes mellitus and was not using her medications regularly, and there were no pathological findings on the cranial imaging performed. Her complaints completely resolved after blood sugar regulation. In some cases, treatment may take longer and can even result in death due to increased hyperviscosity.

The pathophysiology of hyperglycemia-associated hemichorea-hemiballismus has not been fully elucidated. In their study of 18 patients, Chang et al., based on imaging findings, argued that hyperglycemia causes transient ischemia in patients and that this is the cause of the clinical presentation (5). According to the hypothesis developed by Shan et al. in their studies, dysfunction of GABAergic neurons in the caudate nucleus and putamen caused by hyperglycemia or cerebral ischemia leads to non-ketotic hyperglycemia-associated hemichorea-hemiballismus (6). Typical neuroleptics (haloperidol, pimozide, perphenazine, fluphenazine), atypical neuroleptics (olanzapine, quetiapine), sodium valproate, and levetiracetam are used in treatment (4). Valproic acid is chemically similar to GABA, and its mechanism of action is explained by increasing GABA levels as well as blocking voltage-dependent sodium and potassium channels in the brain (7). Therefore, in our presented case, valproic acid infusion was administered to our patient in the emergency department. Clinical improvement was achieved with combination therapy of haloperidol, a D2 receptor agonist, and valproic acid.

Although the pathophysiology is not clear, studies conducted so far have shown that symptoms resolve in most patients after blood sugar regulation. In our case, the findings also regressed and completely resolved on the third day of treatment following blood sugar regulation and combination therapy with haloperidol and valproic acid. However, it has been reported that hemiballismus persists for more than three months in 20% of patients, and medical treatment may be required for a longer duration (2).

Conclusion

In cases of non-ketotic hyperglycemia-associated hemichorea-hemiballismus reported in the literature, no specific findings were detected on cranial CT, but hyperintense foci may be present on T1-weighted MRI. In our case, cranial CT and T1-weighted MRI were normal; additionally, no pathology was detected on electroencephalography examination. Our patient's current clinical picture resolved with blood sugar regulation. This led to the evaluation of our case as non-ketotic hyperglycemia-associated hemichorea-hemiballismus. In patients presenting to the emergency department with gait disturbance and involuntary movements, even if cranial imaging is normal, it should not be forgotten that high and unregulated blood sugar can cause this clinical picture. Emergency physicians should keep in mind that non-ketotic hyperglycemia-associated hemichorea-hemiballismus can be a neurological complication of diabetes.

Author Contributions

Study design and conceptualization: **Taner Şahin;** data collection: **Merve İrem Atıcı and İrem Kömürcü;** analysis and interpretation of results: **Merve İrem Atıcı, Baycan Kuş, and Merva Tuna;** drafting the manuscript: **Merve İrem Atıcı.** The authors reviewed the results and approved the final version of the article.

Ethical Approval

This study is not an experimental or clinical research. As it is a case report, ethical approval was not required. Written informed consent was obtained from the patient for the publication of the case report.

Conflicts of Interest

The authors declare that there is no conflict of interest to disclose.

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