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Case Report

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A case of nonketotic hyperglycemia without movement anomalies: MRI findings

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ABSTRACT

In our case report, we aimed to describe a patient with nonketotic hyperglycemia who was admitted to the emergency department with left basal ganglion involvement presenting with a stroke-like picture without movement anomalies.

Keywords: Hyperglycemia, nonketotic hyperglycemia, MRI

This case report was presented as a printed poster at the 2016 Turkish Magnetic Resonance Society Ankara Congress.

INTRODUCTION

Diabetes mellitus (DM) can cause many neurologic complications such as peripheral neuropathy and encephalopathy. Nonketotic hyperglycemia (NKH) is a serious acute and mortal complication of uncontrolled DM. It is caused by hyperglycemia, hyperosmolality and dehydration due to uncontrolled diabetes.[1-3] Nonketotic hyperglycemic seizures present with high blood glucose levels, normal or increased serum osmolality and negative urine ketone bodies.[4] Rarely, focal seizure activity is also seen in patients.[5] The exact mechanism is still unknown. In this article, we present the clinical and radiologic features of a case diagnosed as nonketotic hyperglycemia.

CASE

A 69-year-old woman who presented to the emergency department with complaints of weakness and syncope had a history of diabetes and hypertension. She was receiving insulin and diuretic treatment. Biochemistry tests revealed a blood glucose level of 714 mg/dl (normal blood glucose levels: 60-109 mg/dl). Urinalysis was ketone negative. Brain CT scan of the patient with stroke symptoms showed hyperdansite in the left basal ganglion (Figure 1). Brain MRI was then performed. Brain MRI showed hypointensity in FLAIR and T2 A series (Figure 2,3), hyperintensity in T1 A series (Figure 4), minimal contrast enhancement (Figure 5) and diffusion restriction (Figure 6a and b) in the left thalamus and lentiform nucleus. The case was treated as nonketotic hyperglycemia.

The patient was discharged after a few days with resolution of symptoms. One month later, basal ganglia were normalized on brain CT.

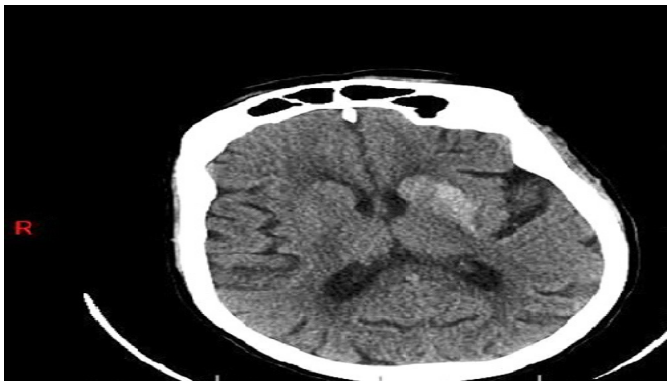

Figure 1. Hyperdensity at the level of the left basal ganglia on brain CT

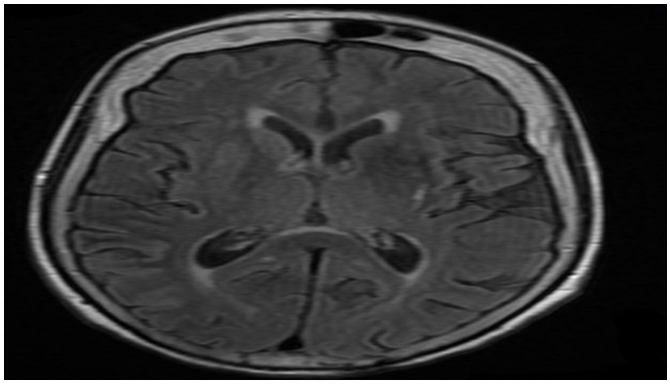

Figure 2. In MRI, hypointensity in FLAIR series in the same area

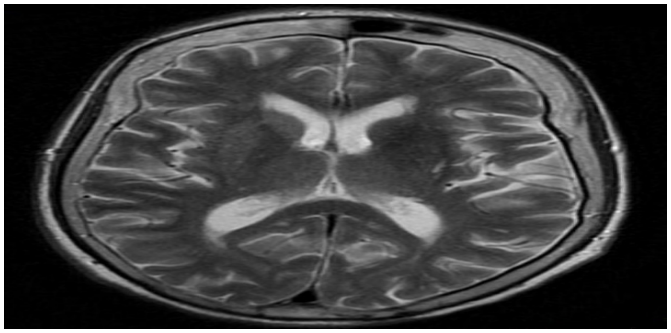

Figure 3. Hypointensity in T2A series

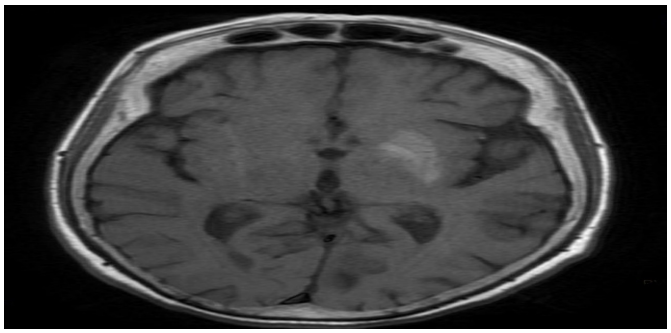

Figure 4. Hyperintensity in T1 A series

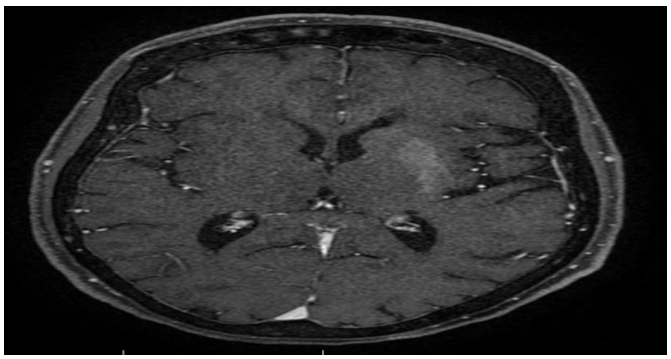

Figure 5. Minimal hyperintensity in contrast-enhanced series

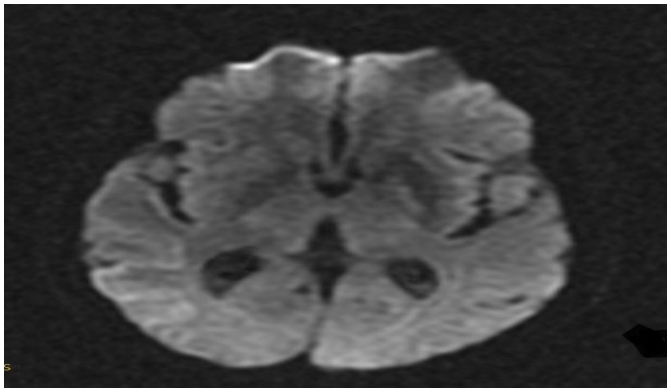

Figure 6 a. There is diffusion restriction in diffusion weighted series

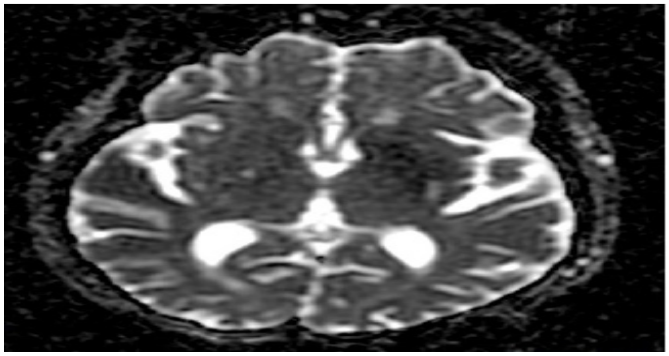

Figure 6 b. Diffusion restriction exists in diffusion weighted series(ADC)

DISCUSSION

Nonketotic hyperglycemia is a complication of DM that cannot be controlled with treatment, and its mechanism and imaging features are still a mystery. There are many different opinions in the literature. On magnetic resonance imaging, T2/FLAIR hypointensity in the subcortical posterior cerebral region is the most characteristic finding.[6] The most commonly reported MR imaging features of seizures associated with NKD are cortical hyperintensity and reversible subcortical hypointensity which may show restriction on diffusion-weighted images. The mechanisms that may cause these findings are reported to be focal cytotoxic edema secondary to cortical focal ischemia or hyperviscosity caused by free radicals and their units.[7,8]

It may also occur with single or bilateral basal ganglion involvement.[5] Hyperintensities of T1-weighted series observed in the basal ganglion are generally thought to be related to petechial hemorrhages.[9] In some series, it has also been suggested that this may be related to demyelination observed in diabetic patients.[10] The follow-up of T2 images and contrast-enhanced series varies in the literature.

Iwata et al.[11] showed contrast enhancement in the globus pallidus. It has been reported that hyperintensity may be seen in the basal ganglia region and most commonly in the putamen, then in the caudate and globus pallidus on T1-weighted images on MRI, whereas T2-weighted images may vary.[12,13]

In patients with unilateral or bilateral basal ganglion involvement, nonketotic hyperglycemia should be kept in mind in the differential diagnosis and questioning of the patient's history and laboratory values should not be neglected.

CONCLUSION

Pathologies other than infarction should be considered in the differential diagnosis of diffusion restriction observed in patients presenting to the emergency room with a stroke-like picture. Basal ganglion involvement may occur in nonketotic hyperglycemia without movement anomalies. Nonketotic hyperglycemia should be kept in mind as a differential diagnosis in patients with uncontrolled diabetes.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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