Case Report

A Case of Bilateral Diffusion-Weighted Imaging Abnormality Due to Profound Hypoglycemia Mimicking Ischemic Stroke

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ABSTRACT

Hypoglycemia: It is a common clinical entity that can present with a wide range of neurological signs and symptoms, from transient focal signs to permanent coma. It can cause DWI abnormalities that can be confused with ischemic infarction or toxic leukoencephalopathy. Here, we present a patient who caused severe hypoglycemia and caused temporary DWI lesions.

Keywords: *Hypoglycemia, Ischemic Stroke, Stupor, Diffusion Weighted MRI.*

ÖZ

İskemik İnmeyi Taklit Eden Derin Hipoglisemiye Bağlı Bilateral Difüzyon Ağırlıklı Görüntüleme Anormalliğinin Gözlendiği Bir Olgu

Hipoglisemi; Klinik olarak geçici fokal bulgulardan kalıcı koma durumuna kadar geniş bir yelpazede nörolojik belirti ve bulgularla prezente olabilen ve sık gözlenen bir klinik antitedir. İskemik enfarktüs ya da toksik lökoensefalopati ile karıştırılabilecek DWI anormalliklerine sebep olabilir. Bizde burada ağır hipoglisemiye bağlı ve geçici DWI lezyonlarına sebep olmuş olan bir olgumuzu sunuyoruz.

Anahtar Sözcükler: *Hipoglisemi, Iskemik Inme, Stupor, Difüzyon Ağırlıklı MRG.*

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Hypoglycemia is a common endocrine emergency that can lead to various neurological signs and symptoms, ranging from transient focal signs to permanent coma (1). Magnetic resonance imaging (MRI) of hypoglycemia patients admitted to the emergency department in a coma might show diffusion restrictions that are similar to those seen in ischemic stroke or toxic leukoencephalopathy (2). This case report emphasizes that severe hypoglycemia can cause diffusion-weighted imaging (DWI) restriction, particularly in certain areas of the brain, and that this may be mistaken for an ischemic stroke.

CASE REPORT

A 75-year-old woman was brought to our emergency department by ambulance due to impaired consciousness. She was unconscious, and her Glasgow Coma Scale score was 9 (E:2, V:4, M:3). Her blood pressure was 110/70 mmHg, pulse rate was 96/min, respiratory rate was 20/min, and oxygen saturation was 90%. Her fingertip glucose was measured at 22 mg/dL, and she was given an immediate 50cc 10% dextrose intravenous bolus and started on infusion therapy. According to the patient's relatives, she was last seen 5 hours ago, and her general condition was good. Her relatives also revealed that she had a medical history of diabetes mellitus, hypertension, and bipolar affective disorder. They mentioned that the lithium medication she was using had been replaced with olanzapine, and her blood glucose increased after starting olanzapine. Additionally, it was noted that the patient had self-adjusted her insulin dose.

Other physical examination findings were normal, and a detailed neurologic examination was performed. The patient was in a stuporous state and exhibited a flexor response to painful stimuli. Pupils were of equal in size and reacted normally to light. Both foot plantar reflexes elicited no response. No abnormal reflexes or signs of neurological improvement were observed after 20 minutes of glucose infusion. Due to the lack of neurological improvement and the patient's risk factors, non-contrast brain tomography and DWI were performed. The DWI and apparent diffusion coefficient (ADC) mapping revealed symmetric bilateral butterfly-shaped

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areas of restricted diffusion in the periventricular white matter near the splenium of the corpus callosum and lateral ventricles (Figure 1).

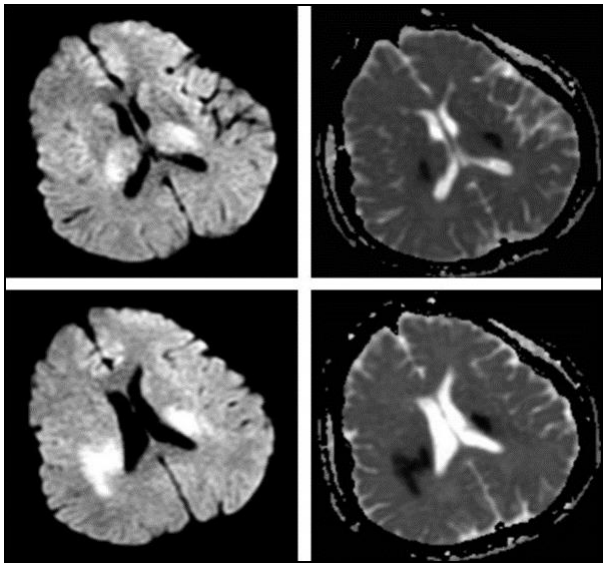

Figure 1. *DWI and ADC imaging taken at the time of admission to the emergency department (Hyperintense lesions are clearly observed).*

The patient was admitted to the intensive care unit and received meticulous monitoring of their blood glucose levels. Dextrose support was no longer necessary after 10 hours, but there was limited progress in the patient's neurological examination until the 48th hour. Following this, a DWI and ADC mapping were conducted, showcasing a complete resolution of the lesions (Figure 2).

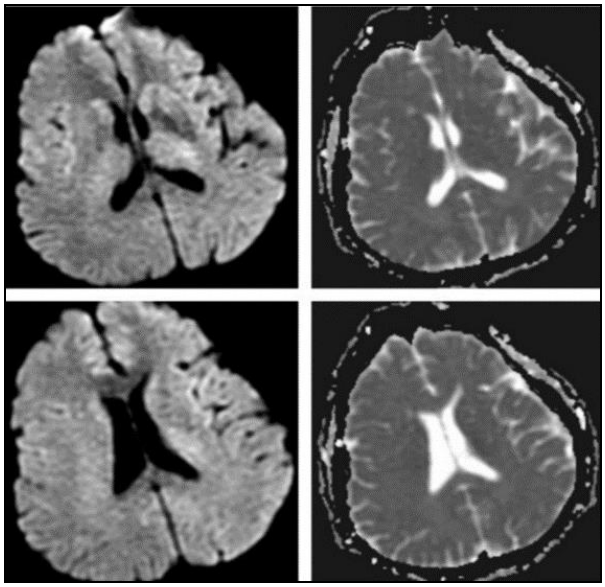

Figure 2. *DWI and ADC imaging of the patient taken 48 hours later (Hyperintense lesions have disappeared).*

Despite the encouraging findings, the absence of neurological improvement prompted concerns about non-convulsive status epilepticus, although the electroencephalography results returned normal. Over the course of 20 days in intensive care, the patient's neurological condition showed gradual improvement, culminating in complete neurological recovery after six months with the aid of physical therapies.

DISCUSSION

Hypoglycemia is defined as a decrease in blood glucose level below 50 mg/dl. Brain cells are the most sensitive cells to glucose deprivation as they are supplied with glucose and are damaged in relation to the low blood glucose level and its duration. Glucose deprivation in brain cells causes a severe cellular energy crisis, leading to a failure of the cell membrane ionic pump activity and consequently to intracellular calcium and water entry, resulting in cytotoxic edema. This pathological process can be visualized using DWI, which appears as a hyperintense lesion similar to those seen in ischemic stroke and has a corresponding ADC mapping counterpart (1, 2).

In contrast to ischemic stroke, hyperintense lesions can also be observed in a variety of other conditions. These include toxicity, withdrawal from antiepileptic drugs, seizures, infectious encephalitis, cerebral edema due to high altitude, alcohol use, hemolytic uremic syndromes, as well as metabolic disorders such as hypernatremia and hypoglycemia (3-5).

Lesions on DWI caused by hypoglycemia can typically appear in different areas, such as the basal ganglia, pons, temporal and occipital cortices, and hippocampus (3, 6, 7). Contrary to what was previously known, a study has shown that white matter was more sensitive to hypoglycemia. However, white matter, gray matter, or co-involvement do not seem to correlate clearly with the clinical outcome (8).

In some rare case series, it has been reported that corona radiata, centrum semiovale, and internal capsule parts of the white matter were involved (1). Considering hypoglycemia in the differential diagnosis is imperative, given the substantial disparities in managing and treating ischemic infarction and other metabolic causes, in order to avert deleterious outcomes (9). In our case, as seen in most cases of hypoglycemia, a symmetric bilateral lesion with more intense involvement of the white matter, hyperintense on DWI, and hypointense on ADC mapping was observed (Figure 1).

In the neurological examination of the patient, metabolic causes were considered in the foreground, and correction of profound hypoglycemia was planned because of the presence of stupor-like disturbance of consciousness, feeble motor response to central painful stimuli, and equal flexor response in four extremities in the absence of lateralizing findings.

However, although the clinical improvement was insufficient at 48 hours, the complete disappearance of the lesions was observed in the new DWI and ADC mapping taken for control (Figure 2). Our patient's clinical recovery occurred after approximately 20 days of in-

tensive care treatment, and complete neurological recovery was achieved after six months. As in the study by Ma et al., the clinical status of our patient at the time of presentation and the observation of restriction in DWI suggested that she was exposed to profound hypoglycemia (8).

As a result, the presence of hyperintense lesions on DWI typically signifies ischemic infarction. Nevertheless, it is essential to recognize that numerous other pathologies can also produce hyperintense lesions, as demonstrated in the present case. When physical examination fails to reveal lateralizing findings and bilateral lesions are observed on DWI, consideration should be given to metabolic etiologies aside from ischemic infarction.

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