Case Report

Isolated reversible involvement of bilateral internal capsule on magnetic resonance imaging in case of acute hypoglycemic encephalopathy: A case report

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ABSTRACT

Hypoglycemic encephalopathy (HE) is a metabolic disorder that can lead to morbid manifestations including motor deficits, transient memory loss, persistent vegetative state, and even deep coma. Due to potential clinical symptom overlap with acute stroke, accurate diagnosis is very crucial. Besides the patient's medical history and routine blood investigations, magnetic resonance imaging (MRI), particularly diffusion-weighted imaging (DWI), plays a valuable role in diagnosing HE. We present a case of acute hypoglycemia with reversible changes in the bilateral internal capsule seen on DWI-MRI.

Key words: Acute hypoglycemic encephalopathy, Diffusion-weighted imaging, Internal capsule, Magnetic resonance imaging

Acute hypoglycemic encephalopathy (HE) is a type of metabolic encephalopathy that can lead to serious morbidities and mortality, if severe and prolonged. The clinical and imaging features of acute HE can mimic those of ischemic stroke [1]. In acute HE, radio-imaging studies have shown reversible changes in magnetic resonance imaging (MRI) in specific locations such as the cerebral cortex, basal ganglia, hippocampus, splenium of the corpus callosum, internal capsule, and corona radiata [2,3]. Simultaneous involvement of multiple anatomical locations is also observed in a few cases [4]. However, isolated involvement of the bilateral posterior limbs of the internal capsule as observed in our case is an exceedingly rare presentation [5]. This unique presentation highlights the importance of considering various differential diagnoses in cases of acute neurological disturbances, particularly in the context of HE, and underscores the need for further investigation and understanding of such atypical manifestations.

CASE REPORT

A 78-year-old male patient was brought to the emergency department with complaints of confusion and loss of consciousness for the past 8 h. He had no history of previous episodes with similar symptoms. He had a history of type 2 diabetes mellitus for the past 12 years and has been on hypoglycemic drugs (Tab. Glipizide IP 5 mg) but with poor compliance. There was no history of hypertension or recent alcohol intake.

On examination, the patient was unconscious with a Glasgow Coma Scale score of 9/15. Doll's eye reflex was present, suggesting intact brainstem function. Pupils were fixed and dilated. However, no neck stiffness was present. His blood pressure was measured as 110/70 mm Hg, pulse rate was 78 beats/min, and temperature was normal.

The patient was referred for an MRI brain with suspicion of acute stroke. Routine investigations including a complete blood count, electrocardiogram, chest X-ray, serum electrolytes, and arterial blood gas were all within normal limits except for a random blood sugar level of 44 mg/dl. The initial MRI of the brain revealed symmetrical areas of hyperintense signals on diffusion-weighted imaging (DWI) (b value=1000 s/mm²) in the posterior limbs of the bilateral internal capsule. A reduction in the apparent diffusion coefficient (ADC) was also observed (Fig. 1a and b). However, no high-intensity signals were seen in the cerebral cortex, hippocampus, basal ganglia, or corpus callosum. There were no abnormal signals observed in the T2 fluid-attenuated inversion recovery sequence. In addition, there were no findings suggestive of acute infarction, recent hemorrhage, or space-occupying lesions.

The patient received an immediate infusion of 100 ml of 25% glucose, and shortly thereafter, he regained consciousness. His neurological symptoms also returned to normal the following day.

A follow-up scan performed after 7 days revealed complete resolution of the previously observed hyperintense signals

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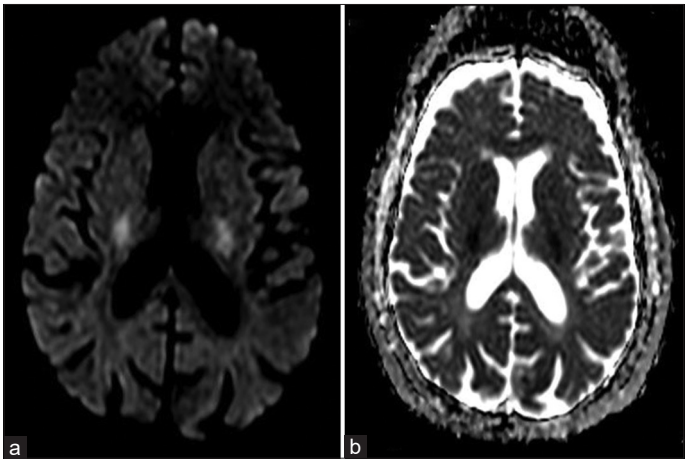

Figure 1: (a and b) Diffusion-weighted imaging in acute hypoglycemic phase showed high-intensity signals in bilateral posterior limbs of internal capsule (b value=1000 s/mm², TE 72 ms, TR 2.9 s) (a) with reduction in apparent diffusion coefficient values (b)

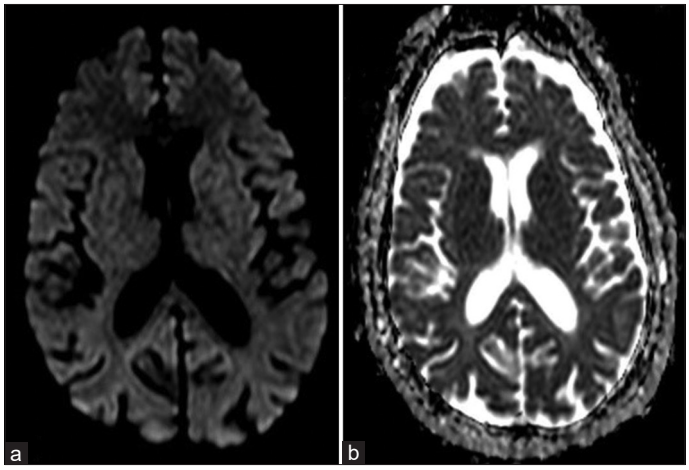

Figure 2: (a and b) Repeat scan performed 7 days after correction of blood glucose level showed complete resolution of hyperintense signals in bilateral posterior limbs of internal capsule on diffusion-weighted imaging (b value=1000 s/mm², TE 72 ms, TR 2.9 s) (a) with normalized apparent diffusion coefficient values (b)

on initial DWI and ADC values also returned to normal (Fig. 2a and b).

DISCUSSION

The neurological symptoms of HE include memory loss, confusion, behavioral disorders, motor function deficits, a persistent vegetative state, deep coma, or even death [6,7]. Clinically, HE can mimic acute ischemic stroke in terms of both symptoms and imaging features on DWI and ADC [1,8]. However, the former is usually reversible with immediate treatment, while the latter can lead to long-term sequelae. Therefore, differentiating between the two and making an accurate diagnosis poses a great challenge for treating clinicians.

Hypoglycemia can occur due to various causes including excessive use of hypoglycemic drugs or insulin, presence of an insulinoma (a tumor that produces insulin), drug intake, and certain medical conditions such as sepsis, renal failure, or hepatic failure.

Brain MRI, particularly the DWI sequence, is a valuable diagnostic tool for identifying acute HE. It measures changes in water diffusion between extracellular and intracellular spaces. Literature reports have shown hyperintense signals in various brain locations such as frontal, parietal, and occipital cortex [3] and basal ganglia, hippocampus, splenium of the corpus callosum, internal capsule, and corona radiata indicating abnormalities in these areas in HE. However, brain stem, cerebellum, and hypothalamus are typically unaffected [2,9].

The exact mechanisms underlying HE are not fully understood. Proposed mechanisms include energy failure, excitotoxic edema, and asymmetrical cerebral blood flow. Severe hypoglycemia disrupts protein synthesis, leading to neurochemical changes and selective neuronal necrosis. Excitotoxic edema involves increased glutamate levels and can affect glial cells. In cases of hypoperfusion, certain brain regions experience decreased cerebral blood flow. However, all these changes in HE are typically transient and abnormalities seen on DWI usually normalize over time with appropriate management [6].

Other conditions such as drug toxicity, seizures, and viral encephalitis [10] can also exhibit reversible diffusion restrictions. However, these conditions are usually accompanied by additional other abnormalities also, making it easier to differentiate them from HE. Our study emphasized that specific brain areas including the internal capsule are susceptible to hypoglycemic injury. Importantly, these lesions can be reversed upon correcting blood glucose levels.

CONCLUSION

We conclude that magnetic resonance findings of acute HE are characterized by high signal intensity on DWI and a decrease in ADC values at multiple specific anatomical locations. However, isolated and reversible involvement of bilateral internal capsules is rarely seen in clinical practice.

Hence, it is important to be aware of the various imaging features of acute HE that may mimic those of acute ischemic stroke, as both conditions have significantly different treatment outcomes. Early diagnosis and timely management of acute HE can save patients from long-term morbidities and mortality.

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