Case Reports

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Reversible Injury of Internal Capsule and Splenium in a Patient with Transient Hypoglycemic Hemiparesis

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Introduction

Diverse neurological manifestations complicating profound hypoglycemia are well-described, ranging from reversible focal deficits to irreversible coma and death [1, 2]. Among these, transient hypoglycemic hemiparesis is rarely reported and frequently misdiagnosed as stroke or transient ischemic attack (TIA), especially in the elderly patients [3]. We report here a patient with transient hypoglycemic hemiparesis in whom sequential MRI examinations showed reversible injury in the internal capsule and splenium. We also discuss its possible pathogenesis and clinical implications based on MRI findings.

Case Report

A 78-year-old woman with no history of stroke was taken to the Guro Hospital emergency department after being found dysarthric and less responsive at home. Her husband noted that on the day of admission she developed diaphoresis and tremulousness after not having two consecutive meals, followed by dysarthria, right-sided weakness, and somnolence. She was diagnosed as having type 2 diabetes 5 years before and has remained on gliclazide 80 mg/day. She had no history of hypertension, migraine, seizure, or drug abuse. On admission, she was somnolent but easily arousable to painful or auditory stimuli. She was confused and disoriented to time and place. Vital signs were normal. Initial blood glucose was 38 mg/dl. Other blood works including complete blood count, arterial blood gases, electrolytes, chemistries, blood urea nitrogen, creatinine, and a drug screen were all within the normal range. Initial diffusion-weighted imaging (DWI) per-

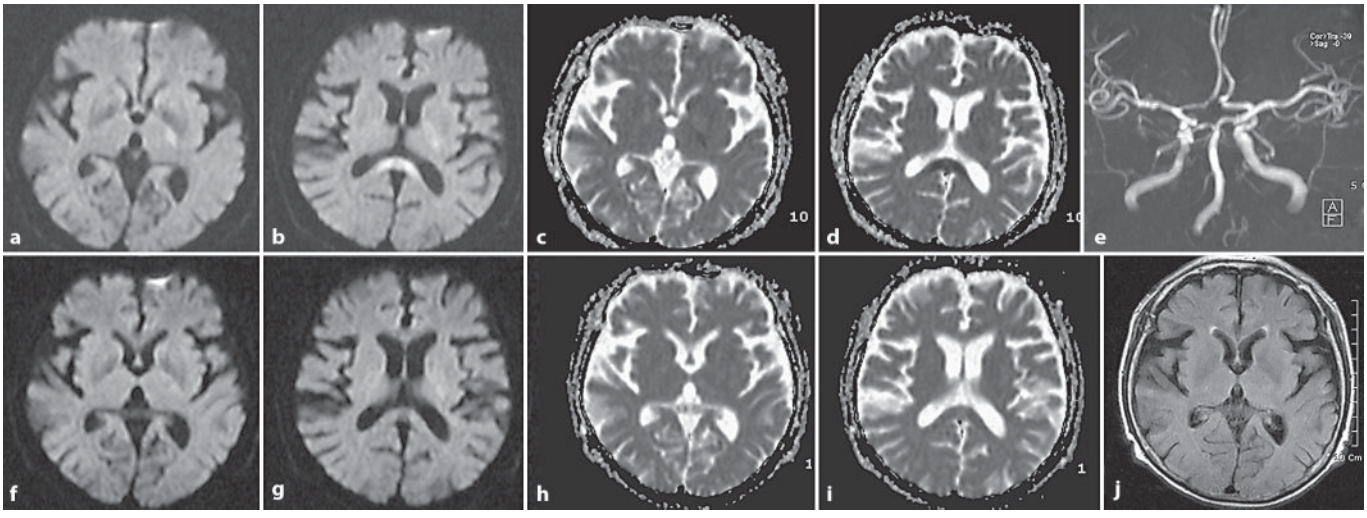

Fig. 1. Initial diffusion-weighted imaging (DWI) at the time of profound hypoglycemia showed hypersignals in the posterior limb of left internal capsule and splenium of corpus callosum with reduced apparent diffusion coefficient (ADC) values (**a–d**). A simultaneous MR angiography showed no evidence of significant stenosis or spasm in the major intracranial arteries (**e**). Repeat DWI performed 12 h after correction of profound showed complete resolution of the lesions with normalized ADC values (**f–i**). Another fluid-attenuated inversion recovery (FLAIR) MRI performed 3 days later showed no residual lesion (**j**).

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formed 8 h after the symptom onset disclosed hypersignals in the posterior limb of left internal capsule and splenium of the corpus callosum with reduced apparent diffusion coefficient (ADC) values (fig. 1a–d). There was no definite stenosis of the major cerebral arteries on a simultaneous MR angiography (fig. 1e). Intravenous dextrose was injected immediately after the first MRI examination, and her mental status had gradually returned to normal within 1 h. She was then found to have mild dysarthria and right hemiparesis (Medical Research Council grade 3) with enhanced tendon reflexes on the right side. Cranial nerve and sensory functions were normal. EKG monitoring and echocardiography failed to show an evidence of embolic source. Blood glucose was increased to 145 mg/dl, and her right hemiparesis and dysarthria had gradually improved to normal over the next 6 h. Repeat DWI performed 12 h after correction of hypoglycemia showed complete resolution of hypersignals observed on initial DWI with normalized ADC values (fig. 1f–i). Another MRI performed 3 days later showed neither visible lesion nor circumscribed hemorrhage close to initial lesions on DWI (fig. 1j). The dose of oral hypoglycemic agent was adjusted and she has remained asymptomatic thereafter.

Discussion

Available MRI reports on hypoglycemic brain injury are limited and manifested by lesions that involve cerebral cortex, basal ganglia, and hippocampus, implying that these areas are more vulnerable to hypoglycemia [4, 5]. However, no consensus has been reached as to selective vulnerability of these areas to hypoglycemia. It is of interest that in our patient the lesions were identified in the internal capsule and splenium that may be responsible for hemiparesis and altered mental status, respectively [6–8]. This confirms recent reports showing lesions in the internal capsule and splenium in patients with profound hypoglycemia [6–10], suggesting that these areas should be added to the list of selective vulnerability to hypoglycemia.

Several hypotheses have been proposed regarding the pathogenesis of hypoglycemic brain injury, but the exact mechanism is unclear to date [11, 12]. Significant vascular spasm or underlying atherosclerosis in long-standing diabetic patients may be implicated in the pathogenesis of transient neurological deficits. In our patient, however, these possibilities could be excluded based on normal MR angiography and the lesion topography that did not conform to vascular distribution.

The first MRI examination in our patient showed hypersignals on DWI with reduced ADC values. The second performed 12 h after appropriate correction of hypoglycemia showed complete resolution of the lesions, as did neurological deficits, indicating the lesions are reversible cytotoxic edema in nature. These DWI/ADC alterations could be encountered not only in focal ischemia but also hypoglycemia itself, but their underlying mechanisms seem distinct. Glucose deprivation leads to severe metabolic energy depletion and subsequent membrane ionic pump failure that result in a restriction of water diffusion, as does ischemia, but the topographical and temporal evolution of hypoglycemic brain injury are different from ischemia [13]. Furthermore, complete or partial recovery of ADC reductions could be demonstrated after glucose supplementation, suggesting that early ADC reductions do not necessarily portend irreversible brain injury [13, 14]. Taken together, we conclude that several brain areas including internal capsule and splenium have selective vulnerability to hypoglycemia, and that the lesions can be reversed potentially with correction of hypoglycemia. This has important clinical implications. The timing of neurological recovery may be related to the duration and severity of the hypoglycemic injury [13]. Therefore the patients, if not treated promptly with glucose intake, may have permanent neurological deficits.

Transient hypoglycemic hemiparesis is a rare but important presentation of hypoglycemia that can be misinterpreted as stroke or TIA. Although the radiological and clinical features may be identical, the underlying mechanisms and treatment strategies are distinct between these two disorders. It is our intention to increase awareness of this disorder which, without early recognition and prompt correction of hypoglycemia, could result in permanent neurological deficits.

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