Transient Hypoglycemia-induced Hemiparesis Mimicking Stroke

– A Case Report –

Seong Hwa Lee, M.D., Ji Ho Ryu, M.D.*, Yong In Kim, M.D.*, Maeng Real Park, M.D.*,
Mun Ki Min, M.D.* and Sun Min Hwang, M.D.*

Department of Emergency Medicine, Pusan National University Hospital, Busan,

*Department of Emergency Medicine, Pusan National University Yangsan Hospital, Yangsan, Korea

Hypoglycemia is caused by poor oral intake, excessive exercise, alcohol abuse and inaccurate use of a hypoglycemic agent or insulin in patients that have history of diabetes mellitus (DM), especially in the elderly. Severe hypoglycemia has a variety of different symptoms or signs from focal neurologic deficits to severe coma, or death. It can be difficult to differentiate hypoglycemia-induced symptoms or signs, and stroke or cardiovascular disease in acute setting. Transient hypoglycemic hemiparesis is an infrequent case in the emergency department (ED), which is frequently misdiagnosed for stroke. When patients with decreased mental status or hemiparesis are admitted to the ED, a routine blood sugar test is essential. Hypoglycemic hemiparesis if unrecognized can result in permanent neurological damage. Therefore, it is important to detect hypoglycemia early and treat it appropriately.

Key Words: hemiparesis, hypoglycemia, stroke.

Hypoglycemia is caused by poor oral intake, excessive exercise, alcohol abuse and inaccurate use of a hypoglycemic agent or insulin in patients that have history of diabetes mellitus (DM), especially of elderly. Severe hypoglycemia has a variety of different symptoms or signs from focal neurologic deficits to severe coma, or death. It can be difficult to differentiate hypoglycemia-induced symptoms or signs and stroke or cardiovascular disease in acute setting. In addition, patients with cardiovascular disease have a DM commonly. Clinicians think easily that hemiparesis is not a symptom of hypoglycemia, but a symptom of stroke.

Therefore, we report two cases experienced that patients with transient hypoglycemic hemiparesis but diagnosis is not acute stroke.

CASE REPORT

A 50-year-old male patient with a 15-year history of DM and hypertension who was receiving oral hypoglycemic agent and antihypertensive agent admitted to an emergency department (ED) via 119 fire officer. He woke up 7 AM, and felt some dysarthria but he slept again. At 4 PM, He felt dysarthria was getting worse and the left arm and the leg weakness and developed progressive decreasing of mental status.

On neurological examination, the patient was drowsy mentality with left hemiparesis (upper extremities motor G4-5/G0-1, lower extremities motor G5/G0-1). Initial blood pressure was 220/160 mmHg and glucose level was 33 mg/dl. He was administered 20% dextrose 50 ml and immediately checked Magnetic resonance imaging (MRI) in the ED. After intravenous administration of glucose, serum glucose level was 77 mg/dl, and the patient showed full recovery of his neurological condition and consciousness. Diffusion-weighted imaging (DWI) of MRI revealed high signal intensity in the posterior rim of the right internal capsule with reduced apparent diffusion coefficient (ADC) (Fig. 1). MR angiography excluded arterial occlusion and stenosis. Follow-up DWI performed 3-days after the first MR showed normalization of signal intensity and

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Correspondence to: Ji Ho Ryu, Department of Emergency Medicine, Pusan National University Yangsan Hospital, Beomeo-ri, Mulgeum-eup, Yangsan 626-770, Korea
Tel: 82-55-360-2143 Fax: 82-55-360-2173
E-mail: pnuyhem@gmail.com

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Fig. 1. Initial diffusion-weighted imaging (DWI) and ADC (apparent diffusion coefficient) of MRI. Initial DWI with high signal intensity and ADC with low signal intensity in the posterior rim of the right internal capsule (white arrow).

Fig. 2. Follow-up diffusion-weighted imaging (DWI) and ADC (apparent diffusion coefficient) of MRI. After 3 days, follow-up DWI and ADC map show nearly normalization of signal intensity in the posterior rim of the right internal capsule.

ADC abnormality (Fig. 2). The patient discharged at 7-admission days without other symptoms and complications.

Other case is a 81-year-old female with 5-year history of DM who was receiving oral hypoglycemic agent. She had no history of other vascular disease. When she admitted to the ED, mental status was decreased and had right hemiparesis on neurological examination. Initial blood pressure was 190/110 mmHg and glucose level was 42 mg/dl. She was administered 20% dextrose 50 ml and checked CT angiography in the ED. After intravenous administration of glucose, serum glucose level 60 mg/dl, and the patient showed recovery of mental status and right hemiparesis. CT angiography excluded arterial occlusion and stenosis. She discharged in the ED after 8 hours without other symptoms or signs and complications.

DISCUSSION

Mechanisms about hypoglycemia induced neuronal damage involves cytotoxic edema, shrinkage of the extracellular space and failure of ionic pumps of the cell membrane after energy

depletion.[1] Another hypothesis is the disruption of protein synthesis in the superficial layers of the cortex, in the basal ganglia and hippocampus.[2,3]

There is a relative rarity of reports about hypoglycemia-induced brain lesions. In newborns, the parieto-occipital region is the most affected. In adults, there is a greater variability, with reports of lesions in basal ganglia, hippocampus, temporal and occipital gray matter, insula cortex, corona radiata and splenium of the corpus callosum.[2,4,5] Available MRI reports on hypoglycemic brain injury are limited and manifested by lesion that involve cerebral cortex, basal ganglia, and hippocampus, implying that these areas are more vulnerable to hypoglycemia but no consensus has been reached as to selective vulnerability of these areas to hypoglycemia.[6] In recent reports, internal capsule and splenium were showing lesions in patients with profound hypoglycemia, that suggesting that these areas should be added to the list of selective vulnerability to hypoglycemia.[7-9]

MRI techniques, particularly diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) mapping, are

the most useful tools in diagnosing early ischemic injury.[7] Aoki et al.[3] described reversible abnormalities in diffusion-weighted MRI 10 days after glucose infusion. The determination of blood glucose levels, the effects of its infusion and the comparison of MRI images on the day of admission to one taken several days after glucose infusion can be useful tools to make differential diagnosis. Diffusion-weighted MR imaging characterized the present case of hypoglycemia as areas of abnormal signal intensity in the splenium of the corpus callosum and/or the posterior limb of the internal capsule which did not confirm to the vascular distributions.[9,10] In our reports, first case is discovered high intensity in the posterior limb of the right internal capsule in diffusion-weighted MRI. After 7 days, clinical manifestations and higher intensity in diffusion-weighted MRI were disappeared. So, we thought that this is characteristic hypoglycemia-induced brain injury.

In conclusion, transient hypoglycemic hemiparesis is an infrequent case in the ED, which is frequently misdiagnosed for stroke. When patients with decreased mental status or hemiparesis are admitted to the ED, a routine blood sugar test is essential. Hypoglycemic hemiparesis if unrecognized can result in permanent neurological damage. Therefore, it is important to detect hypoglycemia early and treat it appropriately.

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