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Evolution of Lesion in Hypoglycemic Hemiplegia: Diffusion-weighted Imaging

Hipoglisemik Hemiplejide Lezyonun Evolüsyonu: Difüzyon-ağırlıklı Görüntüleme

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Dear editor,

An 87-year-old male presented to the emergency department with right arm numbness, inability to speak, blank staring, and suddenly started aimless arm/leg movements. Symptoms lasted for 45 minutes. He had diagnoses of hypertension, diabetes mellitus, atrial fibrillation and coronary artery disease for a for about ten years. His medication included rivaroxaban, irbesartan, pitavastatin, metoprolol, furosemid, gliclazide, and insulin lispro. Neurological examination was found to be normal at admission. Diffusion-weighted imaging (DWI, b=1000) performed with the pre-diagnosis of transient ischemic attack (TIA) was interpreted normal (Figure 1A).

The patient suddenly became right hemiplegic and dysarthric in the emergency room. The blood glucose level was measured 43 mg/dl. In the second DWI performed during this episode, a clinically relevant 0.43 cm^2 lesion characterized by diffusion restriction [apparent diffusion coefficient (ADC): 0.35.10-3 mm^2/sec, left to right ratio: 0.44] was detected in the posterior limb of the left internal capsule (Figure 1B).

The initial DWI, obtained 3.5 hours ago when the patient was asymptomatic, was reevaluated and a punctual (0.06 cm^2) lesion with mild ADC decrease (ADC: 0.55.10-3 mm^2/sec, left to right ratio: 0.65) could be discerned in the same region. With meticulous normalization of blood glucose, clinical improvement was dramatic and the DWI lesion resolved rapidly (Figure 1C, D, E).

Presence of diffusion imaging just before the hypoglycemia episode, occurrence of a repeated episode in the emergency room, and prompt reimaging make this case unique in terms of documenting the temporal course of hypoglycemic lesions.

The pathophysiology of rapidly reversible DWI lesions, that have been clearly documented herein, has not been fully elucidated. The diffusion restriction seen in short episodes of hypoglycemia is not caused by ischemia or epileptic activity. Regional energy deficiency, sodium/potassium pump dysfunction, excitotoxic oligodendrocyte and myelin sheath swelling and local alkalosis may be involved (1,2,3).

The absence of axon damage is the reason for the rapid resolution. Prioritizing areas where axon bundles are concentrated, such as the posterior limb of internal capsule, may also be associated with excitotoxic myelin edema (1,3,4).

The clinical significance of hypoglycemia is that these episodes may be misdiagnosed as TIA or acute stroke. This may lead to undertreatment of hypoglycemia and ultimately in permanent cerebral damage. The way to prevent this is to become familiar with the imaging characteristics associated with hypoglycemia.

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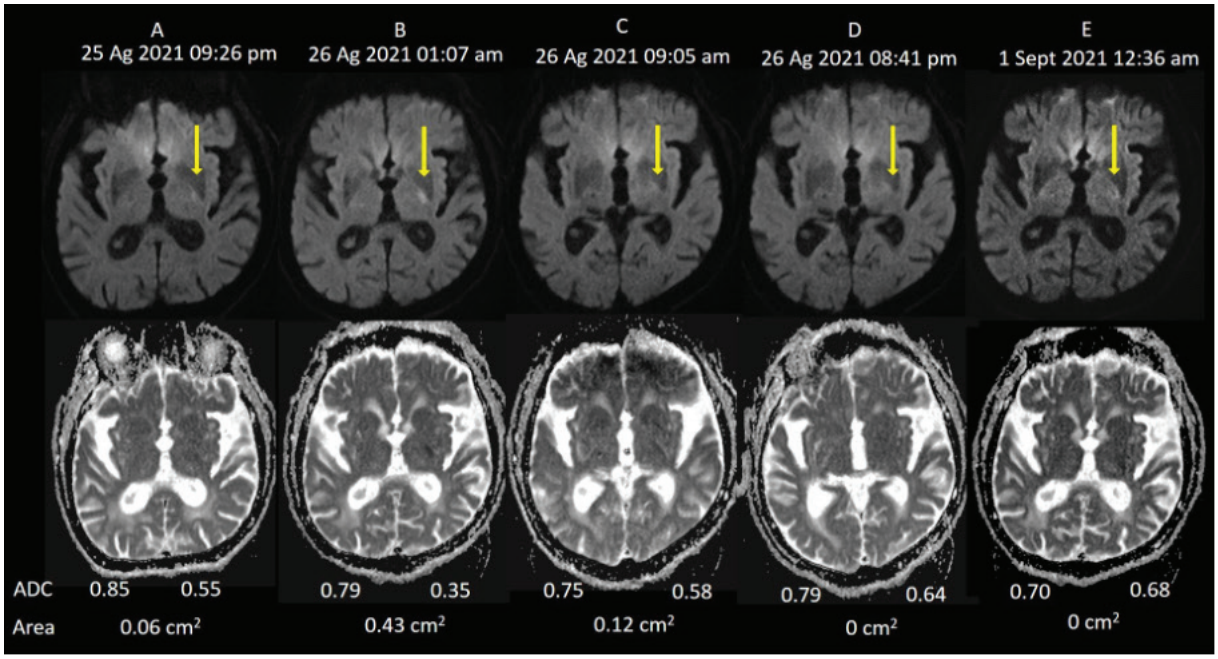

Figure 1. Serial diffusion-weighted imaging obtained with b values 0, 500, and 1000 sec/mm^2 of the patient (top row: Isotropic diffusion images; bottom row: Apparent diffusion coefficient (ADC) maps). The numbers on the bottom of the ADC images demonstrate the ADC values in the posterior limb of the right and left internal capsules (PLIC) and the affected area of the left PLIC

Ethics

Informed Consent: Written consent was obtained.
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Authorship Contributions

Surgical and Medical Practices: İ.G.O., R.G., M.A.T., Concept: İ.G.O., R.G., M.A.T., Design: İ.G.O., R.G., M.A.T., Data Collection or Processing: İ.G.O., R.G., M.A.T., Analysis or Interpretation: İ.G.O., R.G., M.A.T., Literature Search: İ.G.O., R.G., M.A.T., Writing: İ.G.O., R.G., M.A.T.

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