A 73-year-old diabetic man treated with oral hypoglycemic agents presented with dysarthria. He was alert and could obey simple verbal and written commands. His dysarthria was too severe to understand. Muscle tone of his legs was increased. Deep tendon reflexes were generally decreased without pathological reflex or clonus. The blood glucose level was 44 mg/dL. Diffusion-weighted MR imaging (DWI) showed symmetric hyperintensity of the bilateral posterior limbs of the internal capsule (PLIC) (Picture A). The lesions seemed elongated along the pyramidal tracts (Picture B, C). Intravenous glucose injection immediately improved his dysarthria and spasticity. Exitotoxic edema has been proposed as the pathomechanism of hypoglycemic encephalopathy. Glutamate induces glial edema and the myelin sheath might protect axons from intracellular edema and irreversible damage (1). Spatial distribution of the presented case supports this hypothesis and there might be tract susceptibility since PLIC or corona radiata were reportedly involved in 8 of 11 cases of hypoglycemic encephalopathy (1).

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Reference