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LETTER TO THE EDITOR
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Hashimoto Encephalopathy Mimicking Acute Ischemic Stroke: Perfusion-Weighted Magnetic Resonance Imaging

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Dear Editor,

A 61-year-old woman presented with sudden-onset left hemianopia. She had a history of hypertension but no other remarkable medical history, including thyroid disease. The findings of the initial brain computed tomography (CT) were normal, and CT angiography showed no cerebral arterial occlusive lesion. Intravenous tissue plasminogen activator (tPA) was administered at 3 hours after symptom onset due to the impression of an infarction in the right posterior cerebral arterial territory. The visual symptom was not improved after tPA use. Brain magnetic resonance imaging (MRI) performed 6.5 hours after symptom onset showed no acute ischemic lesion, but perfusion-weighted MRI showed perfusion delay at the right parieto-occipital lobe, which was compatible with her visual symptom (Fig. 1A and B). Magnetic resonance (MR) angiography revealed no remarkable stenosis or occlusion (Fig. 1C). A visual field test showed left homonymous hemianopia (Fig. 1D, upper panel). We prescribed an antiplatelet agent, and the patient was discharged with her remaining visual symptom.

Ten days after discharge she was readmitted due to right-sided hypesthesia and mental confusion. Hemianopsia was also detected in a follow-up visual field test. Brain MRI showed no ischemic lesion, and MR angiography findings were normal (Fig. 1E). The findings of a cerebrospinal fluid examination (160 mmH_2O opening pressure, 2 red blood cells/mL, 0 white blood cell/mL, 56.6 mg/dL protein, and 62 mg/dL glucose) and other laboratory tests were unremarkable. Although the findings of a thyroid function test (TFT) were normal [79.5 ng/dL T3 (normal: 71−161 ng/dL), 1.3 ng/dL free T4 (normal: 0.8−1.7), and 1.89 μIU/mL thyroid-stimulating hormone (normal: 0.86−4.69 μIU/mL)], both antithyroglobulin antibody [138.5 IU/mL (normal: 10−124.2 IU/mL)] and antithyroid peroxidase antibody [126.0 IU/mL (normal: 5−13.6 IU/mL)] were elevated. The suspicion of Hashimoto encephalopathy resulted in corticosteroid therapy being started, which gradually improved her neurological symptoms. A visual field test performed after completing steroid therapy showed no visual field defect (Fig. 1D, lower panel).

Hashimoto encephalopathy has been described as a syndrome of encephalopathy and high serum antithyroid antibody concentrations that responds to glucocorticoid therapy.[1] Although it presents with various clinical manifestations, two subtypes have been previously proposed: a vasculitic type and a diffuse progressive type.[2] Several pathogenic mechanisms underlying Hashimoto encephalopathy have been proposed: 1) autoimmune-mediated cerebral vasculitis, with or without immune complex deposition and 2) an antineuronal antibody-mediated mechanism.[2-4] Brain single-photon-emission CT and positron-emission tomography scans have revealed perfusion abnormalities in several cases: either focal or global hypoperfusions.[1,5-7] However, there has been no report of a perfusion delay in brain MRI dur-

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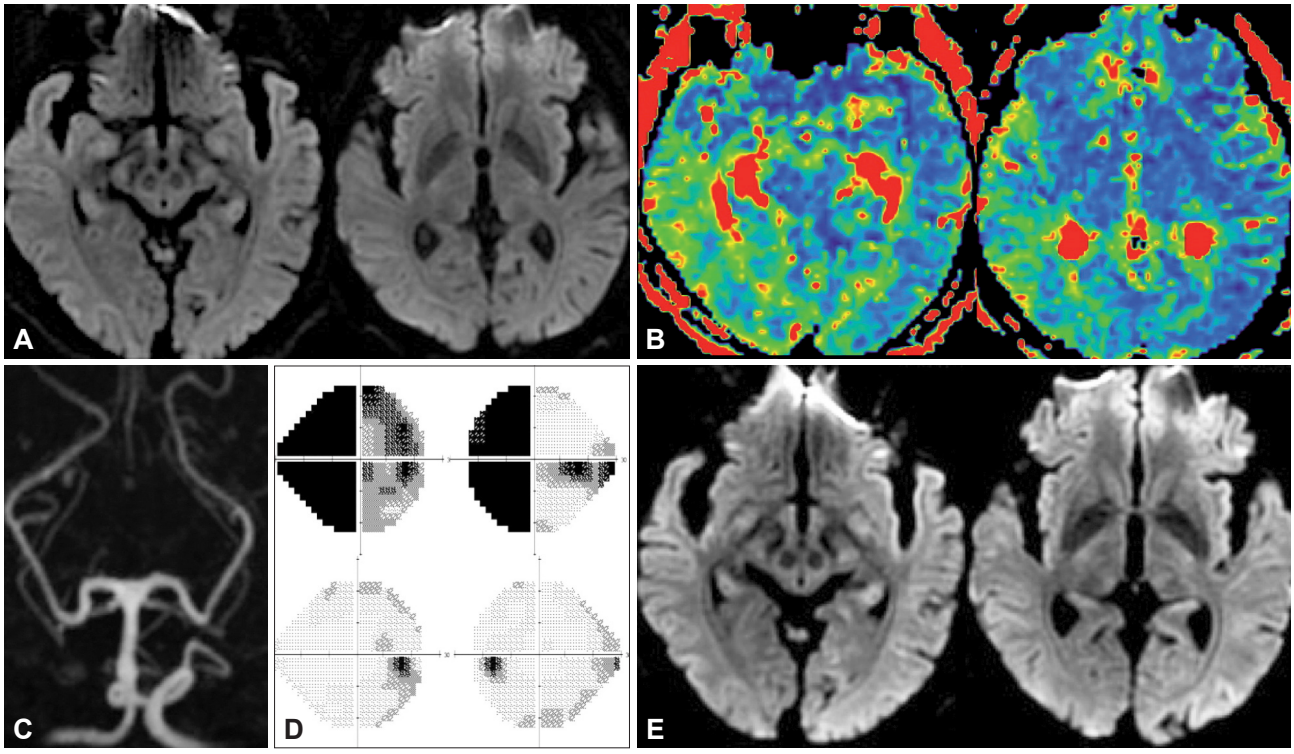

Fig. 1. Brain MRI and visual field examination of the patient. A: Initial brain diffusion-weighted MRI shows no acute ischemic lesion. B: The mean transit time is delayed in the right parieto-occipital lobe in perfusion-weighted MRI. C: Magnetic resonance angiography shows that there is no significant steno-occlusive lesion in the posterior cerebral artery. D: A left homonymous field defect is noted in the initial visual field examination (upper panel), and this is normalized after steroid therapy (lower panel). E: Follow-up MRI shows no ischemic lesions. MRI: magnetic resonance imaging.

ing the hyperacute period of cerebral symptoms. In the present case we performed perfusion-weighted MRI several hours after the onset of cerebral symptoms, which resulted in the perfusion delay being misinterpreted as a true ischemic stroke lesion. The absence of a diffusion-restriction lesion in two serial brain MRI scans helped to rule out ischemic stroke as a cause of the neurological symptom. With suspicion of other etiologies of cerebral symptoms, an antithyroid antibody test was performed, for which the results were abnormal. In agreement with the present case, normal TFT results have been reported in 22% of patients with Hashimoto encephalopathy.[1] We speculated that the neurological sign in the present patient resulted from a vasculitic lesion of the brain, based on the relatively abrupt onset of the neurological symptom and the focal hypoperfused cortical regions.

Conflicts of Interest

The authors have no financial conflicts of interest.

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