Magnetic Resonance Imaging of Herpes Simplex Virus Encephalitis: Reversible Asymmetric Basal Ganglia Lesions

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We report a patient with herpes simplex virus type 1 encephalitis (HSE) who showed abnormal magnetic resonance imaging (MRI) signals in the basal ganglia. The lesions were asymmetric and became apparent with relapse of the neurological symptoms, but they completely disappeared, concomitantly with improvement of the illness.

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Key words: putamen, thalamus, acyclovir, adenine arabinoside (Ara-A)

Introduction

Herpes simplex virus type 1 encephalitis (HSE) is the most common sporadic, acute focal encephalitis, and characteristically involves the medial temporal lobe, inferior frontal lobe and limbic system. Magnetic resonance imaging (MRI) is more sensitive than computed tomography (CT) in demonstrating early edematous changes in the involved regions. Recent neuroradiological studies have demonstrated an atypical form of HSE, presenting with prominent involvement of the thalamic nuclei (1), lateral geniculate body (2), cingulate gyrus (3) or occipital lobe (4). We report here a patient with HSE who showed strikingly asymmetric high signal intensity of the basal ganglia on T2-weighted MRI.

Case Report

A 41-year-old man with a one-week history of fever, abnormal behavior and convulsions was transferred from another hospital. At the time of transfer, the patient was confused (score on the Japan Coma Scale [JCS]: 10), inattentive and forgetful, and had a temperature of 38.4°C. There was no prior history of neurologic or psychiatric illness, except for mild lumbar disc hernia. On neurological examination, he exhibited nuchal rigidity, dysarthria and mild left hemiparesis. Cerebrospinal fluid (CSF) obtained by lumbar puncture contained 152 cells/mm³, which were all lymphocytes, and 170 mg/dl of protein. The titer of anti-herpes simplex virus type 1 (HSV-1) IgM antibody measured by EIA was not significantly increased in CSF.

Cranial MRI (1.0 T, Siemens Magnetom) performed 4 days after admission revealed marked high signal intensity at the right temporal lobe in the T2-weighted images. Since HSE was suspected, acyclovir was initiated at a dose of 1,500 mg/day. Other drugs to control seizures and brain edema were also administered. He became afebrile and his consciousness started to improve gradually (to JCS 2-3).

Eight days after admission, his consciousness worsened again (JCS 100) and mild right hemiparesis appeared. The cell count in CSF increased to 282 cells/mm³, mostly lymphocytes, and the protein level remained high (116 mg/dl). The CSF titer of anti-HSV-1 IgM antibody was elevated to 3.07 EIA units. T2-weighted MRI obtained at this time showed new high signal intensity at the bilateral putamen and the left temporal lobe, in addition to the high signal intensity at the right temporal lobe that had been observed at admission. High signal intensities were also found in parts of the bilateral thalami, but these signals were weaker than those in the putamen and temporal lobe. The lesions in the putamen were asymmetric, with higher signal intensity on the left (Fig. 1, right). The T1-weighted image showed linear high signal intensity involving the medial side of the left putamen (Fig. 1, left). Nine hundred mg per day of adenine arabinoside (Ara-A) was added to acyclovir for ten days. He gradually became alert and the paresis of the limbs disappeared within three weeks. MRI obtained two months later demonstrated decreased high signal intensity in the putamen and the thalami, and follow-up MRI performed ten months later revealed residual high signal intensity in the bilateral temporal lobes, but complete resolution of the high signal intensity in the putamen and the thalami (Fig. 2). The cell count and protein level in CSF were normalized and the anti-HSV-1 IgM titer in CSF was significantly decreased (1.26 EIA units) within two months. During his illness, involuntary movements such as choreoathetosis or dystonia were not observed.

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Figure 1. Right: T2-weighted MRI (TR: 3,000 msec, TE: 90 msec) performed 4 days after admission reveals high signal intensity in the putamen with an asymmetric distribution. Parts of the bilateral thalami showed weaker high signal intensity. Left: A part of the putaminal lesions is seen as linear high signal intensity on the T1-weighted image (TR: 600 msec, TE: 25 msec). TR: repetition time, TE: echo time.

Figure 2. T2-weighted MRI (TR: 3,000 msec, TE: 90 msec) performed ten months later. High signal intensity in the temporal lobes is apparent (Left), but that in the putamen and thalami has almost disappeared (Right).
Discussion

The diagnosis of HSE in our patient was supported by the clinical features, typical temporal lobe involvement visualized by MRI and raised CSF anti-HSV-1 IgM antibody titers. Although HSE causes severe selective damage to the basal frontotemporal region and limbic system as a necrotizing hemorrhagic encephalitis, basal ganglia involvement is extremely uncommon (5). If such involvement is noted, it is usually unilateral and mild (6). Extrapyramidal movement disorders such as chorea, dystonia, choreoathetosis and ballistic movements have been reported in association with HSE, usually presenting as the first sign of relapse (7–9). However, imaging of the brain in many of these studies disclosed no abnormalities in the basal ganglia, except for one male infant with HSE presenting with myoclonus who showed calcification at the bilateral thalami and basal ganglia (10). Acute bilateral basal ganglia lesions are often observed in patients with hypoxia, hypoglycemia, carbon monoxide poisoning, hemolytic-uremic syndrome or osmotic myelinolysis (11), but such complications were not seen during the course of our patient’s illness.

The richly vascularized basal ganglia, with their end-vessel vascular supply, are prone to infections carried via the blood, and lesions of basal ganglia are occasionally observed in viral or non-viral encephalitis (12–14). However, the high signal intensity at the basal ganglia in our patient characteristically appeared in association with the symptoms of relapse, and was almost resolved within two months concomitantly with clinical improvement associated with additional antiviral therapy. This is distinct from the abnormal signals in the temporal lobes, which remained for more than ten months. These observations make it unlikely that the lesion in the basal ganglia were due to direct invasion by HSV-1 and associated inflammation. This hypothesis is supported by the fact that brain biopsy at the time of relapse rarely gives evidence of an active viral process, in terms of either detection of viral particles by electron-microscopy or culture for HSV-1 (7). Encephalitis may result in vascular compromise, hypoxic change, and subsequent hemorrhagic infarction (10). Thus, the high signal intensity on the T2-weighted image and that on the T1-weighted image are considered to reflect vascular damage with circulation disturbance, rather than a special affinity of HSV-1 for the basal ganglia (15).

Although our patient received the standard dosage of acyclovir, relapse of neurological symptoms after initial improvement occurred after 8 days of treatment. Incomplete antiviral therapy may often contribute to the relapse of HSE, and relapse is a predictor of poor prognosis for HSE patients (9). In our patient, early addition of Ara-A promoted recovery and may have prevented severe ultimate deficit. This patient shows that HSE may produce focal findings in cerebral regions other than the basal frontotemporal lobe areas. The possibility of such an atypical appearance of lesions of basal ganglia should be recognized when recurrence of symptoms is observed during the course of HSE. It should also be emphasized that HSE, which is considered to be acute encephalitis, may take a subacute or chronic course in some patients.

References