Nonconvulsive Partial Status Epilepticus Mimicking Recurrent Infarction Revealed by Diffusion-weighted and Arterial Spin Labeling Perfusion Magnetic Resonance Images

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"Non-convulsive" partial status epilepticus (SE) is an important pathologic condition that should be differentiated from cerebral infarction. Herein, we reported 2 patients who had partial SE associated with old infarction in the right parietal lobe. Each patient had 2 episodes of left hemiparesis and hemisensory disturbance without convulsion. On diffusion-weighted magnetic resonance images (DW-MRI), a hyperintense lesion was noted in the cortex around the old infarction lesion, and recurrent infarction was suspected. Although electroencephalography (EEG) failed to reveal ictal discharges or interictal paroxysmal activities in 3 of 4 episodes, perfusion images with arterial spin labeling (ASL) clearly demonstrated ictal hyperperfusion in the area corresponding to the cortical hyperintense lesion on DW-MRI. After appropriate anticonvulsant treatment based on the diagnosis of partial SE, clinical symptoms were completely improved. These data stress the importance of cortical hyperintensity on DW-MRI and ictal ASL hyperperfusion, even when SE cannot be determined from EEG. **Key Words:** Stroke mimics—ictal hyperperfusion—nonconvulsive status epilepticus—diffusion-weighted image—arterial spin labeling.

Epilepsy is the most common disorder that can mimic signs of stroke.[1] Although "convulsive" status epilepticus (SE) is easily recognized, inhibitory seizures such as "non-convulsive" prolonged partial epilepsy and partial SE are difficult to differentiate from stroke.[2] In acute stroke, diffusion-weighted magnetic resonance images (MRI) (DW-MRI) and perfusion MRI (PI) are typically used, although recent studies demonstrated that these techniques provide information in the peri-ictal phase in epilepsy patients.[3-5] In SE, the epileptogenic cortex is in an electrophysiologically extreme state, and the activated cortex exhibits increased glucose and oxygen usage, thereby causing compensatory regional hyperperfusion. Arterial spin labeling (ASL) is a noninvasive and repeatable PI technique, which uses magnetically labeled blood water as an endogenous tracer.[6] Recent reports have described the appearance of "ictal hyperperfusion" with ASL.[6-10] When the hyperperfusion is no longer sufficient to supply the hyperactive cortical area, pathophysiologic changes leading to cytotoxic edema in epileptic cortical neurons can occur, which appear as an abnormal high signal in the cortical lamina (cortical hyperintensity) on DW-MRI.[3,7,11-15] These MRI findings of low apparent diffusion coefficient and high signals on DW-MRI in SE resemble those of acute ischemic stroke, indicating changes attributable to both cytotoxic and vasogenic edema.[4,7] Such ictal changes on ASL and

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DW-MRI were reversible in most cases.[5,7] In the present study, using serial MRI including DW-MRI and ASL and electroencephalography (EEG) recording, we examined 2 patients with nonconvulsive partial SE clinically resembling recurrent infarction around the old infarction.

Methods

Magnetic Resonance Image

Brain MRI with routine protocols and PI were performed using a 3-T magnetic resonance unit (Signa HDxt 3.0T, version 23; GE Healthcare, Milwaukee, WI). Routine protocols included axial diffusion-weighted echo planar sequences (b value = 1500 seconds/mm^2; repetition time (TR)/echo time (TE), 6000/min), T1 fluid-attenuated inversion recovery (T1-FLAIR) sequences (TR/TE/TI, 2050/16.1/AUTO), and T2-weighted fast spin-echo sequences (TR/TE, 4400/100) and T2-FLAIR sequence (TR/TE/TI, 9000/140/AUTO).

ASL was prepared using 3-dimensional, spiral, fast spin-echo sequence with background suppression for perfusion imaging covering the entire brain. A pulsed continuous scheme was employed. Other acquisition parameters were as follows: 4 arms with 1004 points in each spiral arm, phase encoding in the z direction = 32, section thickness = 4 mm, TR = 4728(AUTO) seconds, postlabel wait = 1.525 seconds, and number of excitations = 3.

Electroencephalography

Routine EEG recordings were obtained from an 18-channel digital EEG machine (Neurofax; Nihon-Kohden, Tokyo, Japan) with electrode placement according to the International EEG 10-20 system. The EEG recordings were performed for at least 30 minutes for each patient in resting conditions.

Results

Case 1

A 74-year-old female developed left hemiparesis and was admitted to us. She was alert on arrival (day 1). She had left hemiparesis and sensory disturbance in the left extremities, although convulsive seizures were not apparent. Emergency MRI with FLAIR revealed multiple old infarctions in the right parietal lobe and deep white matter on the both side (Fig 1, A). The cortex of the old infarction area in the right parietal lobe exhibited cortical laminar necrosis. On DW-MRIs, a hyperintense lesion was noted in the cortex around the old infarction site in the right parietal lobe (Fig 1, B). The precentral gyrus was not involved in this cortical hyperintensity. The tentative diagnosis was recurrent infarction around the old right parietal infarction, and an antithrombin agent (argatroban hydrate) and a free radical scavenger (edaravone) were administrated intravenously. The left hemiparesis and hemisensory disturbance were gradually improved, and DW-MRI on day 4 demonstrated dramatic disappearance of cortical hyperintensity in the right parietal lobe (Fig 1, C). However, ASL clearly showed hyperperfusion in the corresponding area to that of the cortical hyperintensity on DW-MRI of day 1 (Fig 1, D). Diagnosis of symptomatic partial epilepsy was made and intravenous phenytoin followed by oral carbamazepine was administered. On day 5, she completely recovered from left hemiparesis and sensory disturbance, and EEG demonstrated intermittent focal slow wave on the right centroparieto-occipital region (Fig 2, A).

Unfortunately, the patient chose to discontinue carbamazepine treatment when she was transferred to the orthopedic department. Ten months later, she again developed left hemiparesis and hemisensory disturbance (day 1′, day 1 of the second episode). On DW-MRIs, the cortical hyperintense lesion was again noted around the old infarction in the right parietal lobe (Fig 1, E). ASL clearly showed hyperperfusion in the corresponding area to that of the cortical hyperintensity on DW-MRI (Fig 1, F). These DW-MRI and ASL findings were quite similar to those at the first episode. Thus, intravenous fosphenytoin followed by oral carbamazepine was administered. Although EEG failed to reveal ictal discharges, frequent interictal paroxysmal activities were noted on the right centroparietal region (Fig 1, B; P4 and C4), which was identical to the area of cortical hyperintensity on DW-MRI and hyperperfusion on ASL. On day 2′, she completely recovered from left hemiparesis and sensory disturbance, and EEG of day 6′ demonstrated occasional low-amplitude paroxysmal activities in the right centroparietal region (Fig 2, C). With monotherapy of carbamazepine, she was free from epilepsy during the 2 years following her second episode. The cortical hyperintensity on DW-MRI was transient, and subsequent T2 prolonged lesion was not demonstrated on the follow-up images.

Case 2

A 74-year-old female with a past history of infarction in the right parietal lobe developed left hemiparesis and sensory disturbance (day 1). She was alert on admission. She had left hemiparesis and sensory disturbance in the left extremities and dressing apraxia, although convulsive seizures were not apparent. FLAIR revealed old infarctions in the right parietal lobe (Fig 3, A). Perilesional gliosis was also noted. On DW-MRI, a gyriform cortical hyperintense lesion was noted around the old infarction in the right parietal lobe (Fig 3, B). The precentral gyrus was not involved. Her abnormal neurological findings were gradually improved. Although epilepsy was highly suspected, antiepileptic drug was not administered as EEG failed to reveal paroxysmal discharges (Fig 4, A).