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Case Report

Severe and prolonged ictal paresis in an elderly patient

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ABSTRACT

We report an 84-year-old female who showed a rare manifestation of epilepsy, ictal paresis, a type of simple partial seizure presenting with focal motor dysfunction. While the patient exhibited severe left hemiplegia which lasted for a week, cranial diffusion-weighted MRI demonstrated slightly high intensity in the right posterior quadrant, and electroencephalography (EEG) showed continuous epileptiform discharges located mainly in the right parieto-occipital area, strongly suggesting that the patient was in an ictal state. ^{99m}Tc-hexamethylpropylene amine oxime-single photon emission computed tomography (HMPAO-SPECT) showed markedly high blood perfusion in the right parieto-temporo-occipital areas. Considering the distribution of EEG epileptiform activities and HMPAO-SPECT hyperperfusion, it is most likely that the ictal paresis of our patient was associated with epileptic activities at the sensorimotor area which caused either direct or indirect activation of an inhibitory system. Careful clinical consideration of the possibility of ictal paresis is needed in elderly patients, especially in those with preexisting dementia, because paresis can be as severe as complete flaccid hemiplegia and can last as long as for a week.

1. Introduction

Ictal paresis or inhibitory motor seizures are a relatively rare nonconvulsive manifestation of epileptic seizures characterized by an inability to initiate or continue movements usually with preservation of muscle strength and consciousness [1–9]. Here, we report an elderly female patient with dementia who manifested with severe and prolonged left hemiplegia.

2. Case report

An 84-year-old female had convulsive seizures for the first time in her life out of sleep (Day 1). On arrival at our emergency room, she had left clonic hemiconvulsions including the neck in association with conjugate deviation of the eyes to the left. The convulsion, sustained for more than 45 min as witnessed by her family, stopped following injection of diazepam and phenytoin (PHT). Prior to the convulsion, she did not speak voluntarily because of dementia and was bedridden most of the time, although she had no hemiparesis. Laboratory examinations were unremarkable, only showing slight leukocytosis and slight elevation of lactate dehydrogenase and ammonia. Cerebrospinal fluid examination was also unremarkable.

At 10 h after the admission (Day 2), her consciousness recovered to the previous level, and she could keep her eyes open visually pursue objects around around her bed. However, she had left flaccid hemiplegia, bilateral symmetrical brisk deep tendon reflexes, and Babinski signs. Cranial diffusion-weighted MRI demonstrated slightly high intensity in the right posterior quadrant (Fig. 1), which did not indicate cerebral infarction in terms of signal intensity and distribution. Electroencephalography recorded while she had left hemiplegia demonstrated continuous epileptiform discharges located mainly in the right parieto-occipital areas and occasionally evolving to the right central area, indicative of focal non-convulsive status epilepticus (Fig. 2). ^{99m}Tc-hexamethylpropylene amine oxime-single photon emission computed tomography showed markedly high blood perfusion in the right parieto-temporo-occipital areas (Fig. 1). On the basis of the diagnosis of nonconvulsive status epilepticus manifesting with ictal paresis, intravenous PHT and oral sodium valproate were administered. Her left hemiplegia improved gradually, although seven days were needed to obtain full voluntary movements.

3. Discussion

In the present elderly patient with dementia, the ictal paresis consisted of severe complete flaccid hemiplegia and lasted for a week,

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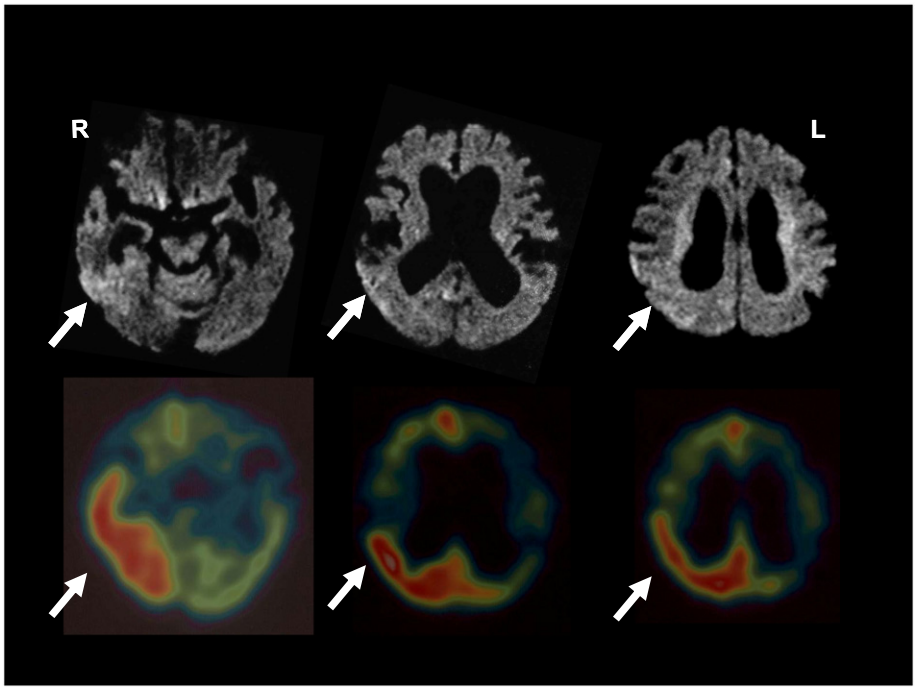

Fig. 1. Axial views of diffusion-weighted MRI and ^{99m}Tc-hexamethylpropylene amine oxime-single photon emission computed tomography (HMPAO-SPECT) almost at the same level while the patient exhibited left hemiplegia. The cranial MRI shows slightly high intensity in the right posterior quadrant (arrows). HMPAO-SPECT demonstrates marked high blood flow in the right parieto-temporo-occipital area delineating the symptomatogenic zone.

whereas, in previous reports, paresis is usually described as mild [1–9]. Murahara et al. reported an elderly male whose ictal paresis, though in mild degree, prolonged for a week similar to that of the present patient [8]. Patients with dementia are known to be at increased risk of developing seizures and epilepsy, which may also be in association with seizure severity [10]. Therefore, age and preexisting dementia, as in the present patient, can be possible factors associated manifestation of severe ictal paresis.

There are two postulated pathophysiologies of ictal paresis: epileptic activities in negative motor areas which are located anterior to the face region of the primary or supplementary sensorimotor areas may affect voluntary movements [9], and epileptic activities in the sensorimotor

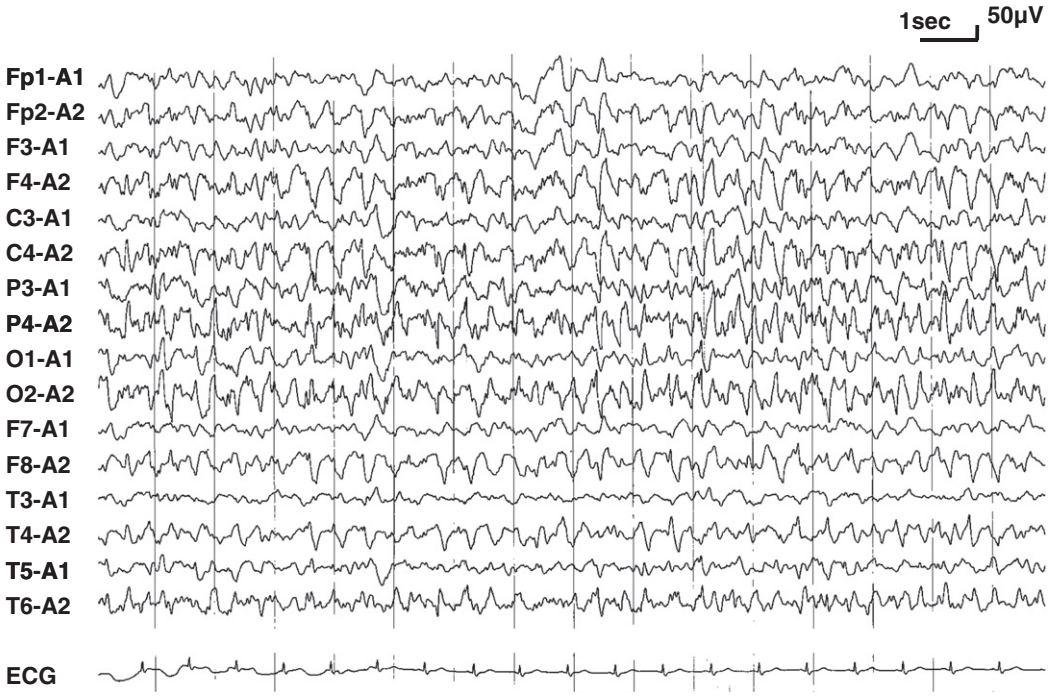

Fig. 2. Electroencephalography recorded at Day 2 while the patient exhibited left hemiplegia without convulsion. Note continuous epileptiform discharges mainly in the right parieto-occipital area.

area may activate an inhibitory system [1,11]. Considering the distribution of EEG and SPECT findings, it is most likely that the ictal paresis of our patient was caused by the latter mechanism.

In summary, ictal paresis can be as severe as complete flaccid hemiplegia and can last as long as for a week, especially in elderly patients with preexisting dementia. Careful clinical consideration of the possibility of ictal paresis is needed, as this is potentially a treatable condition.

Conflict of interest

The authors declare that they have no conflict of interest.

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References

[1] Matsumoto R, Ikeda A, Hitomi T, Aoki T, Hanakawa T, Miki Y, et al. Ictal monoparesis associated with lesions in the primary somatosensory area. Neurology 2005;65:1476–8.
[2] Matsumoto R, Ikeda A, Ohara S, Kunieda T, Kimura K, Takahashi JB, et al. Nonconvulsive focal inhibitory seizure: subdural recording from motor cortex. Neurology 2000;55:429–31.
[3] Noachtar S, Lüders HO. Focal akinetic seizures as documented by electroencephalography and video recordings. Neurology 1999;53:427–9.
[4] Bussière M, Pelz D, Reid RH, Young GB. Prolonged deficits after focal inhibitory seizures. Neurocrit Care 2005;2:29–37.
[5] Villani F, D'Amico D, Pincherle A, Tullo V, Chiapparini L, Bussone G. Prolonged focal negative motor seizures: a video-EEG study. Epilepsia 2006;47:1949–52.
[6] Calarese T, Ferlazzo E, Daquin G, Genton P, Di Bella P, Villeneuve N. Ictal paresis associated to PLEDS in two children: a video-EEG study. Seizure 2008;17:735–9.
[7] Tseng YL, Chen YF, Lai SL. Ictal paralysis with tonic eye gazing mimicking a pontine infarction. Seizure 2006;15:637–42.
[8] Murahara T, Kinoshita M, Usami K, Matsui M, Yamashita K, Takahashi R, et al. Prolonged ictal monoparesis with parietal periodic lateralised epileptiform discharges (PLEDs). Epileptic Disord 2013;15:197–202.
[9] Ikeda A, Hirasawa K, Kinoshita M, Hitomi T, Matsumoto R, Mitsueda T, et al. Negative motor seizure arising from the negative motor area: is it ictal apraxia? Epilepsia 2009;50:2072–84.
[10] Hesdorffer DC, Hauser WA, Annegers JF, Kokmen E, Rocca WA. Dementia and adult-onset unprovoked seizures. Neurology 1996;46:727–30.
[11] Ikeda A, Ohara S, Matsumoto R, Kunieda T, Nagamine T, Miyamoto S, et al. Role of primary sensorimotor cortices in generating inhibitory motor response in humans. Brain 2000;123:1710–21.