Three Cases of Aphasic Status Epilepticus: Clinical and Electrographic Characteristics

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ABSTRACT: Aphasic status epilepticus (ASE) is unusual and has clinical characteristics similar to those of other disorders. Herein, we report 3 cases of ASE. A left-handed man (patient 1) showed continuous aphasia after the administration of flumazenil. He had underlying alcoholic liver cirrhosis and traumatic brain lesions in the right hemisphere. Electroencephalography (EEG) revealed periodic epileptiform discharges in the right frontotemporal area, which were intervened by rhythmic activity with spatiotemporal evolutions. A right-handed woman (patient 2) showed recurrent aphasia. Blood tests revealed a high blood glucose level (546 mg/dL) and high serum osmolality (309 mMol/L). Her EEG showed rhythmic activity in the left frontotemporal area with spatiotemporal evolutions on a normal background rhythm. She became seizure-free after the administration of an antiepileptic drug and strict glucose regulation. A right-handed woman (patient 3) developed subacute aphasia a week before hospital admission. She had a gradual decline of cognition 1 year before. Her EEG showed intermittent quasi-rhythmic fast activity in the frontotemporal area bilaterally, with fluctuating frequency and amplitude. The patient became seizure-free after the administration of an antiepileptic drug. Brain single-photon emission tomography performed after seizure control showed decreased perfusion in the left frontotemporal area. After discharge, her cognitive function gradually declined to a severe state of dementia. ASE can be caused by diverse etiologies; it is usually caused by cerebral lesions and less frequently by non-lesional etiologies or degenerative disorders. Adequate treatment of underlying disorders and seizures is critical for curing the symptoms of ASE.

KEYWORDS: Status epilepticus, aphasia, magnetic resonance image, single photon emission tomography, electroencephalography

Introduction

Aphasic status epilepticus (ASE) is a rare form of non-convulsive status epilepticus (NCSE). Aphasia is the sole manifestation of seizure in patients with this disorder; therefore, other symptoms of NCSE, such as consciousness alterations, behavioral symptoms, and psychiatric symptoms, are absent.[1,2] ASE can arise from various neurological or metabolic disorders.[3-6] It should be differentiated from prolonged postictal aphasia or organic brain disorders, such as stroke. ASE is usually treatable with antiepileptic drugs, but diagnostic delays may cause irreversible neuronal damage.[7,8] Herein, we describe 3 patients with ASE and discuss their clinical and electrographic characteristics.

Case Report

Patient 1

A 71-year-old left-handed man was brought to the emergency department with the complaints of drowsiness and confusion. He had underlying alcoholic liver cirrhosis and traumatic cerebral hematoma and subarachnoid hemorrhage in the right frontotemporal area. He had taken 15 mg of flurazepam for 2 days to relieve insomnia. He could speak when he presented to the emergency department, but he was drowsy, confused, and disoriented. Deducing benzodiazepine toxicity, 0.5 mg of flumazenil was administered to reverse the symptoms. He subsequently became alert but could not speak properly; he could only say "eung" ("yes" in Korean) and "ireum" ("name" in Korean). He could not obey any command given by medical personnel. The serum level of ammonia, creatinine, and total bilirubin were 98 μg/dL (normal value: less than 86 μg/dL), 0.7 mg/dL (normal value: 0.6-1.2 mg/dL), and 0.6 mg/dL (normal value: 0.2-1.2 mg/dL), respectively. Clinically, asterixis, myoclonus, and jaundice were absent. Despite treatment with a lactulose enema, the symptoms persisted.

After consulting the Department of Neurology, electroencephalography (EEG) and brain magnetic resonance imaging (MRI), including diffusion-weighted imaging (DWI), were performed. On EEG, continuous seizure activity was observed in the right frontotemporal area, including continuous periodic sharp waves intervened by rhythmic activity with spatiotemporal evolutions (Figure 1a and b), which met the Salzburg Consensus Criteria for NCSE, including periodic epileptiform discharges (PEDs) with rhythmic activity with spatiotemporal evolutions.[9] The patient showed continuous aphasia with confusion during the recording period (30 min). Brain MRI, including DWI, was performed 18 h after visiting Emergency Department, which revealed high signal intensities with diffusion restriction in the right insular cortex (Figure 1d and f). An apparent diffusion coefficient (ADC) map showed low signal intensities in the area of diffusion restriction (Figure 1e). Subsequently, he was treated with
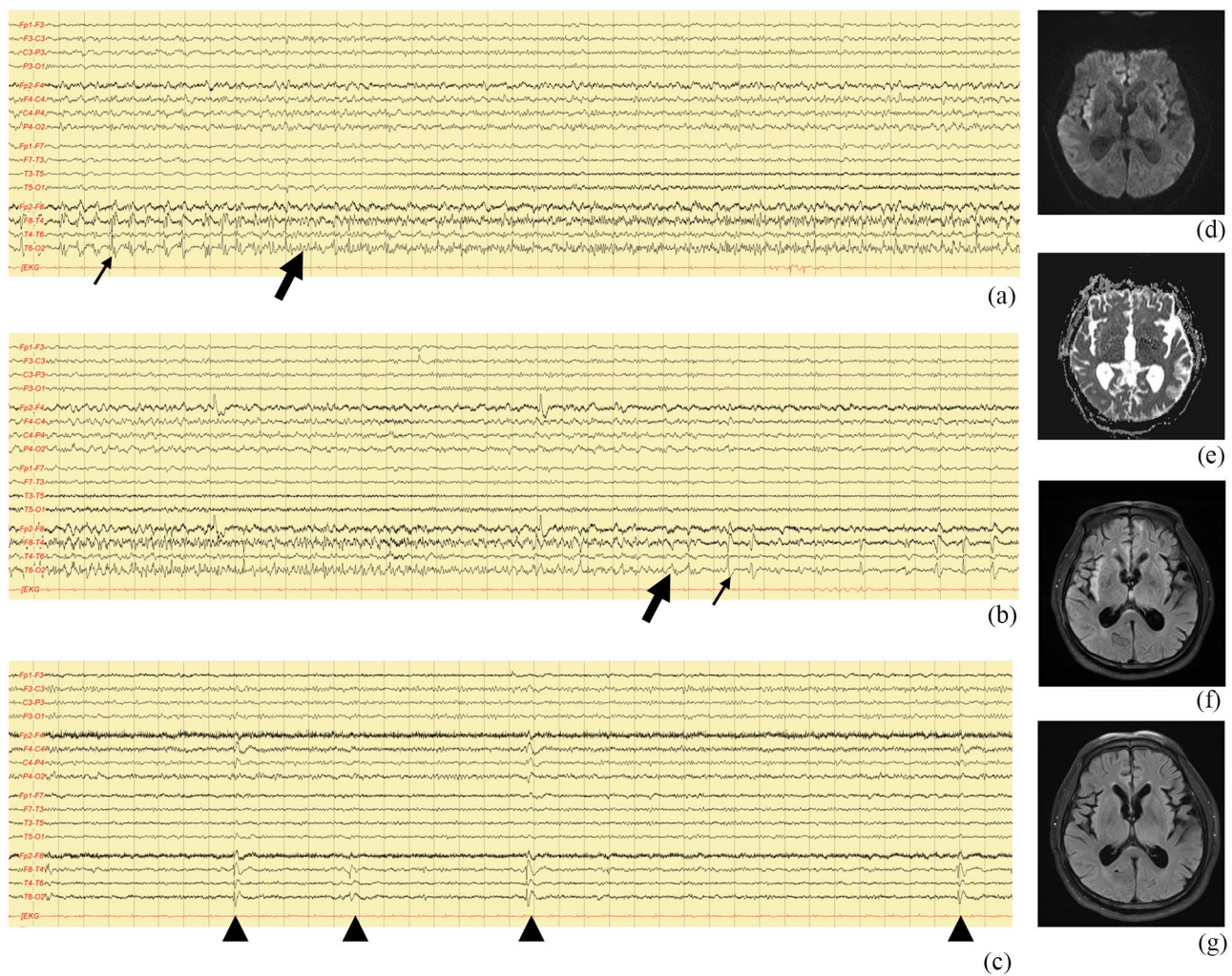
Figure 1. The bipolar montages (double banana) of electroencephalography (EEG) and magnetic resonance imaging (MRI) of patient 1: (a), (b) periodic epileptiform discharges (thin arrows) are observed in the right frontotemporal area, followed by paroxysmal fast activity (thick arrows), evolving to medium- to high-voltage rhythmic theta and delta activity. The sensitivity of the EEG is 70 μV/cm and the display speed is 15 mm/s. The high frequency filter is set to 70 Hz and the low frequency filter to 1.0 Hz, (c) after 2 weeks of treatment, the EEG became normal, except for inter-ictal spikes in the right temporal area (arrowheads). The axial view of (d) diffusion-weighted imaging, (e) apparent diffusion coefficient map, and (f) fluid-attenuated inversion recovery (FLAIR) shows high signal intensities in the right insular cortex, and (g) Follow-up axial FLAIR imaging shows no abnormality.

controlled-release carbamazepine (loading dose of 600 mg, followed by maintenance doses of 200 mg twice a day) and levetiracetam (1500 mg twice a day). He recovered gradually and become nearly free of aphasia after 2 weeks of treatment. The seizure activity normalized, except for inter-ictal spikes in the right temporal area (Figure 1c, arrowheads). The follow-up brain MRI, which was performed 2 months after the initial evaluation, was also normal (Figure 1g). The patient remained symptom-free for 3 years.

Patient 2

A 56-year-old right-handed woman was brought to the emergency department with the complaints of recurrent speech arrest of 2 to 3 minutes' duration. After such incidents, she could remember the events and told her daughter that she wanted to speak but could not make any sound. During those events, she could understand her daughter's speech. While

being examined at the emergency department, she developed motor aphasia of 2 to 10 minutes' duration more than 10 times. Between those events, she was alert and remembered the speech of the medical personnel.

Routine blood tests revealed a blood glucose level of 546 mg/dL (normal value: 70-110 mg/dL) and a serum osmolality of 309 mMol/L (normal value: 285-295 mMol/L). The levels of serum electrolytes, ionized calcium, blood urea nitrogen, and creatinine were within normal limits. Ketone bodies were not detected in her serum or urine. Her hemoglobin A1c level was 9.5% (normal value: 4.0%-5.8%). However, she had not been previously diagnosed with diabetes mellitus. On brain MRI, including DWI, no abnormal findings were observed. Recurrent ictal activity was observed in the left frontotemporal area on EEG (Figure 2a and b); rhythmic delta activity with spatiotemporal evolutions that gradually ceased was observed. The EEG findings met the Salzburg Criteria for NCSE.[9] Background rhythm showed normal activity without any
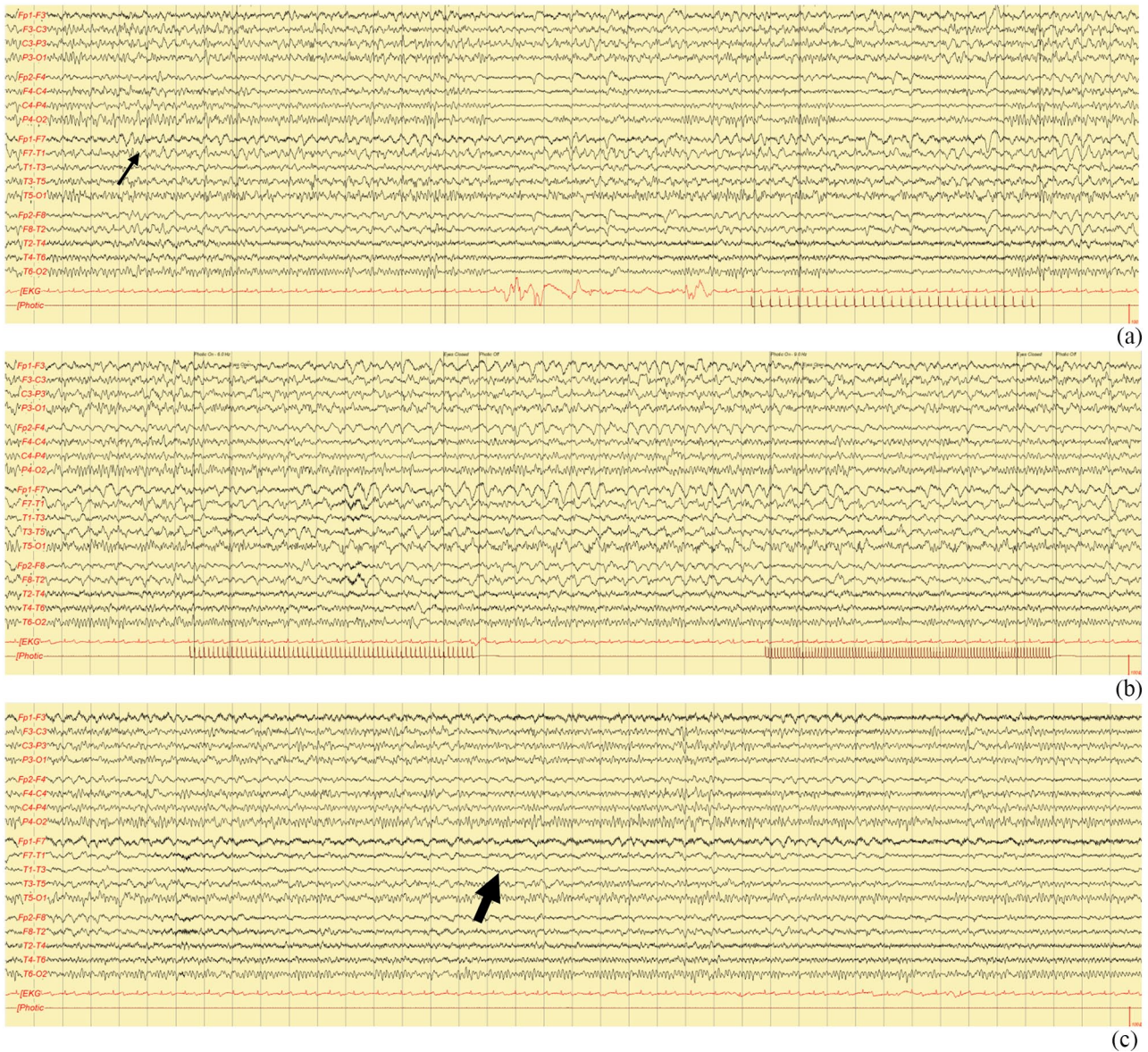
Figure 2. The bipolar montages (double banana) of electroencephalography (EEG) of patient 2: (a), (b) intermittent rhythmic delta activity is observed in the left temporal area (thin arrow), evolving to high amplitude rhythmic delta activity and spreading to the right frontal area, and (c) the amplitude of seizure activity gradually decreases and disappears in the left temporal area (thick arrow). Background EEG shows no abnormality. The sensitivity of the EEG is 70 μV/cm and the display speed is 15 mm/s. The high frequency filter is set to 70 Hz and the low frequency filter to 1.0 Hz.

inter-ictal epileptiform discharges. Rhythmic activity was observed 7 times during the recording period (30 min). The patient was continuously aphasic during this period.

Despite the administration of fosphenytoin (loading dose of 20 mg phenytoin sodium equivalent per kg and maintenance doses of 5 mg phenytoin sodium equivalent per kg), the symptoms persisted. After strict glucose control with insulin, her symptoms and EEG abnormalities disappeared in a day (Figure 2c). Fosphenytoin was discontinued from day 7 of administration. The seizures did not recur during the 6-month follow-up period.

Patient 3

An 82-year-old right-handed woman developed speech disturbance a week before visiting the Department of Neurology.

During the neurological examination, she was alert and could obey one-step verbal commands. However, she could not speak spontaneously. Physical examination revealed no abnormalities. Her medical history, as reported by her husband, revealed that she had experienced a gradual decline of memory and cognition over the past year, but she could speak with other people and perform normal activities without any difficulty until a week previously.

Routine laboratory tests revealed no abnormalities. On EEG, intermittent rhythmic delta activity superimposed by quasi-rhythmic fast activity was observed in the frontotemporal area bilaterally, with fluctuating frequency and amplitude (Figure 3a), which was compatible with possible NCSE.[9] The patient was continuously aphasic during the recording period (30 min). On brain MRI, diffuse atrophy of
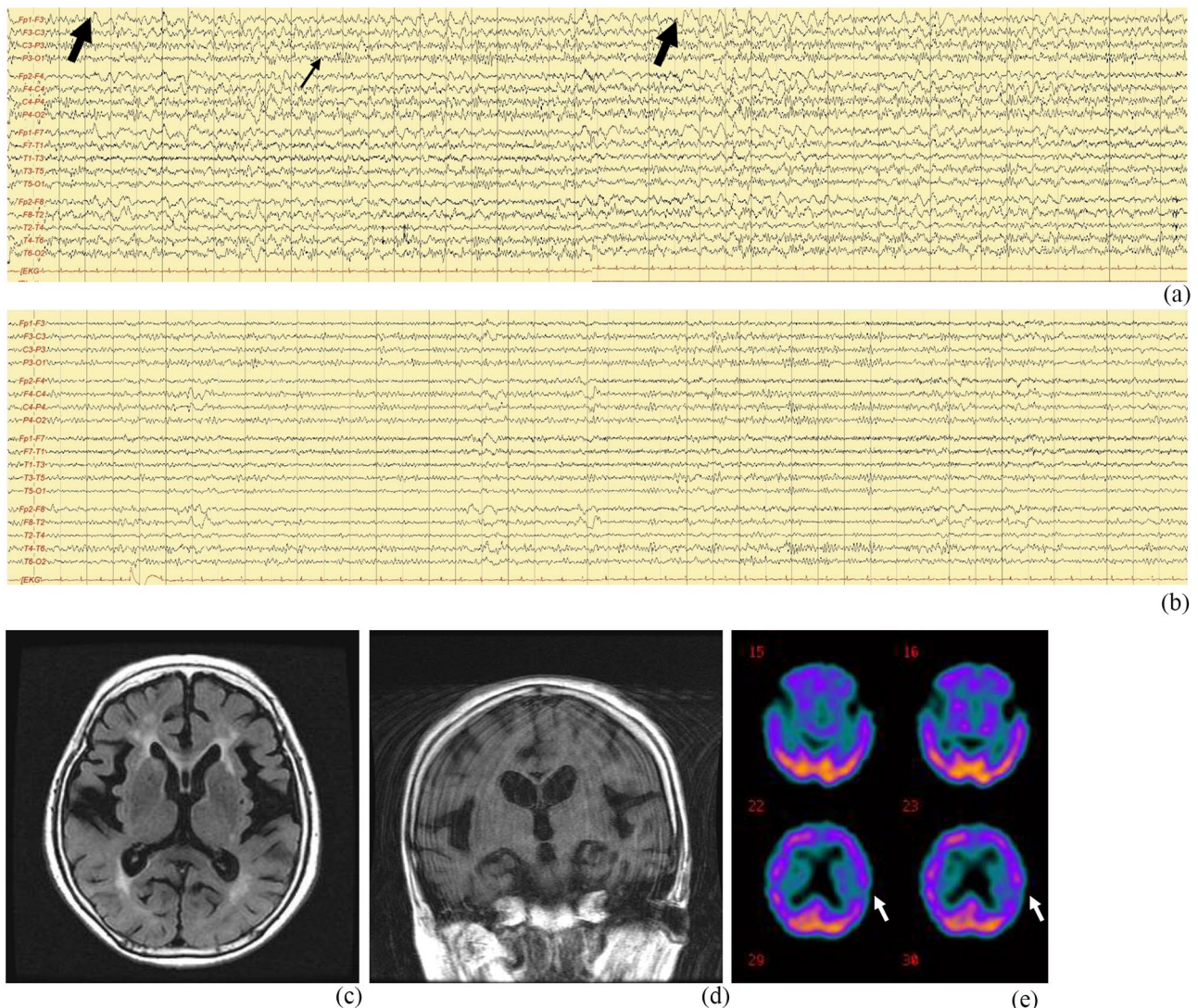
Figure 3. The bipolar montages (double banana) of electroencephalography (EEG), brain magnetic resonance image (MRI), and single-photon emission tomography (SPECT) of patient 3: (a) intermittent rhythmic delta activity superimposed by quasi-rhythmic fast activity is observed in the frontotemporal area bilaterally, with fluctuating frequency, and amplitude (thick and thin arrows), (b) the rhythmic activity disappears after the administration of an antiepileptic drug. The sensitivity of the EEG is 70 μV/cm and the display speed is 15 mm/s. The high frequency filter is set to 70 Hz and the low frequency filter to 1.0 Hz, (c) the axial fluid-attenuated inversion recovery and (d) the coronal T1 MRI shows the diffuse atrophy of bilateral cortices and hippocampi, and (e) the SPECT image obtained after seizure control shows decreased perfusion in the left frontotemporal area (white arrows).

the cerebral cortex was observed (Figure 3c and d). The symptoms and EEG abnormalities disappeared in a day after the administration of valproate (loading dose of 25 mg per kg and maintenance doses of 6 mg per kg) (Figure 2b). She could remember nearly all events during the seizure periods. Brain single-photon emission tomography performed after the control of ASE showed decreased perfusion in the left frontotemporal area (Figure 3e). Her score on the Korean version of the Mini-Mental Status Examination (K-MMSE) was 19 (0/3 in memory registration, 1/5 in attention and calculation, 0/1 in repetition, 4/5 in time orientation, and 3/5 in place orientation), and her Global Deterioration Scale (GDS) score was 4. A detailed neuropsychological test (Seoul Neuropsychological Screening Battery) showed global deterioration, particularly in the domains of memory registration,

language, and frontal executive function, which was compatible with Alzheimer's disease. The patient was seizure-free for 5 years after discharge, but her cognitive function gradually declined to a severe state of dementia. Follow-up K-MMSE and GDS scores were 3 and 6, respectively.

The clinical features of our patients are summarized in Table 1.

Ethics

No ethical approval was required due to the retrospective nature of the study. Further, consent to participate was not required, as this is a case report. Written informed consent was obtained from the patients to publish the findings of their cases without revealing their identities.

Table 1. The clinical features of patients.

PATIENT	PRESENTING SYMPTOMS	NEUROIMAGING FINDINGS	EEG FINDINGS	TREATMENTS	OUTCOMES
Patient 1	Global aphasia	Old traumatic cerebromalacia, HSI in the insular cotex on DWI and FLAIR image.	PEDs intervened by RA with STE in the right FT area.	CBZ-CR, LEV	Gradual improvement of aphasia after 2 weeks of treatment. Nearly free of aphasia
Patient 2	Motor aphasia	No specific finding	RA with STE in the left FT area on the normal background EEG.	Fos-PHT, Strict glucose control	Free of aphasia in a day
Patient 3	Motor aphasia	Diffuse cortical atrophy, Perfusion defect in the left FT area on inter-ictal brain SPECT.	Intermittent quasi-rhythmic FA in the FT area bilaterally.	VPA	Free of aphasia in a day

Abbreviations: CBZ-CR, control-release carbamazepine; DWI, diffusion-weighted image; EEG, electroencephalography; FA, fast activity; FLAIR, fluid attenuated inversion recovery; Fos-PHT, fosphenytoin; FT, frontotemporal; HSI, high signal intensity; LEV, levetiracetam; PEDs, periodic epileptiform discharges; RA, rhythmic activity; STE, spatiotemporal evolusion; SPECT, single photon emission computed tomography; VPA, valproic acid.

Discussion

Aphasia is a common neurological manifestation, but it rarely presents as seizure or status epilepticus. Furthermore, ASE is rarely reported. De Pasquet et al.[2] first reported a case of ASE in 1976. Thereafter, some cases have been reported in the literature. The types of aphasia are variable: Broca's, Wernicke's, or global aphasia are all possible types of ASE.[10-13] Lesional cases constitute most instances of ASE, but non-lesional cases, though rare, have also been reported. In non-lesional cases, metabolic disturbances, such as nonketotic hyperglycemia (NKH) and aggravated uremia, or drugs, such as cefepime, are reportedly the cause of ASE.[4-6,13] There are some reports of cases with combined factors, that is, epileptogenic lesions along with metabolic disturbances.[10,13–15] Metabolic disturbances may act as acute triggering factors for status epilepticus, lowering the seizure threshold in the epileptogenic area. Most patients respond well to antiepileptic drugs, but some are intractable to antiepileptic drugs and need other treatment, such as resective surgery.[16,17]

In patient 1, ASE was precipitated by a seizure threshold-lowering drug (flumazenil) in the presence of an epileptogenic lesion and hepatic comorbidity. In patient 2, ASE was caused by a metabolic disturbance, specifically NKH. In patient 3, ASE was manifested during the progression of degenerative disorder, probably Alzheimer's disease. Acute triggering factors were present in patients 1 and 2, but not in patient 3. Degenerative processes may have contributed to the generation of ASE in patient 3.

A previous study reported that flumazenil is beneficial for in a subset of patients with liver cirrhosis because the reversing effect of the benzodiazepine may improve the neurological score.[18] However, flumazenil is associated with a risk of lowering the seizure threshold and must be used cautiously in patients at risk of seizures. The chronic cerebral lesions of our patient may have been involved in the generation of seizures.

Diagnosing patients with NCSE is challenging, especially when they have comorbid liver cirrhosis or other serious hepatic diseases.[19] It is difficult to differentiate NCSE from hepatic coma using clinical and laboratory findings only. EEG can be useful in elucidating the cause of the altered mental state in these patients.

The EEG patterns in the 3 patients were diverse. The EEG of patient 1 had focal PEDs in the background that were sometimes intervened by focal rhythmic ictal discharges with spatiotemporal evolutions. The EEG of patient 2 showed rhythmic ictal discharges with spatiotemporal evolutions on a normal background rhythm. The EEG of patient 3 showed a fluctuating delta activity superimposed by fast rhythm without definite spatiotemporal evolutions. Patients 2 and 3 responded rapidly to antiepileptic drugs, but patient 1 responded slowly.

In a previous study, PEDs were present in 5 out of 9 cases of lesional ASE, which presented as ictal or inter-ictal discharges.[17] PEDs can be observed in patients with various cerebral lesions without clinical or electrographic seizures.[20-22] In patient 1, PEDs may have reflected lesions in the right insula that were related with continuous seizure activity or with an old lesion caused by traumatic brain injury. The symptoms gradually regressed with the disappearance of PEDs.

Metabolic disturbance can cause ASE without epileptogenic lesions. NKH can induce an increase in gamma-aminobutyric acid (GABA) metabolism, which causes seizures by lowering the level of GABA in cerebral neuronal tissues.[23,24] Ketoacidosis, which activates glutamate decarboxylase and increases the level of GABA, is usually not observed in patients with NKH.[23] Focal motor status epilepticus is the most frequent type of status epilepticus, but other types of status epilepticus can occur in this disorder. Other metabolic disturbances, such as uremia, may cause ASE.[25] The reason for focality in non-lesional status epilepticus is still unknown. A previous study reported that a majority of patients with NKH-associated focal motor status epilepticus showed evidence of localized structural cerebral lesions.[26] However, other studies failed to demonstrate structural lesions in such patients.[27-30] It is postulated that small chronic or acute

lesions could have been missed on computed tomography or routine MRI. EEG findings in patients with ASE caused by metabolic disturbances usually include spatiotemporal evolution patterns on the normal or minimally abnormal background rhythm; PEDs or other inter-ictal epileptiform discharges are usually absent.[6,25]

In patient 3, EEG findings were borderline, that is, possible NCSE according to the Salzburg Criteria for NCSE. However, the clinical symptoms and electrographic abnormalities responded well to treatment with antiepileptic drugs, which means that they were compatible with NCSE.

Degenerative disorders, such as Alzheimer's disease, may cause aphasic seizure.[31] The causes of seizures in Alzheimer's disease are diverse, including extrasynaptic glutamate spillover; tau-induced enhancement of presynaptic glutamate release; reduced axonal and dendritic transport of mitochondria, which regulate neuronal excitability; selective impairment of GABAergic interneurons in the hippocampus; altered expression of postsynaptic AMPA and NMDA receptors; altered amounts of voltage-gated ion channels in neurons; alterations in NMDA activity; shortened dendrites, which lowers the threshold for action potential generation; impaired cortical input to the reticular thalamic nucleus, which subsequently disinhibits thalamic relay nuclei; and increases in cholinergic tone before the degeneration of cholinergic pathways.[32] Subclinical epileptiform activity is often detected in patients with Alzheimer's disease, in whom the progression of cognitive decline is known to be faster.[32] In patients showing cognitive fluctuation or rapidly progressing cognitive decline, epileptiform activity and silent seizures should be investigated by EEG.[33] Seizures may occur in the early stages of Alzheimer's disease.[34,35]

Intravenous lorazepam and intramuscular midazolam are first-line treatments for patients with status epilepticus.[36] We treated our patients with other antiepileptic drugs because our patients were old and had other comorbidities that may have facilitated side effects of benzodiazepines, and the sedation induced by benzodiazepine could have made clinical follow-up of recovery more difficult.

Diffusion restriction is usually observed in patients with stroke and can be observed in patients with status epilepticus. The pattern of diffusion restriction of status epilepticus is different from that of stroke; the area of diffusion restriction may not be compatible with the territory of vascular supply, and an increase in ADC value can be observed in the area of diffusion restriction.[37] The above findings are helpful in differentiating between stroke and status epilepticus. The MRI changes are secondary to the ASE because the MRI lesions were reversible and not compatible with structural diseases.

ASE is usually a benign disorder, as it can be reversed by antiepileptic drugs. However, prolonged seizure activity may cause neuronal damage.[7,8,17] The prolonged duration of seizure is associated with poor outcomes[8] and MRI alterations,[38] which reflect neuronal damage.

ASE can be caused by diverse etiologies. It is usually caused by cerebral lesions and less frequently by non-lesional etiologies or degenerative disorders, such as Alzheimer's disease. EEG patterns are diverse, including PEDs with rhythmic spatiotemporal evolutions, rhythmic discharges with spatiotemporal evolutions, and fluctuations of quasi-rhythmic discharges. Diagnosis and treatment should be accurate and prompt to prevent irreversible cerebral damage. Adequate treatment of underlying disorders is also critical.

Patient Consent for Publication
Written informed consent was obtained from the patients to publish the findings of their cases without revealing their identities.

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Availability of data and materials
Not applicable.

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