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Myoclonic Status Epilepticus of Unknown Etiology in an Elderly Patient

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Dear Editor,

Myoclonic status epilepticus (MSE) is defined as prolonged continuous or discontinuous clusters of epileptic myoclonus, with or without impairment of consciousness.[1] MSE is a heterogeneous condition that has been described in generalized epilepsy syndromes, neurodegenerative diseases, toxic-metabolic states, and following anoxic brain injury.[2] Acute symptomatic MSE usually occurs after prolonged anoxia or metabolic insults.[1] Except for acute symptomatic seizures, MSE is rarely reported in patients who do not have a history of epilepsy.[1] This case study explores MSE of unknown etiology in an elderly patient who did not have a history of seizures.

A 76-year-old woman presented with sudden clusters of irregular myoclonic twitching without impairment of consciousness, which had developed 1 day prior to seeking treatment. The myoclonic jerks had first appeared in the lower face and spread bilaterally to the shoulders and upper arms. They occurred with a high frequency (more than dozens of times per minute), and each cluster lasted a few seconds. The patient denied any history of seizures or morning jerks. She suffered from ischemic heart disease and had taken medications including nicorandil, aspirin, clopidogrel, and pravastatin several years previously. She did not report any other medical illness or additional medications, and denied any history of withdrawal from alcohol or benzodiazepines.

The patient was alert and oriented during her myoclonic jerks. She had experienced mild memory difficulties during the previous several years, but she reported no impairment in the activities of daily living. Laboratory findings were unremarkable, including the levels of glucose, electrolytes, blood urea nitrogen, creatinine, and the findings of liver function tests, thyroid function tests, and arterial blood gas. Diffusion-weighted imaging revealed no acute abnormality (Fig. 1A). Fluid-attenuated inversion recovery (FLAIR) imaging showed bilateral subcortical ischemic lesions (Fig. 1B). Gradient echo imaging did not reveal any hemorrhagic lesions or microbleeds (Fig. 1C). Electroencephalogram (EEG) displayed abundant bursts of high-voltage generalized polyspikes with a normal background. In addition, generalized asymmetric spikes were observed between or preceding the bursts (Fig. 1E). Myoclonic movements were time-locked with the bursts of generalized polyspikes.

The patient was initially treated intravenously with a loading dose of valproic acid (800 mg), followed by the oral administration of valproic acid (900 mg/day) beginning the next day. The myoclonic seizures almost completely resolved after single intravenous valproic acid infusion. The patient received oral valproic acid for 1 year and remained free of myoclonic seizures after valproic acid was discontinued up to the follow-up examination conducted after 3 years.

This case was an elderly patient who presented with late-onset MSE without any previous history of epilepsy. Except in generalized epilepsy syndromes (e.g., juvenile myoclonic epilepsy and progressive myoclonic epilepsy), MSE is usually associated with anoxic ischemic injury,

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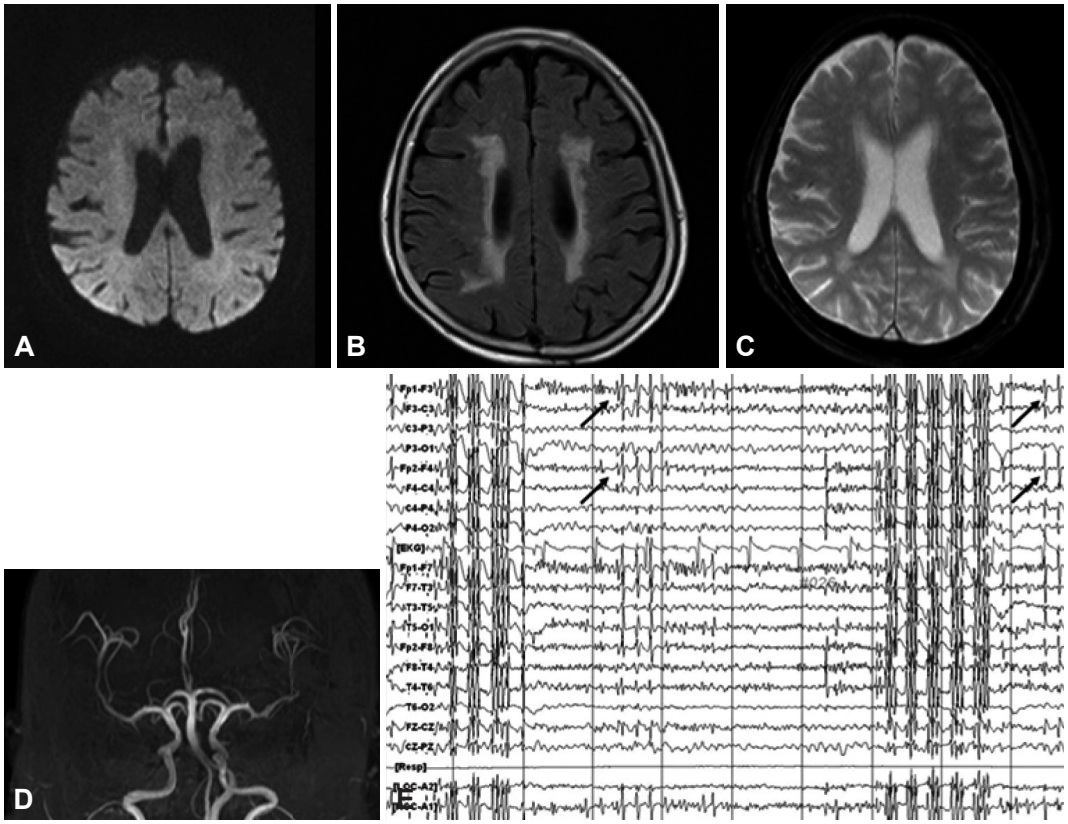

Fig. 1. Diffusion-weighted imaging (A) revealed no acute lesion and FLAIR imaging (B) revealed bilateral ischemic lesions in the subcortical areas. Gradient echo imaging (C) and MRA (D) revealed no specific abnormality. EEG showed abundant bursts of high-voltage generalized polyspikes with a normal background. Some isolated interictal spikes (arrows) were evident between or preceding the bursts (E). EEG: electroencephalogram, FLAIR: fluid-attenuated inversion recovery, MRA: magnetic resonance angiography.

metabolic encephalopathy such as uremic or hepatic encephalopathy, or taking several specific drugs.[1,2] The present patient did not have an identifiable etiology, nor was she taking any drugs associated with MSE. However, the existence of unknown acute symptomatic etiologies cannot be ruled out since a thorough etiological evaluation (e.g., lumbar puncture and other toxicology screening) was not conducted.

Cerebrovascular disease (including small-vessel disease and lacunar infarct) might play a role in the occurrence of late-onset unprovoked seizures. MRI assessment of the present patient revealed subcortical ischemic lesions. Another patient who did not have an identifiable cause of MSE showed multifocal chronic ischemic lesions and subacute ischemic infarct in MRI.[2] The relationship between seizures and small subcortical infarcts remains uncertain, with the associated seizure frequency estimated to range from 0% to 23%.[3] Subcortical lacunar infarcts might be associated with an increased risk of epileptic seizures.[4] Cortical-subcortical diaschisis[4] and global cerebral dysfunction[5] are possible underlying pathophysiological mechanisms.

Conflicts of Interest

The author has no financial conflicts of interest.

REFERENCES

1. Panayiotopoulos CP. *A clinical guide to epileptic syndromes and their treatment.* 2nd ed. London: Springer, 2007;63-64.
2. Baysal Kirac L, Aydogdu I, Acarer A, Alpaydin S, Bayam FE, Onbasi H, et al. Myoclonic status epilepticus in six patients without epilepsy. *Epilepsy Behav Case Rep* 2012;1:10-13.
3. Bladin CF, Alexandrov AV, Bellavance A, Bornstein N, Chambers B, Coté R, et al. Seizures after stroke: a prospective multicenter study. *Arch Neurol* 2000;57:1617-1622.
4. Schreiner A, Pohlmann-Eden B, Schwartz A, Hennerici M. Epileptic seizures in subcortical vascular encephalopathy. *J Neurol Sci* 1995;130:171-177.
5. De Reuck J, Nagy E, Van Maele G. Seizures and epilepsy in patients with lacunar strokes. *J Neurol Sci* 2007;263:75-78.