Epilepsy and Takotsubo Cardiomyopathy: A Case Report

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Abstract

A 60-year-old woman with a history of symptomatic seizures secondary to a subarachnoid hemorrhage was admitted to hospital because of a generalized seizure. The following day, her electrocardiogram showed negative T waves in II, III, V₁, and V₂-V₆, and the echocardiogram showed an impaired left ventricular ejection fraction with ventricular apical akinesia. Head magnetic resonance imaging showed no acute brain injury, but single photon emission computed tomography (SPECT) showed hyperperfusion which affected the left temporal cortex in particular. Hyperactivity of the temporal lobe might cause autonomic nervous system dysfunction and might be related to takotsubo cardiomyopathy.

Key words: epilepsy, SPECT, takotsubo cardiomyopathy, temporal lobe, insular cortex


Introduction

Takotsubo cardiomyopathy is characterized by left ventricular apical ballooning, electrocardiographic (ECG) changes without coronary artery disease, and improvement within weeks in most cases. Takotsubo cardiomyopathy may be caused by catecholamine-induced myocardial stunning and has been associated with intense physical or psychological stresses (1, 2). Acute illnesses, including neurological disorders such as subarachnoid hemorrhage, brain infarction, subdural hematoma, and metastatic brain tumors, have been associated with takotsubo cardiomyopathy (3-7).

Epilepsy has also been reported as a cause of takotsubo cardiomyopathy (8-10). In past case reports, a generalized tonic-clonic seizure was associated with takotsubo cardiomyopathy, and epileptic sharp wave activity in the temporal lobe was occasionally seen in some patients. Findings of single photon emission tomography (SPECT) after epilepsy associated with takotsubo cardiomyopathy have not been previously reported. A patient with takotsubo cardiomyopathy after developing epilepsy who was assessed with SPECT is described.

Case Report

A 60-year-old woman with symptomatic epilepsy secondary to a subarachnoid hemorrhage treated with zonisamide was brought to our emergency room by ambulance because of an epileptic seizure at night. She had a generalized tonic seizure at home and in the ambulance. She was independent in her activities of daily living, and had an epileptic seizure 1 year prior to this attack. She had a history of mitral valve replacement at 38 years of age and had been on warfarin since then. She had a subarachnoid hemorrhage (SAH) and clipping of a ruptured left internal carotid artery-posterior communicating artery aneurysm at the age of 57 years. She did not have hypertension, diabetes mellitus, dyslipidemia, or coronary artery disease.

When she was admitted 24 minutes after onset, her seizure disappeared, but her consciousness was impaired. Her height was 155.5 cm, her weight was 30.1 kg, her temperature was 36.5°C, and her peripheral oxygen saturation was 99% with oxygen flow of 6 L/min via face mask. Her blood pressure (BP) was 97/44 mmHg, though it had been 150/66 mmHg when the rescue team first measured it. Her blood pressure was 97/44 mmHg, though it had been 150/66 mmHg when the rescue team first measured it. Chest X-rays showed cardiomegaly, and arterial blood gas analysis showed a metabolic acidosis (pH 7.099, PaO₂ 197.0 mmHg,

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PaCO₂ 42.2 mmHg, HCO₃⁻ 12.8 mmol/L, B.E. -16.3 mmol/L). After she was brought to our emergency room, she had a generalized tonic seizure with conjugate deviation of the eyes to the right. The seizure was terminated with intravenous diazepam, after which she was given intravenous phenytoin.

On the day following admission (day 2), she became alert and had no neurological deficits. An electrocardiogram on day 2 showed negative T waves in II, III, aVF, and V₂-6 (Fig. 1A). An echocardiogram showed a decreased left ventricular ejection fraction of 39% with ventricular apical akinesia. She never complained of chest pain during the hospital stay. An electroencephalogram on day 2 showed no epileptic spikes. T2-weighted magnetic resonance (MR) imaging on day 2 showed a high intensity area mainly in the left frontal lobe that was caused by the subarachnoid hemorrhage and clipping surgery (Fig. 1B), but diffusion-weighted MR imaging showed no acute brain damage. N-isopropyl-p-¹²³I-iodoamphetamine (IMP) single photon emission computed tomography (SPECT) on day 3 showed hyperperfusion affecting the left temporal cortex in particular (Fig. 1C, D).

The patient had no further epileptic seizures during hospitalization other than the seizure in the emergency room. Her blood pressure gradually improved without intensive care, and the negative T waves on the electrocardiogram also improved gradually (Fig. 1A). On day 13, an echocardiogram showed a normal left ventricular ejection fraction of 60% without ventricular apical akinesia. She was discharged home on day 14.

Discussion

A patient with a generalized epileptic seizure who developed hypotension on admission and was found to have takotsubo cardiomyopathy on echocardiography and extensive hyperperfusion in the temporal lobe on SPECT is described. The incidence of takotsubo cardiomyopathy associated with epilepsy remains unknown, but that associated with neurological disorders was reported to be 1.2% of SAH patients and 1.2% of ischemic stroke patients (4, 5). Although the precise mechanism of takotsubo cardiomyopathy is unknown, it appears to be a toxic effect of excessive endogenous catecholamine (1, 2). The intense physical stress caused by a prolonged generalized epileptic seizure itself might cause takotsubo cardiomyopathy. The present patient had severe metabolic acidosis on admission, indicating that she had had a severe epileptic seizure.

However, not every generalized epileptic seizure causes takotsubo cardiomyopathy (11). This fact might indicate that a mechanism other than the physical stress caused by the prolonged generalized epileptic seizure itself may be in-
volved. In a few patients with epilepsy and takotsubo cardiomyopathy who underwent ECG, an echocardiographic sharp wave was present in the temporal lobe (8-10). Temporal lobe epilepsy is considered to be associated with alteration of autonomic function. In fact, the temporal cortex has been shown to be related to the stellate ganglion, which regulates the heart in a rat model (12). In addition, the insular cortex adjacent to the temporal cortex also plays an important role in autonomic control of cardiac activity, and damage to the insular cortex has been shown to cause heart dysfunction (13-15). Takotsubo cardiomyopathy that was related to brain infarction at or near the insular cortex and limbic encephalitis has been thought to be caused by changes in central autonomic function because of the affected insular cortex (5, 16, 17). In an animal model, neurogenic cardiac damage occurred in mice with adrenalectomy to reduce circulating catecholamines, although this damage was attenuated compared to that of non-adrenalectomized mice (18). In the clinical setting, partial status epilepticus that originated in the temporal lobe, not a generalized seizure, was related to takotsubo cardiomyopathy (19). These results might suggest that not only catecholamine release because of the stress from the generalized epileptic seizure but also stimulation of the central nervous system including the temporal lobe might cause takotsubo cardiomyopathy.

Postmenopausal women have also been reported to be at higher risk for takotsubo cardiomyopathy. The reason is unclear, but estrogen changes are believed to be involved in the mechanism (20). In fact, the present patient was a postmenopausal woman, and most patients with takotsubo cardiomyopathy after epilepsy in previous studies were postmenopausal women (8, 9, 20).

The present patient did not need intensive care, and she gradually recovered. As in this case, most patients with takotsubo cardiomyopathy improve in a few weeks. However, there have been some patients with poor outcomes due to a severe cardiac event, such as cardiac rupture (8). Therefore, careful observation is necessary after a seizure.

This case report had some limitations: first, a coronary angiogram was not performed, and serum creatine kinase levels were not measured. However, this patient had no vascular risk factors, including hypertension, diabetes mellitus, and dyslipidemia. Secondly, plasma catecholamine levels were not measured, the coefficient of variation of R-R intervals was not calculated, and 123I-metaiodobenzylguanidine cardiac scintigraphy to investigate cardiac autonomic function was not performed. Thirdly, SPECT was not performed in the recovery phase. Finally, SPECT showed hyperperfusion in a vast area of the left temporal cortex; therefore, the critical area related to takotsubo cardiomyopathy could not be identified.

The authors state that they have no Conflict of Interest (COI).

References