Case Report
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Thalamic Hyperintensity on Diffusion-Weighted MRI in a Patient with Nonconvulsive Status Epilepticus

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We present a 70-year-old woman with nonconvulsive status epilepticus (NCSE) with thalamic hyperintensity on diffusion-weighted MRI (DWI). She had no previous history of epilepsy. Her altered mentality was not normalized though we successfully controlled the ictal activity by standard treatment. Initial DWI showed diffuse hyperintensity in the right thalamus, which raised the possibility of seizure-related change. At the follow-up DWI, more localized high signal intensity lesion was present in the right pulvinar area. There was no apparent cause of her NCSE despite our extensive work-ups. The authors suggest that transient ischemia is a possible causative pathomechanism in this case. **(2013; 3:32-34)**

Key words: Nonconvulsive status epilepticus, Diffusion-Weighted MRI, Thalamus

Introduction

Nonconvulsive status epilepticus (NCSE) is a condition that may result in serious morbidity and mortality.[1] NCSE has been easily overlooked, because of the absence of distinct behavior in contrast to convulsive status epilepticus. However, it is important to demonstrate the etiology of NCSE and to correct that immediately. At this point, diffusion-weighted MRI (DWI) may be helpful to identify an epileptogenic zone. We recently experienced a case with NCSE with right thalamic hyperintensity on DWI. Thus, we report this case.

Case report

A 70-year-old woman with altered mentality and unresponsiveness arrived at our emergency room. She had a medical history of Alzheimer's disease, hypertension and chronic obstructive pulmonary disease, but no seizure or stroke. Her vital signs were stable. On the initial neurologic examination, she was drowsy and mute. She had left homonymous hemianopsia and right gaze preponderance. She showed symmetric withdrawal response to noxious stimuli. Electrocardiogram revealed atrial fibrillation and the cardiac enzymes were normal. Since eight hours before arrival, she had a cluster of generalized tonic clonic seizures at an interval of about ten minutes without full recovery of consciousness for just prior two hours. The convulsive seizure subsided with intravenous lorazepam injection. However, her drowsiness and unresponsiveness did not return to the baseline normal status. We took the DWI and the EEG to identify the cause of sustained abnormal mentality. The DWI revealed an ill-defined high signal intensity in the right thalamus and subtly in the right temporal area, in which the values of apparent diffusion coefficient (ADC) were reduced (Fig. 1A). In the MR angiography, there was no significant steno-occlusive lesion. These DWI findings suggested a possibility of a clustered seizure-related change or an acute ischemic stroke. EEG recording showed a right temporal ictal onset zone with persisting ictal discharges with rhythmic alpha to theta activity (Fig. 2A).

After the confirmation of ictal EEG, we applied intravenous phenytoin to control the NCSE. Oral topiramate and intravenous levetiracetam were added to suppress remnant ictal discharges. Finally, ictal discharges disappeared (Fig. 2B). Though she was gaining alertness, her mutism sustained. Follow-up DWI at the 5th day clarified a brighter lesion localized to the pulvinar (Fig.1B). Transthoracic and transesophageal echocardiographies revealed a small echogenic nodular mass (7.5 × 3.5 mm sized) attached to the posterior mitral valve leaflet and a severely dilated left atrium. Other studies including CSF examination and serologic markers for autoimmune and paraneoplastic diseases were normal. The cause of status epilepticus remained unknown despite extensive work-ups. We suspected a ringt posterior cerebral artery (PCA) infarction when we took her atrial fibrillation and the infarction territory into account.

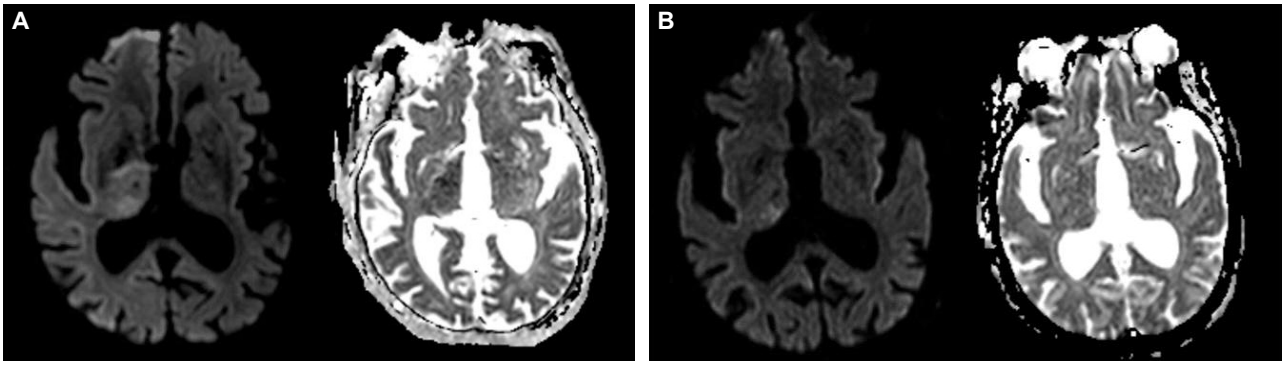

Figure 1. Diffusion-weighted MR images (DWI) and apparent diffusion coefficient (ADC). (A) Initial DWI showed high signal intensity in the entire right thalamus, in which the value of ADC was reduced. (B) High signal intensity was well-localized on the right pulvinar area in the follow-up DWI.

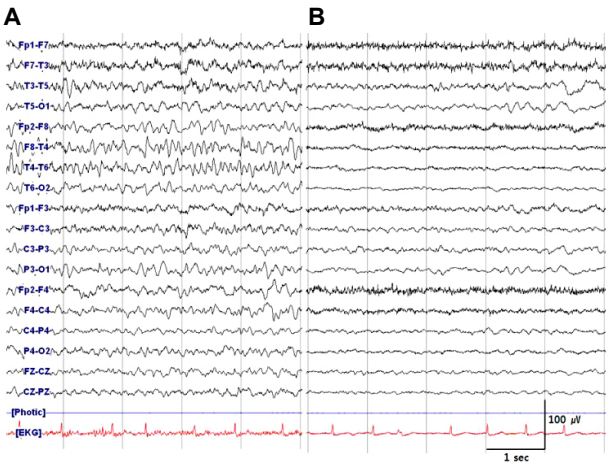

Figure 2. EEG in this patient. (A) In the first EEG, persisting ictal discharges of alpha frequency in the right temporal area were noted. (B) Follow-up EEG showed suppressed rhythms in the right hemisphere after intravenous antiepileptic drugs.

As CHA_2DS_2-VASc score was 3, it was reasonable to start anticoagulation irrespective of the feasible mechanism of the seizure.[2] Altered mentality with global aphasia persisted despite the adequate treatment.

Discussion

We report a case of NCSE associated with a unilateral and diffuse thalamic hyperintensity in the DWI, which was localized to the pulvinar area in the follow-up DWI on day 5. In this patient, the possible mechanism of NCSE might be a transient ischemic attack or a stroke in the right PCA territory because of the cardiac arrhythmia she had. We authors suggest a decreased perfusion in the right PCA territory provoked status epilepticus.

NCSE is defined as a change in behavior and/or mental processes from baseline associated with continuous epileptiform discharges in the EEG.[3] It is unclear which brain structures are engaged in the development of NCSE. One study suggested that thalamic dysfunction might participate in the pathogenesis of status epilepticus.[4] In line with advanced neuroimaging techniques, higher resolution MR scan with DWI is helpful to identify etiologic factor in status epilepticus. Transient focal hyperintensity on DWI with corresponding decrease of the ADC is an increasingly-recognized phenomenon in the peri-ictal phase of epileptic seizures or acute stroke.[5,6] It has been described in experimental models and human subjects with status epilepticus.[5,7] The DWI changes in status epilepticus may be associated with enhanced energy metabolism, hyperperfusion and cell swelling as a consequence of increased ictal activity.[8,9] In a large series of patients with poststroke seizures, 9% had status epilepticus. Changes in cerebral blood flow, hypoxia, involvement of the cerebral cortex by hemorrhages or infarcts, and the development of epileptogenic changes in cortical neurons, their connections, or their environment have been proposed as potential mechanisms underlying seizures in patients with stroke.[5]

We experienced a case of NCSE with a right thalamic DWI hyperintensity, the mechanism of which is presumed to be a transient ischemic attack or a stroke based on the risk factor, involved terrotory, and acceptable causal relationship. Serial EEG and DWI measures are helpful to delineate the time course and to infer the underlying diagnosis.

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