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
Dae Lim Koo, et al. Thalamic Hyperintensity on Diffusion-Weighted MRI in a Patient with Nonconvulsive Status Epilepticus

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 CHA2DS2-VASc score was 3, it was reasonable to start anticoagulation irrespective of the feasible mechanism of the seizure. 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 based on the risk factor, involved territory, and acceptable causal relationship. Serial EEG and DWI measures are helpful to delineate the time course and to infer the underlying diagnosis.

NCSE is defined as a change in behavior and/or mental processes from baseline associated with continuous epileptiform discharges in the EEG. 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. 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. It has been described in experimental models and human subjects with status epilepticus. The DWI changes in status epilepticus may be associated with enhanced energy metabolism, hyperperfusion and cell swelling as a consequence of increased ictal activity. 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.

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 territory, 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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