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Antiepileptic treatment for anti-NMDA receptor encephalitis: the need for video-EEG monitoring

To the Editor,

In a recently published clinical commentary (Dericioglu *et al*., 2013), two patients with anti-NMDAR encephalitis were referred to the neurological ICU with a diagnosis of status epilepticus. Prolonged video-EEG monitoring, however, showed that, for both patients, dyskinesias were not associated with ictal EEG changes. Antiepileptic therapy was not increased and the patients eventually improved with immunotherapy. The authors advised caution in order to avoid mistaking dyskinesias for seizures, and suggested that video-EEG monitoring be used to help differentiate between the two.

We recently reviewed the video-EEGs of all our anti-NMDAR encephalitis patients who had been admitted to our institution between 2009-2012 (ethics approval was obtained). Ten patients were identified; all were female and all had confirmatory evidence of serum anti-NMDAR antibodies (assays performed at Neuroimmunology Laboratory, John Radcliffe Hospital, Oxford, UK). The mean number of EEG recordings was 4.3 (SD±2.1; range: 2-8) per patient. The most common EEG pattern seen in stupor was generalised rhythmic 1.5-2-Hz delta activity; the EEG of three patients showed an extreme delta brush pattern (Schmitt *et al*., 2012). All episodes of oro-lingual-facial or limb dyskinesias, that were captured during EEG recordings in eight patients, were not associated with any changes in the EEG pattern.

Electrographic seizures were recorded in two patients. One patient had a complex partial seizure during her convalescence period on Day 56, manifesting with gaze deviation to the right and head turn to the right, followed by right-sided facial twitching. The corresponding ictal EEG showed paroxysmal fast alpha activity over the left frontal region. During the same period of hospitalisation, she underwent a two-day (Days 18-19) period of video-EEG monitoring in the ICU with no evidence of epileptiform activity during recorded episodes of lip smacking and limb dyskinesias.

A second patient had four electrographic seizures seven days after disease onset. Clinically, she was obtunded; she received treatment for non-convulsive status epilepticus. On Day 14, she developed gaze deviation with limb dyskinesias but did not have EEG changes to suggest seizures. Both patients received antiepileptic treatment guided by video-EEG findings.

In the seminal 100-patient case series of anti-NMDAR encephalitis patients (Dalmau *et al*., 2008), 76 patients were described to have clinical seizures, but documented epileptic activity was only present in 21 patients. In contrast, dyskinesias were described in 86 patients. Dyskinesias may be misinterpreted as seizures, or *vice versa,* and may lead to under- or over-treatment with antiepileptic drugs. Our series confirms that patients with anti-NMDAR encephalitis may have seizures, dyskinesias, or both seizures and dyskinesias during the course of the disease. Multiple short-term or prolonged video-EEG recordings may be necessary to distinguish between seizures and movement disorders, as well as to identify patients with subtle non-convulsive seizures. We therefore strongly agree with the authors that video-EEG monitoring is an important investigation for anti-NMDAR encephalitis patients with suspected status epilepticus. We also suggest that clinicians should consider expanding the use of video-EEG monitoring to patients without suspected status epilepticus whenever it is challenging to distinguish clinically between seizures and dyskinesias. In such cases, accurate diagnosis of seizures or dyskinesias using video-EEG monitoring can help guide clinicians in their therapeutic decision-making, particularly with regards to antiepileptic therapy. □

Disclosures.

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Dr. Kevin Tan has received personal compensation from Novartis and Biogen Idec for consulting services and *honoraria* for speaking from Biogen Idec and Merck Serono.

All three authors have no conflicts of interests to declare.

Yee-Leng Tan, Kevin Tan, Nigel CK Tan

Department of Neurology
National Neuroscience Institute, Singapore
<yee-leng_tan@nni.com.sg>

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