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Letter to the Editor

Awake-induced increase of epileptiform discharges in a case with poststroke epilepsy

In the management of epilepsy, the diagnostic value of the sleep electroencephalogram (EEG) is high. Epileptiform discharges that are not present in awake can be detected by sleep EEG. However, recent studies have demonstrated more frequent epileptiform discharges during awake than in sleep in patients with benign adult familial myoclonus epilepsy (BAFME) and Unverricht-Lundborg disease (ULD) (Hitomi et al., 2018; Ferlazzo et al., 2007). Here we evaluated the changes in epileptiform discharges in relation to awake/sleep stage in conventional EEG of a patient with poststroke epilepsy.

An 83-year-old Japanese woman with poststroke epilepsy, who developed the last episode of epileptic seizure 2 months previously, was investigated. EEG was performed as a part of a clinical evaluation of her condition. Five years ago, she developed her fourth stroke event consisting of motor-dominant aphasia, right hemispatial neglect, and right hemiparesis. Diffusion-weighted magnetic resonance imaging showed high signal intensity lesions scattered in the left middle cerebral artery territory, and magnetic resonance angiography showed a stenosis of the distal portion of the horizontal segment of the artery (Fig. 1A). She was treated with percutaneous transluminal angioplasty and on the next day showed non-convulsive status epilepticus (NCSE). Since then, she had infrequent seizures under treatment with antiepileptic drugs (AEDs). The current case report was exempt from the need for local ethics committee approval and informed consent, on the basis of the anonymous and noninvasive study design.

Conventional EEG was recorded continuously for 20 min in the shield room at 10:00 in the morning after breakfast (Table 1). Sleep record was obtained by spontaneous natural sleep. All the wave forms were reviewed offline, time constant of 0.3 s, and high-cut filter of 50 Hz. Each parameter was determined by the agreement of the two board-certified researchers (M.N. and M.K.). The awake/sleep stage was classified according to the AASM manual version 2.5 (AASM, 2018), reviewing EEG 30 s/screen as one epoch. The frequency and amplitude of epileptiform discharges were analyzed by reviewing EEG 10 s/screen. Because the epileptiform discharges distributed in the parasagittal areas and did not activate the earlobe electrodes, a monopolar derivation referenced to the ipsilateral earlobe electrode was used. An epileptiform activity was defined as a clearly outstanding transient sharp activity with the duration less than 200 ms, followed by a slow wave (Fig. 1B). For the statistical analysis of frequency and amplitude of epileptiform discharges, values obtained from each 30 s epoch were arbitrarily considered as independent variables. Nonparametric group comparison was performed among awake/sleep stages.

The first epoch contained calibration and impedance checks and thus the rest of 39 epochs were analyzed. The frequency of posterior dominant rhythm was 7–8 Hz. Intermittent irregular slow waves, sharp waves, and spikes distributed in the bilateral parieto-occipital regions (Fig. 1C). The percentage of stage W, N1, and N2 was 46%, 51%, and 3%, respectively. Frequent epileptiform discharges were noticed during awake and also during the brief arousal periods (Fig. 1D). The number of epileptiform discharges per 30 s epoch in stage W and N1 was 3.17 ± 2.36 (mean ± SD) and 0.65 ± 0.79, respectively, and the amplitude of epileptiform discharges was 97.5 ± 35.5 μV and 71.9 ± 24.8 μV, respectively. No epileptiform discharge was seen in stage N2. Both frequency and amplitude were significantly higher in stage W than in N1 (Fig. 1E).

To the best of our knowledge, this is the first case report with poststroke focal epilepsy that showed awake-induced increase of epileptiform discharges. EEG findings suggest that epileptic foci and focal abnormalities reside in the bilateral parieto-occipital region. Similar change in epileptiform discharges along with vigilance has been reported in familial generalized myoclonus epilepsy syndromes, i.e., BAFME and ULD, whose epileptic activities are dominant in posterior part of the brain (Hitomi et al., 2018, Ferlazzo et al., 2007). In this case, as well as these syndromes, the neural networks involved in maintenance of vigilance may have been altered and play a role in acquisition of epileptogenicity. Moreover, attention closely relates to alertness. Attentional functions are comprised of alerting: enhancing and maintaining response readiness for anticipated stimuli, orienting: selecting from multiple sensory information, and executive: processing and executing tasks involving contradictions, and the parietal lobe is particularly relevant to orienting (Posner and Petersen, 1990; Raz, 2004). Epileptiform discharges in our patient distributed posterior to cerebral infarcts in the middle cerebral artery territory. Arterial stenosis of the main trunk of the major artery, which causes chronic hypoperfusion not only in the arterial territory but also in the watershed areas, is possibly an additional factor. Further studies are needed to elucidate relationship among vigilance, attention, and epileptogenesis in poststroke patients.

In critically ill patients with unexplained consciousness disturbance, early diagnosis of NCSE is crucial for immediate AED therapy and determination of length of continuous EEG (cEEG) monitoring. In a previous retrospective study including 570 patients who underwent cEEG monitoring, seizures were recorded in 110 (19%) patients, and in 56% of them the first seizure was detected within 1 h of recording (Claassen et al., 2004). A recent prospective study including 50 consecutive patients with clinical suspicion of NCSE showed that recurrent electrographic seizure activity was detected in 5 patients (10%); in 3 of them it was within initial 30 min of cEEG recordings, and in the rest of 2 patients retrospective intensive analysis of cEEG revealed extremely discrete electrographic seizure activity in the initial 30 min of recordings (Krøigård et al., 2019). Additional information

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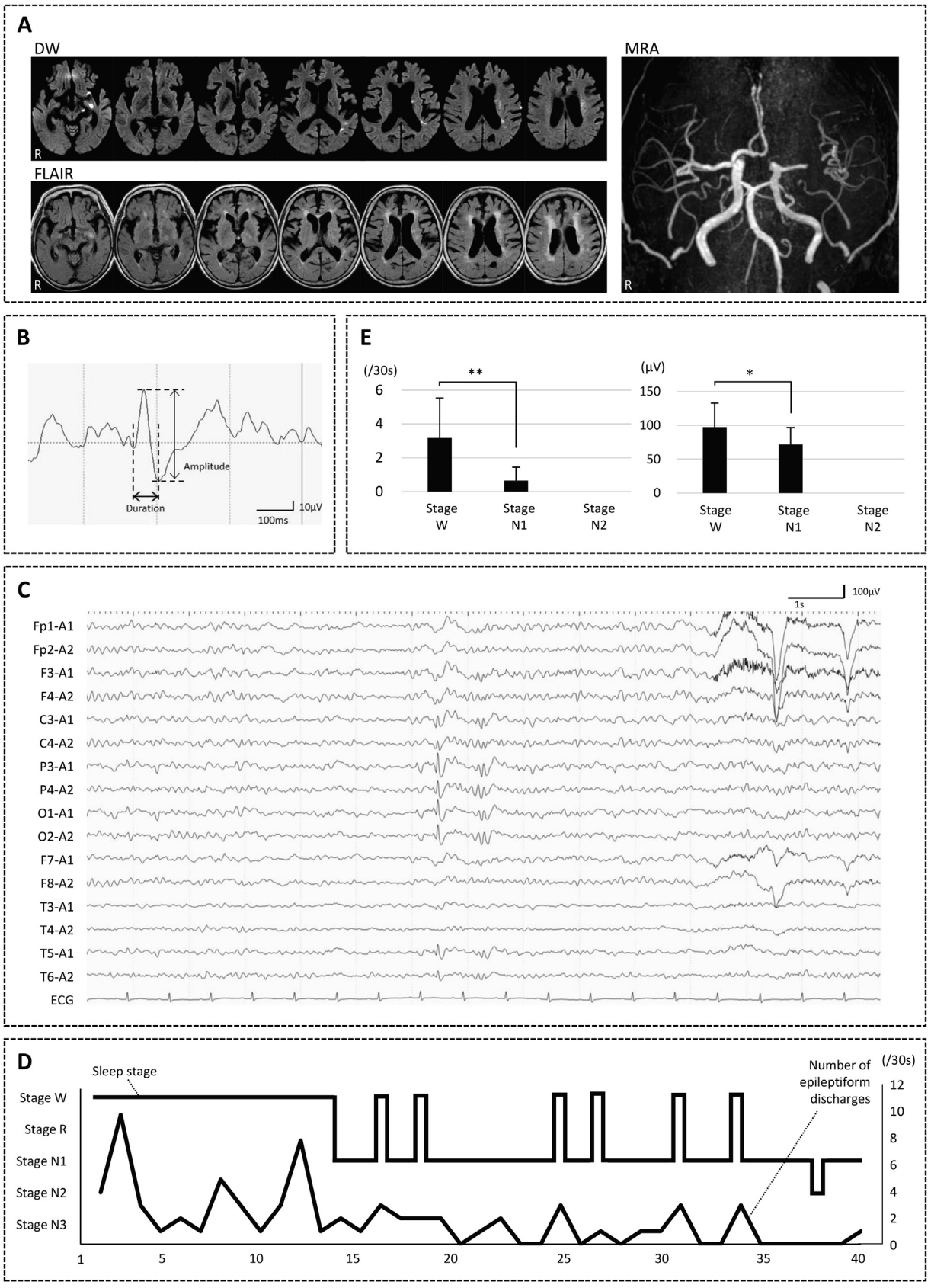

Fig. 1. (A) Magnetic resonance imaging and magnetic resonance angiography. Acute cerebral infarcts are scattered in the left hemisphere. Cessation of flow signal is seen at the horizontal segment of the left middle cerebral artery. (B) An epileptiform activity. A clearly outstanding (i.e., with the amplitude higher than 120% of the maximum amplitude of posterior dominant rhythm), transient sharp activity with negative pointed peak, with the duration less than 200 ms, with steeper ascending negative slope than descending positive slope, with more positive descending trough than the baseline, and followed by a slow wave. Peak-to-peak amplitude was measured. (C) A representative epileptiform discharge during awake. (D) Hypnogram and frequency of epileptiform discharges (ordinate) against epochs (abscissa). (E) A comparison among stages W, N1, and N2 of the frequency (left) and amplitude (right) of epileptiform discharges (mean + SD); none in N2. Mann-Whitney *U* test, $^{**}p = 0.0000164$, $^{*}p = 0.0011347$.

Table 1
Recording condition.

Electrodes	Silver/silver chloride shallow cup electrodes
Sampling rate	500 Hz
Bandpass filter for data acquisition	0.08–120 Hz
Scalp electrodes placement	The International 10–20 system
Electrooculogram electrodes placement	1 cm above the right external canthus, 1 cm below the left external canthus
Electrode impedance	Below 20 kΩ
Activation	Eye-opening test: done Photic stimulation, hyperventilation, or medication: not done
Machine	EEG-1218, Nihon Kohden, Tokyo, Japan

obtained from precise evaluation of conventional EEG records, especially in relation to physiological change in waveforms as shown in the present patient, can be further utilized.

Conflict of interest

None of the authors have potential conflicts of interest to be disclosed.

References

American Academy of Sleep Medicine. AASM manual for the scoring of sleep and associated events, version 2.5. 2018.
Claassen, J., Mayer, S.A., Kowalski, R.G., Emerson, R.G., Hirsch, L.J., 2004. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. Neurology 62 (10), 1743–1748.
Ferlazzo, E., Magaudda, A., Striano, P., Vi-Hong, N., Serra, S., Genton, P., 2007. Long-term evolution of EEG in Unverricht-Lundborg disease. Epilepsy Res. 73 (3), 219–227.
Hitomi, T., Inouchi, M., Takeyama, H., Kobayashi, K., Sultana, S., Inoue, T., Nakayama, Y., Shimotake, A., Matsuhashi, M., Matsumoto, R., Chin, K., Takahashi, R., Ikeda, A., 2018. Sleep is associated with reduction of epileptiform discharges in benign adult familial myoclonus epilepsy. Epilepsy Behav. Case Rep. 11, 18–21.
Krøigård, T., Forsse, A., Bülow, K., Broesby, J., Poulsen, F.R., Kjaer, T.W., Høgenhaven, H., 2019. The diagnostic value of continuous EEG for the detection of non-convulsive status epilepticus in neurosurgical patients - a prospective cohort study. Clin. Neurophysiol. Pract. 4, 81–84.
Posner, M.I., Petersen, S.E., 1990. The attention system of the human brain. Annu. Rev. Neurosci. 13 (1), 25–42.
Raz, A., 2004. Anatomy of attentional networks. Anat. Rec. B New Anat. 281 (1), 21–36.

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