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SHORT COMMUNICATION/COMMUNICATION BRÈVE

Not everything that shakes is a seizure... Role of continuous EEG in the intensive care unit

Toutes les secousses ne sont pas des crises... Un intérêt de l'EEG continu dans les services de réanimation

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Summary Treatment of status epilepticus often requires highly sedative drugs with risk of side effects. Correct diagnosis is mandatory in order to prevent introduction of usefulness treatments. We report a case of suspected myoclonic status epilepticus. A thalamic lesion secondary to an osmotic demyelination syndrome was found to be the likely etiology of the myoclonus. Electrophysiological data (electroencephalography and electromyography) provided evidence for a subcortical origin of myoclonus and use of continuous EEG allowed monitoring of drug withdrawal.

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MOTS CLÉS
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Syndrome de démyélinisation osmotique ;
État de mal épileptique ;

Résumé Le traitement des états de mal épileptique fait appel à des traitements sédatifs, qui ne sont pas dénués d'effets secondaires. Une certitude diagnostique est nécessaire afin d'éviter l'introduction de traitements inutiles. Nous rapportons le cas d'une patiente suspecte d'état de mal myoclonique. Le bilan mis en évidence une lésion thalamique, secondaire à une myélinolyse extra-pontine, probablement à l'origine des myoclonies. Le bilan électrophysiologique (EEG, EMG) apporta des éléments en faveur d'une origine sous-corticale de ces myoclonies, et l'utilisation de l'EEG continu permit un arrêt des traitements en toute confiance.

Case report

A 52-year-old woman, with past medical history of chronic hypertension, alcohol abuse and recent potomania, was admitted to the emergency department following impaired consciousness. On admission, Glasgow coma scale was 13, without focal neurological deficit. She was hemodynamically stable and apyrexial. Initial laboratory examinations showed serum sodium level of 107 mmol/L (normal 135—145), without other abnormalities. Following water restriction and sodium infusion, serum sodium level rose to 126 mmol/L after 24 hours, was normalized the following days, and remained stable (Fig. 1). Her neurological state concomitantly improved to Glasgow coma scale 15 at day two.

Three days later, she became confused, and a motor deficit with pyramidal hypertonia appeared. A first brain MRI, performed at day 10, showed increased signal in the pons, both lenticular nuclei, external capsule and posterior frontal cortex on DWI sequences, also just visible on FLAIR sequences, consistent with osmotic demyelination syndrome (Fig. 2a). On day 19, she presented right-sided limb myoclonus, suggestive of seizures. IV clonazepam was first administered. As myoclonus persisted, treatment was immediately switched to IV phenytoin and IV levetiracetam, without significant clinical effect. Status epilepticus was suspected; the patient was sedated with propofol, and phenobarbital was added. She was then referred the same day to our tertiary neurology service.

On admission, Glasgow coma scale was 3 during sedation with propofol and she still had right-sided myoclonic jerks. These were rhythmic, permanent, at rest and had no reflex character. They predominated in proximal upper limbs, were also seen in lower limbs, but spared the face (video). Monitoring with continuous EEG was started and revealed

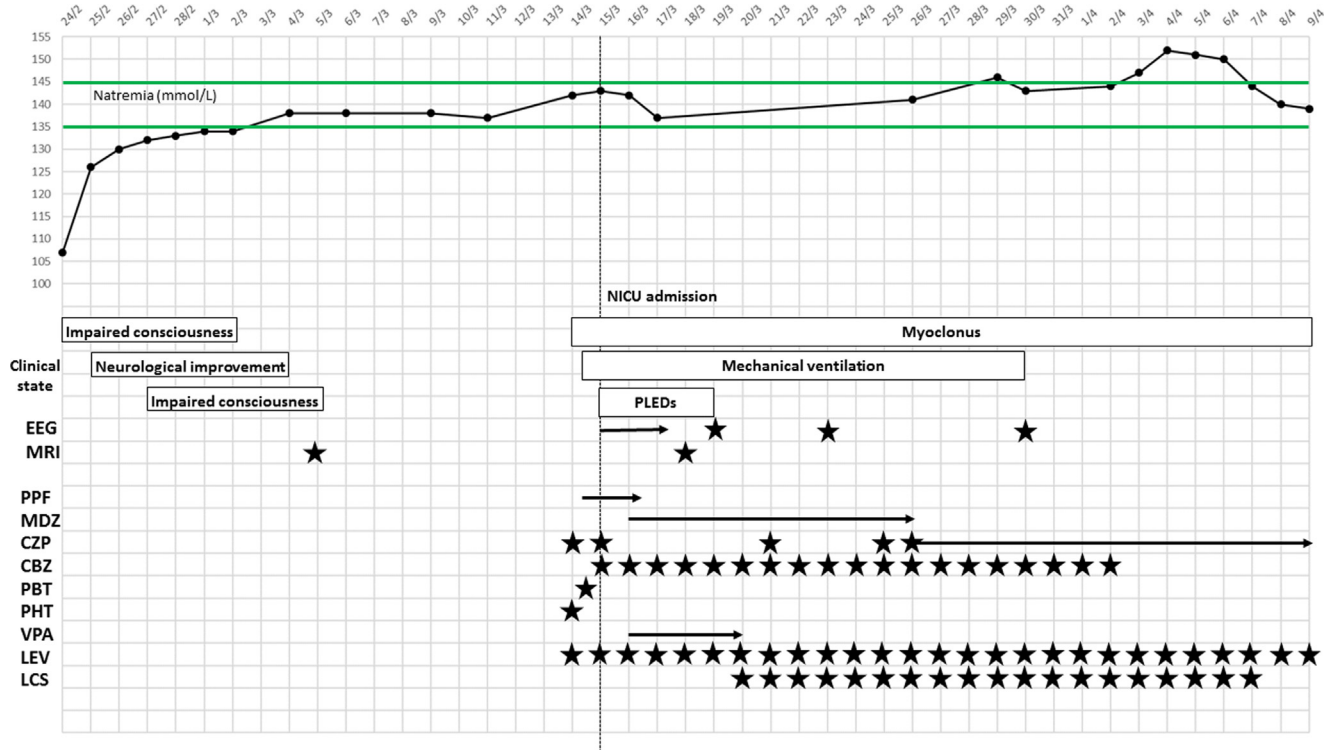

Figure 1 Evolution of the natremia, and clinical events (normal values of natremia are beyond green lines). PPF: propofol; MDZ: midazolam; CZP: clonazepam; CBZ: carbamazpine; PBT: phenobarbital; PHT: phenytoin; VPA: valproate; LEV: levetiracetam; LCS: lacosamide.