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ORIGINAL ARTICLE/ARTICLE ORIGINAL

Anti-NMDA-R encephalitis: Should we consider extreme delta brush as electrical status epilepticus?

*Encéphalite à anticorps anti-NMDA-R : l'*extreme delta brush *est-il une forme d'état de mal épileptique ?*

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Summary Seizures are common clinical manifestations in anti-*N*-methyl-D-aspartate receptor (anti-NMDA-R) encephalitis, among other neurological and psychiatric symptoms. During the course of the disease, some specific EEG patterns have been described: generalized rhythmic delta activity (GRDA) and extreme delta brush (EDB). In comatose patients, the association of these EEG abnormalities with subtle motor manifestations can suggest ongoing non-convulsive status epilepticus (NCSE). We report the case of a 28-year-old woman admitted for a clinical presentation typical of anti-NMDA-R encephalitis, which was confirmed by CSF analysis. She was rapidly intubated because of severe dysautonomia and disturbed consciousness. Clinical examination revealed subtle paroxysmal and intermittent myoclonic and tonic movements,

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correlated on video-EEG with GRDA and/or EDB. NCSE was then suspected, but electroclinical manifestations persisted despite many anti-epileptic drugs combinations, or reappeared when barbiturate anesthesia was decreased. In order to confirm or dismiss the diagnosis, intracranial pressure (ICP) and surface video-EEG monitoring were performed simultaneously and revealed no ICP increase, thus being strongly against a diagnosis of seizures. Sedation was progressively weaned, and clinical condition as well as EEG appearance progressively improved. Literature review revealed 11 similar cases, including 2 with focal NCSE. Of the nine other cases, NCSE diagnosis was finally excluded in 5 cases. NCSE diagnosis in association with anti-NMDA-R encephalitis is sometimes very difficult and its occurrence might be overestimated. Video-EEG is highly recommended and more invasive techniques may sometimes be necessary.

MOTS CLÉS
Activité delta ;
Encéphalite auto-immune ;
État de mal épileptique ;
Récepteur *N*-méthyl-D-aspartate

Résumé Les encéphalites auto-immunes à anticorps anti-récepteurs-NMDA (Ac anti-NMDA-R) se manifestent typiquement par des troubles neurologiques, psychiatriques, et des crises d'épilepsie. À l'EEG, des patterns particuliers ont été décrits : activités lentes delta rythmiques (OLDR) diffuses et *extreme delta brush* (EDB). La présence chez des patients comateux de ces anomalies EEG rythmiques, associées à des manifestations motrices erratiques, fait suspecter un état de mal épileptique non convulsivant (EMENC). Nous rapportons le cas d'une patiente de 28 ans adressée pour un tableau typique d'encéphalite à Ac anti-NMDA-R, confirmé par l'analyse du LCR. Devant la dysautonomie sévère et les troubles de conscience, la patiente est rapidement intubée. L'examen clinique objective des mouvements myocloniques et toniques intermittents. L'EEG retrouve des OLDR, puis un aspect d'EDB typique. Ces éléments électro-cliniques, temporellement corrélés sur la vidéo-EEG, conduisent à une escalade de la thérapeutique anti-épileptique, en plus du traitement spécifique. Devant le caractère réfractaire et prolongé de l'EMENC, une mesure de la pression intracrânienne est réalisée, venant remettre en cause le diagnostic initial. La levée de la sédation, à 2 mois du diagnostic, est suivie d'une amélioration clinique et électro-encéphalographique. Après analyse de la littérature, nous avons retrouvé 11 cas similaires, dont 2 sont des états de mal partiel. Le diagnostic d'EMENC fut finalement réfuté dans 5 cas sur les 9 restants. Diagnostiquer un EMENC dans un contexte d'encéphalite à Ac anti-NMDA-R s'avère délicat. L'usage de la vidéo-EEG est fortement recommandé, et des techniques plus invasives peuvent parfois être nécessaires.

Introduction

Anti-*N*-methyl-D-aspartate receptor encephalitis is a recently described autoimmune and paraneoplastic encephalitis [5]. It is the second most common immune-mediated encephalitis, after acute disseminated encephalomyelitis, and represents 4% of all encephalitis. Women are more frequently affected than men, and an underlying tumor (mostly ovarian teratoma) is observed in 60% of cases. The clinical picture is stereotyped [5], usually starting with psychiatric and neurological symptoms: acute psychosis episode, hallucinations, seizures, dyskinesia, vegetative (hemodynamic and respiratory instability), and/or cognitive impairment. Brain MRI shows non-specific abnormalities in 50% of the cases, in various locations: hippocampus, cerebellum, fronto-basal cortex, insula and/or basal ganglia. Cerebrospinal fluid (CSF) is abnormal in 80% of patients [5]. Diagnosis is based on detection of Ig G antibody against the NR1 sub-unit of NMDA receptor [5].

Specific treatment of any underlying tumor (surgical resection) and immunotherapy are associated with a slow recovery without sequellae in about 75% of cases [5]. Decrease of antibody level against NMDA-R seems to be a good indicator of treatment efficacy and favorable outcome [5]. In order to rapidly initiate the treatment, tests other than antibody detection are necessary.

EEG abnormalities are observed almost constantly in NMDA-R encephalitis [5], but they are usually non-specific (focal or diffuse polymorphic slow-waves). However, some particular patterns that could help to suggest the diagnosis have been recently described: generalized rhythmic delta activity (GRDA); excessive beta frequency activity; and their co-occurrence that gives rises to, a peculiar pattern named extreme delta brush, which is considered highly specific of the disease [14].

Epileptic seizures are very common in NMDA-R encephalitis, occurring in 76% of the patients. Initially considered quite rare [5], a growing number of non-convulsive status epilepticus (NCSE) cases have been published over the last few years [1,2,6—11,13]. They are usually diagnosed electrically, because of rhythmic EEG abnormalities more or less associated with erratic motor manifestations in a comatose patient.

We report the case of a female patient with a typical clinical presentation. EEG showed prolonged rhythmic slow-waves associated with intermittent motor symptoms during

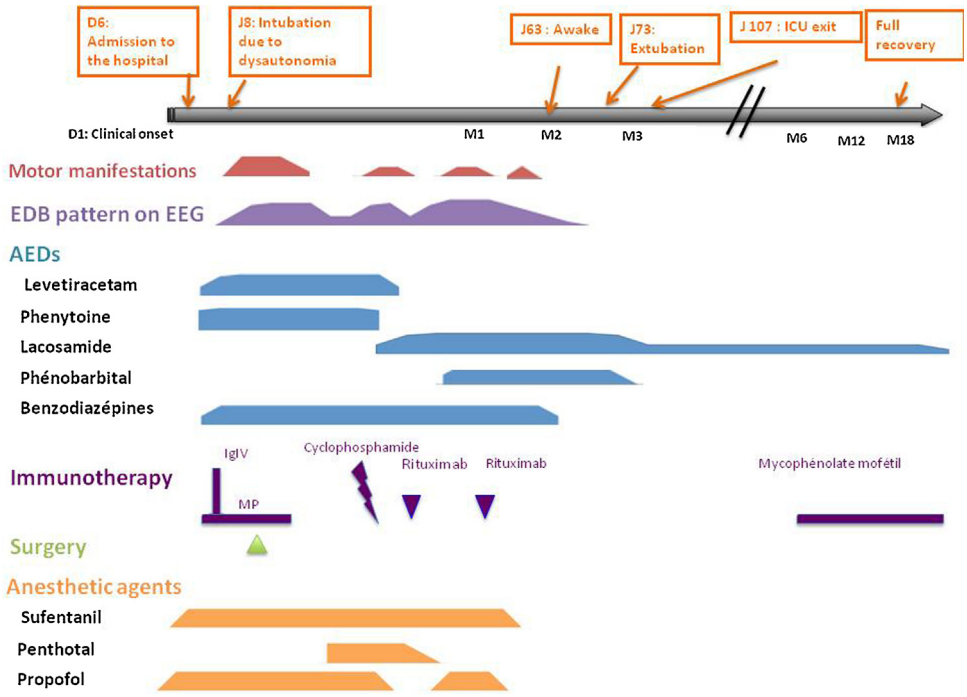

Figure 1 EEG, clinical and therapeutic events during the course of the disease.

a prolonged stay in the intensive care unit (ICU). This case provides the opportunity to discuss NCSE diagnostic criteria in the particular setting of NMDA-R encephalitis.

Case report

Clinical data

A 28-year-old woman complained of unusual headache accompanied by nausea and slight fever. She had no medical history except for dysimmune hypothyroidism and left ovarian cyst. The next day, she was admitted to the psychiatric unit because of abnormal behavior including disinhibition, euphoria, auditory hallucinations, and logorrhea. On day 6, clinical examination revealed orofacial dyskinesia, stiff neck and signs of encephalitis: sleepiness and confusion, mutism and signs of pyramidal irritation (diffuse hyperreflexia, positive Babinski sign). General clinical examination was normal with apyrexia. Shortly after, dysautonomia signs appeared (bradycardia, apnea), and the patient was transferred to the ICU and intubated.

Paraclinical data

Initial brain imaging [contrast computerized tomography (CT) scan at day 6 and magnetic resonance imaging (MRI) at day 7] was normal, as were repeated MRI examinations performed a few days and 3 months later. Cerebrospinal fluid (CSF) analysis revealed lymphocytic meningitis (48 leucocytes/mm^3, 94% lymphocytes) and raised CSF protein (0.54 g/L) with intrathecal synthesis of IgG. Tests for multiple viral, bacterial and mycological pathogens were negative. Thoraco-abdominal CT, echography and pelvic MRI revealed a dermoid aspect of the ovarian cyst.

Specific NMDA-R encephalitis treatment

Triple anti-infectious therapy with meningeal diffusion (comprising aciclovir, ceftriaxone and amoxicillin) introduced at day 6 was stopped at day 13, after reception of positive results for NMDA-R antibody (identified by immunohistochemistry and cell-based assay in the French national reference center of paraneoplastic neurological syndromes). Despite early immunological treatment (methylprednisolone on day 7, IV immunoglobulin on day 9) and surgical resection of the ovarian cyst (day 10), no improvement was observed. Diagnosis of mature teratoma was confirmed by pathological examination. Cyclophosphamide was introduced on day 23, followed by rituximab on days 34 and 44 (see Fig. 1). Immunosuppressive therapy was discontinued for a long time due to severe complications including febrile aplasia and peritonitis caused by jejunal perforation.

EEG, seizures and paroxysmal motor signs

The first EEG at day 7 showed high voltage and monomorphic delta slow-waves, with an intermittent rhythmic organization.

On days 8 and 9, the patient developed erratic myoclonic movements in the arms and a generalized tonic-clonic seizure occurred, leading to continuous benzodiazepines IV administration. EEG showed no paroxysmal discharges during the myoclonic jerks, but rather monomorphic delta waves with some bursts of beta activity.

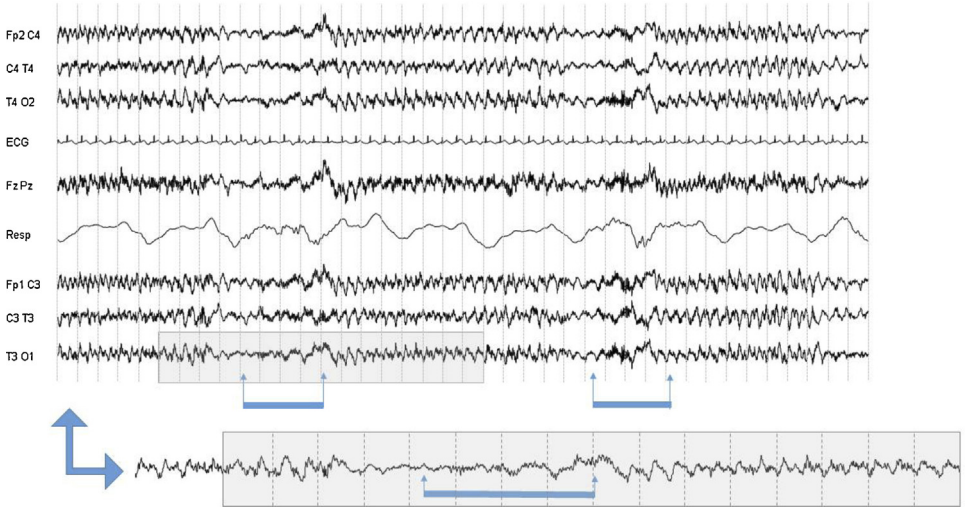

Figure 2 EEG at day 15 visualized with a compressed time window, in order to show the paroxysmal occurrence of GRDA, accompanied and proceeded by excess beta activity. The figure displays 40 seconds of EEG activity, the interval between each vertical dotted line corresponding to 1 second. The horizontal bar indicates the occurrence of motor symptoms (tonic and/or vibratory movements of the limbs). On the lower part of the figure, the grey insert show the T3-01 derivation without time compression, allowing a better appreciation of the dynamical evolution/intertwining of the beta/delta rhythms and their correlation with motor symptoms.

From day 10, the GRDA pattern on EEG was fully formed, the association with beta rhythm became more evident, and a typical EDB pattern progressively emerged. Over periods of several hours, rhythmic slow-waves organized in short sequences were observed, interrupted during a few seconds by more pronounced recruiting beta rhythms. Clinically, some orofacial automatisms and erratic tonic and vibratory movements appeared concomitantly with the rapid rhythms (see Fig. 2). These electroclinical data were suggestive of subtle non-convulsive status epilepticus, and a more aggressive anti-epileptic management was adopted.

While the patient was on sodium thiopental (introduced at day 16), EEG showed the expected burst-suppression pattern, but every attempted subsequent decrease of barbiturate anesthesia level led to reappearance of the rhythmic activity, as well as the motor manifestations.

After many ineffective anti-epileptic drug (AED) combinations, we decided to perform invasive intracranial pressure monitoring, in order to confirm or firmly dismiss the NCSE diagnosis. We performed simultaneous intracranial pressure monitoring (ICP) and continuous surface video-EEG monitoring during 48 hours, and no elevated ICP was observed during the EDB or GRDA sequences on the EEG.

The decision was then taken to progressively wean the sedation level (day 55), and progressive awakening occurred, allowing extubation 11 days later. EEG rhythmic activities disappeared progressively from day 64, and motor manifestations were still noted during a few days. AED (lacosamide and phenobarbital) was maintained during a few months, and progressively stopped during the recovery process.

Chronic phase of the disease

The patient was dismissed from the ICU 3 months and 2 weeks after the onset of the disease. At 4 months, neuropsychological testing revealed a moderate dysexecutive syndrome, with disinhibition and attentional deficit. At 6 months, CSF analysis was normal, but positive detection of antibody against NMDA-R led us to prescribe mycophenolate mofetil. The neuropsychological examination showed a spectacular improvement, and EEG was normal. At the last visit, one year and a half after the beginning of the disease, the recovery process was complete, and the patient was back full time to her previous job. AEDs were stopped, as well as immunosuppressive agents in spite of NMDA-R antibodies still being present in the CSF.

Discussion

We report a clinically typical case of NMDA-R encephalitis in a 28-year-old woman in whom EEG rhythmic abnormalities associated with subtle motor manifestations appeared very suggestive of prolonged NCSE.

Epileptic seizures are very frequent in anti-NMDA-R encephalitis, observed in 80% of the cases [5]. These have been reported as generalized tonic-clonic seizures in 45% of cases, partial complex in 10%, and status epilepticus in 6% of cases. Since this first large series, many status epilepticus have been reported [1,2,6–11,13], and some authors have pointed out the risk of underestimating or overestimating this diagnosis [4,8,6,13]. Indeed, a large variety of movement disorders are frequently described in these patients, associated or not with authentic seizures. Differential diagnosis is sometimes very difficult, particularly in comatose patients. Almost all kinds of dyskinesia have been observed: mastication, grimacing, ballistic or dystonic movements, stiffness, myoclonus, and so on [5].

In that context, EEG is sometimes very difficult to interpret, and could even be erroneously suggestive of NCSE. Indeed, GRDA associated or not with excessive beta activity are described in 40% of anti-NMDA encephalitis cases [14].

Table 1 Review of the 11 potential NCSE cases described in the literature.

	Sex, age (years)	Seizures before NCSE	Clinical semiology of NCSE	AEDs administered	EEG abnormalities	NCSE duration	Management in ICU	Treatment considered efficient	Clinical evolution after NCSE
Bayreuther et al., 2009 [3]	F, 25	Yes. Staring, unresponsiveness, oro-alimentary automatisms	Dystonic posturing of left hemibody, orofacial dyskinesias, hypersalivation, opisthotonus	CBZ, BZD, LVT, thiopental	''Generalized pseudo-periodic complex of about 3 Hz''	1—2 months	Yes	Spontaneous recovery	Improvement accelerated after Ig IV administration and surgical resection of the tumour. Full recovery in 6 months
Johnson et al., 2010 [4]	F, 35	No	Unresponsiveness to external stimuli	PHT, LVT, VPA, BZD, propofol, PHB	''Cyclical pattern of moderate to high voltage generalized anteriorly based 2 HZ alternating with 5—6 Hz sharp wave activity''	6 months	Yes	Surgical resection of the tumour	At 6 months: mild deficit on naming and memory tests without functional impairment
Kirkpatrick et al., 2011 [5]	F, 19	Yes — not described	Coma, left beating nystagmus, lip smacking	BZD, PHT, LVT, VPA, thiopental, OXCZ, PHB	''Continuous generalized rhythmic delta activity with an evolution in voltage frequency and field consistent with NCSE, more apparent when EEG was compressed''	Not stated	Yes	Felbamate	Full recovery in 10 months

Table 1 (*Continued*)

	Sex, age (years)	Seizures before NCSE	Clinical semiology of NCSE	AEDs administered	EEG abnormalities	NCSE duration	Management in ICU	Treatment considered efficient	Clinical evolution after NCSE
Gataullina et al., 2011 [6]	M, 8	Yes — paroxysmal attacks of agitation, terror	Multiple daily paroxysma episodes of agitation, neurovegetative disorders	VPA, CBZ, TPM, LVT, KD, BZD	''Diffuse rhythmic delta waves with particularly sudden onset and cessation (....) which persisted throughout and after paroxysmal episodes of agitation''	3 months	Not stated	Plasma exchanges	Progressive but incomplete recovery at 6 months
Goldberg et al., 2011 [7]	F, 8	No	Headache, nausea, somnolence, right hemianopsia, increased tone in right arm	BZD, LVT, fos-PHT, PHB	Nearly continuous seizures consisting of 12—13 Hz sharply contoured activity emanating from the left occipital region	< 24 h	No	Fos-PHT	Neurological worsening (mild right hemiparesis, expressive aphasia, dystonia) followed by incomplete recovery after 1 year
Dericioglu et al., 2013 (case 1) [8]	F, 25	Yes — not described	Orofacial dyskinesia, hypersalivation, hyperventilation	PHT, LVT, CBZ, VPA, ZNS, BZD, propofol, thiopental	''Diffuse rhythmmic delta activity. No correlation with dyskinetic movements''	2 months	Yes	Surgical resection of the tumour + Ig IV	Incomplete progressive recovery but has been living independently for 1 year
Dericioglu et al., 2013 (case 2) [8]	F, 26	Yes — partial seizures with oral automatisms	Unresponsiveness, stereotypic movements, blinking, severe attacks of dysautonomia	PHT, LVT, BZD	''General diffuse slowing down in theta-delta range, sometimes accompanied by rhythmic delta waves without epileptiform activity''	1—3 weeks	Yes	Ig IV	Progressive but full recovery after 1 year

Table 1 (*Continued*)

	Sex, age (years)	Seizures before NCSE	Clinical semiology of NCSE	AEDs administered	EEG abnormalities	NCSE duration	Management in ICU	Treatment considered efficient	Clinical evolution after NCSE
Finné Lenoir et al., 2013 [9]	M, 17	Yes — head deviation to the right evolving to partial status epilepticus and some generalized seizures	After resolution of clinical, SE, no clinical manifestations but persistence of electrical SE	VPA, LVT, PHT, thiopental, TPM, ketamine, BZD	''Rhythmic delta waves with a left predominance''	2 months 1/2	Yes	Spontaneous, or late-onset efficacy of plasma exchange + Ig Iv	Progressive but full recovery after 1 year
Barros et al., 2014 [10]	M, 7	Yes — generalized and partial seizures (eyes and head deviation to the right)	Recurrent partial seizures and lately no manifestations but persistence of electrical SE	PHT, VPA, propofol, BZD, PHB, thiopental, LVT, VGB, CBZ, LMT, ZNS, felbamate, KD	''Left parieto-occipital almost continuous activity''	3 months 1/2	Yes	Left occipital lobectomy	Right hemianopsia, psychomotor delay, pharmaco-resistant epilepsy
Kadoya et al., 2015 [11]	F, 48	No	NCSE — no detailed description	LVT, PHB, TPM, LMT, propofol, BZD	''Generalized rythmic delta activity''	2 months	Yes	Rituximab + cyclophosphamide	Full recovery at 8 months
Probasco et al., 2014 [12]	F, 34	Yes — generalized and partial seizures	Fluctuating aphasia and right hemiparesis, correlated with EEG abnormalities	LVT, PHT, VPA, LCS, BZD, propofol, KD	''Temporal delta activity that would progress and evolve asymmetrically and rhythmically in bursts of 10 s''	Not clearly stated. > 6 months?	Yes	Rituximab	Progressive but incomplete recovery at 6 months

BZD: benzodiazepine; CBZ: carbamazepine; LMT: lamotrigine; LVT: levetiracetam, OXCZ: oxcarbazepine; PHB: phenobarbital; PHT: phenytoine; TPM: topiramate; VPA: valproate; VGB: vigabatrin; ZNS: zonisamide; DC: cetogen diet.

In comatose patients, the presence of EEG rhythmic slow-waves, if these are focal or recruiting (spatial or temporal modification in frequency and voltage), fulfil the criteria for NCSE diagnosis [3]. In anti-NMDA-R encephalitis, some authors claim that it is necessary to compress the time base in order to see clearly this dynamic variation of the EEG traces [12]. The other proposed criteria for ''electrical diagnosis'' of NCSE are:

- subtle clinical manifestations time-locked with rhythmic theta/delta activity;
- clinical and electrical clear-cut reactivity after a benzodiazepine trial [3].

In our patient, 2 out of 3 of these criteria were present: frequency or voltage changes if the time base was compressed for visualization, and intermittent tonic-vibratory movements of the lower limb that seemed correlated with EEG modification (see Fig. 2). However in terms of the 3rd criteria, these clinical signs proved to be resistant to AED, benzodiazepines included. On the other hand, in the case reported by Kirkpatrick et al., administration of felbamate, a glutaminergic antagonist drug, was followed 48 hours later by EEG improvement, which was considered in favor of NCSE diagnosis even though many drugs had been previously tried and found ineffective [12]. In other reported cases, NCSE was finally dismissed after video-EEG analysis because of no temporal correlation between EEG and clinical manifestations [6,8].

The absence of increased intracranial pressure during the EEG sequences with GRDA or EDB goes against NCSE diagnosis in our patient. Such increase has been frequently and even systematically observed when seizures occur, and is strongly in favour of status epilepticus [15]. The full recovery of the patient is also surprising after such a long NCSE, with the time to recovery of one year being similar to previous observations in anti-NMDA encephalitis, even without NCSE [5]. Finally, we found another case report in which invasive investigation was also used to reach a definite diagnosis, with a similar conclusion: even though surface EEG was highly suggestive of NCSE, intracranial EEG recordings found no epileptic discharges, allowing reduction of AED and aggressive sedation [13].

After literature analysis, we found 10 articles, reporting a total of 11 patients suffering from status epilepticus during Anti-NMDA-encephalitis (see Table 1). Two of these cases are well-documented focal status epilepticus, one of which required surgical resection to be controlled. The EEG abnormalities of the nine other cases seem quite similar to the GRDA and EDB described by Schmitt et al. in this disease [14]. All of them were initially diagnosed as a super-refractory NCSE lasting 1 to 6 months, but this diagnosis was finally revised and rejected in 5 of the 9 patients [2,8,6,11,13]. EDB is associated with a more prolonged illness [14], but the reason for this is unclear. Currently, it is impossible to determine if EDB directly reflects a more aggressive phenotype of the disease, or if its presence might induce more aggressive management and consequently a higher risk of complications in these patients. EEG and clinical improvements resulted from immunotherapy and/or surgical resection in most previously reported cases (64%).

To conclude, EEG is essential for diagnosis and management in anti-NMDA-R encephalitis, but its interpretation is sometimes very difficult, particularly when rhythmic EEG patterns are associated with motor manifestations. These difficulties should be taken into account, in order to avoid over-diagnosis of NCSE, and thus iatrogenic complications due to over-prescription of AED and anaesthetic drugs. Video-EEG is highly recommended, but if diagnostic doubt remains then invasive techniques (intracranial pressure monitoring, intracerebral EEG recordings) should be considered in these complex situations.

Disclosure of interest

The authors declare that they have no competing interest.

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