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Delta Brush Pattern Is Not Unique to NMDAR Encephalitis: Evaluation of Two Independent Long-Term EEG Cohorts

Betul Baykan[1], Ozlem Gungor Tuncer[2], Ebru Nur Vanli-Yavuz[1,3], Leyla Baysal Kirac[1], Gokcen Gundogdu[4], Nerses Bebek[1], Candan Gurses[1], Ebru Altindag[2], and Erdem Tuzun[5]

Abstract

Purpose. Although its specificity has not previously been investigated in other cohorts, delta brush pattern (DBP) is increasingly reported in the EEGs of patients with anti-*N*-methyl-D-aspartate receptor (NMDAR) encephalitis. *Methods*. We aimed to investigate the DBP in the EEGs of 2 cohorts; patients with change in consciousness for various causes monitored in the intensive care unit (ICU) (n = 106) and patients with mesial temporal lobe epilepsy (MTLE) with or without antineuronal antibodies (n = 76). *Results*. These patients were investigated for the presence of DBP, defined as an EEG pattern characterized by delta activity at 1 to 3 Hz with superimposed bursts of rhythmic 12- to 30-Hz activity. Two investigators blindfolded for the clinical and immunological data independently analyzed the EEGs for recognition of this pattern. An EEG picture compatible with DBP was observed in 4 patients; only 1 of them (1.3%) belonged to the MTLE group. She did not bear any of the investigated autoantibodies and was seizure-free after epilepsy surgery. In the ICU group, there were 3 additional patients showing DBP with various diagnoses such as hypoxic encephalopathy, brain tumor, stroke, and metabolic derangements. All of them had died in 1-month period. *Conclusions*. Our results underlined that DBP is not unique to NMDAR encephalitis; it may very rarely occur in MTLE with good prognosis after surgery and second, in ICU patients who have high mortality rate. Therefore, the presence of this pattern should alert the clinician for NMDAR encephalitis but other possible etiologies should not be ignored.

Keywords

electroencephalography, delta brush pattern, anti-*N*-methyl-D-aspartate receptor encephalitis, long-term video-EEG monitoring, intensive care unit, mesial temporal lobe epilepsy

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Introduction

After the first description by Schmitt et al[1] in 7 out of 23 patients who underwent continuous EEG monitoring, delta brush pattern (DBP) is increasingly reported in the EEGs of patients with anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis.[1] Many small retrospective studies and case reports emphasized its actual importance for the diagnosis of this highly specific and newly recognized type of autoimmune encephalitis.[2-5] This impressive EEG finding, named as delta brush because of its resemblance to waveforms seen in premature infants, was found to be associated with a more prolonged hospitalization and increased days of monitoring in the patients with anti-NMDAR encephalitis.[1]

NMDAR encephalitis often presents with limbic symptoms (amnesia, psychosis), temporal lobe seizures and neuroimaging findings indicating involvement of mesial temporal lobes. In line with these clinical findings, experimental studies have shown reduced hippocampal NMDAR expression in the animal model of NMDAR encephalitis.[6] Disorders of consciousness is also frequently encountered in this disease.[7] Thus, DBP could be a marker of underlying hippocampal dysfunction and/or accompanying alterations in consciousness. However, there are no studies investigating the presence of this unique and under-recognized EEG pattern in other groups of patients with alteration in consciousness monitored in neurological intensive

[1]Department of Neurology, Istanbul Faculty of Medicine, Istanbul University, Istanbul, Turkey
[2]Department of Neurology, Istanbul Bilim University, Istanbul, Turkey
[3]Department of Neurology, School of Medicine, Koc University, Istanbul, Turkey
[4]Department of Pathology, Istanbul Faculty of Medicine, Istanbul University, Istanbul, Turkey
[5]Department of Neuroscience, Institute of Experimental Medical Research, Istanbul University, Istanbul, Turkey

Corresponding Author:
Ebru Nur Vanli-Yavuz, Department of Neurology, Istanbul Faculty of Medicine, Istanbul University, 34093 Capa/Fatih, Istanbul, Turkey.
Email: ebruvanli@gmail.com
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care unit (ICU) or in adult patients with long-term video-EEG monitoring studies for epilepsy surgery. Thus, the specificity of this pattern that created recent alertness is not exactly known due to the lack of large cohort studies.

Mesial temporal lobe epilepsy with hippocampal sclerosis (MTLE-HS) is another disease with prominent hippocampal involvement. Moreover, the presence of anti-NMDAR antibodies was shown in some patients with chronic epilepsy forms such as cryptogenic focal epilepsy or MTLE-HS, which is the prototype of drug-resistant epilepsy syndromes undergoing presurgical evaluation with long-term EEG monitoring.[8,9] To investigate the specificity of "delta brush pattern," we investigated this finding in patients with alteration in consciousness for various causes monitored with continuous EEG in ICU and in consecutive patients with MTLE-HS with or without antineuronal antibodies.

Materials and Methods

Patients and EEG Investigations

Two independent cohorts with long-term EEG investigations available for reevaluation were included and the local ethical committee approved this retrospective observational study.

Delta brush was defined as an electroencephalographic pattern characterized by delta activity at 1 to 3 Hz with superimposed bursts of rhythmic 12- to 30-Hz beta frequency activity "riding" on each delta wave.[1]

Patients With Change in Consciousness Monitored in ICU. We retrospectively identified all critically ill patients, older than 18 years with change of consciousness (Glasgow Coma Score [GCS] ≤14) that underwent continuous EEG (cEEG) monitoring in the ICU during a 2-year period. Clinical and demographic data, all radiology and laboratory findings were investigated and necessary information for the diagnosis and prognosis was gathered from review of inpatient medical notes, neuroimaging studies, reports, and discharge summaries. One patient with endometrial cancer diagnosed as nonconvulsive status epilepticus of unknown cause was excluded because her exact etiology could not be found and her antineuronal antibody tests could not be performed.

cEEG was recorded using 21 MR-compatible silver chloride electrodes, affixed to the scalp according to the International 10-20 System by a certified EEG technologist. Electrodes were checked by both EEG technologist and the health personnel twice a day. The video recording was done simultaneously by a microphone system and cameras. cEEG monitoring studies were interpreted by 2 certified clinical neurophysiologists experienced in EEG (one of them was blinded for clinical findings), independently. Any disagreements between them were resolved by consensus meeting after reviews.

Patients With MTLE-HS. We included 76 consecutive patients diagnosed with MTLE fulfilling the MRI criteria for HS and had long-term video-EEG studies, available. Written informed consent was obtained from all patients. MRIs were performed with a 1.5-T scanner (Magnetom Siemens Symphony, Erlangen, Germany) with thin coronal in addition to sagittal and axial planes including T1, T2, and fluid-attenuated inversion recovery (FLAIR) images to visualize mesial temporal regions optimally and were investigated for the verification of HS diagnosis. The presence of atrophy on T1 and high signal changes on T2 and FLAIR series in any one or more parts of the hippocampus were considered as the major criteria necessary to establish the neuroradiological diagnosis of HS. Those patients with HS on MRI but with dual pathologies were excluded to obtain a more homogenous group.

All available EEGs (routine, video-EEG, and invasive monitoring) were evaluated by 2 investigators independently. Investigators were blind to the antibody profile of the patients.

Antibody Detection

The methods and results of the antineuronal antibody testing of this group were reported previously.[9] Briefly, sera of MTLE-HS patients were kept at −80°C until assayed and tested for antibodies against NMDAR, voltage-gated potassium channel (VGKC) complex antigens, contactin-associated protein-like 2 (CASPR-2) and leucine-rich glioma inactivated 1 (LGI1), glutamate decarboxylase (GAD), glycine receptor (GLY-R), alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAR), and type A gamma aminobutyric acid receptor ($GABA_AR$) as described previously. Antibody investigation could not be performed in ICU patients due to unavailability of sera because of the retrospective nature of this study. Descriptive statistics were computed using statistical software (SPSS 20).

Results

A total of 106 patients were included and monitored in the ICU with long-term EEG. The etiologies were stroke in 32 patients (26 of them had ischemic stroke and 6 had hemorrhagic stroke), metabolic-toxic causes in 22 patients, central nervous system neoplasia in 10 (4 patients had brain metastases, and 6 patients had brain tumors), hypoxic-anoxic encephalopathy in 28 (11 had received cardiopulmonary resuscitation), head trauma in 6 (2 had traumatic subarachnoid hemorrhage, 4 had subdural hemorrhage), central nervous system infection in 3 (one with acute bacterial meningoencephalitis, one with acute viral encephalitis of unknown cause and the last one with Creutzfeldt-Jakob disease as diagnosed with typical clinical, cerebrospinal fluid, MRI findings, and follow-up data), 2 possible autoimmune encephalitis (antineuronal antibody tests of NMDAR, AMPAR, GABAAR, LGI1 and CASPR-2 with commercially available ELISA kits were negative), dementia in 2 patients and the last patient had brain surgery for Parkinson's disease. Because of the retrospective nature of the study, we could not test for other neuronal autoantibodies in the ICU group. The

Table 1. Clinical and Laboratory Findings of Patients With Delta Brush Pattern on Their EEG.

Patients	1 (Figure 1)	2 (Figure 2)	3 (Figure 3)	4 (Figure 4)
Age (y) at index EEG/Sex	86/Female	38/Male	83/Female	20/Female (age at seizure onset: 17 y)
Seizure types	Myoclonic seizures, nonconvulsive seizures	Focal motor seizures (started 6 months ago), nonconvulsive seizures	Myoclonic jerks in the face, arms, and legs	Focal seizures with loss of consciousness (temporal onset)/2 clusters per month
Etiology	Hypoxic ischemic encephalopathy, dyspnea	Nonconvulsive status epilepticus related to systemic derangements, nonspecific infection, and stroke	Brain tumor and nonspecific upper respiratory tract infection	Mesial temporal lobe epilepsy
MRI findings	Cortical atrophy (no strokes)	Bilateral occipital L > R DWI restriction suggestive of stroke	Bilateral parietal mass lesion with contrast enhancement	Hippocampal sclerosis
Follow-up duration	30 days	7 days	7 days	6 years
Other EEG findings	Right sided PLEDs, nonconvulsive seizures, then bi-PLEDs	Nonconvulsive seizures from both occipital regions, GPED	Left frontocentral focus, Bi-PLED, L frontocentrotemporal seizure activity, triphasic waves, GPED	Right temporal interictal focus and TIRDA
Rhythmic delta activity	TIRDA	TIRDA	—	TIRDA and nonspecific short delta waves
Fast activity (independent)	Present	Present	Present	Present
Prognosis	Exitus	Exitus	Exitus	R selective amygdalahippocampectomy (2012)
Drugs used during DB	Levetiracetam 1500 mg, valproic acid 2000 mg	Prednisone, sotalol, entacavir, tigecycline, amikacin, midazolam, valproic acid 800 mg, levetiracetam 2000 mg	Levetiracetam 1000 mg, steroid for brain edema	Carbamazepine 600 mg, topiramate 100 mg
The percentage of DB	2.7	1.7	6.6	4.0
Current treatment	Not applicable	Not applicable	Not applicable	None (antiepileptic drugs were gradually discontinued)
PET findings	—	—	—	PET: bilateral R > L temporal hypometabolism
Past history	Congestive heart failure	Renal failure and renal transplantation	Diabetes, dementia	Unremarkable
Other notes	Entubation	Entubation	No respiratory support	Paternal grandmother with epilepsy Depression
Neurological status	Coma	Coma	Somnolence	Normal, mild attention deficit in neuropsychological testing
Biochemistry	Unremarkable	Uremia and elevated infection parameters, pancytopenia	Normal except elevated infection parameters	Unremarkable

Abbreviations: BiPLED, bilateral independent periodic lateralized epileptiform discharges; DB, delta brush; GPED, generalized periodic epileptiform discharges; TIRDA, temporal intermittent rhythmic delta activity; PET, positron emission tomography; PLED, periodic lateralized epileptiform discharges; VEM, video-EEG monitoring.

current ages of the included patients were 68 ± 15.4 years and 56 of them were women. The evaluation of the EEG data revealed rhythmic periodic patterns in 47 of the patients, 23 being of ictal and 15 nonictal and 9 probably ictal records, according to Salzburg Consensus criteria.[10] Rhythmic delta activity was observed in 5 out of these 106 patients and fast rhythmic activity was seen in 11 patients. DBP was determined only in 3 patients (2.8% in the whole ICU group) and their characteristics are summarized in Table 1. The EEG examples were also shown in Figures 1-3.

The MTLE-HS group included 76 consecutive patients (44 women) fulfilling our inclusion criteria. The mean follow-up duration and ages of patients were 120 months (±99.5) and 38 (±8.7) years, respectively. Mean video EEG recording time of the

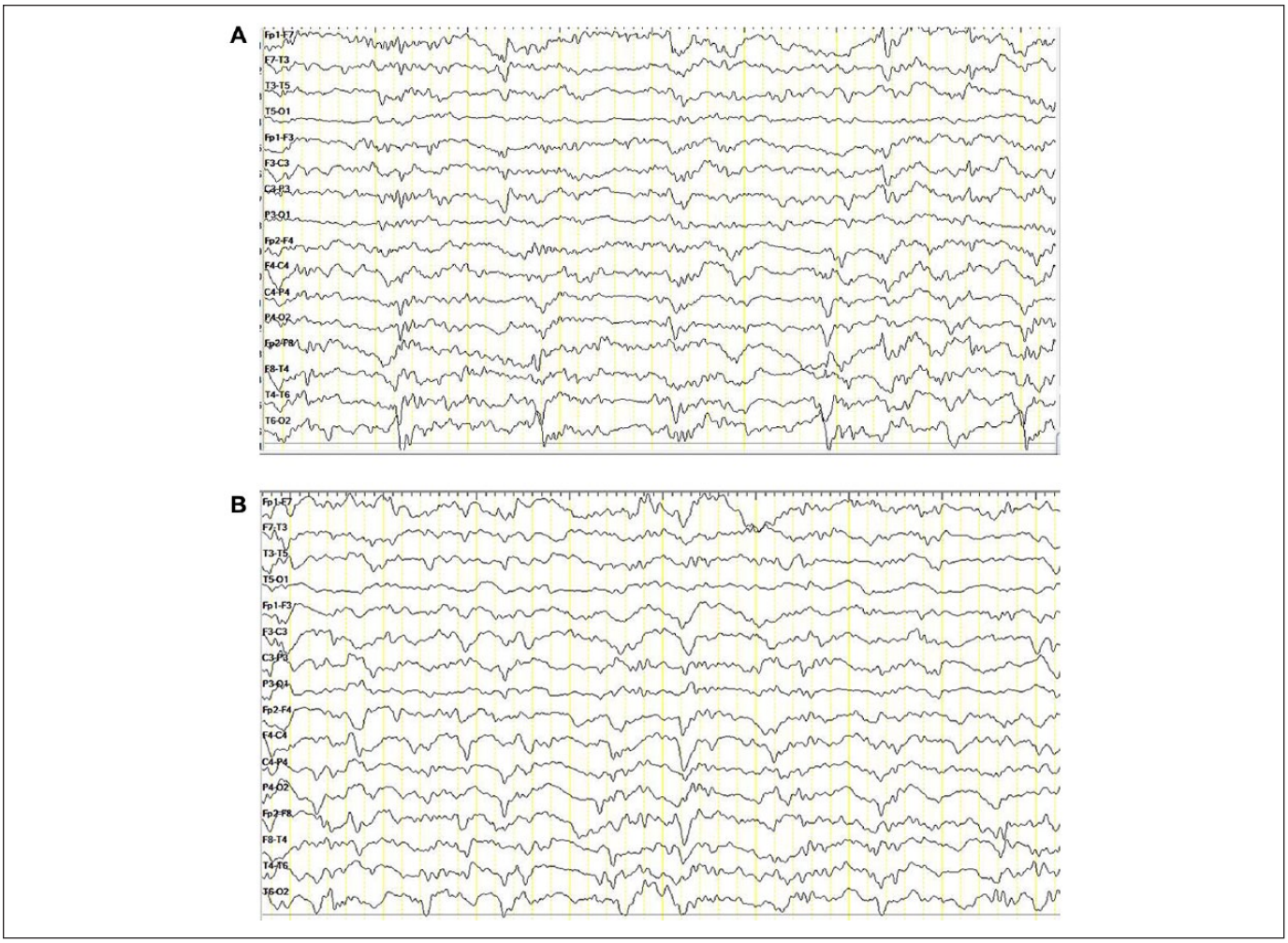

Figure 1. (A, B) An 86-year-old female patient diagnosed with hypoxic ischemic encephalopathy. Diffuse delta brush activity predominantly in bilateral frontal regions.

group was 183.41 hours (±107.60). There were only 4 patients with NMDAR autoantibodies and various other autoantibodies were found in 21 patients (antibodies against CASPR-2 in 11 patients, uncharacterized VGKC complex antigens in 4 patients, glycine receptor in 5 patients, and $GABA_AR$ in 1 patient) as recently reported.[9] In a total of 102 video-EEG investigations including 404 ictal recordings, there were no differences regarding background activity, focal slowing epileptic activity between the seropositive and seronegative group. The rates of the patients with frontal intermittent rhythmic delta activity, temporal intermittent rhythmic delta activity and fast activity were 46 (60.5%), 11 (14.5%), and 31 (40.8%), respectively.

There was only one patient judged to have an asymmetric delta brush by 2 investigators (Figure 4). This patient did not show NMDAR antibodies or any of the investigated autoantibodies and was not under treatment with a drug that could be responsible for the fast activity. Pathological examination of the MTLE-HS patient with delta brush pattern showed hippocampal atrophy as expected. Hematoxylin and eosin staining did not reveal any inflammatory infiltrates.

Discussion

Our results indicate that DBP of premature newborns could also be observed with a rate of 2.8% in long-term EEGs of critically ill patients followed-up in ICU having various etiologies. Although extremely rare (0.9%), it could also appear in patients with MTLE-HS in an asymmetrical manner. Schmitt et al,[1] who brought extreme DBP into attention, already underlined the fact that the specificity of this pattern is unclear; its presence should only raise consideration of anti-NMDAR encephalitis.

The classical DBP of premature neonates (a combination of delta waves with superimposed 8-20 Hz fast symmetrical rhythms) is not a well-known EEG picture among the adult neurologists. They can be seen in any head region, are less commonly reported in the frontal regions, present both during sleep and wakefulness and known to disappear by one month of age.[11] Therefore, this unique pattern which was not reported in other neurological conditions evoked great interest among the electroencephalographers as a "pathognomonic" marker of

Figure 2. (A) A 38-year-old male patient with a history of renal transplantation has acute renal failure associated with nonconvulsive and convulsive seizures. Delta brush activity is asymmetric and intermixed with nonconvulsive seizure activity characterized by spike and waves in the left occipital region (B) Asymmetric delta brush pattern (Right > Left) seen together with bi-occipital rhythmic delta activity (Left > Right) in the same patient.

NMDAR encephalitis. However, this does not seem to be appropriate according to our results. The determination of DBP as "extreme" in the relevant literature is not a clear definition which needs further elaboration. There are some case reports of patients with NMDAR encephalitis showing DBP lasting up to several hours.[12] In our study, we observed DBP for shorter periods around a few seconds to minutes and usually less than 5% of the total recording time. There are also patients with relatively short periods of DBP; we observed in a recent study 2 patients with NMDAR Ab positivity showing rhythmic delta waves superimposed with beta frequency activity resembling "delta brush" pattern, like the ones presented here, appearing in relatively shorter periods.[13]

Although previously reported in about one-third of adult patients, DBP was seen only in one of 20 pediatric patients with NMDAR encephalitis, indicating its predominance in adults.[2,14]

A recent case report of a previously normal 4-year-old boy who presented with an explosive onset of drug-resistant prolonged seizures, ataxia, and encephalopathy responsive to immunotherapy showed atypical "delta brush–like" waves on the EEG. The patient was found to be positive for GLY-R antibodies indicating that this EEG pattern is not specific for NMDAR antibodies, only.[15]

We investigated our MTLE-HS group for GLY-R autoantibodies, found 5 patients with these newly described antibodies, but none of them showed delta brush–like patterns in their investigated EEGs.

It was reported that patients with this pattern were hospitalized longer and DBP pattern may be a marker of more severe disease and perhaps worse outcome.[1] There is a report of a patient with early evidence of extreme DBP and persistence of this pattern many weeks associated with little clinical improvement.[4] In our study, 3 of the 4 patients having this

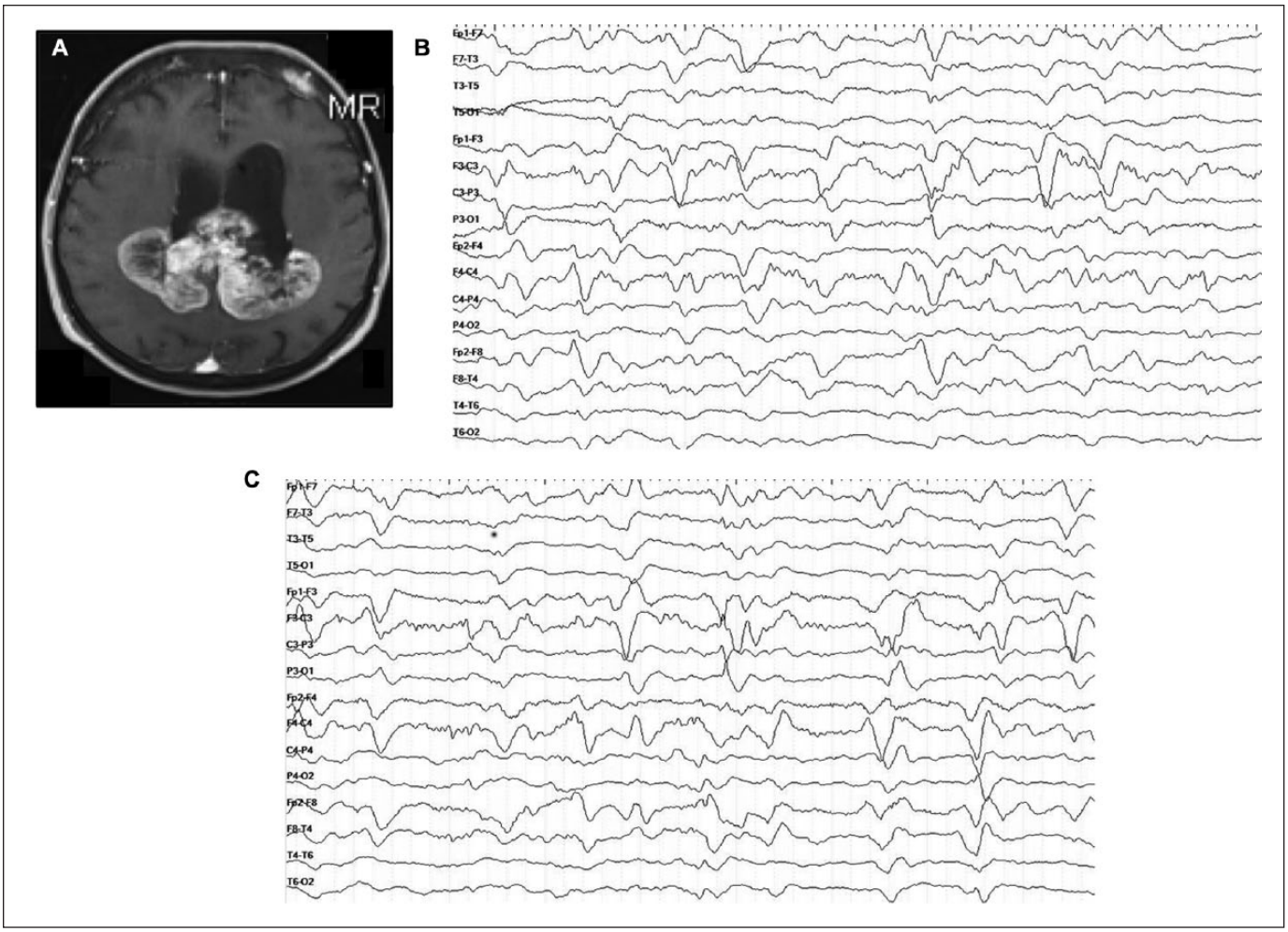

Figure 3. (A) An 83-year-old female patient who has biparietal mass lesion suggestive of glioblastoma with contrast enhancement in magnetic resonance imaging. (B, C) Delta brush activities prominent in the frontocentral regions.

pattern were deceased indicating a trend toward a worse outcome also with other etiologies of this pattern, as seen in Table 1. But our patient with MTLE had a favorable prognosis, indicating the importance of the underlying etiology.

The mechanisms underlying the delta brushes remain still unclear and require further investigation. As a cellular mechanism, modulation of NMDAR-mediated currents was proposed by altering rhythmic neuronal activity and leading to this pattern.[1] Furthermore DBP was recently found (4%) among intracranial EEG seizure-onset patterns related exclusively to focal cortical dysplasia in a series including HS in one-third of the investigated patients with different epileptogenic lesions defined by high-frequency oscillation correlates of each seizure pattern.[16] Another recent study analyzed the most representative seizure type of 37 patients with drug-resistant focal epilepsy who underwent invasive EEG and reported that delta brushes had the highest densities of both ripples and fast ripples.[17] Our study added different etiologies like brain tumors to this spectrum.

Regarding the ictal versus interictal nature of this pattern, in the original study by Schmitt et al,[1] none of the patients responded clinically or electrographically to trials of intravenous antiepileptic drugs. Therefore, these authors suggested that it does not qualify as ictal by proposed definitions of nonconvulsive seizures, and it may lie on the ictal-interictal continuum. On the other hand, there are video-EEG studies indicating that DBP represented an ictal pattern and even could be an evolutive pattern of status epilepticus in NMDAR encephalitis.[2,18] On the contrary other authors supported the view that to avoid unnecessary treatment, this pattern should not be interpreted as indicative of ictal activity, unless there is evidence of its ictal nature. The importance of serial EEGs and prolonged EEG monitoring is also underlined.[19] We observed DBP as an interictal EEG pattern in 3 patients, whereas in the last one, this pattern was closely related to an ictal recording.

Benzodiazepines and barbiturates typically induce excess beta activity, which could create a similar picture when overlapping with delta waves related with coma or sleep, and so on. In one of our cases, DBP appeared during use of midazolam, but the beta activity occurring in periodic short form

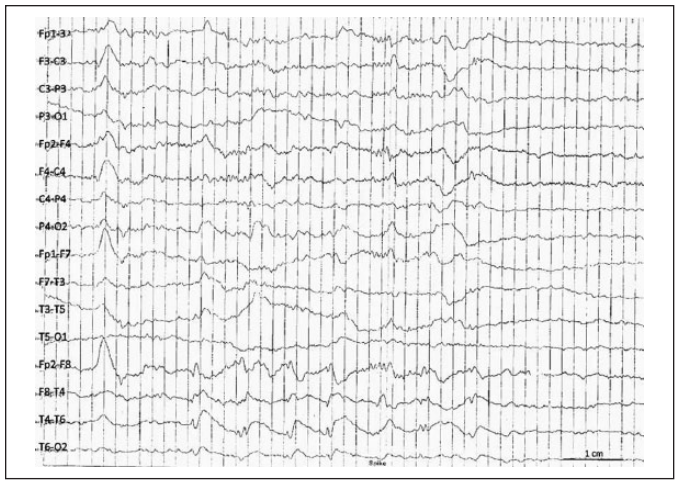

Figure 4. A 20-year-old female patient with typical mesial temporal lobe epilepsy associated with right-sided hippocampal sclerosis on her magnetic resonance imaging showed this asymmetrical pattern of delta brushes in her presurgical video-EEG monitoring that lasted 19 days. During this EEG example, she was neurologically normal, awake, and was using only carbamazepine and topiramate with lowered doses for capturing her seizures. Her interictal EEGs showed independent, temporal intermittent rhythmic delta waves, nonspecific delta paroxysms prominent on frontal regions besides interictal epileptic activity over right temporal area. Seven seizures originating from the right mesial temporal lobe were recorded with concordant semiological characteristics including preserved speech during automatisms, postictal nose wiping, and mild smiling. This patient benefited from epilepsy surgery (please also check patient 4 in Table 1).

synchronized with diffuse, frontally predominant rhythmic delta activity could not be explained by sedative drug use alone. Moreover, our MTLE-HS patient and 2 other patients did not use these drugs.

In conclusion, delta brush pattern on the EEG may rarely observed with various etiologies in adult patients followed up in ICU or with MTLE-HS and therefore it is not pathognomonic for NMDAR encephalitis. But its presence still may guide early recognition and prompt immunotherapy in NMDAR encephalitis, especially in cases with suitable clinical features when other possible etiologies are excluded.

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Declaration of Conflicting Interests

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