# AUTOIMMUNE ENCEPHALITIS: A PRACTICAL EEG APPROACH

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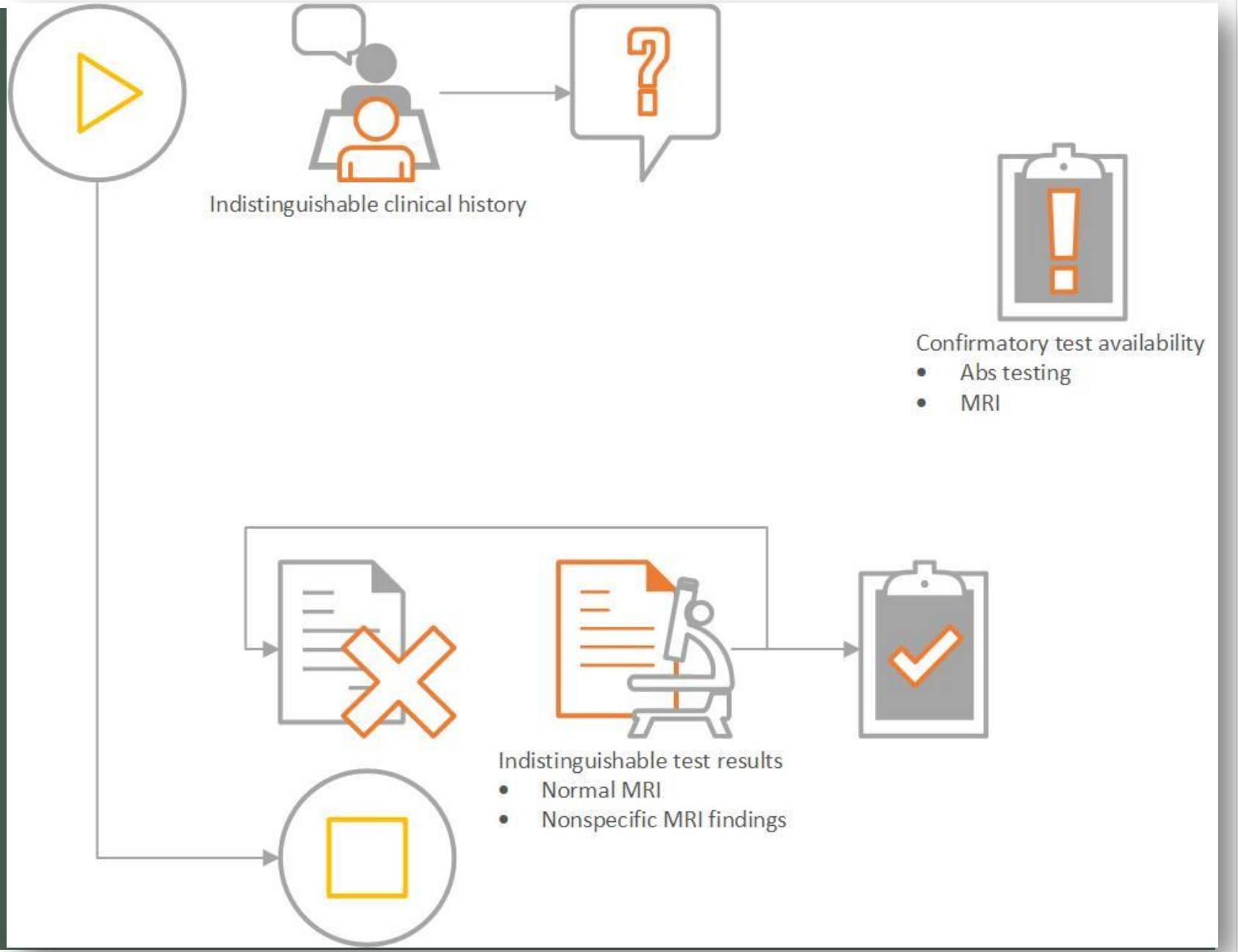
# Why is the EEG a promising tool in autoimmune encephalitis ?



# Talk overview

- Clinical clues
- Reported EEG features in specific autoimmune encephalitis
- EEG features distinguishing autoimmune encephalitis from metabolic encephalopathy

# CLINICAL CLUES

# Clinical features suggestive of autoimmune seizures/epilepsy

| | Most likely antibodies |
|---|---|
| **Typical syndromes** | |
| - **Limbic encephalitis, in part resulting in hippocampal sclerosis** | LGI1, GAD65 |
| - **Faciobrachial dystonic seizures** | LGI1 |
| - **Encephalopathy with seizures, that is, seizures in the context of multifocal, even multi-level involvement of the CNS, manifests as rapidly evolving cognitive impairment, partly with additional myoclonus, neuropsychiatric features, or focal neurological deficits** | NMDAR, LGI1 |
| **Demographic features, personal or family history** | |
| - **New-onset epilepsy in young females** | NMDAR |
| - **Early AED resistance** | Several |
| - **Personal or family history of autoimmunity** | Several |

# Clinical features suggestive of autoimmune seizures/epilepsy

| | Most likely antibodies |
|---|---|
| **Seizure types or patterns** | |
| - Recurrent temporal lobe seizures with onset in adult life without obvious reason for an acquired cause | LGI1, GAD65 |
| - New-onset status epilepticus or extraordinary high seizure frequency from beginning | Several |
| - Multifocal seizures from beginning | |
| - Pilomotor seizures | NMDAR<br>VGKC complex (probably LGI1) |
| **Paraclinical findings** | |
| - MRI: encephalitic lesions | Several (except NMDAR) |
| - EEG pattern "Extreme delta brush" | NMDAR |
| - CSF: elevated cell count, unmatched oligoclonal bands | Several (LGI1 antibodies often detected without CSF abnormalities) |
| - Histopathology : "chronic encephalitis" | Several |

Bien CG; Epilepsia 2013

# REPORTED EEG FEATURES IN SPECIFIC AUTOIMMUNE ENCEPHALITIS

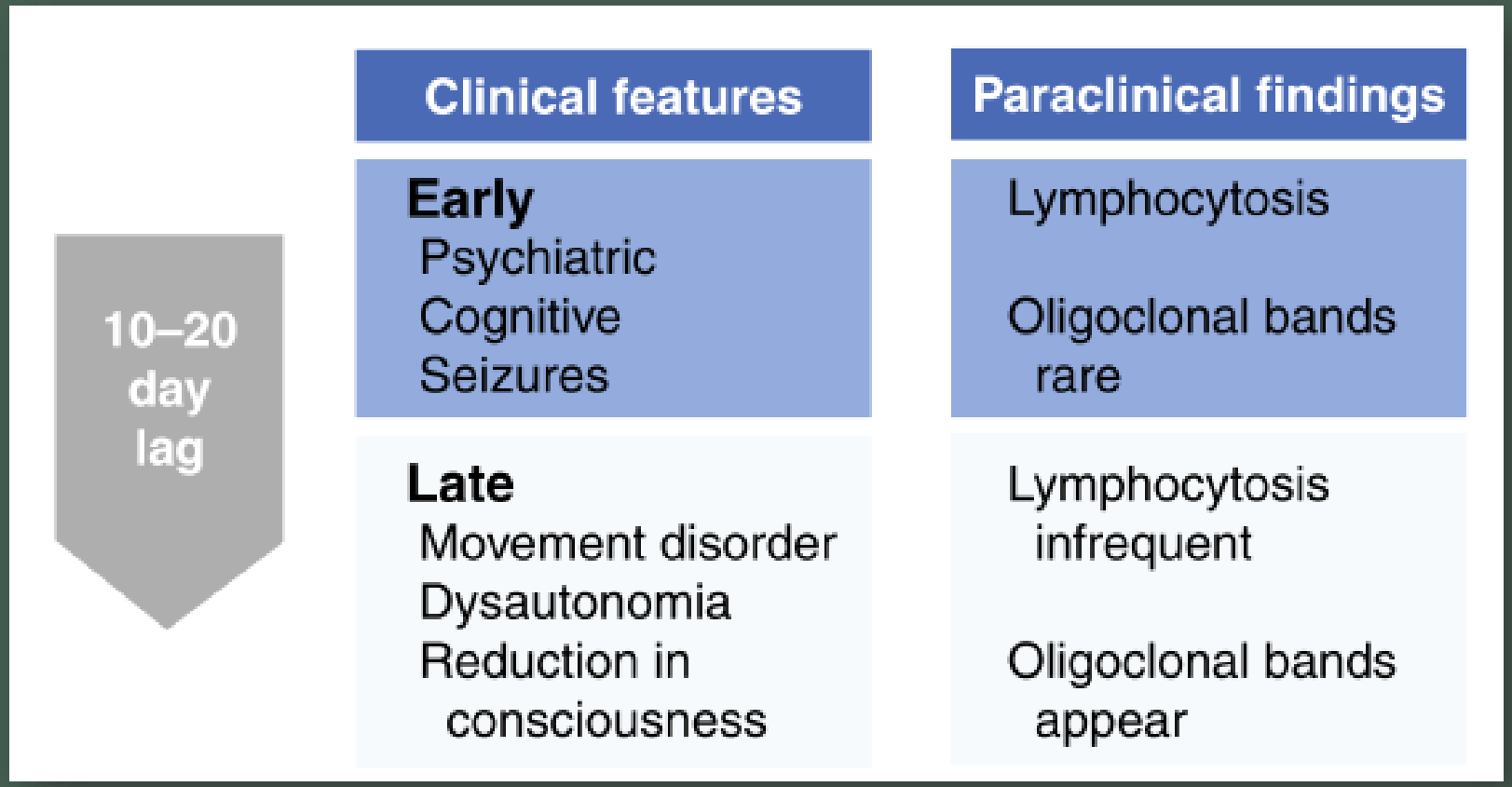
How can the EEG findings be distinguishable the autoimmune encephalitis from metabolic encephalopathy/ other common structural abnormalities?

Specific region of the brain involved

Specific pathophysiology creates unique EEG waves

Epileptogenic level

# Anti-NMDA encephalitis




*Irani SR and Vincent A; Curr Neurol Neurosci Rep 2011*

# Case

**14 yo girl**

**CC:** Acute afebrile seizures, 3 wks before admission

**PI:** - 3 wks PTA "weird feeling" without LOA

- 1-2 wks PTA, developed GTCs with frequent staring episodes

**"Staring episode"**

- lasted 10-30 seconds
- non-verbal
- partially responsive towards the end
- resumed her baseline immediately

# Case (cont.)

1 wk PTA admitted in outside hospital (LOS 5 days)

**MRI: unremarkable**

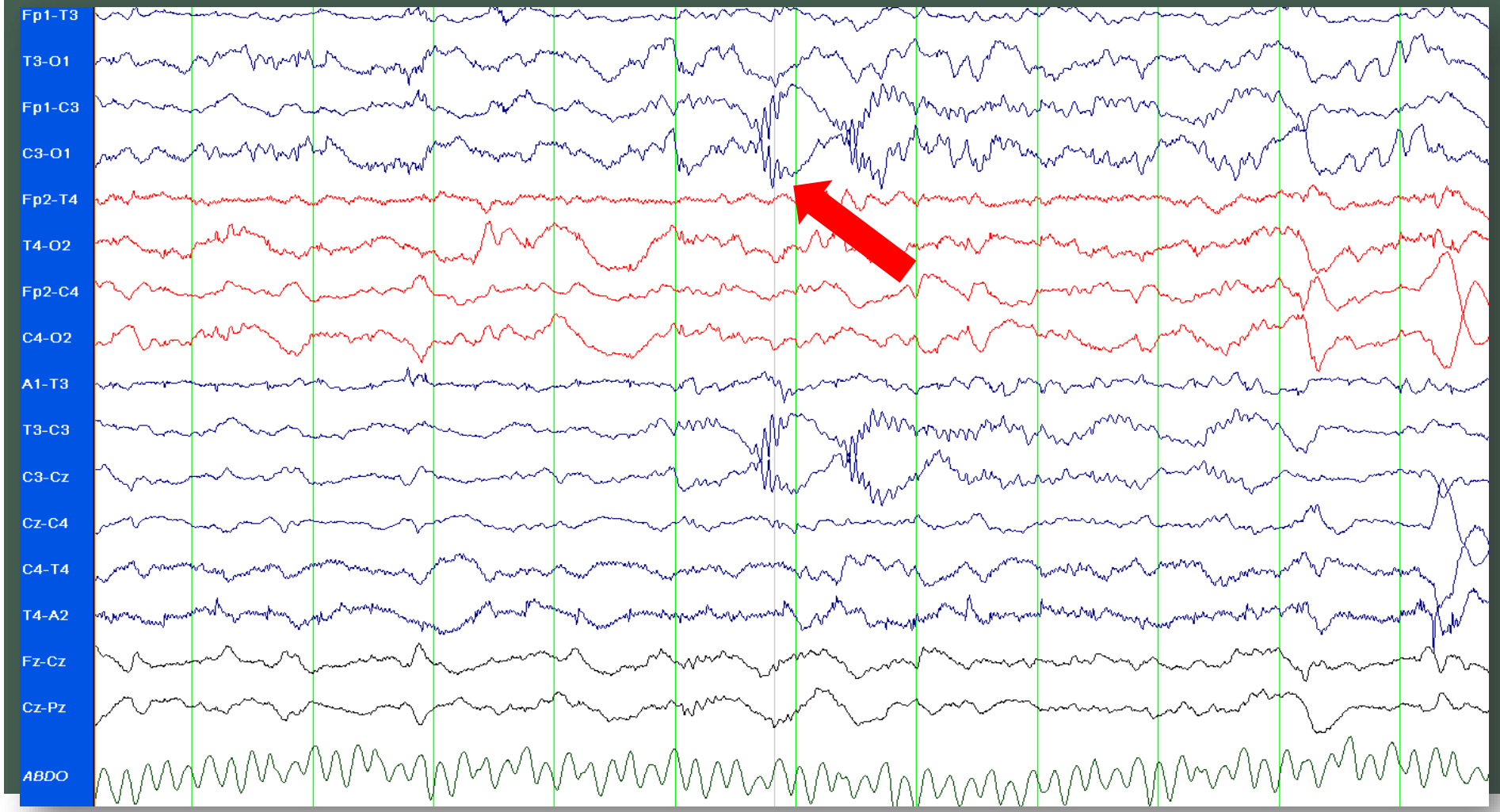
**CSF: WBC 2, RBC 567, glucose 3.8** (2.8-4.4 mmol/L)

**protein 0.65** (0.18-0.4 g/L)(mild elevation)

**HSV-1, HSV-2 PCR DNA: negative**

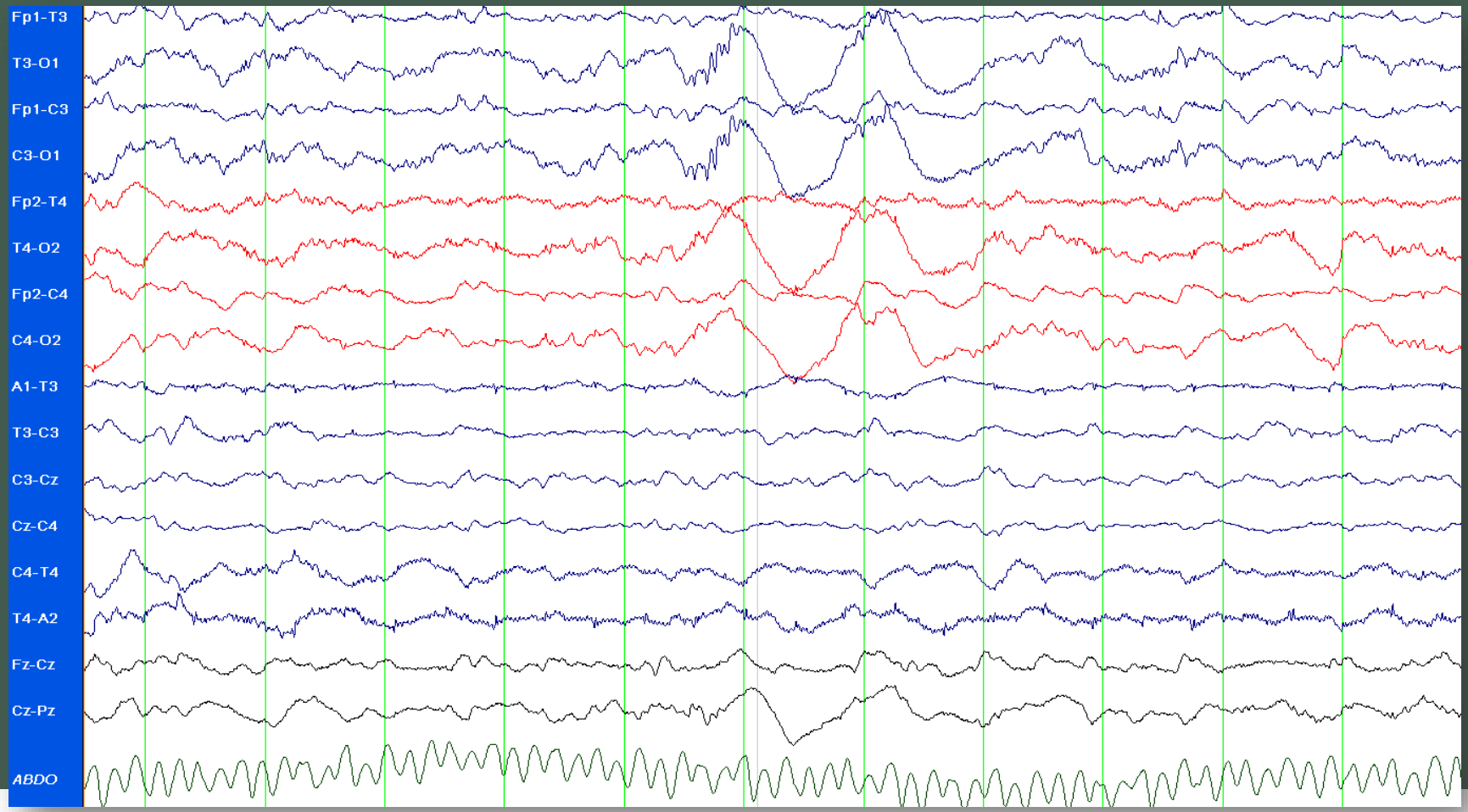
- Frequent episodes of combative and aggressive behaviors (paranoid delusion)
- D/C with LVT 500 mg BID, without staring episodes

# Central delta brushes in quiet sleep in neonates (delta brush CA 26-38 weeks)

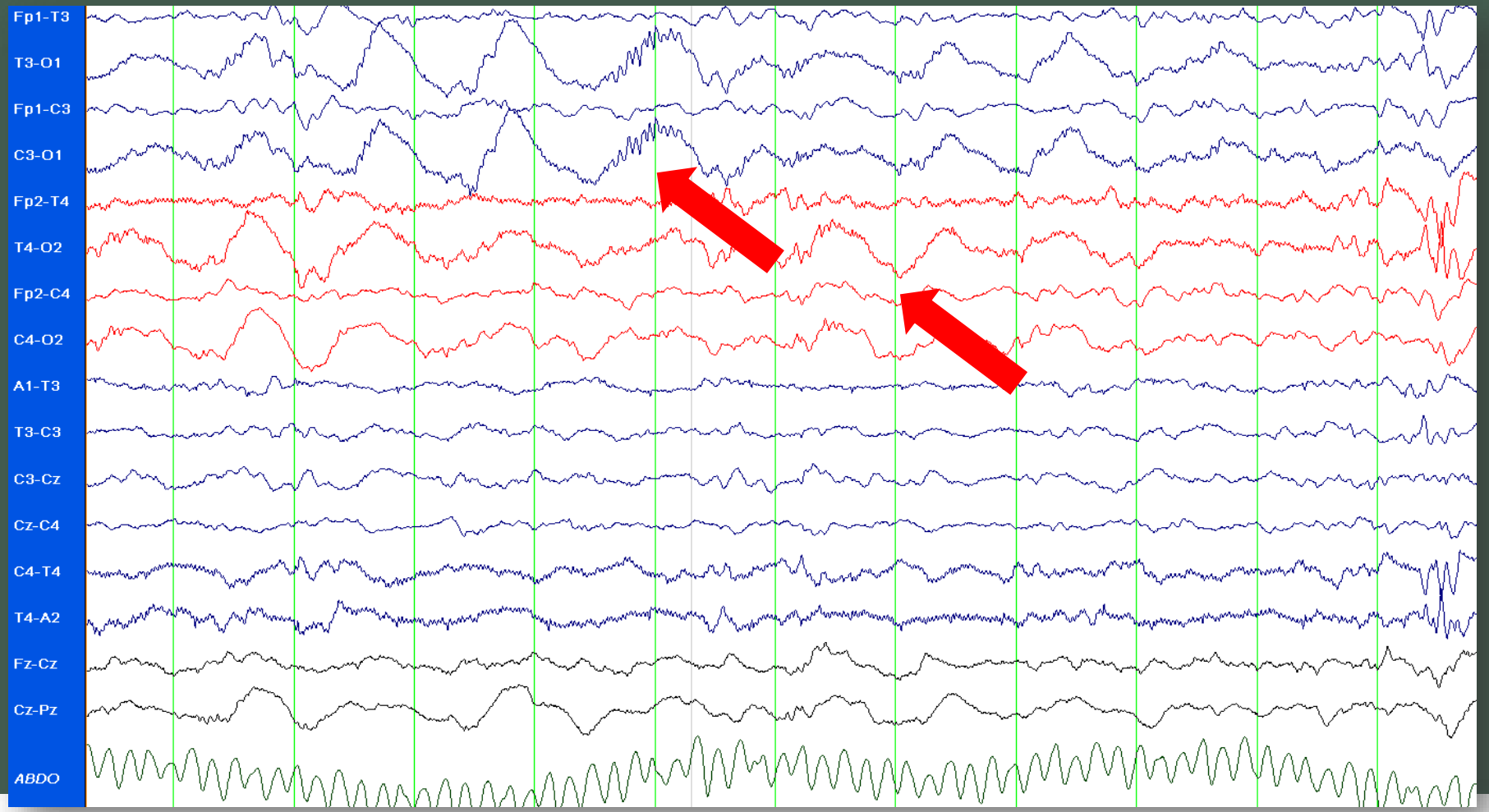



Beta component:
8-12 Hz and
18-22 Hz
(more common)

# Occipital delta brushes in QS (after 30 weeks, occipito-temporal > central)



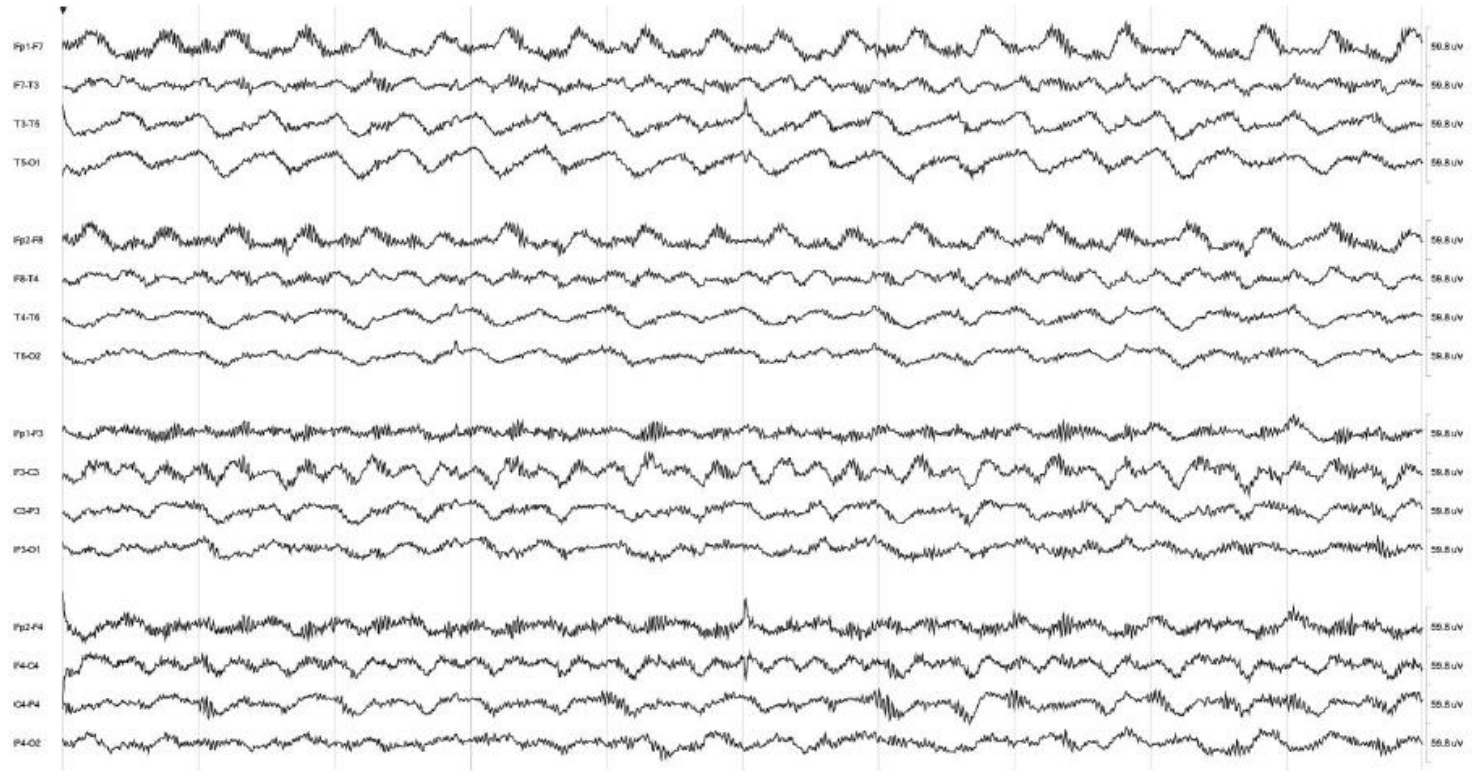
# Occipital delta brushes in QS, asynchronous and asymmetric (normal)



# “Extreme delta brush”

**Figure 2** Continuous EEG recording in a 19-year-old man with anti-NMDA receptor encephalitis associated with dyskinesias, seizures, and coma

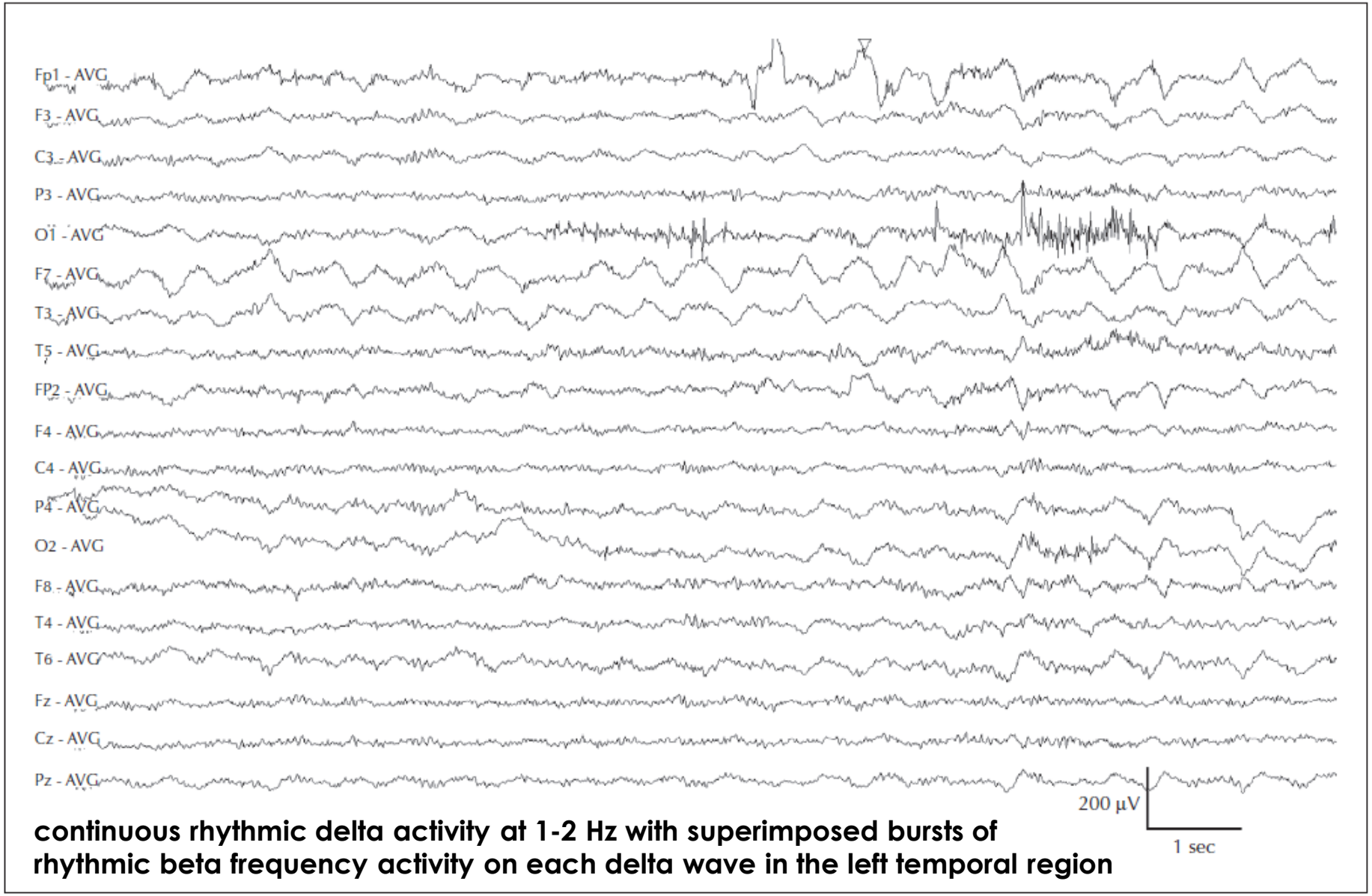
***Schmitt SE et.al., Neurology 2012***



The initial EEG demonstrates generalized rhythmic delta frequency activity at 2–2.5 Hz with superimposed rhythmic beta frequency activity.

- ✓ **7/23 patients (30%)**
- ✓ Rhythmic **delta activity at 1-3 Hz** with superimposed bursts of **rhythmic 20–30 Hz beta frequency** activity “riding” on each delta wave
- ✓ EDB pattern may be a marker of **more severe disease and perhaps worse outcome**

(not confirmed by subsequent studies by Li H Zhonghua Er Ke Za Zhi 2016 and Zhang 2017)




**continuous rhythmic delta activity at 1-2 Hz with superimposed bursts of rhythmic beta frequency activity on each delta wave in the left temporal region**

**Figure 1.** Electroencephalogram with interictal rhythmic continued delta activity with superimposed beta activity riding on each delta wave. Low and high frequency filters were set at 1 and 70, respectively.

*Capua DD et.al., Epileptic Disord 2013*

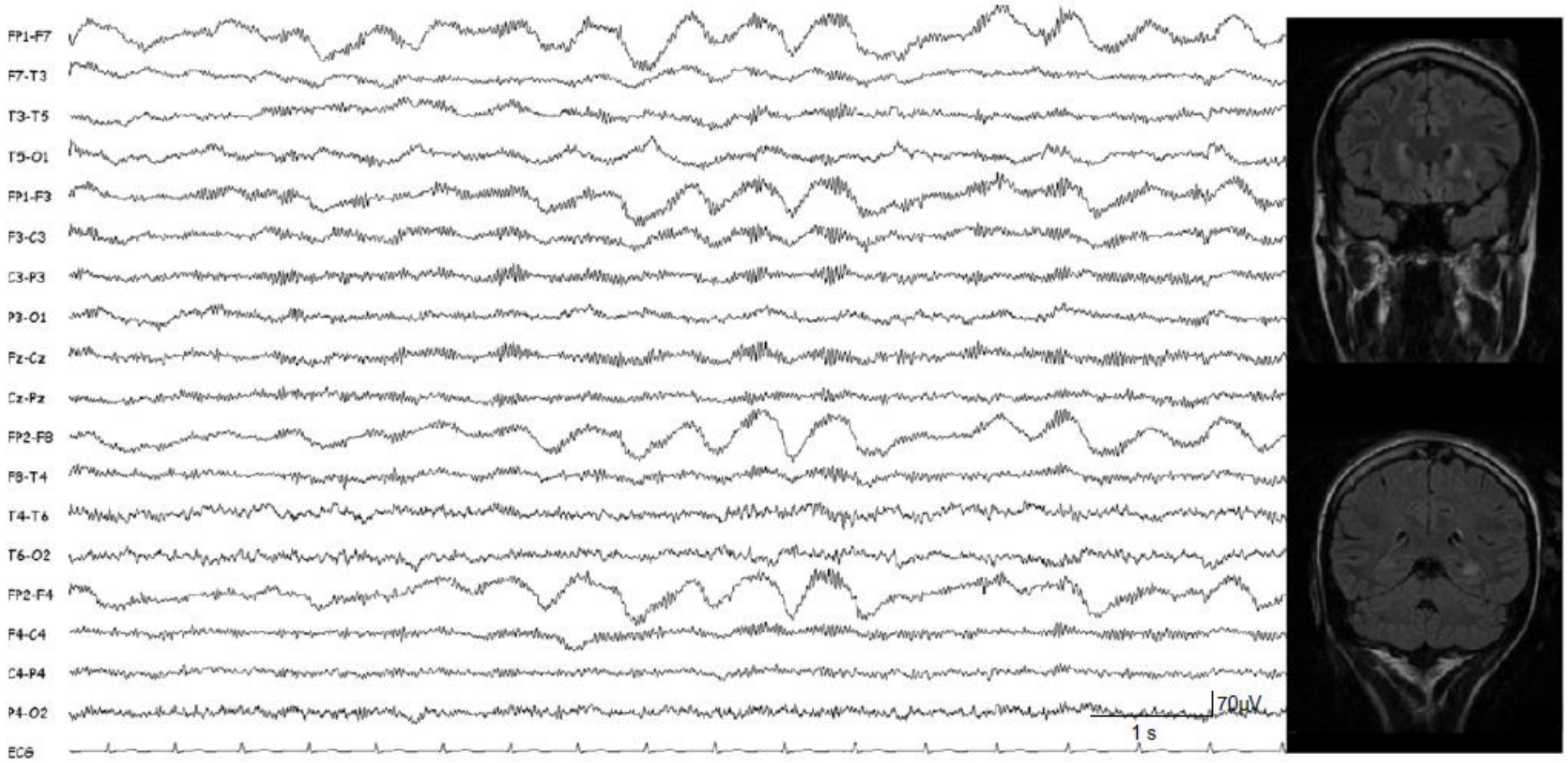



**Fig. 2.** Extreme delta brush. EEG of the same patient as in Figs. 3 and 4 the day after the EDB pattern without electroencephalographic seizures appeared. MRI showed left hippocampal hyperintensity and left frontosubcortical hyperintensity. High pass filter: 0.5 Hz; low pass filter: 70 Hz.

*Veciana M et.al., Epilepsy & Behav 2015*

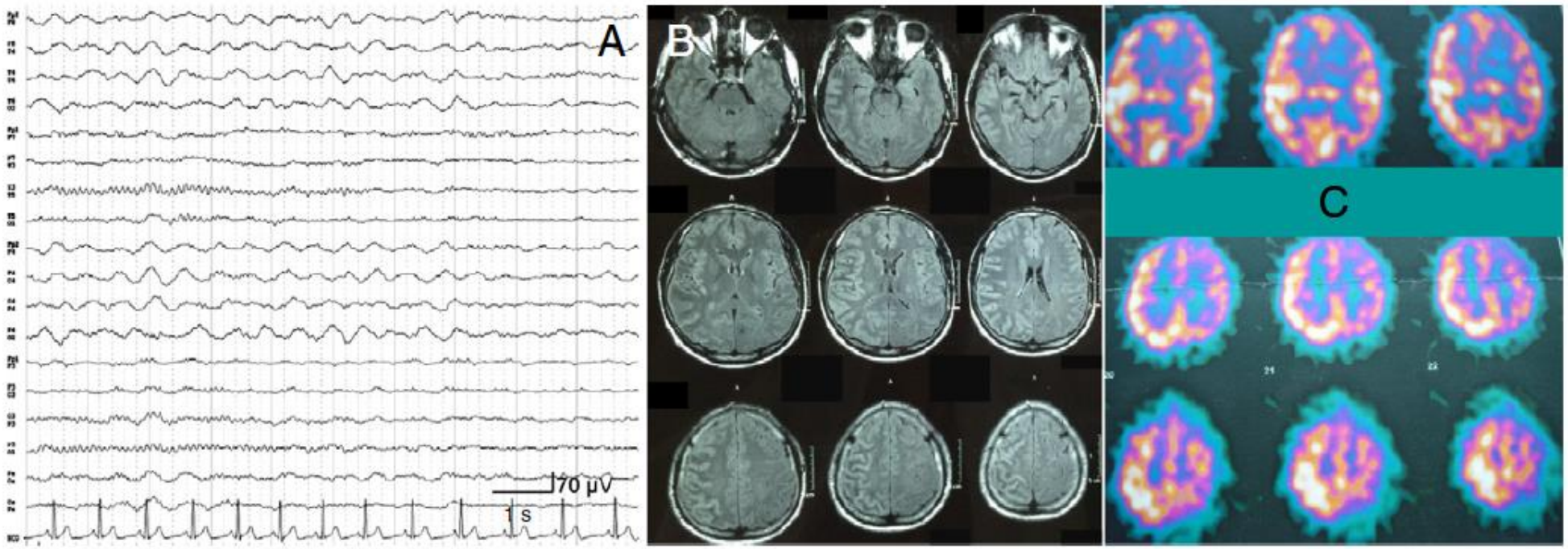
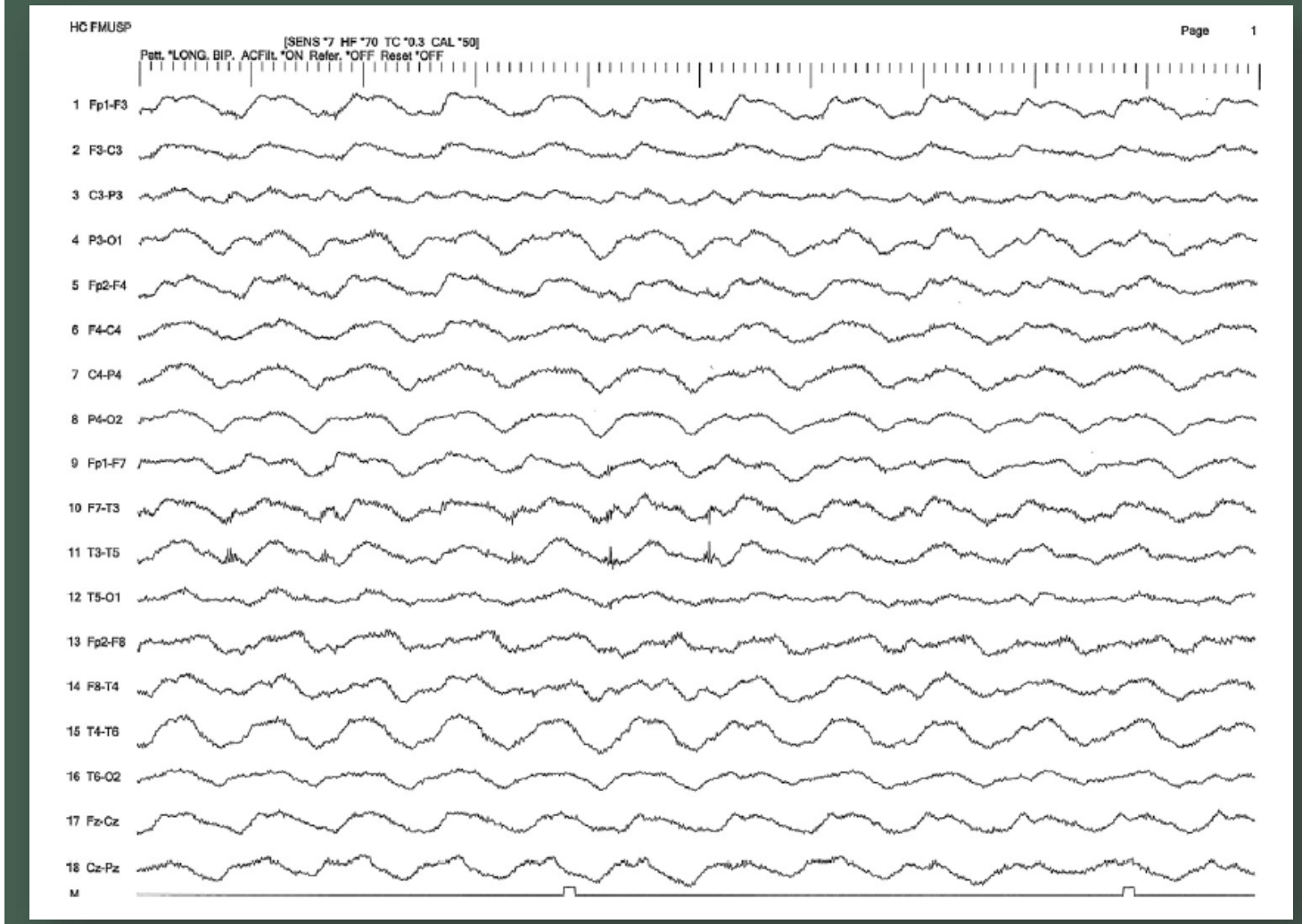



**Fig. 1.** Neuroradiological findings and EEG correlations. EEG of a 36-year-old man showing nearly continuous right hemisphere delta slowing. He had headache, left hemiparesis, and speech disturbances. MRI showed extensive right hemisphere cortical hyperintensities and SPECT revealed right hemispheric hyperperfusion. High pass filter: 0.5 Hz; low pass filter: 70 Hz.

*Veciana M et.al., Epilepsy & Behav 2015*

# Serial and prolonged EEG recording in anti-NMDA encephalitis




**Generalized rhythmic delta activity (GRDA)**

- Delta frequency ranged from 1 to 4 Hz
- GRDA could occur continuously on 30-min recordings, but not on continuous EEG monitoring, comprising **about 20% of the 24 h EEG recording**

*Schmitt SE et.al., Neurology 2012*

**Table 1** Summary of cEEG findings

| EEG findings | No. | % |
|---|---|---|
| Normal EEG | 2 | 8.7 |
| Mild polymorphic diffuse slowing | 2 | 8.7 |
| Moderate polymorphic diffuse slowing | 10 | 43.5 |
| Severe polymorphic diffuse slowing | 9 | 39.1 |
| Focal slowing | 8 | 34.8 |
| Hemispheric | 5 | 21.7 |
| Frontal | 6 | 26.1 |
| Central | 4 | 17.4 |
| Temporal | 6 | 26.1 |
| Parietal | 4 | 17.4 |
| Occipital | 4 | 17.4 |
| Other | 4 | 17.4 |
| Generalized rhythmic delta frequency activity without extreme delta brush | 4 | 17.4 |
| Excess beta frequency activity without extreme delta brush | 5 | 21.7 |
| Electrographic seizures | 14 | 60.1 |
| Left | 0 | 0 |
| Right | 6 | 42.9 |
| Bilateral/generalized | 4 | 28.6 |
| Unknown | 4 | 28.6 |
| Electrographic seizures without clinical correlate | 9 | 39.1 |
| Clinical seizures | 14 | 60.9 |
| Extreme delta brush | 7 | 30.4 |

Diffuse slowing (mild, moderate, severe) (91%)

GRDA (17%)

Excessive beta activity (22%)

Extreme delta brush (30%)

# Significance of EEG findings in anti-NMDA encephalitis

- **62 cases** of anti-NMDAR encephalitis patients
- **At the peak stage**: Most common EEG findings: **Diffuse slowing (40.3%)**
  - ❖ **extreme delta brush (EDB) (10 cases, 16.1%)**
- Normal background, epileptiform discharges, polymorphic delta rhythm, and diffuse beta activities at the peak stage might suggest favorable long-term prognosis

**Table 2**
The EEG presentations in different clinical stages of anti-NMDAR encephalitis patients.

*Zhang Y et.al., Clin Neurophysiol 2017*

| EEG | Clinical stage | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Initial stage | | Peak stage | | Improvement stage | | Recovery stage | |
| | $n = 10$ | Percentage | $n = 62$ | Percentage | $n = 13$ | Percentage | $n = 4$ | Percentage |
| Normal | 2 | 20.0 | 1 | 1.6 | 1 | 7.7 | 2 | 50.0 |
| Epileptiform discharges | 2 | 20.0 | 11 | 17.7 | 3 | 23.1 | 0 | 0 |
| Diffuse slowing | 4 | 40.0 | 25 | 40.3 | 7 | 53.8 | 0 | 0 |
| Focal slowing | 1 | 10.0 | 5 | 8.1 | 0 | 0 | 1 | 25.0 |
| Polymorphic delta rhythm | 0 | 0 | 6 | 9.7 | 0 | 0 | 0 | 0 |
| Diffuse beta activities | 0 | 0 | 4 | 6.5 | 0 | 0 | 1 | 25.0 |
| EDB | 1 | 10.0 | 10 | 16.1 | 2 | 15.4 | 0 | 0 |

# Pathophysiology of delta bursting during wakefulness

- **Delta oscillations** normally occur throughout the cortex during **slow-wave sleep**
- Their occurrence in the **awake state** in frontal and temporal regions: producing **cognitive abnormalities associated with schizophrenia**
- **GRDA in anti-NMDAR encephalitis** may represent the effect of the antibody against the NMDAR, leading to **reduced NMDA function**
- **NMDAR blockage in the nucleus reticularis (nRT)** of the thalamus switches neuronal cell firing mode from tonic to rhythmic bursting mode, in the **delta frequency range**

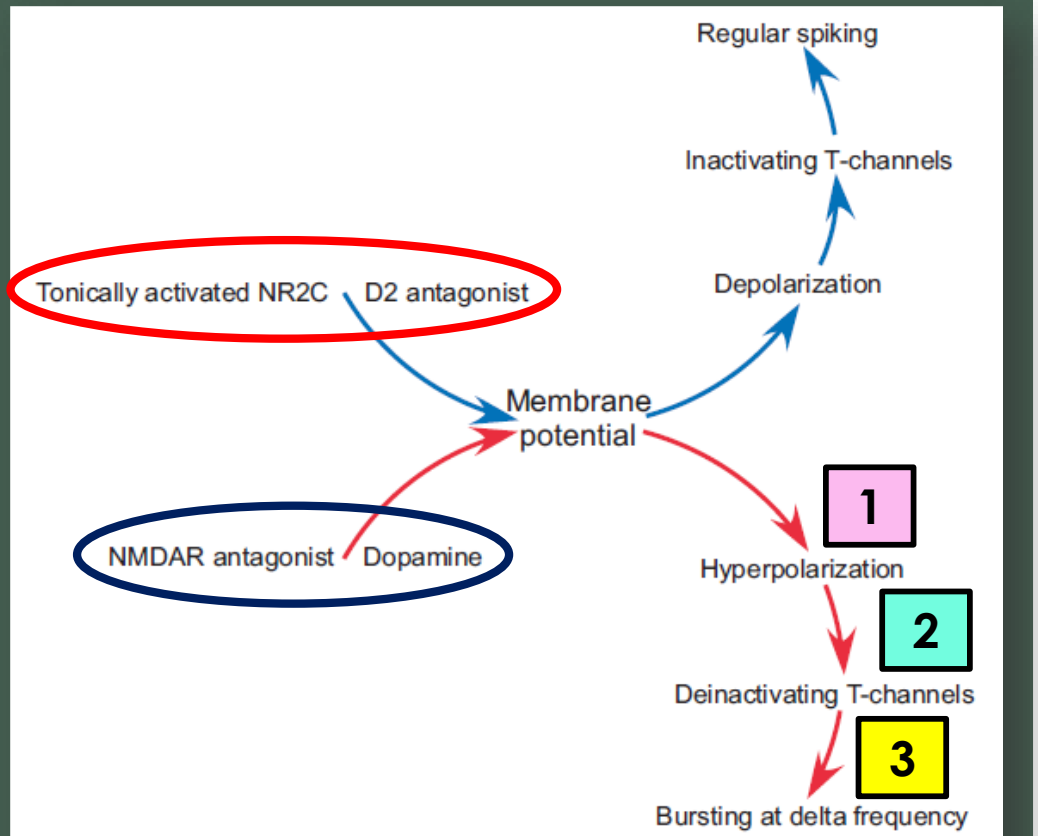



**FIGURE 7 | A diagram summarizing the role of NMDAR and dopamine in intrinsic conductance and firing pattern of nRT neurons.**

*Zhang Y et.al., Front Neural Circuits 2009*

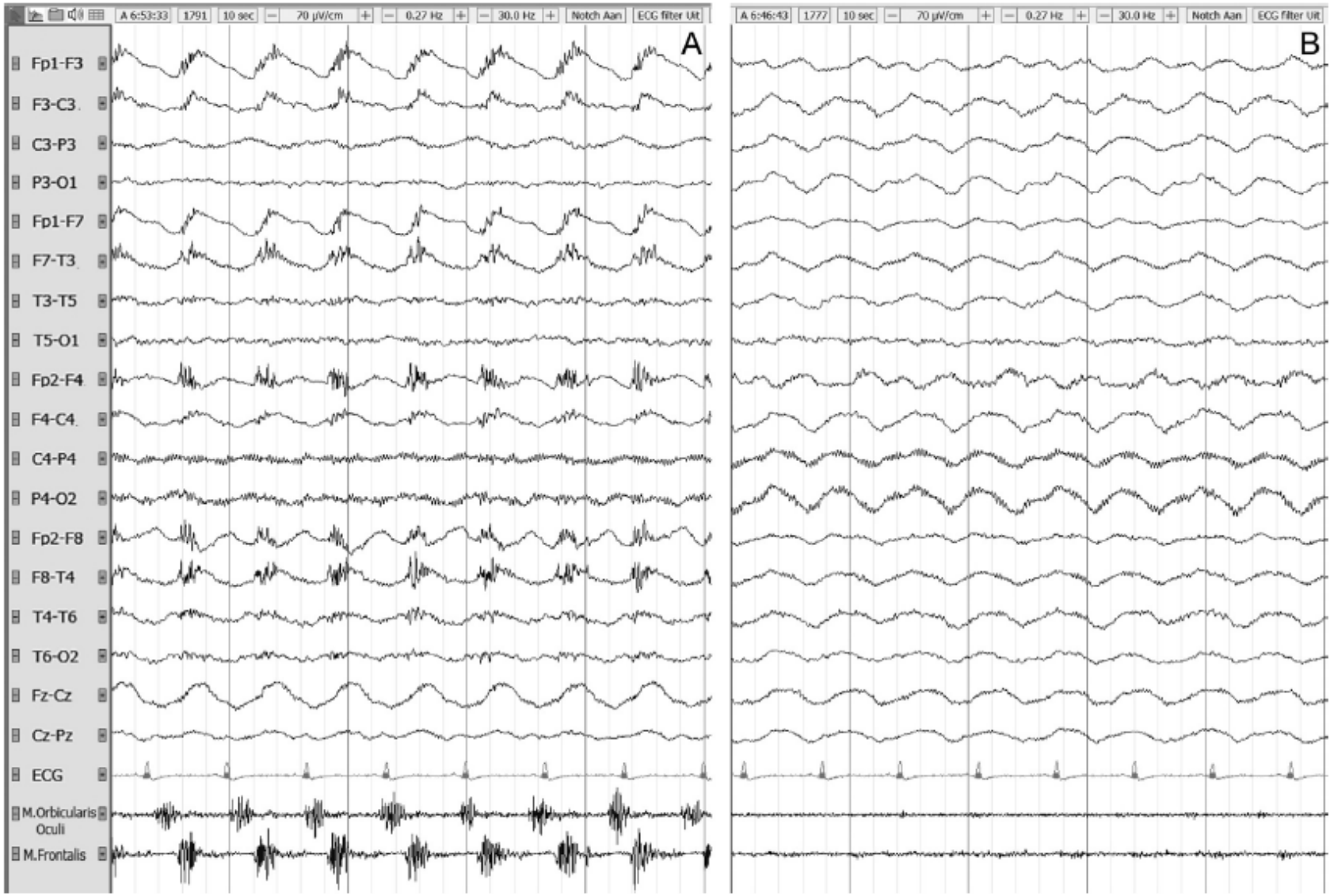
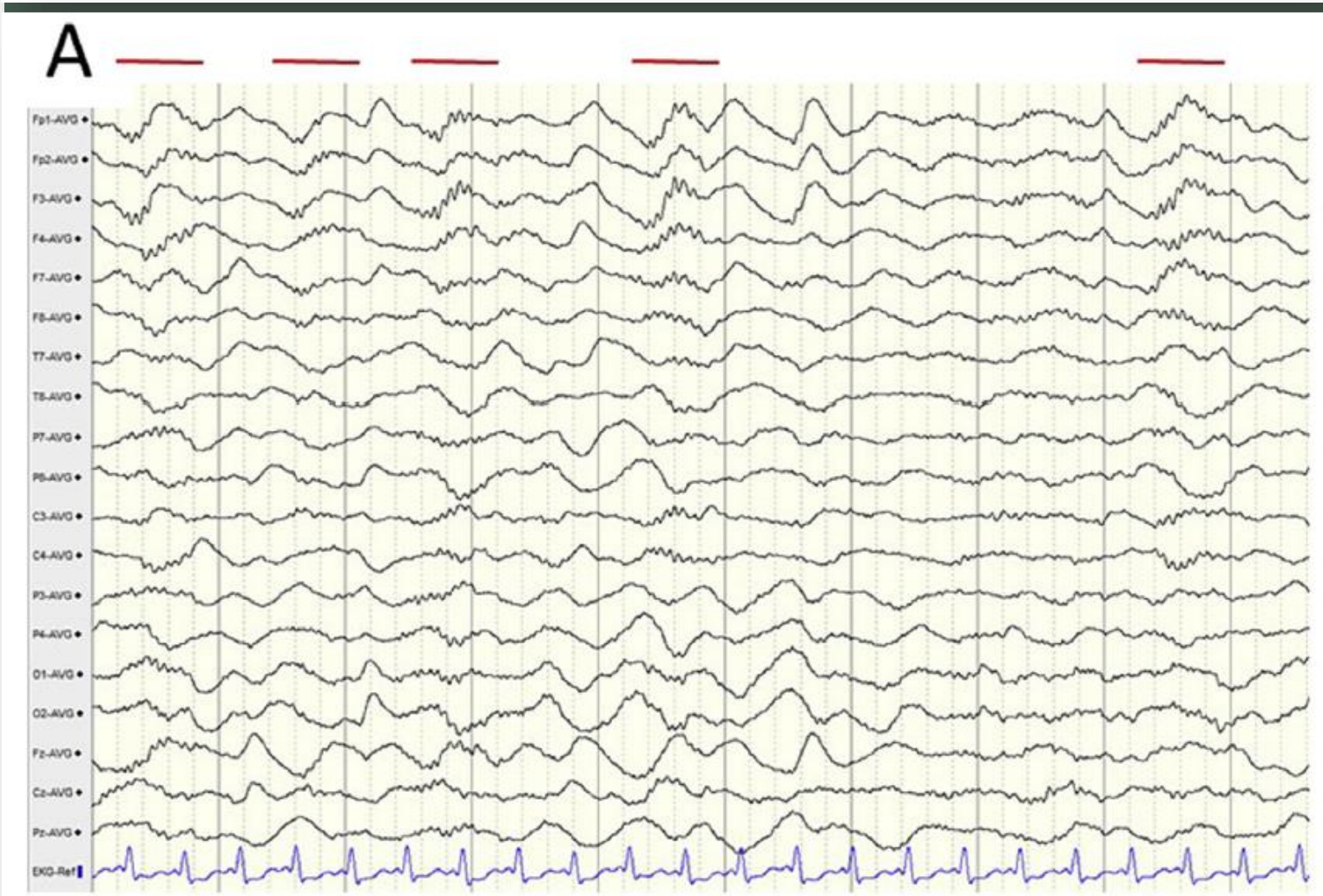



**Fig. 1.** EEG with video and orbicularis oculi and frontalis muscle EMG coregistration during a five second episode of orofacial dyskinesias showing a delta brush pattern (A). The M.frontalis EMG channel was derived from the frontopolar EEG electrodes. EMG activity of the orbicularis oculi and facial videorecording including the frontalis muscles was rhythmic with the same frequency, with contraction of these muscles that were mostly asynchronous and in anti-phase, and of which the frontalis muscle activity coincided with the brush component of the delta brushes. During a five second episode without EMG activity, the delta brushes are absent (B).

The ‘‘brush’’ may alternatively represent an **EMG artifact due to rhythmic contractions of the frontalis muscle,** occurring in synchrony with frontal delta activity

*Van der Meulen AAE et.al., Clin Neurophysiol 2017*

# EEG with extreme delta brush in young female with methotrexate neurotoxicity supports NMDA receptor involvement




Frontal 1-2 Hz activity with overlying 13-16 Hz activity

**MTX interferes with potentially neurotoxic amino acid and neurotransmitter pathways causing accumulation of homocysteine and its metabolites with strong excitatory effect on NMDA receptors**

*Schmidt LS et.al., Eur J Ped Neurol 2017*

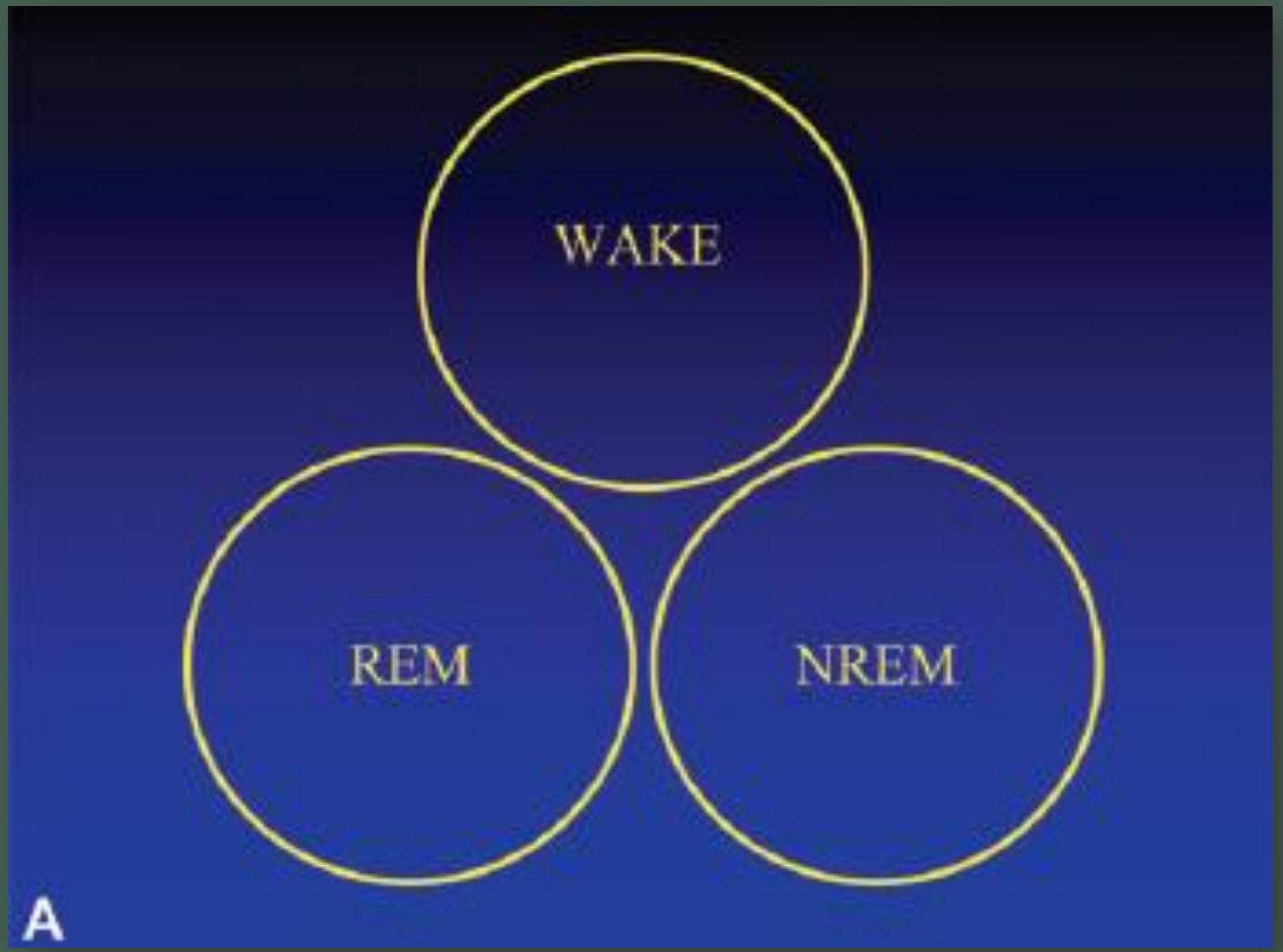
“State dissociation”
“Status dissociatus”

**Status dissociatus**:

The extreme expression of states dissociation is characterized by the asynchronous occurrence of the various components of the different states that prevents the recognition of any state of being.

*Antelmi E et.al;; Sleep Med Rev 2016*

# State boundary



| State dissociation | Conditions |
| --- | --- |
| **Dissociation from prevailing wakefulness** | - hypnagogic or hypnopompic hallucinations<br>- automatic behaviors<br>- sleep drunkenness<br>- cataplexy sleep paralysis |
| **Dissociation from REM sleep** | - RBD<br>- lucid dreaming |
| **Dissociation from NREM sleep** | - disorders of arousal |

*Antelmi E et.al;; Sleep Med Rev 2016*

# Status dissociatus

**Occurs only at night time or intermittently**

- Autoimmune encephalopathies
- Narcolepsy type 1
- Anti-IgLON5 parasomnia

**Occurs nearly continuously with complete loss of any conventionally defined state of being, and of the circadian pattern**

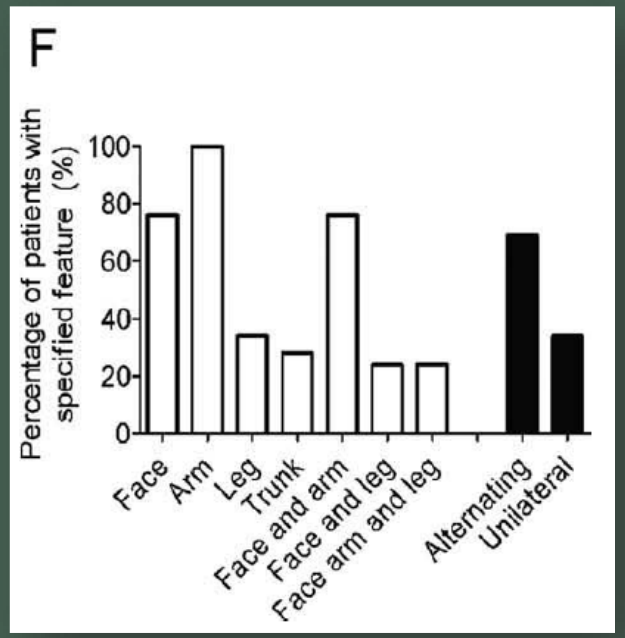
- Agrypnia excitata
  - ✓**Fatal familial insomnia** and other prion diseases
  - ✓**Autoimmune encephalopathies (anti-LGI1, anti-Ma2)**
  - ✓Brain lesions (hypothalamus, thalamus, brainstem)
  - ✓Neurodegenerative diseases (PD, DLB)
  - ✓Rare congenital, probably genetic conditions: Mulvihill-Smith syndrome
  - ✓Misc: GBS, narcolepsy, alcohol/drug abuse/withdrawal

*Antelmi E et.al;; Sleep Med Rev 2016*

# LGI1-antibody–mediated encephalitis

- **Limbic encephalitis** and tonic seizures involving the face, arm, and/or leg, which have been called "faciobrachial dystonic seizures (FBDS)"

- "**Faciobrachial dystonic seizures**" (77%) precede LGI1 antibody limbic encephalitis (amnesia, confusion)




Very brief (< 3 s)
Very frequent (median of 50/ day)

- ✓ No cognitive impairment
- ✓ Normal sodium level and normal MRI

*Irani SR et.al., Ann Neurol 2011*

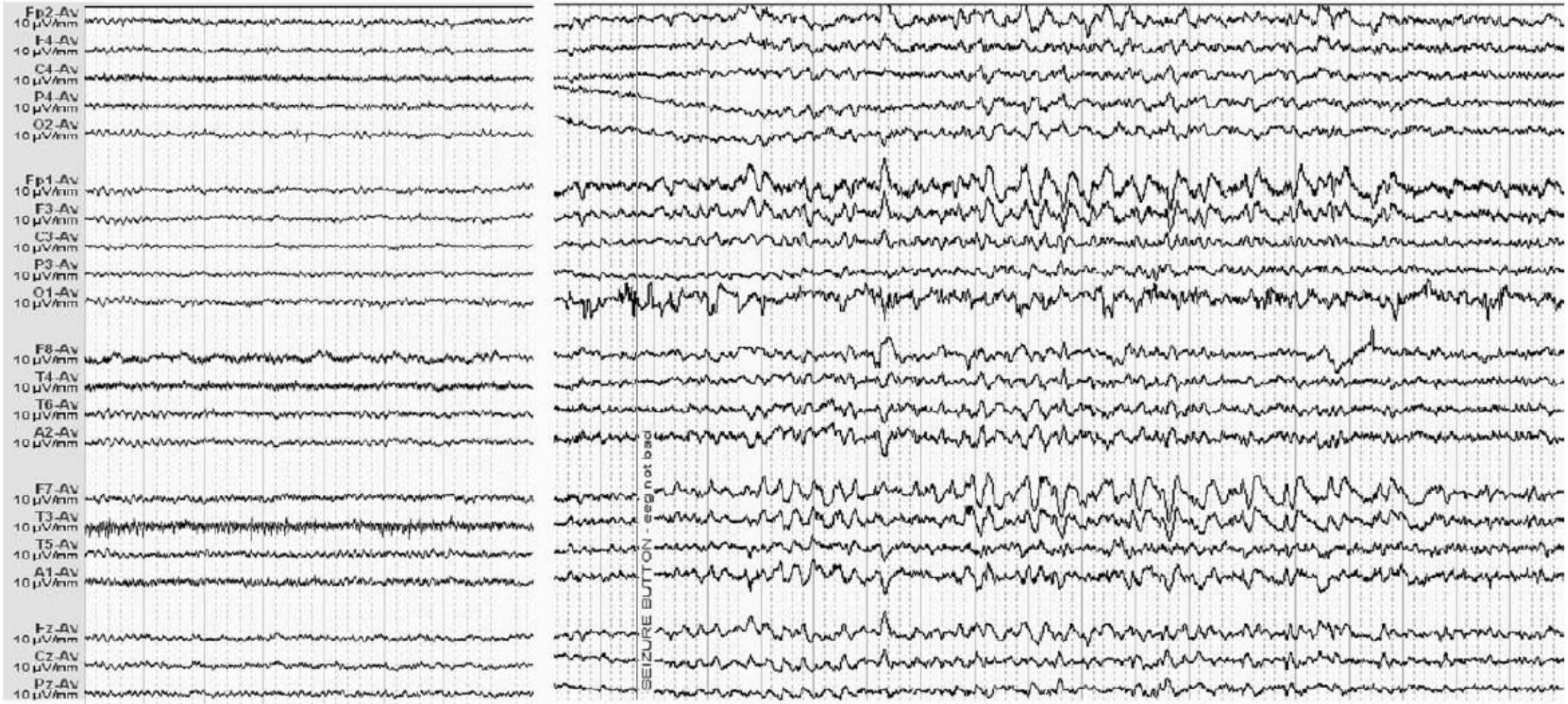
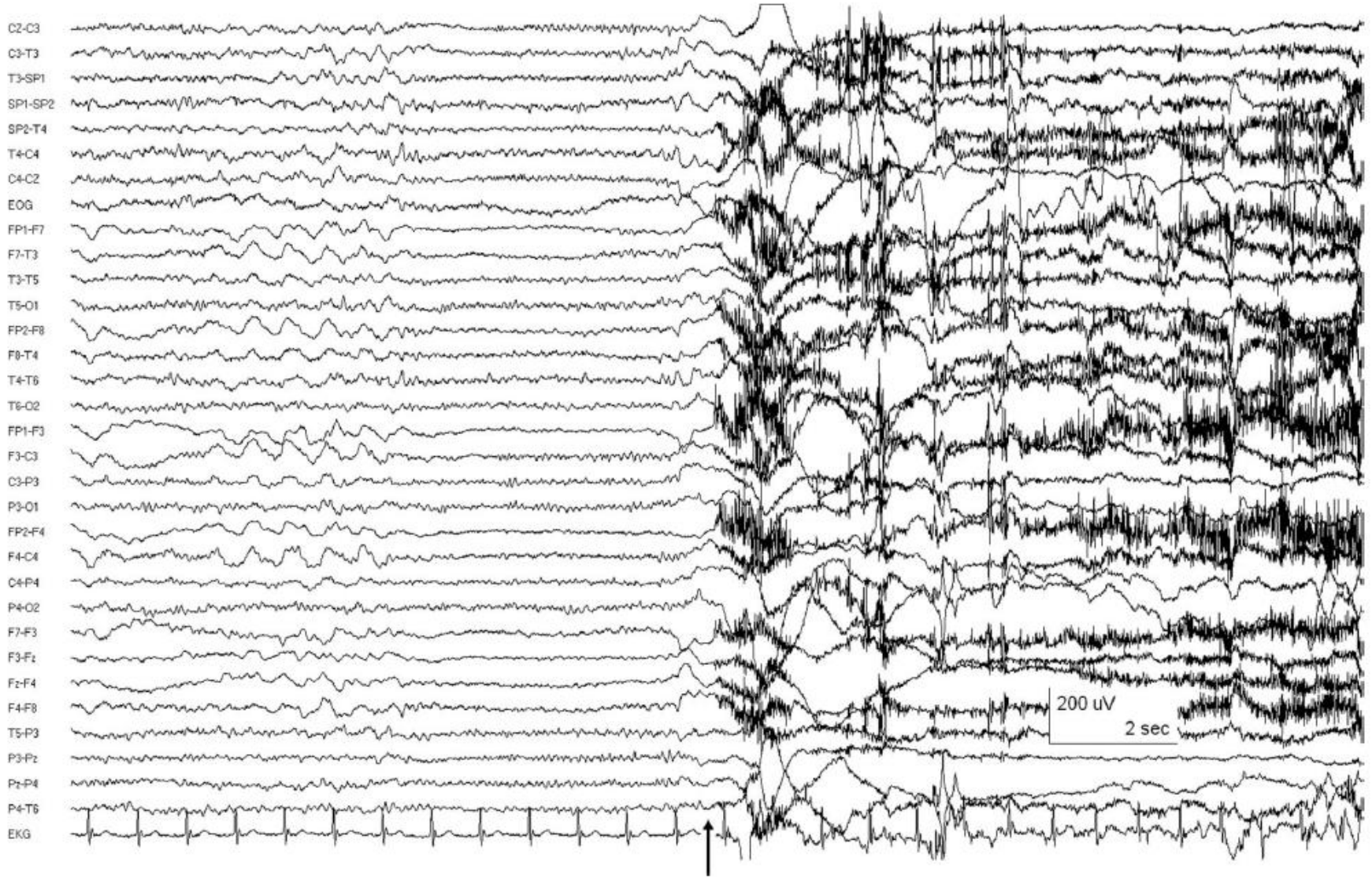


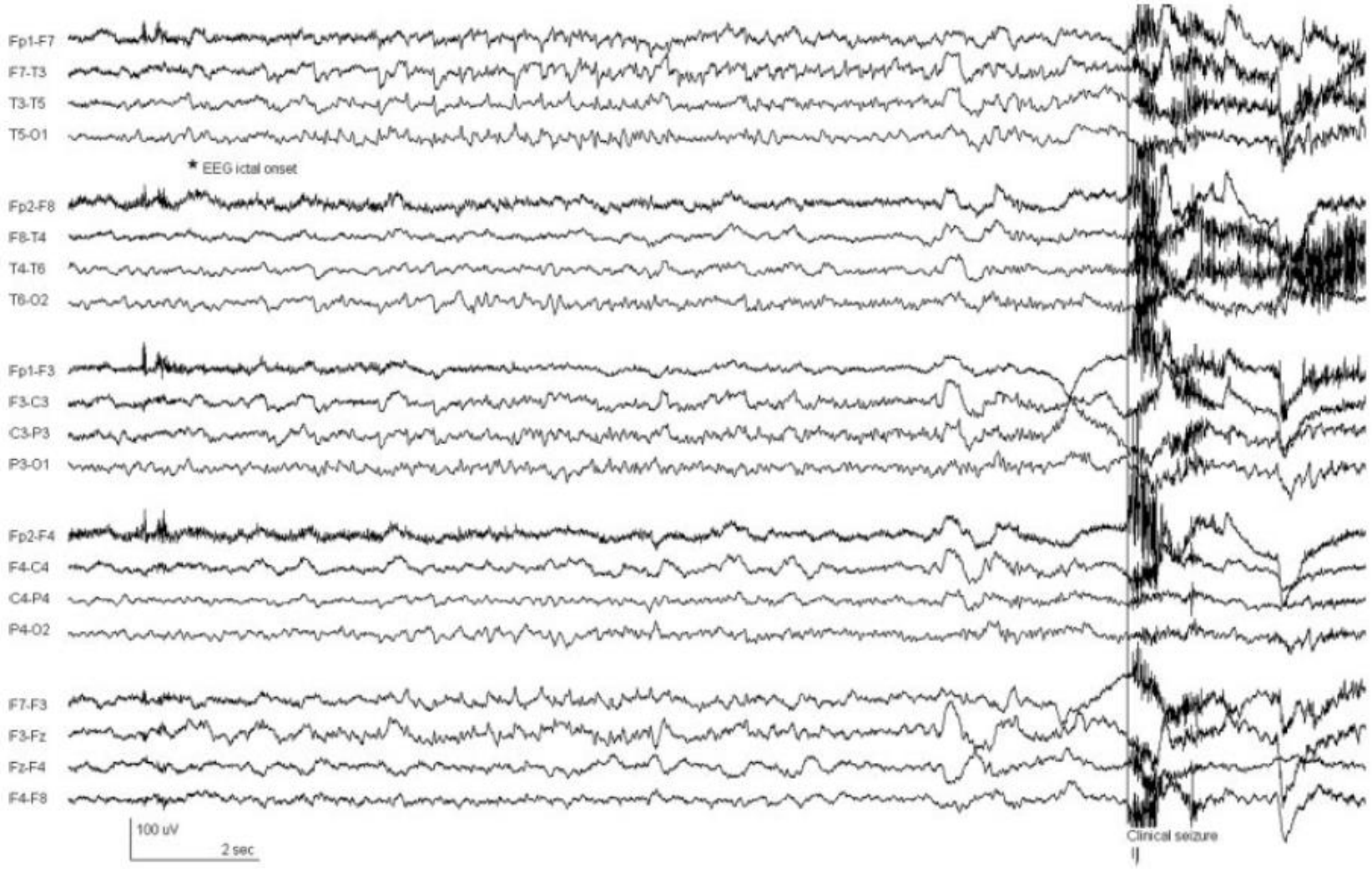

FIGURE 2: Electroencephalography. (A) Interictal and (B) ictal electroencephalograms (EEGs) were recorded during a faciobrachial dystonic seizure in a 39-year-old woman. On this occasion, ictal 2 to 4Hz spike-wave activity was noted of maximal amplitude over the left frontotemporal region. The interictal EEG was normal. Ictal epileptiform activity was seen in 7 of 29 cases. Bar = 1 second.

# TONIC SEIZURES: A DIAGNOSTIC CLUE OF ANTI-LGI1 ENCEPHALITIS?



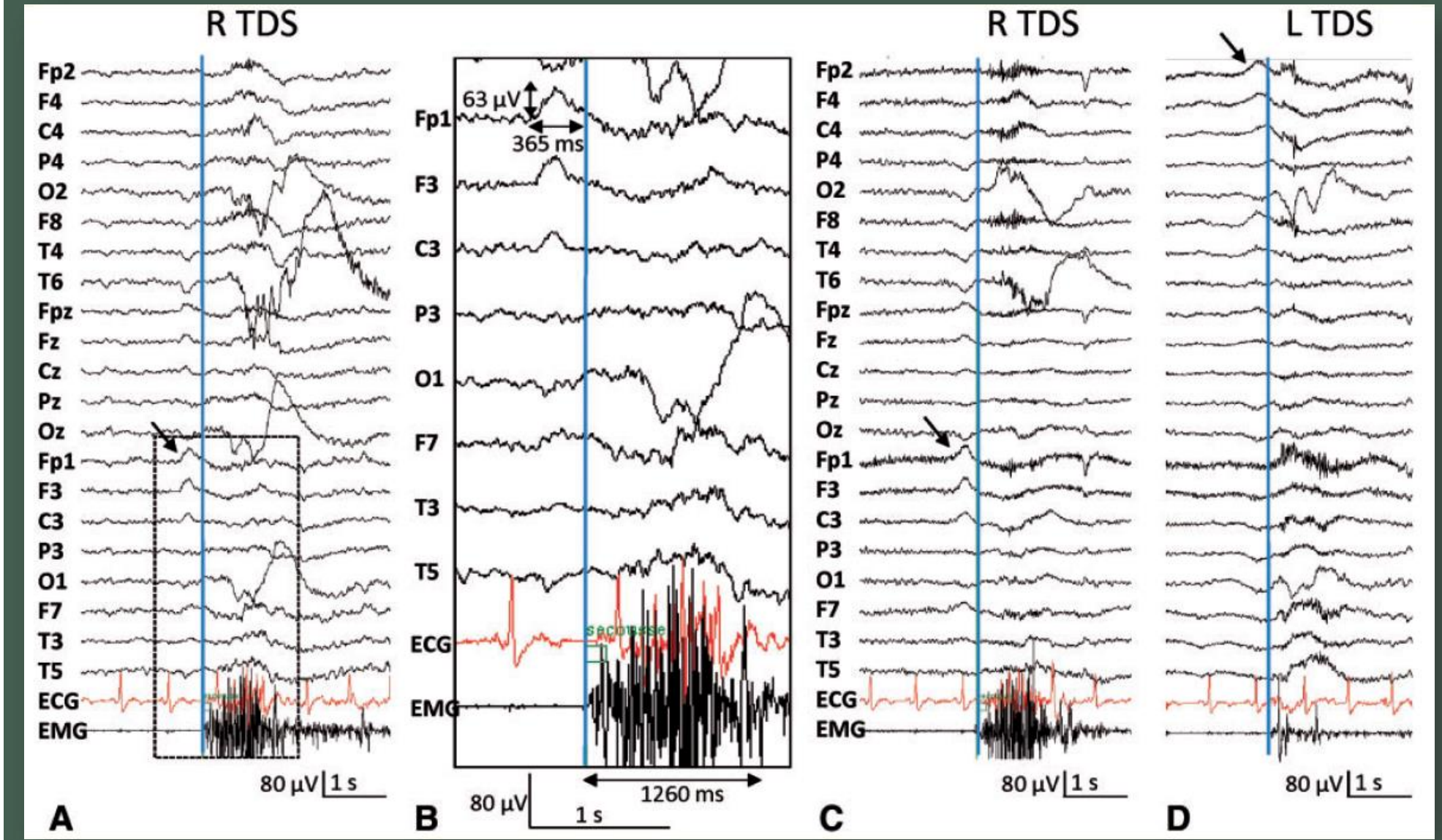

*Andrade DM et.al., Neurology 2011*

**Figure 2** EEG showing a subclinical left lateral temporal electrographic seizure (onset at *) and a later clinical tonic seizure (vertical line)




**In a small minority of seizures, a tonic seizure occurred shortly after the onset of a lateralized subclinical electrographic seizure**

*Andrade DM et.al., Neurology 2011*




A focal slow wave on frontal EEG electrodes (arrow) consistently preceded EMG activity.

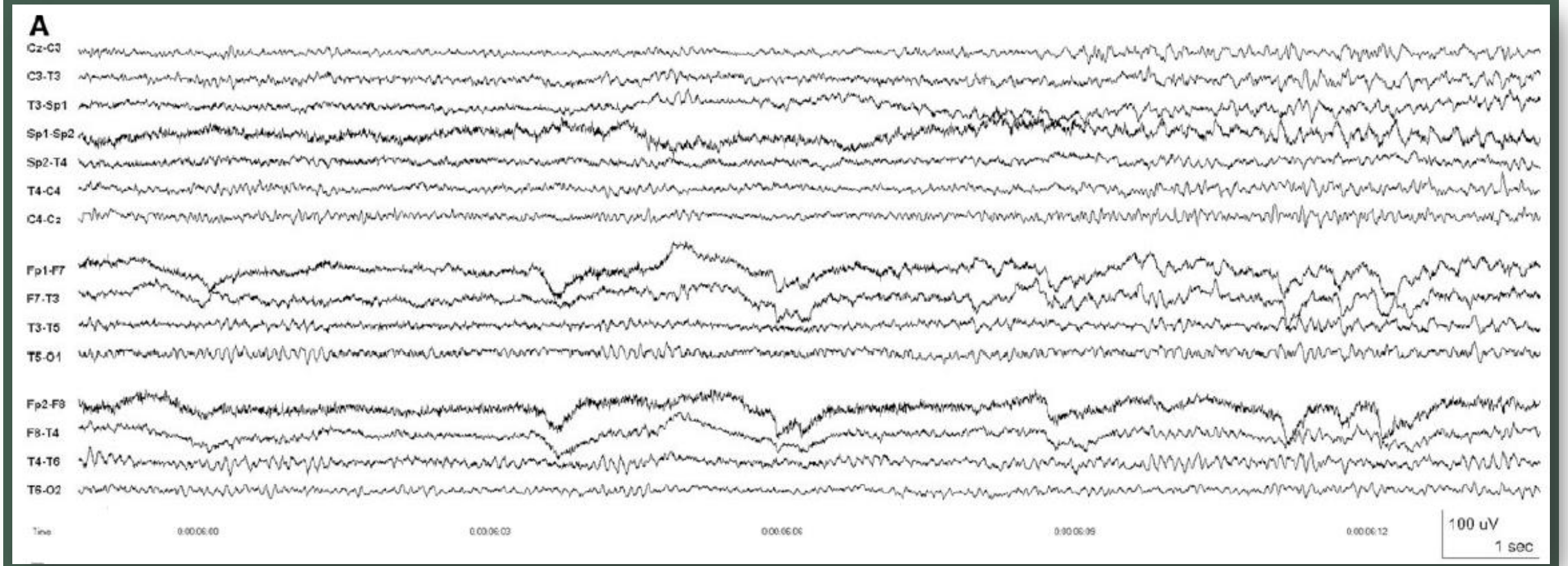
*Narvarro V et.al., Brain 2016*

# Subclinical temporal EEG seizure pattern in LGI1-antibody–mediated encephalitis

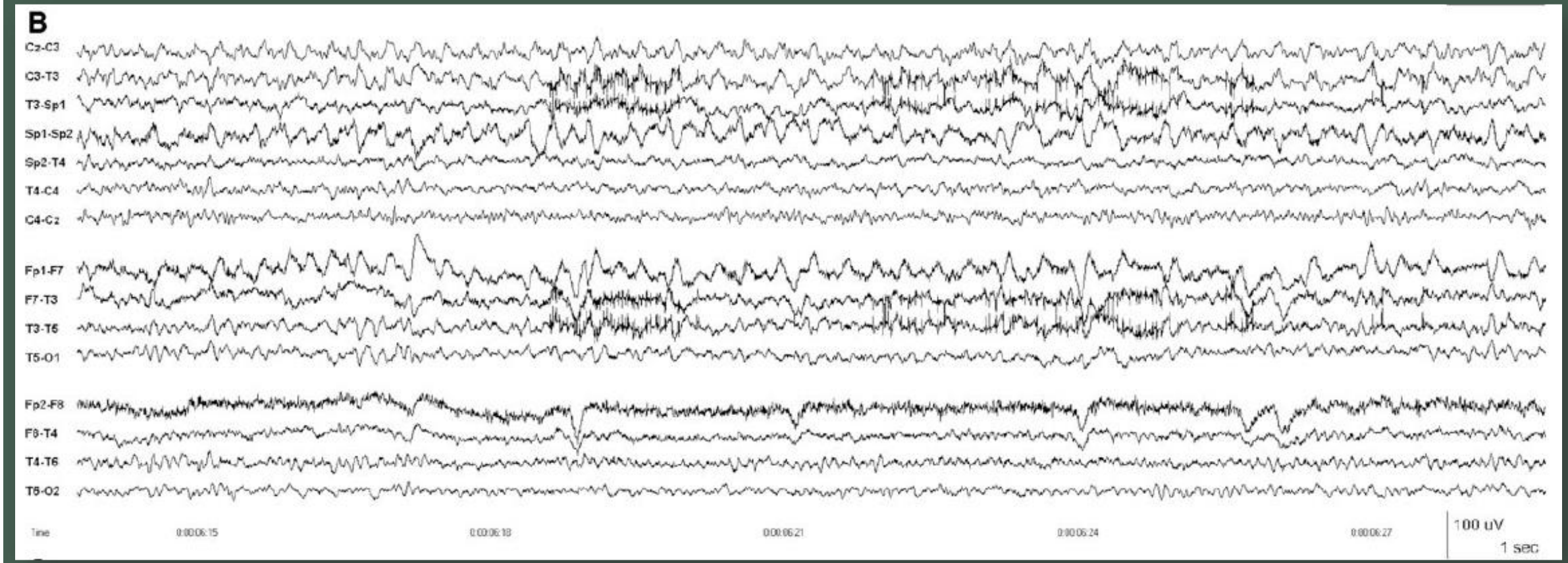
- A distinct, **unusual subclinical EEG pattern** was seen in **5/9 patients (56%)**
- In two patients, the electrographic events were consistently **triggered by hyperventilation**
- **Remarkable aspect of the EEG recordings in these patients was the profound lack of IEDs, despite the frequent ictal events**

*Steriade C et.al., Epilepsia 2016*

# Seizure onset maximal over the left anterior–mid-temporal area (F7-T3, T3-SP1)



# Continuation of subclinical seizure and hyperventilation



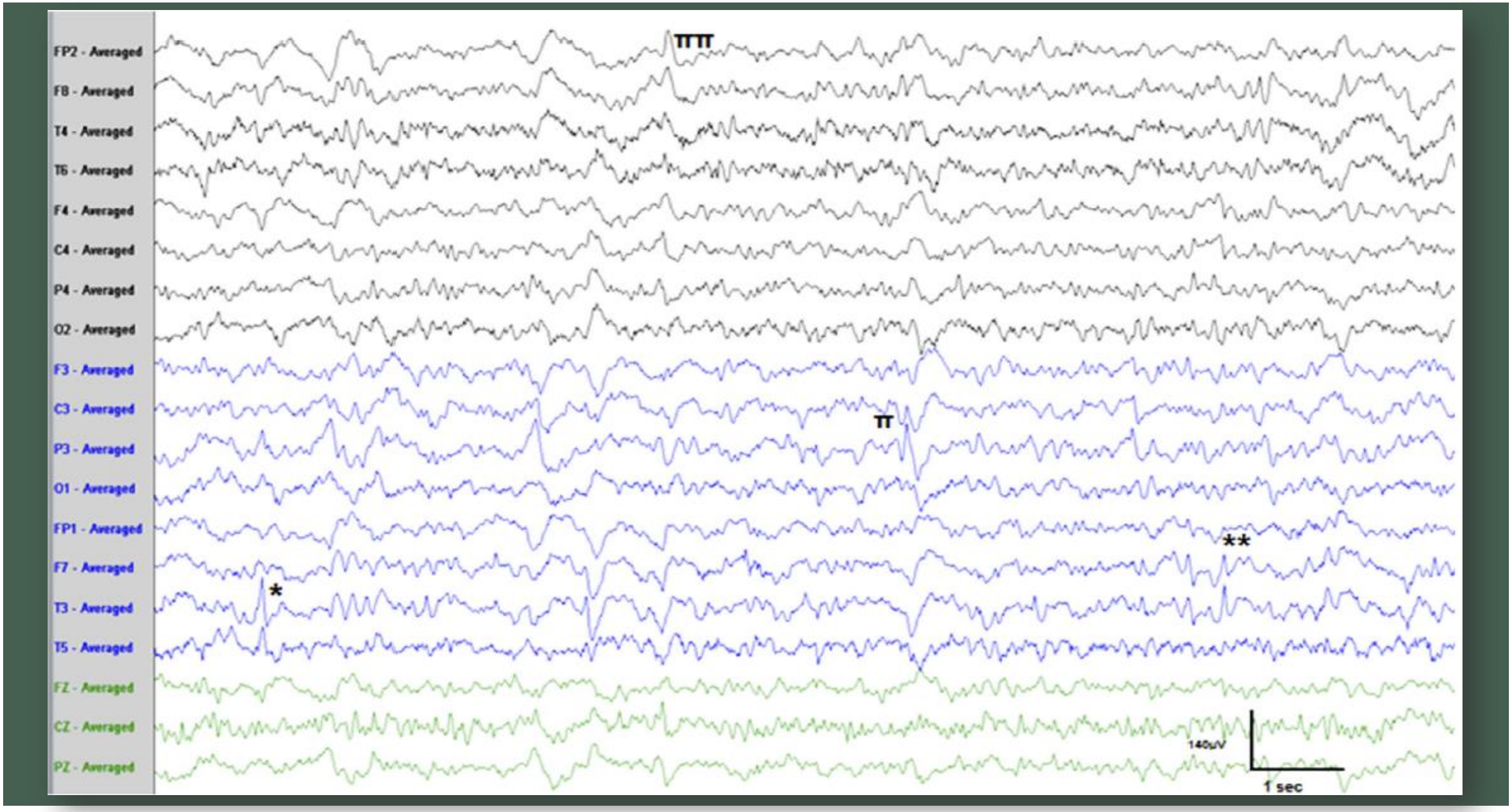
# LGI1-antibody encephalitis is characterised by frequent, multifocal clinical and subclinical seizures

**16 pts** with LGI1-antibody encephalitis

❖Seizure semiology
- **FBDS** (5/86 with ictal EEG changes)
- 11/16 with **other seizure types**
  - ✓Subclinical seizures
  - ✓Motor seizure: automatism, vocalization
  - ✓Sensory seizure: thermal sensation or body-shuddering

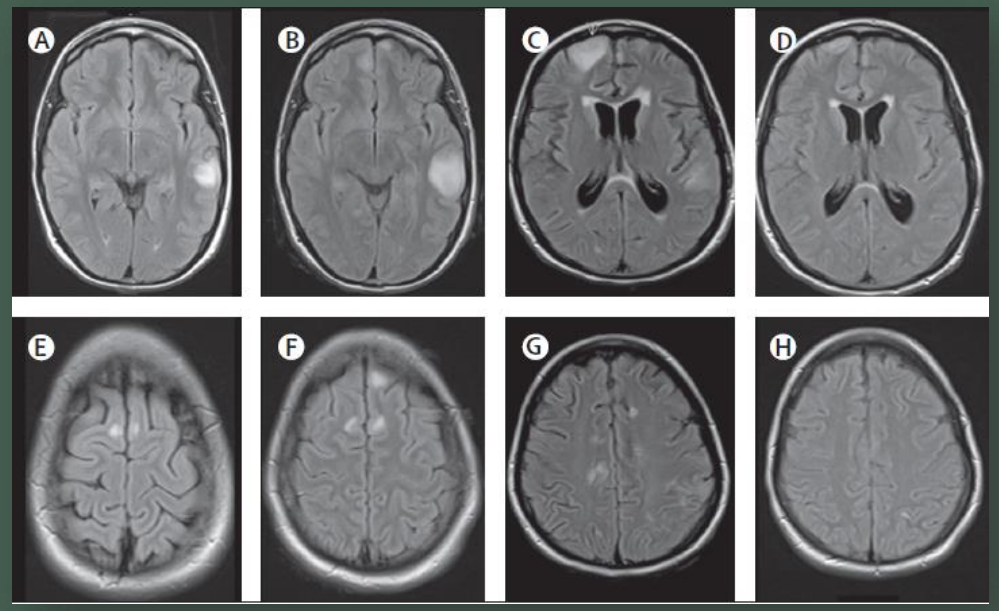
❖IEDs, seen 4/16 (25%)
  - ✓**Multifocal IEDs**: temporal, frontal, parietal foci

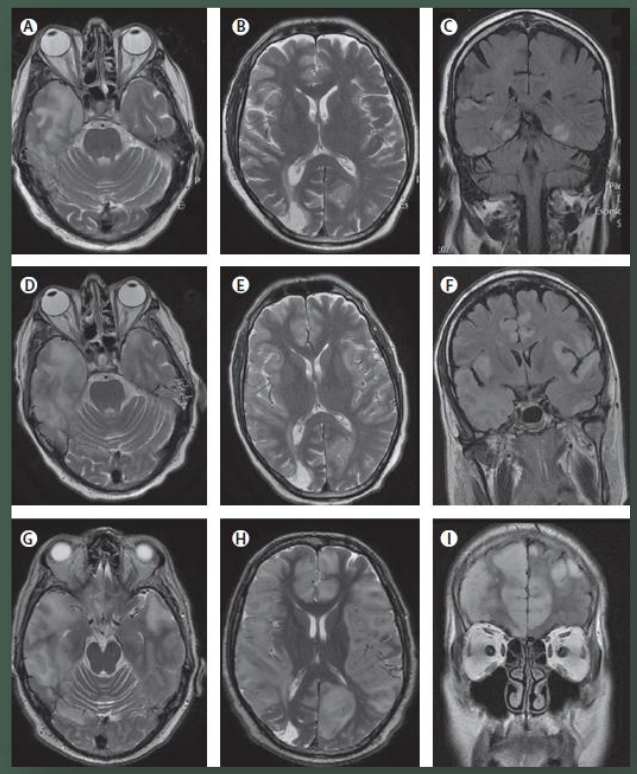
*Aurangzeb S et.al., Seizure 2017*


FP2 - Averaged
F8 - Averaged
T4 - Averaged
T6 - Averaged
F4 - Averaged
C4 - Averaged
P4 - Averaged
O2 - Averaged
F3 - Averaged
C3 - Averaged
P3 - Averaged
O1 - Averaged
FP1 - Averaged
F7 - Averaged
T3 - Averaged
T5 - Averaged
FZ - Averaged
CZ - Averaged
PZ - Averaged
140uV
1 sec

# Anti-$GABA_A$ Ab-mediated encephalitis

- **6 cases** with high titres of serum and CSF $GABA_A$ receptor antibodies are associated with a severe form of encephalitis with seizures, refractory status epilepticus, or both
- **EEG findings**: Generalized slowing, focal seizures, GPDs
- **MRI findings**: Progressive multifocal abnormalities




*Petit-Pedrol M et.al., Lancet Neurol 2014*

# Anti-GAD65 Abs-mediated encephalitis

**Table 3: Clinical features**[14-44]

| | |
|---|---|
| Seizures | 97% (56/58) |
| Cognitive impairment | 66% (38/58) |
| Memory | 59% (34/58) |
| Other than memory | 40% (23/58) |
| Orientation | 7 cases |
| Language | 7 cases |
| Executive functions | 3 cases |
| Attention/concentration | 3 cases |
| Praxia | 2 cases |
| Confabulation | 1 case |
| Psychiatric symptoms | 28% (16/58) |
| Depression | 6 cases |
| Change in behavior or personality | 6 cases |
| Psychomotor agitation | 5 cases |
| Perception disorder (hallucinations, delusions) | 4 cases |
| Anxiety | 2 cases* |
| Fever | 14% (8/58) |
| Dysautonomia | 12% (7/58) |
| Cerebellar manifestations | 7% (4/58) |
| Headache | 5% (3/58) |

*More than one psychiatric symptoms in five cases.

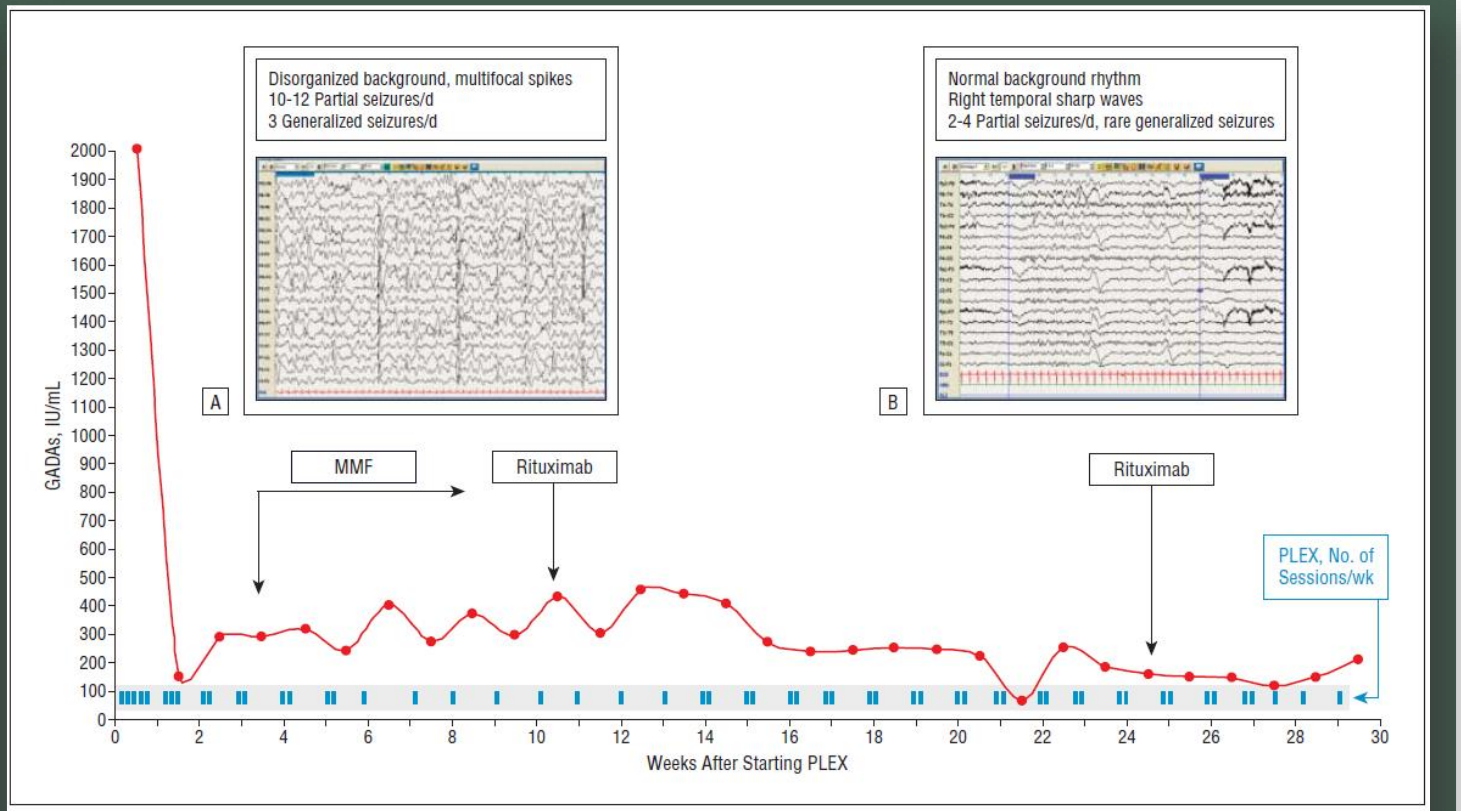
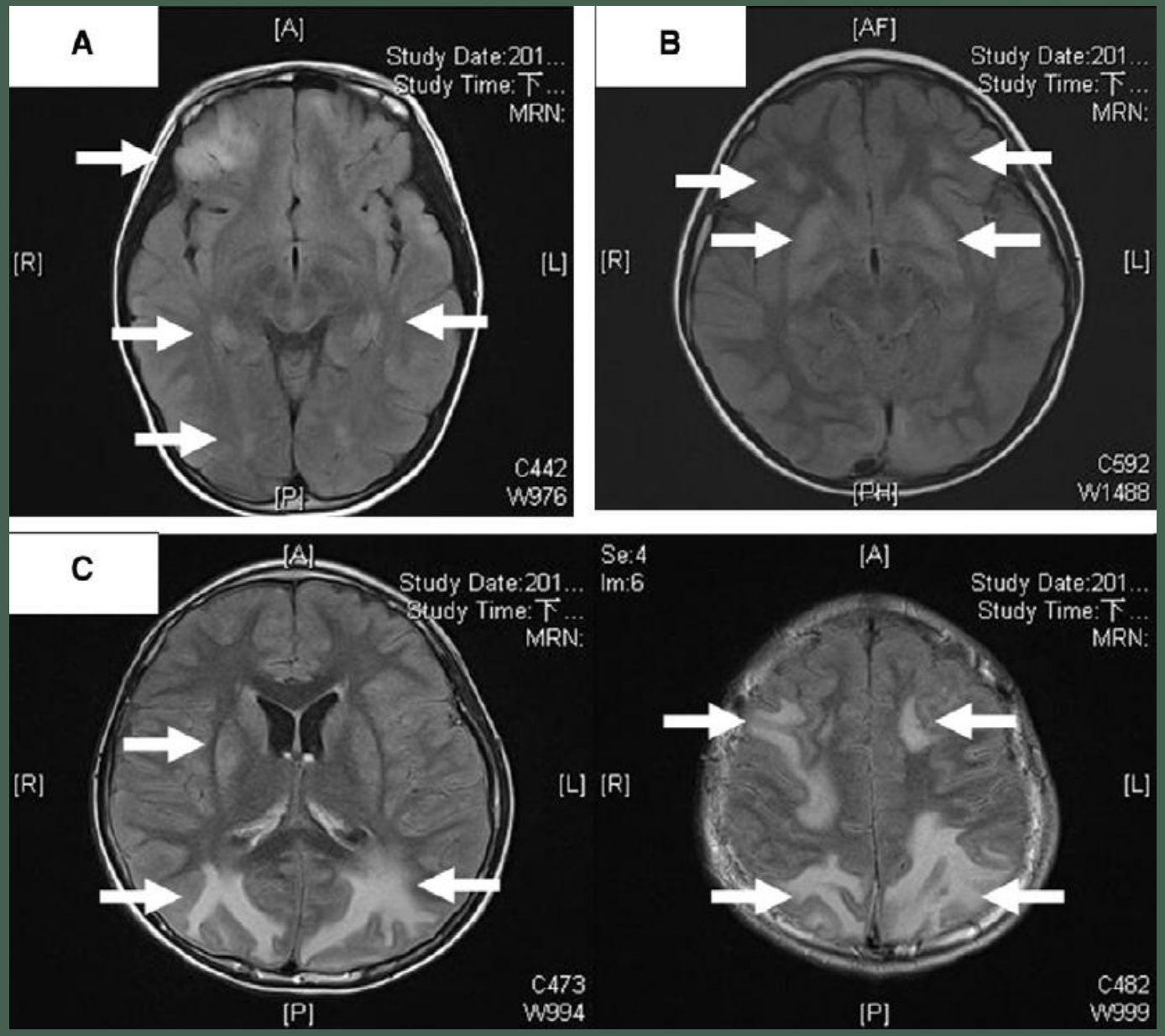



**Figure 2.** Evolution of glutamic acid decarboxylase autoantibodies (GADAs), with drug treatment indication, electroencephalographic findings, and average number of seizures per day from the time of initial plasmapheresis (PLEX). MMF indicates mycophenolate mofetil.

27/35 (77%) with epileptiform discharges

- 19/27 (70%) with **temporal involvement**
- 6/27 (22%) with multifocal EDs
- 2/27 (7%) with generalized EDs

*Korff CM et.al., Arch Neurol 2011 Gagnonl M and Savard M, Can J Neurol Sci 2016*




## MRI findings in Anti-GAD65 encephalitis

45/58% (78%): abnormal

- **34/58 (59%): temporal**
- 9/58 (16%): multifocal

*Gagnonl M and Savard M, Can J Neurol Sci 2016; Lin J et.al.,, Ped Neurol 2012*

# EEG FEATURES DISTINGUISHING AUTOIMMUNE ENCEPHALITIS FROM METABOLIC ENCEPHALOPATHY

# Are There Any Specific EEG Findings in Autoimmune Epilepsies?

**Table 2.** Comparison of EEG Findings of Patients With and Without Antineuronal Antibodies.

| | Seropositive Patients (n = 20) | | Seronegative Patients (n = 21) | | *P* Value for Seropositive vs Seronegative |
|---|---|---|---|---|---|
| | Epilepsy n = 13 (%) | Encephalitis n = 7 (%) | Epilepsy n =13 (%) | Encephalopathy n = 8 (%) | |
| No. of EEGs | 41 | 27 | 45 | 13 | |
| No. of abnormal EEGs | 38 | 27 | 42 | 13 | |
| Intermittent theta/delta slowing | 5 (38) | 2 (29) | 8 (61) | 6 (75) | 0.06 |
| Continuous theta/delta slowing | — | 5 (71) | — | 2 (25) | 0.24 |
| Focal slowing | 6 (46) | 5 (71) | 8 (61) | 2 (25) | 0.76 |
| FIRDA | 4 (31) | 4 (57) | 3 (23) | 2 (25) | 0.33 |
| Periodic discharges | 1 (8) | 2 (29) | — | 1 (12) | 0.34 |
| Epileptiform discharges | 12 (92) | 6 (86) | 9 (69) | 4 (50) | 0.06 |
| Delta brush | — | 2 (29) | — | — | ND |
| Fast activity | 4 (31) | 2 (29) | 4 (31) | 2 (25) | 1.0 |
| Focal seizures | 6 (46) | 1+focal status | 6 (46) | — | 1.0 |
| NCSE | — | 3 (43) | — | — | ND |

Abbreviations: NCSE, nonconvulsive status epilepticus; FIRDA, frontal intermittent rhythmic delta activity; ND, not determined.

*Baysal-Kirac L et.al., Clin EEG Neurosci 2016*

# Predictive values and specificity of electroencephalographic findings in autoimmune encephalitis diagnosis

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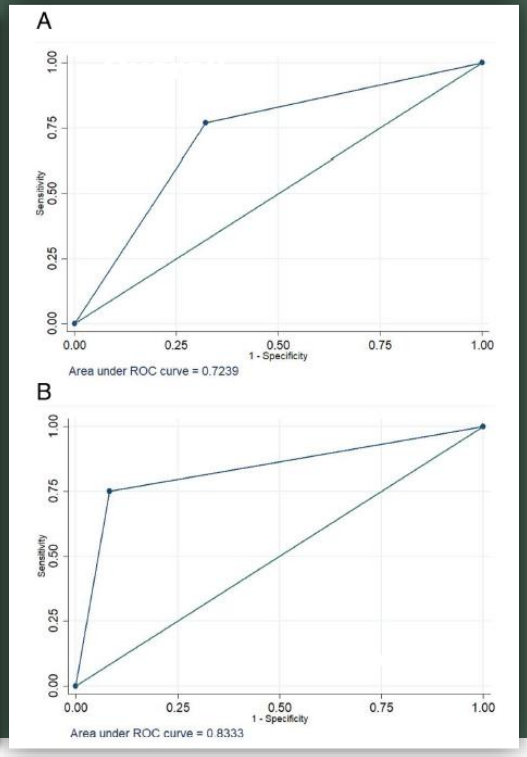
- Twenty AE cases and 40 controls (matched with age and level of consciousness)

**Table 2**
Laboratory findings at time of EEG study, immunomodulation therapy, and clinical course after treatment in patients with autoimmune encephalitis.

| Laboratory tests/immunomodulation therapy/clinical course after treatment | Results |
|---|---|
| Nonparaneoplastic autoimmune encephalitis antibody | 9 |
| Anti-NMDA | 8 [7 in both serum and CSF; 1 in CSF (serum test not performed)] |
| Anti-LGI1 | 1 (both in serum and CSF) |
| Paraneoplastic antibody | 11 |
| Anti-GAD65 | 1 (serum) |
| Anti-Ma2 | 1 (both serum and CSF) |
| Anti-GAD65 and anti-Ma2 | 1 (serum) |
| Anti-GAD65 and anti-neuroendothelium | 1 (serum) |
| Anti-neuroendothelium | 2 (2 in serum) |
| Anti-SOX1 | 2 (1 in serum; 1 in both serum and CSF) |
| Anti-recoverin | 2 (2 in serum) |
| Anti-CV2 | 1 (1 in serum) |




Poorly sustained PDR was significantly associated with AE (p=0.007) and even more predictive in anti- NMDA encephalitis

None of the cases had normal EEG nor Grand Total EEG (GTE) score < 4 (negative predictive value (NPV) of 100%).

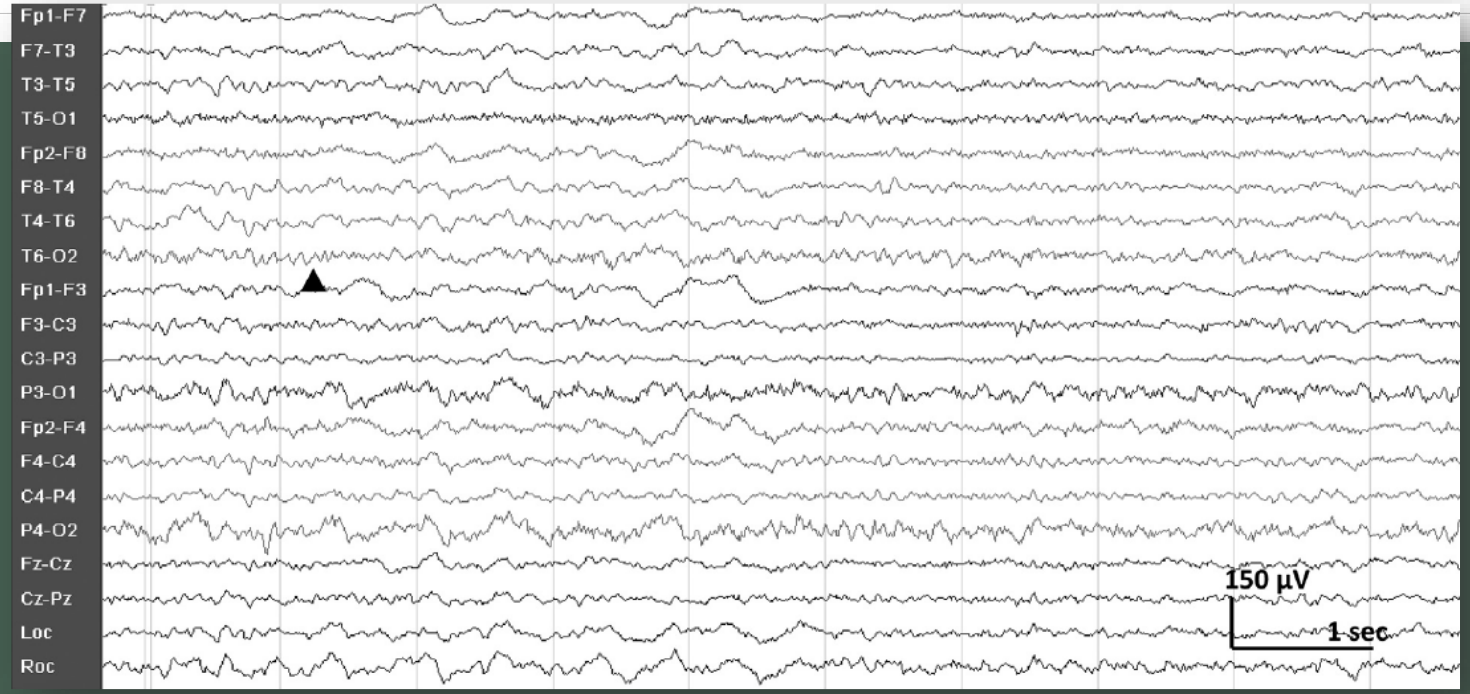
*Limotai C et.al., Epilepsy & Behav 2018*

**Supplementary table**

Visual assessment of the electroencephalogram with the grand total of EEG (GTE) score

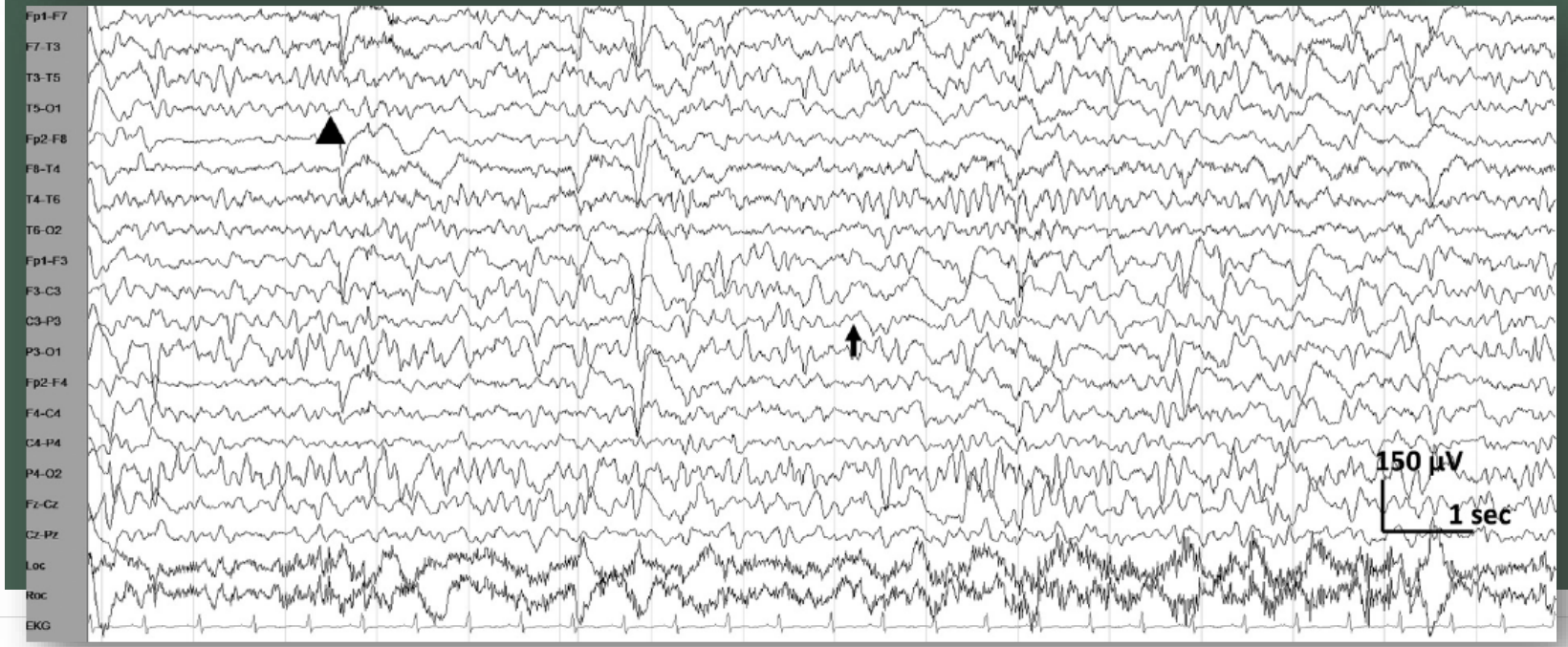
| |
|---|
| **1. Frequency of rhythmic background activity** |
| 0 = > 9.0 Hz |
| 1 = 8-9 Hz |
| 2 = 7-8 Hz |
| 3 = 6-7 Hz |
| 4 = 4-6 Hz |
| 5 = none |
| **2. Diffuse slow activity** |
| 0 = none |
| 1 = intermittent theta |
| 2 = intermittent theta + sporadic delta |
| 3 = continuous theta + intermittent delta |
| 4 = continuous theta + delta |
| 5 = continuous delta |
| **3. Reactivity of the rhythmic background activity** |
| 0 = normal reactivity |
| 1 = diminished on eye opening |
| 3 = absent on eye opening |
| 3 = no reaction to somatosensory stimulation |
| 4 = no reaction to auditory stimulation |
| 5 = absence of all reactivity |
| **4. Paroxysmal activity** |
| 0 = none |
| 3 = paroxysmal slow activity |
| 5 = FIRDA |
| **5. Focal disturbances** |
| 0 = no focal disturbances |
| 1 = mild disturbances unilateral |
| 2 = mild disturbances bilateral |
| 3 = severe unilateral and mild contralateral |
| 4 = severe bilateral |
| 5 = multifocal |
| **6. Sharp wave activity** |
| 0 = none |
| 2 = sporadic sharp waves |
| 3 = frequent sharp waves |
| 4 = triphasic waves |
| 5 = Creutzfeldt-Jakob complexes or PLEDS |
| **Grand total of EEG = (sum 1-6) + 1** |

# Grand Total EEG (GTE) score
# Score 1-31 (most severe)




**GTE score = 11**

**Anti-NMDA encephalitis**




**GTE score = 20**

**Anti-Ma2**

# In summary

| Encephalitis syndrome | EEG hallmarks |
|---|---|
| Anti-NMDA | - Diffuse slowing<br>- "Extreme delta brush"<br>- GRDA or LRDA<br>- Excessive beta or excessive spindle-like activity<br>- "Status dissociatus" |
| Anti-LGI1 | - Frequent subclinical temporal lobe seizures<br>- Rare or multifocal IEDs |
| Anti-$GABA_A$ | - Refractory status epilepticus<br>- Focal seizures |
| Anti-GAD65 | - Prominent temporal IEDs |

How can the EEG findings be distinguishable the autoimmune encephalitis from metabolic encephalopathy/ other common structural abnormalities?

Specific region of the brain involved

Anti-GAD65
Anti-LGI1

Anti-LGI1 (subclinical)
Anti-$GABA_A$ (RSE)

Specific pathophysiology creates unique EEG waves

Anti-NMDA

Epileptogenic level

# THANK YOU FOR YOUR ATTENTION