EST 30th ANNIVERSARY ANNUAL MEETING

Phramongkutklao Comprehensive Pediatric Epilepsy Center of Excellence

Integration • Passion • Wisdom

Autoimmune Epilepsy

Diagnostic Challenges, Treatment Strategies, and Case Presentation

Assoc. Prof. Piradee Suwanpakdee, MD
Pediatric Neurology Division
Department of Pediatrics
Phramongkutklao Hospital and College of Medicine

EST 30th ANNIVERSARY ANNUAL MEETING

Outline

Terminology & Core Concepts

Understand the critical clinical distinction between

- Acute Symptomatic Seizures (ASS)
- Autoimmune-associated epilepsy (AAE)
- Autoimmune Encephalitis-Associated Epilepsy (AEAE)

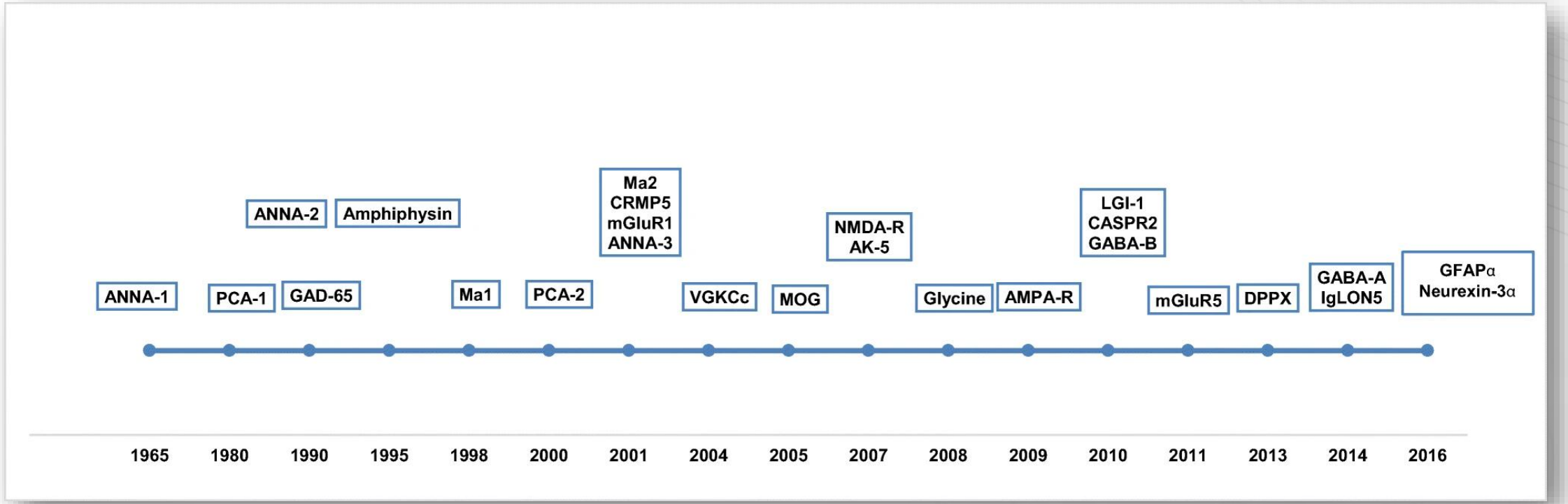
Case-Based Presentations

- Autoimmune-associated epilepsy (AAE)
  - Antibodies against intracellular antigens (GAD65)
  - Rasmussen's encephalitis
- Autoimmune Encephalitis-Associated Epilepsy (AEAE)

Practical Bedside Strategy

- Clinical clues
- Investigations
- Treatment

Timeline of autoimmune epilepsy



Pre-2020: 'Autoimmune epilepsy' (broad concept)

Epilepsia 2020: Autoimmune-associated epilepsy (AAE)

Epilepsia 2023: Autoimmune encephalitis-associated epilepsy (AEAE)

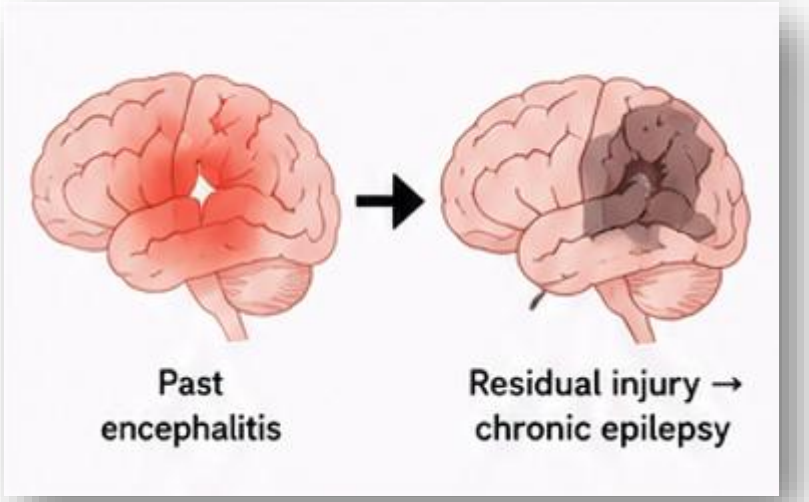
Husari et al, Neurotherapeutics 2019; Steriade et al. Epilepsia 2020; Rada A, Bien CG. Epilepsia. 2023

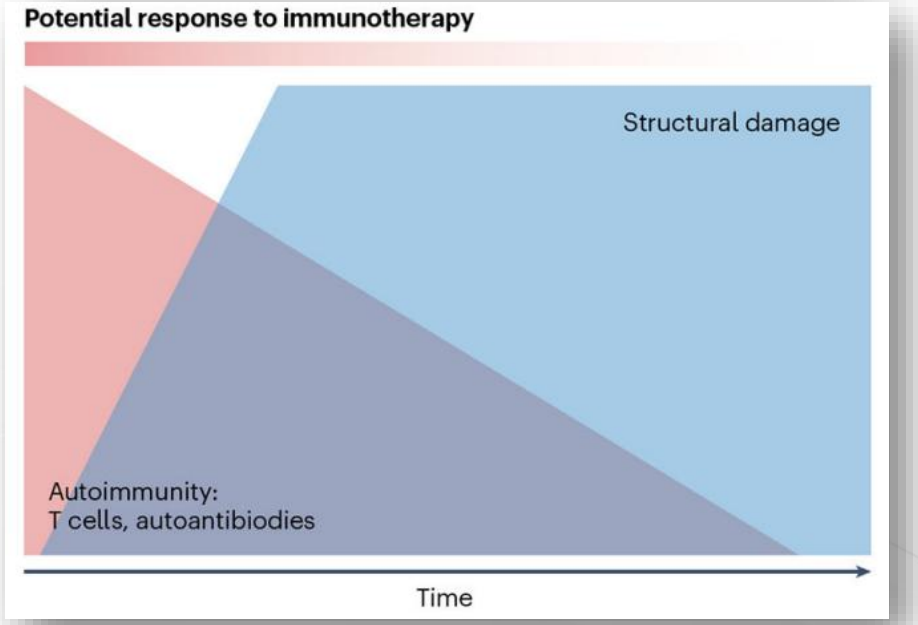
Acute symptomatic seizures secondary to autoimmune encephalitis vs. autoimmune-associated epilepsy

	Acute symptomatic seizures secondary to autoimmune encephalitis	Autoimmune-associated epilepsy (AAE)
Underlying antibodies or conditions	Antibodies against - surface antigens (NMDAR, LGI1, CASPR2, GABABR, GABAAR, mGluR5, DPPX, AMPAR) - intracellular antigens (onconeural, GAD65)	• **Antibodies against intracellular antigens** (onconeural,GAD65) • **Rasmussen encephalitis** • **Persistent epilepsy** after acute autoimmune encephalitis
Hypothesized pathophysiology	**Antibody**-mediated ictogenesis	Epileptogenesis due to **structural** postencephalitic pathology **and/or** ongoing T-cell–mediated brain inflammation
Therapy	**Immunotherapy** Antiseizure medications (usually ineffective in isolation)	Antiseizure medications (often ineffective) Epilepsy surgery (usually with incomplete response) **Immunotherapy (usually with poor response)**
Outcome	Seizures usually terminate with **remission** of encephalitis Potential for antiseizure medication discontinuation Potential enduring cognitive deficits	**Drug-resistant focal epilepsy** common Potential enduring cognitive deficits

Steriade et al. *Epilepsia* 2020

Autoimmune Encephalitis-Associated Epilepsy (AEAE)





- **All five** criteria proposed for AEAE-surface-antibody AE

1. Previous AE: NMDAR, LGI1, CASPR2 or GABABR
2. Persisting seizures > 2 years after immunotherapy initiation
3. No signs of encephalitis on MRI; hippocampal sclerosis allowed and no FDG-PET hypermetabolism
4. Normal CSF cell count
5. Substantial decrease of antibody titers

*The distinction between Autoimmune encephalitis (AIE) and AEAE also has practical consequences:

- In AIE, immunotherapy is usually highly beneficial, whereas anti-seizure medication has little effect.
- In AEAE, immunotherapy is less promising and the usual antiseizure interventions are preferable

Rada A, Bien CG. Epilepsia. 2023; Steriade, et al. Nat Rev Neurol. 2025

Three conditions considered to be autoimmune encephalitis-associated epilepsies (AEAE)

	GAD-TLE	High-Risk Paraneoplastic	Post-Surface AEAE (LGI1)
Demographics	Female 84-95% (24-50 yrs)	Older adults, variable	Males 60% (~60 yrs)
Pathophysiology	Cytotoxic T-Cell	Cytotoxic T-Cell	Complement Cascade / Structural Injury
Systemic Comorbidities	Type 1 Diabetes, Thyroid (55%)	Cancer (63%, e.g., Small Cell Lung)	None typical
Seizure Semiology	Déjà-vu, autonomic, musicogenic	Extralimbic, epilepsia partialis continua	Pilomotor, Faciobrachial Dystonic (historical)
MRI Progression	Mesial temporal swelling -> Atrophy	Variable, often extralimbic	50-58% develop Hippocampal Sclerosis

Steriade et al. Nat Rev Neurol 2025

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Practical Bedside Strategy for Suspecting Autoimmune Epilepsy

Step 1. Identify "Red Flags"

New-onset focal epilepsy (<12 months)
(esp. adult or adolescent onset)

Rapid progression (days–weeks)

High seizure frequency

Early drug resistance

New-onset status epilepticus/NORSE/FIRES

Faciobrachial dystonic seizures (LGI1)

Multifocal seizures

Associated Neurological Symptoms:
- Memory impairment/ Psychiatric symptoms/ Movement disorders

Step 2. Look for Autoimmune Clues

History

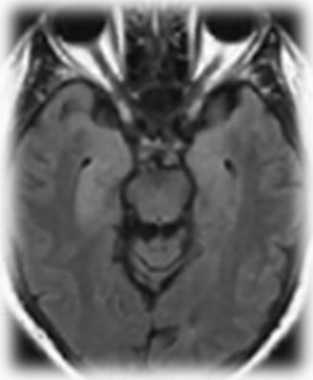
✓ Personal autoimmune disease
- Thyroiditis
- Type 1 diabetes
- Lupus
- Celiac disease

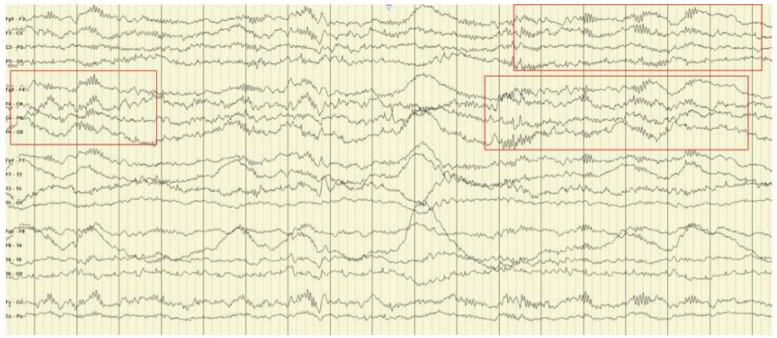
✓ Family history of autoimmune disease
✓ Recent viral illness
✓ History of cancer
✓ Hyponatremia (particularly LGI1)

Graus F et al. *Lancet Neurol.* 2016; Dubey et al. epilepsia 2019; De Bruijn et al. Ann Neurol 2021

Think about the clinical contexts

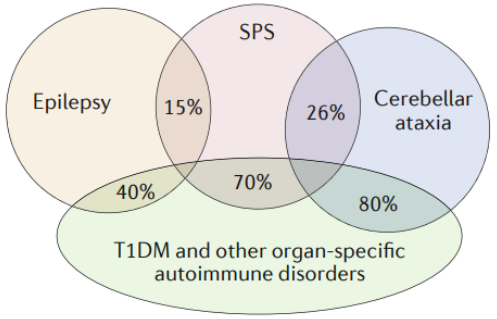
LGI1: Male predominance
Memory impairment, **faciobrachial dystonic seizure**,
Frequent focal seizures
MRI: normal or T2 hyperintensities in Mesial temporal
EEG: temporal onset seizures
CSF: mild pleocytosis, protein elevate, 50% normal





NMDA:Female predominance
Psychiatric and cognitive symptoms, **dyskinesia**,
dysautonomia, Focal to bilateral tonic-clonic seizures
MRI normal in 70% or T2 hyperintensities
EEG: gen. rhythmic delta activity, extreme delta brush
CSF: mild pleocytosis, protein elevate, Ab (+)
Associated with teratoma

GAD 65: Female predominance
Type 1 DM, thyroid disease, ataxia,
temporal lobe seizures (Déjà vu, musicogenic) and
limbic encephalitis
MRI normal or T2 hyperintensities in Mesial temporal
EEG: temporal onset seizures
CSF: typically normal

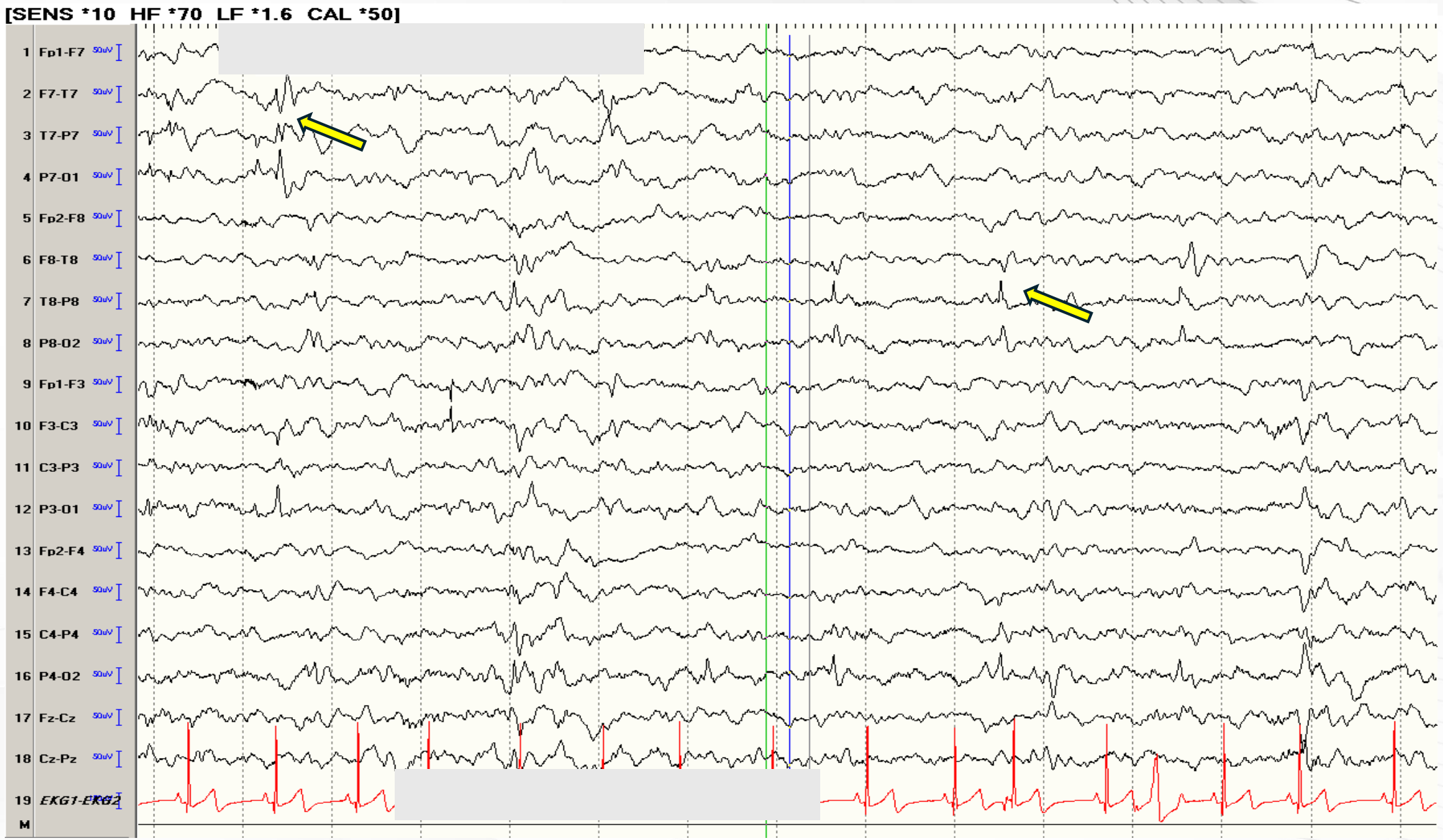


Smith et al. Epileptic Disorder 2024

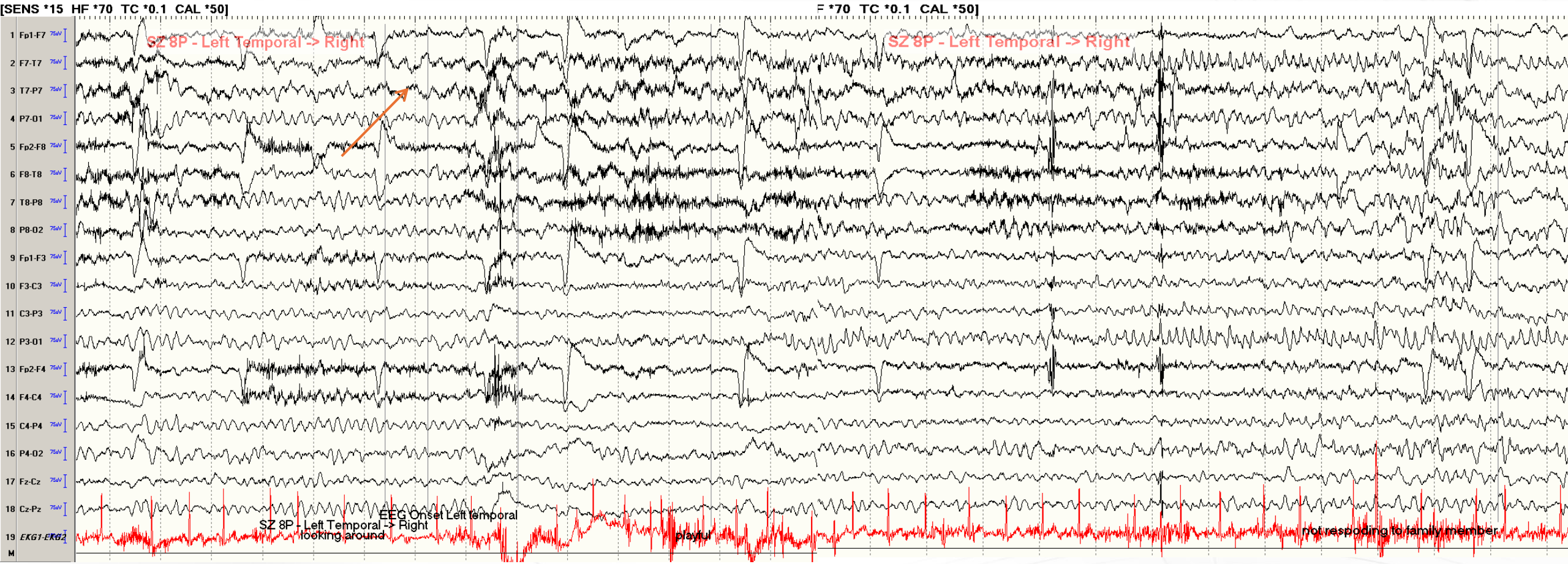
Case 1: A 6-year-old girl, RH

- Age of seizure onset: 4 years
- Seizure type: gelastic seizures with impaired consciousness-> Ictal deafness
  - Description: She has sudden episodes of laughing and crying along with unresponsiveness. She would abruptly stop and may say "I am not hearing. I am not hearing". This may last 10 to 20 min.
  - Frequency: two times per week
- ETIOLOGY : Unknown
- Epilepsy Risk Factors: None
- Antiseizure Medications: VPA, LEV , Clobazam

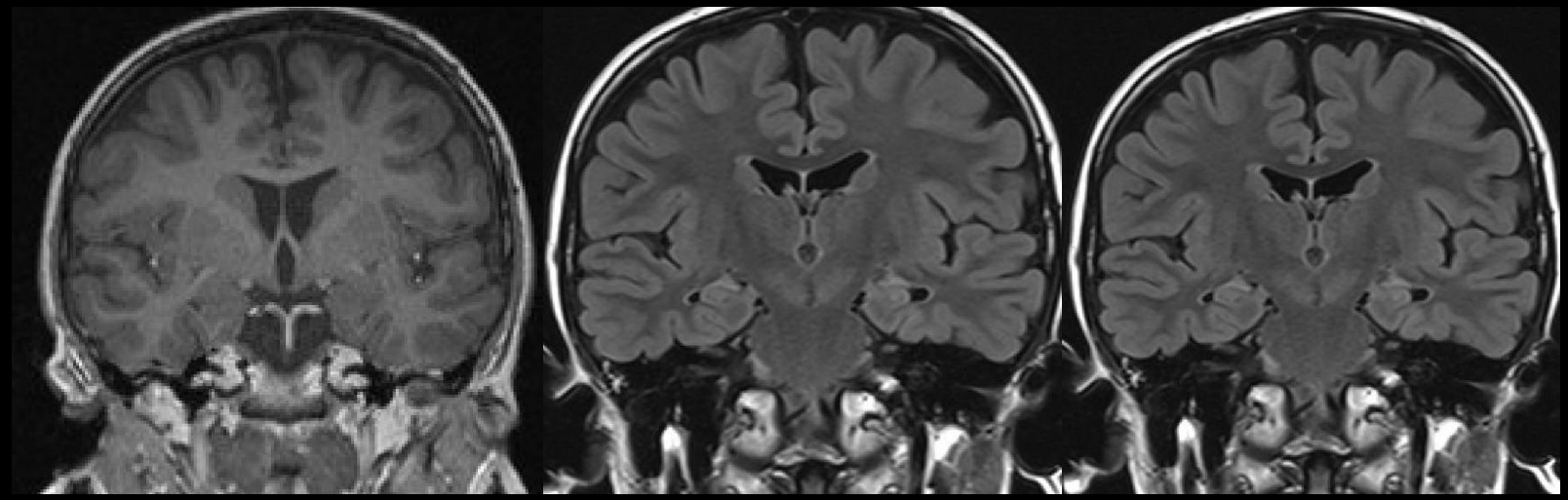
Interictal EEG: Left and Right Temporal



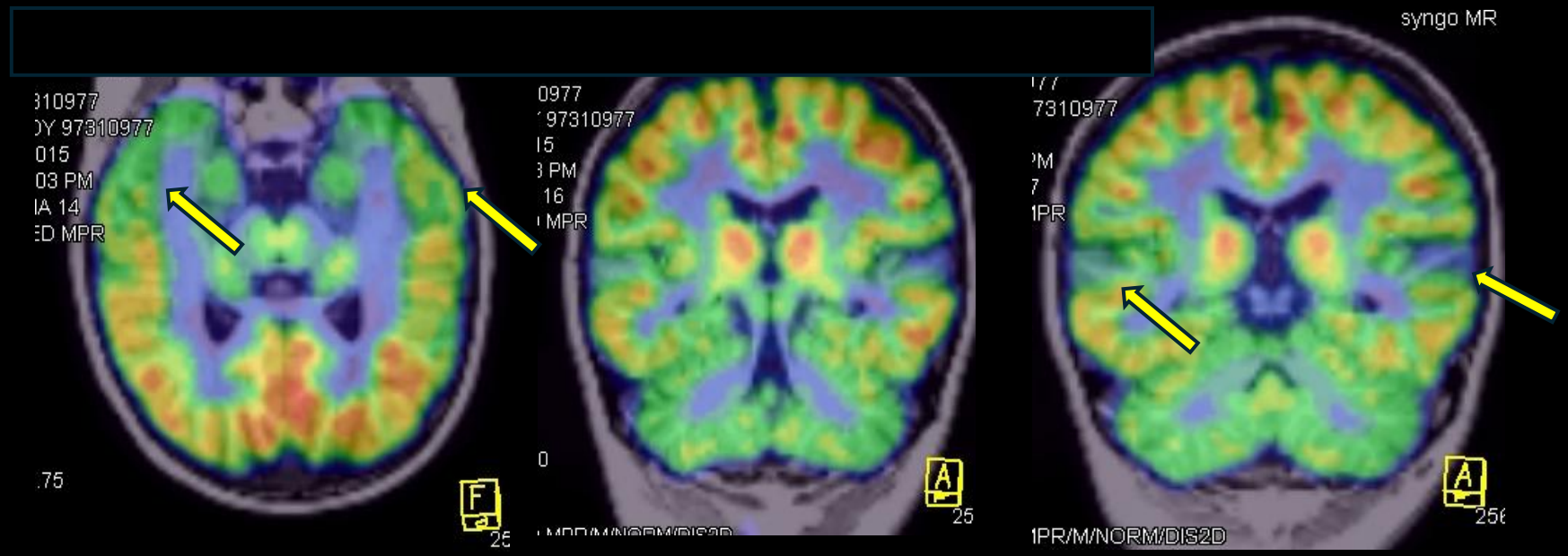
Ictal EEG: Left temporal



MRI brain: Symmetrical hippocampal volume loss



PET : hypometabolism of mesial temporal and superior temporal gyrus



Summary

- 6-year-old girl, RH
- Chronic focal drug-resistant epilepsy from unknown cause
- Seizure type: Gelastic/dacrystic seizure with impaired consciousness-> Ictal deafness
- Interictal EEG: Bilateral temporal
- Ictal EEG: Left temporal
- MRI: Symmetrical hippocampal volume loss
- PET : hypometabolism of mesial temporal and superior temporal gyrus

Refractory chronic epilepsy associated with neuronal auto-antibodies: could perisylvian semiology be a clue?

Lisa Gillinder [1,2], Linda Tjoa [1,2], Basil Mantzioris [1,2], Stefan Blum [1,2], Sasha Dionisio [1,2]

[1] Mater Advanced Epilepsy Unit, Mater Adults Hospital,
[2] Princess Alexandra Hospital, Department of Neurology, Brisbane, Australia

Table 1. Summary of the semiological features of perisylvian seizures, which are largely classified into five groups.

SOMATOSENSORY*	VISCERO-SENSITIVE	AUDITORY	LANGUAGE	AUTONOMIC
Parasthesias	Pharyngeal discomfort	Auditory hallucinations	Dysarthria	Tachycardia
Pain	Abdominal discomfort**	Palinacousis	Dysphasia	Bradycardia
Warmth	Thoracic sensations	Tinnitus		Hyperventilation
	Taste	Vertigo		Hypoventilation
	Tenesmus			Piloerection
				Flushing
				Sweating
				Salivation

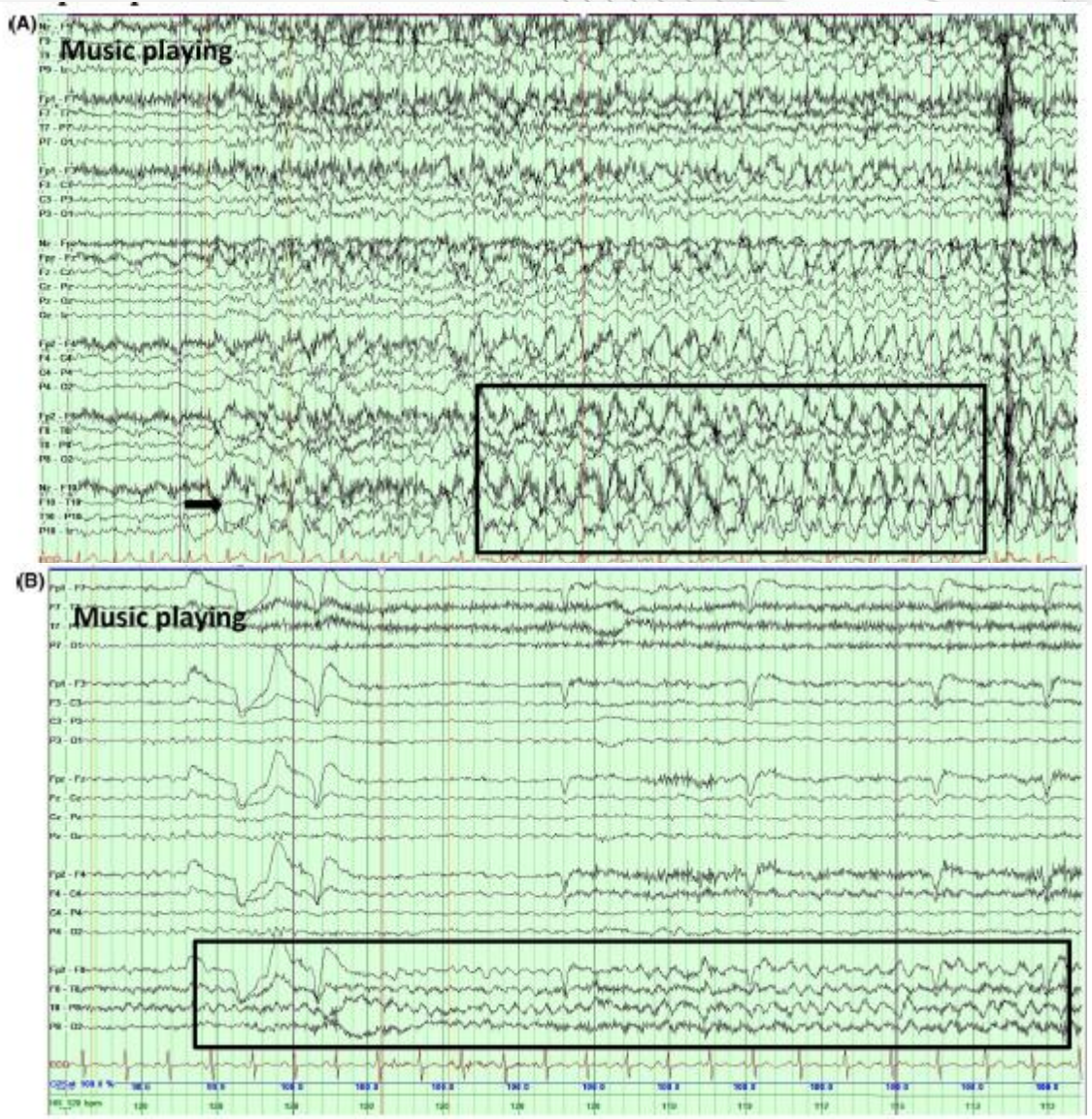
- Retrospective study
- Chronic epilepsy (>2 years) + antibody positive
- N 10, mean age 37 y (22-43)
- 50% GAD65, 30% GLY-R, 10% NMDAR, 10% LGI1

Non-lesional epilepsy with perisylvian semiology -> high level of suspicion for autoimmune epilepsy

Gillinder et al. Epileptic Disord 2017

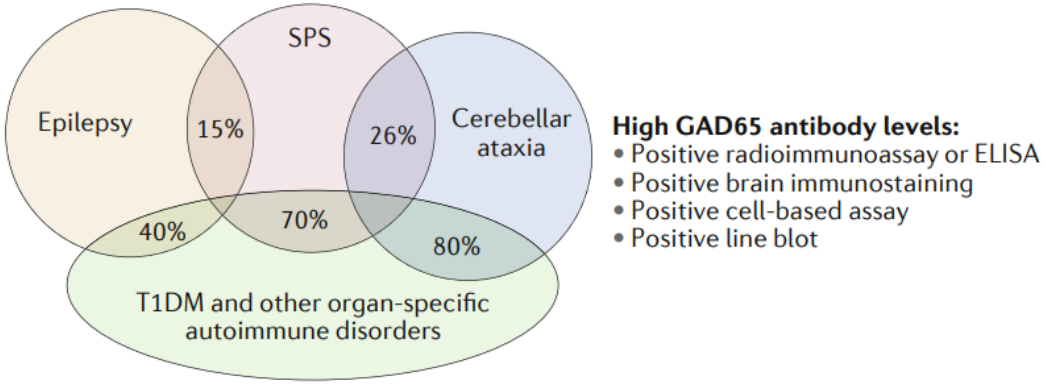
GAD 65: Musicogenic epilepsy

- 9/16 musicogenic epilepsy patients were GAD 65- IgG positive
- All patients had temporal lobe epilepsy with frequent bilateral EEG abnormalities
- Seizures were most often right temporal lobe-driven (75%) during music triggers
- Treatments showed limited benefit; VNS and trigger avoidance were most helpful

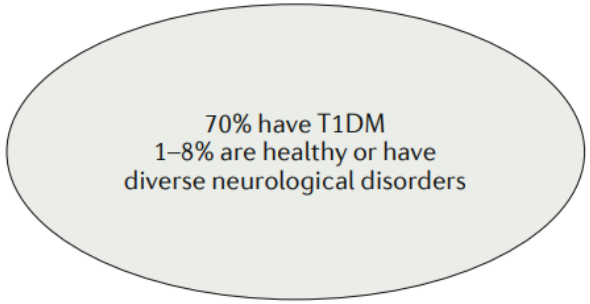


Smith et al. *Epilepsia* 2021

GAD 65- IgG



High GAD65 antibody levels:
- Positive radioimmunoassay or ELISA
- Positive brain immunostaining
- Positive cell-based assay
- Positive line blot



Low GAD65 antibody levels:
- Positive radioimmunoassay or ELISA
- Negative brain immunostaining
- Negative cell-based assay
- Positive or negative line blot

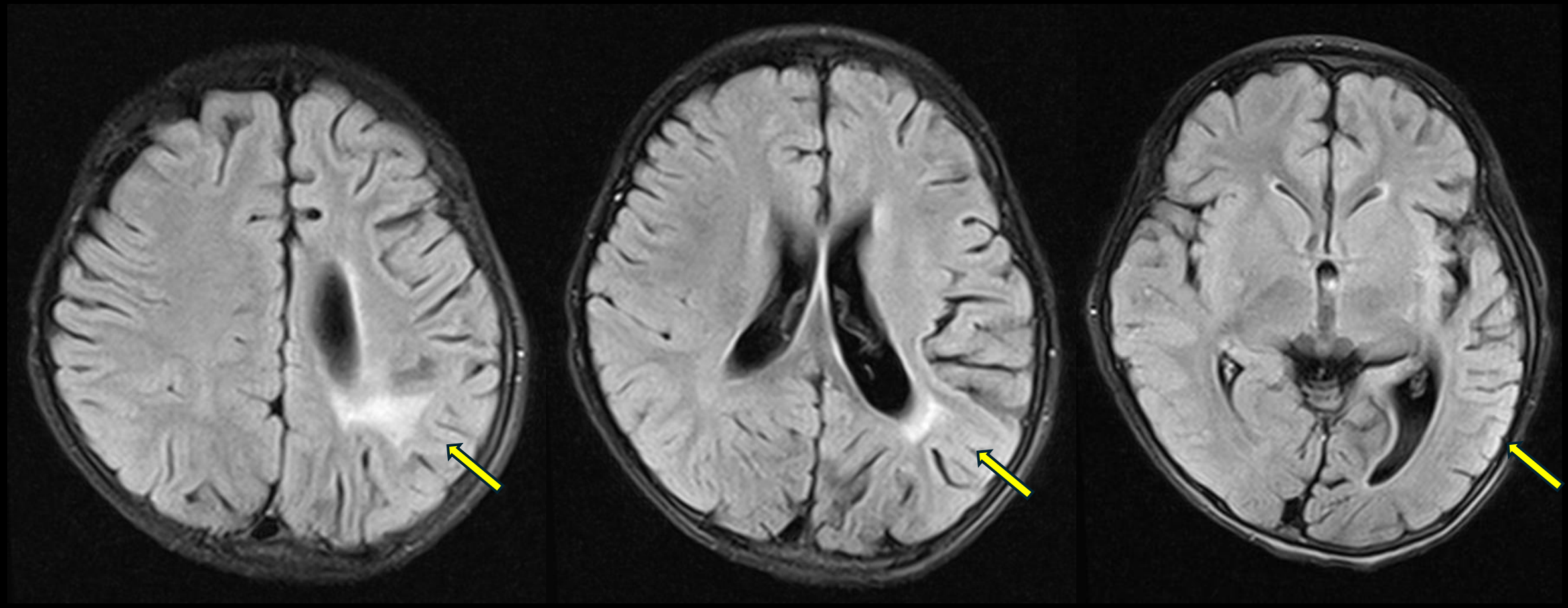
- Women are more frequently affected than men
- Median age is 30 years (range, 5–80 years)
- Can present with SPS, ataxia, epilepsy, acute encephalitis
- Autoimmune disorder (57%): T1DM, thyroid
- Intracellular Ab
- Seizure types: Focal aware seizure (common), temporal lobe seizures (Déjà vu, musicogenic) and limbic encephalitis
- EEG: temporal onset seizures
- CSF is most specific
- RIA titer > 20 nmol/L ELISA > 1000 U/mL
- MRI: vary over the course
- Poorly responsive to immunosuppression, controlled with ASMs, Epilepsy surgery

Graus et al. Nat Rev Neurol 2020

Case 2: A 4-year-old girl, LH

- Age of seizure onset: 2 years
- Seizure type: **EPC of the face/right arm/leg**
  - Description: She initially had focal clonic movements of the right arm
  - 5 months later, the seizures progressed to right face clonic and right leg clonic. She remained conscious during the seizures
  - Frequency: all day
- Perinatal history : NL, term, BW 3,200 gm, no complication
- G&D: regression following seizure onset
- Antiseizure Medications: TPX, LEV , CBZ, Clobazam
- Physical and neuro examination: drowsiness and right-sided hemiparesis

MRI Brain



Left hemispheric atrophy with hyperintensity in T2W FLAIR at left parieto-occipital region

Rasmussen's encephalitis

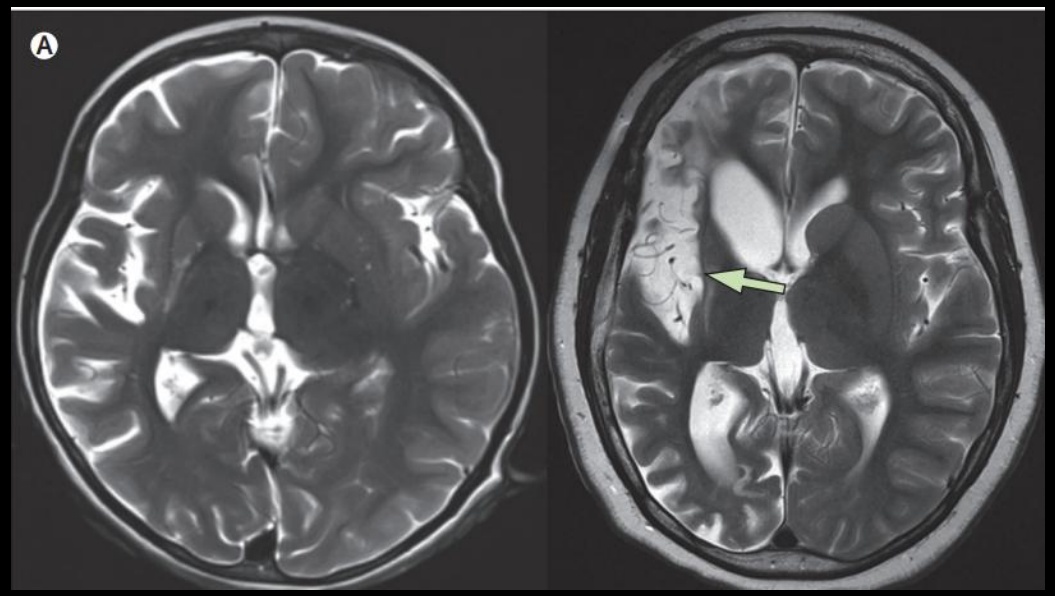
- A rare severe immune-mediated brain disorder leading to unilateral hemispheric atrophy
- The age at onset is between 6 and 8 years(range 1–13 years)
- Diagnostic criteria:

Part A (all three)	Part B (two of three)
Clinical: Focal seizures (with or without EPC) and unilateral cortical deficits	**Clinical:** EPC or progressive* unilateral cortical deficits
EEG: Unihemispheric slowing with or without epileptiform activity and unilateral seizure onset	**MRI:** Progressive* unihemispheric focal cortical atrophy
MRI: Unihemispheric focal cortical atrophy + at least one of the following: • Grey or white matter T2/FLAIR hyperintense signal • Hyperintense signal or atrophy of the ipsilateral caudate head	**Histopathology:** T-cell-dominated encephalitis with activated microglial cells typically, but not necessarily, forming nodules and reactive astrogliosis; numerous parenchymal macrophages, B cells, or plasma cells or viral inclusion bodies exclude the diagnosis of Rasmussen's encephalitis

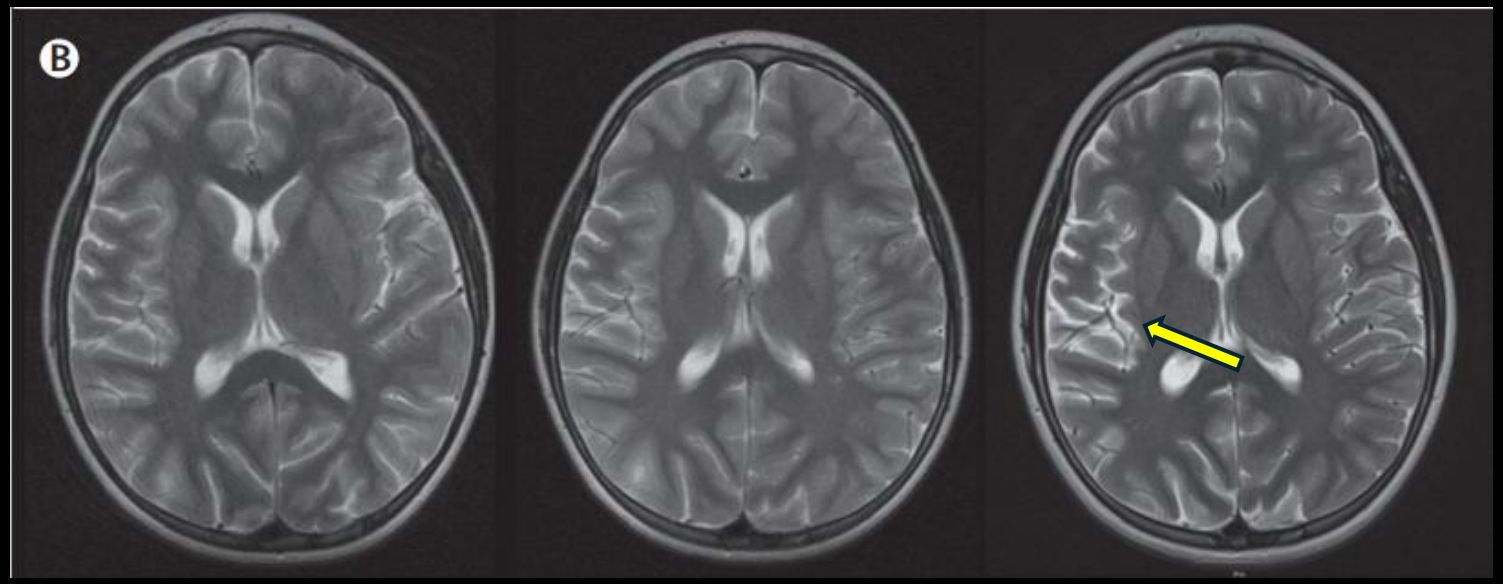
*At least two sequential clinical examinations or MRI studies are needed to meet the respective criteria.

Varadkar S, *Lancet Neurol* 2014

Neuroimaging in Rasmussen's encephalitis



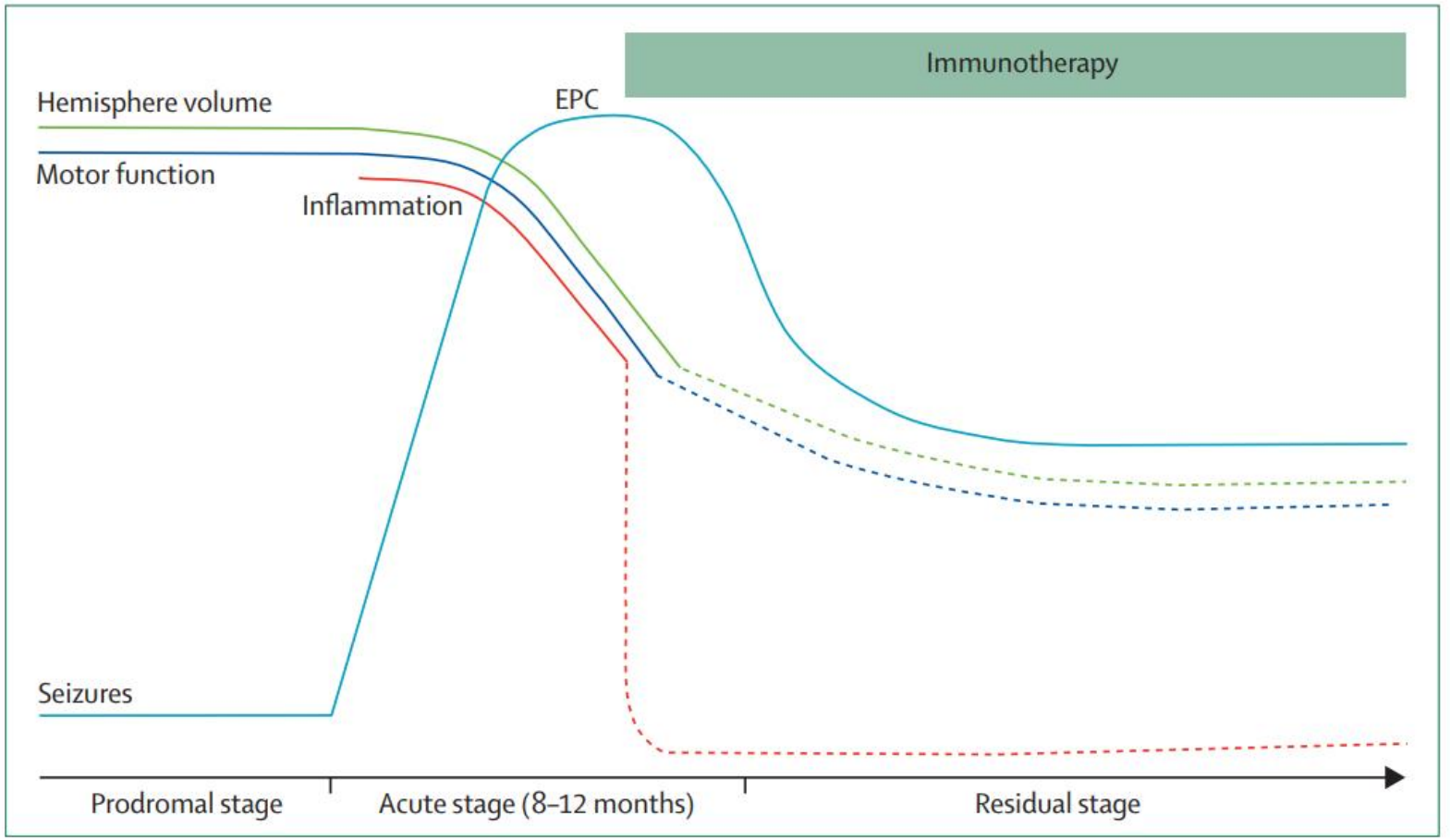
Progressive right hemisphere atrophy,
high signal and basal ganglia loss over 1 year



Slowly progressive disease with
more subtle right hemisphere atrophy

Varadkar S, *Lancet Neurol* 2014

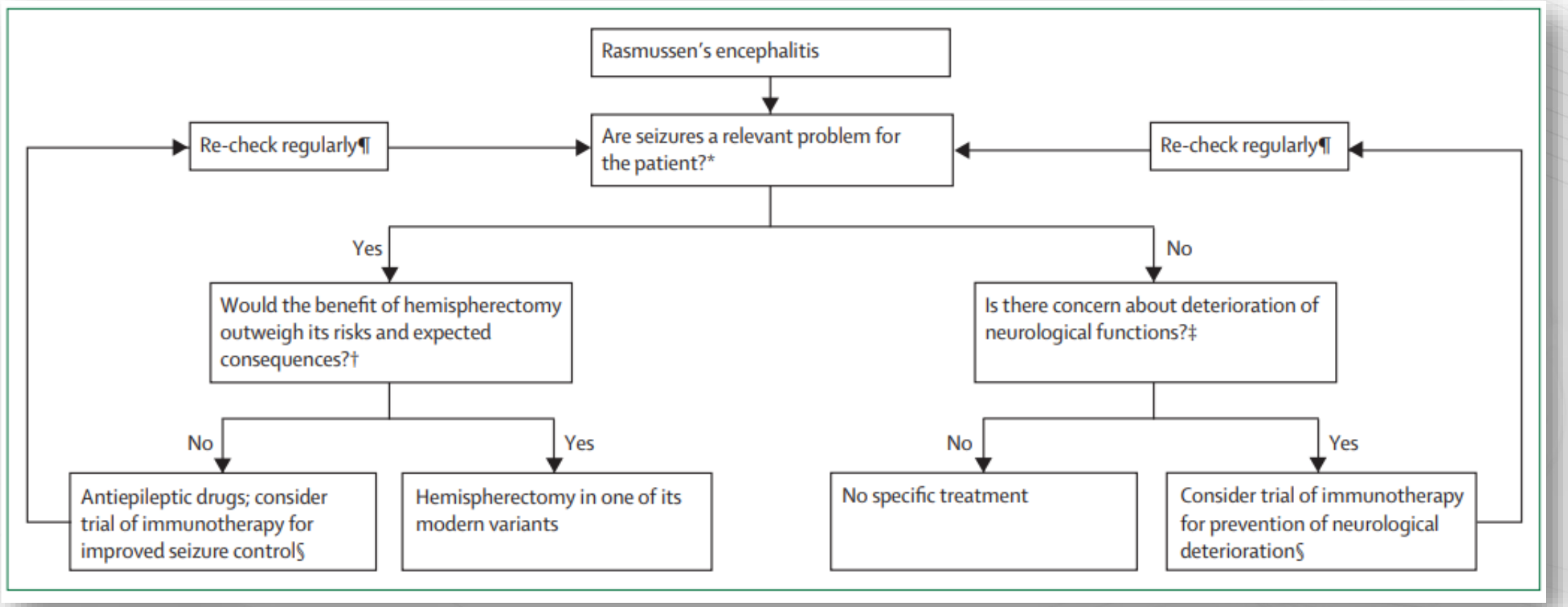
Rasmussen's encephalitis



Clinical	Infrequent focal seizures ± mild hemiparesis	Frequent focal seizures / EPC, progressive hemiparesis, aphasia, cognitive decline	Fixed neurological deficit + chronic refractory epilepsy

Varadkar S, *Lancet Neurol* 2014

Management of Rasmussen's encephalitis



Varadkar S, *Lancet Neurol* 2014

Case 3: A 4-year-old boy, RH

อายุ 2 ปี ไข้ชัก

SZ type: focal clonic Left and right

PE: V/S: T 38.2°C, Drowsiness

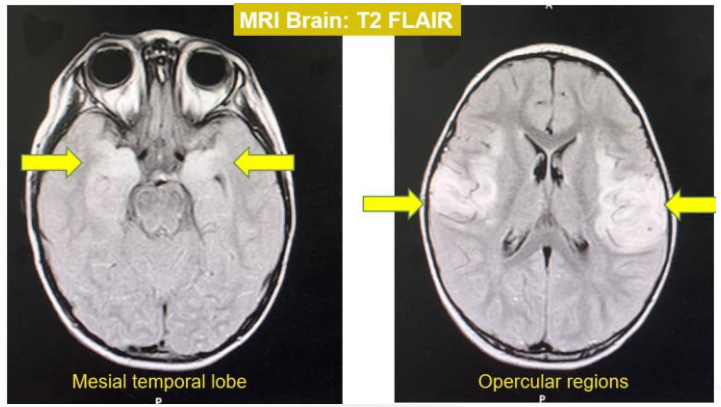
Lab: LP: RBC 10, WBC 90 (PMN 4%, Lymphocyte 90%), protein 55 mg/dl, sugar 72 mg/dl, DTX 104

CSF gram stain: not seen organism

CSF culture: no growth

PCR for HSV1: positive

Dx: Herpes simplex encephalitis



Management:
- Specific treatment: acyclovir x 4 weeks
- Supportive treatment: controlled seizures: phenobarbital, levetiracetam

Day 27 after acyclovir IV

The pt. had new onset focal seizures and dyskinesia

DDX: Relapse herpes encephalitis

Autoimmune encephalitis

Investigation:

CSF for Herpes 7 in 1: negative

CSF for anti-NMDAR: positive

Serum for anti-NMDAR: positive

Dx: Anti-NMDAR encephalitis

Management: IVIG
controlled seizures: phenobarbital, levetiracetam

อายุ 4 ปี

sz type: impaired awareness

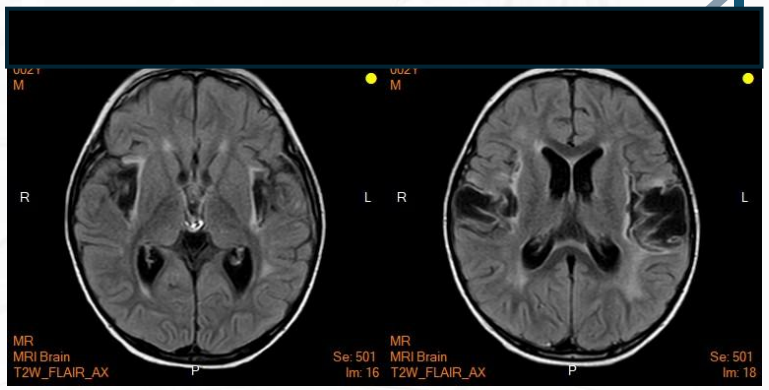
Frequency: 2-3 times/ day

Lab: LP: no RBC, no WBC

CSF for anti-NMDAR: negative

Serum for anti-NMDAR: negative

Dx: ?



Treatment plan?

Case 3: A 4-year-old boy, RH

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Autoimmune encephalitis

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Serum for anti-NMDAR: positive

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อายุ 4 ปี

sz type: impaired awareness

Frequency: 2-3 times/ day

Lab: LP: no RBC, no WBC

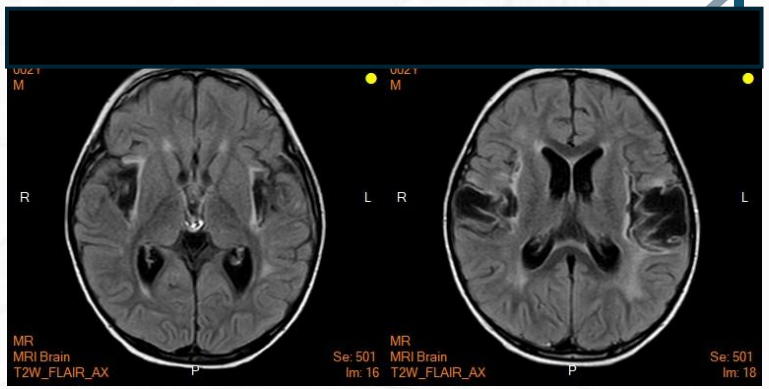
CSF for anti-NMDAR: negative

Serum for anti-NMDAR: negative

Dx: AEAE

- **All five** criteria proposed for AEAE-surface-antibody AE

1. Previous AE: NMDAR, LGI1, CASPR2 or GABABR
2. Persisting seizures > 2 years after immunotherapy initiation
3. No signs of encephalitis on MRI; hippocampal sclerosis allowed and no FDG-PET hypermetabolism
4. Normal CSF cell count
5. Substantial decrease of antibody titers



Management: controlled seizures
LEV 60 mg/day
TPX 10 mg/day
VPA 35 MKD
Clona 0.25 mg/day

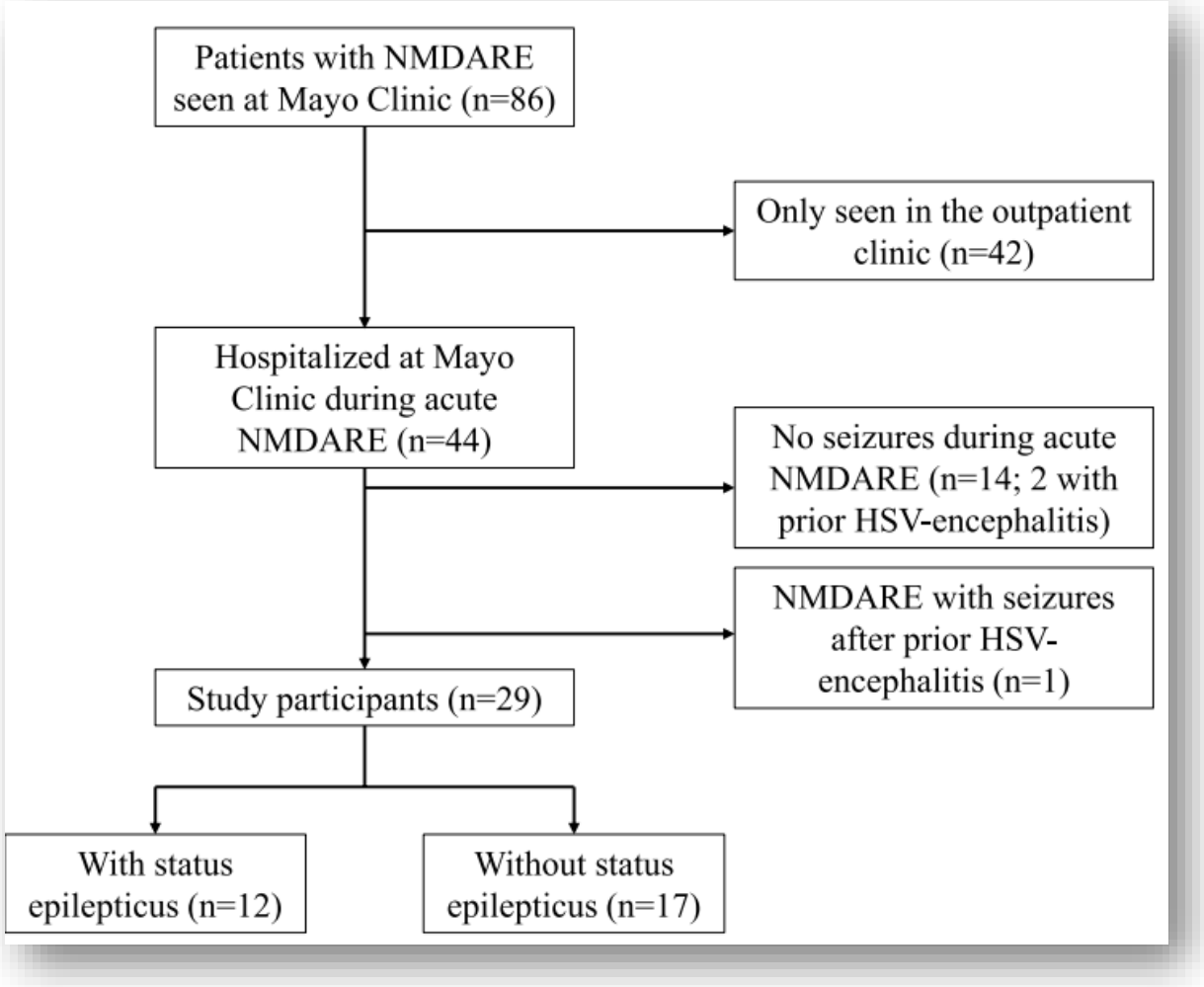
Anti NMDAR encephalitis

Frequency	Most common antibody target in pediatric AE
Clinical	Encephalitis with movement disorder, seizures, psychiatric symptoms, reduced verbal output/mutism, developmental regression (in young children), sleep dysfunction (mainly insomnia) and autonomic instability
MRI	Normal in at least 65% of pt.; T2/FLAIR lesions may be identified in the cortex, white matter, cerebellum or basal ganglia; reversible cerebral atrophy is a late finding
EEG	Abnormal in over 90% of patients-most have generalized slowing, but may see focal epileptiform activity, focal slowing or " prolonged spindles/ delta brush pattern"
Other	CSF antibody testing preferable to serum Increased association with tumors in females and in patients older than 12 Y

Cellucci T, et al. Neurol Neuroimmunol Neuroinflamm 2020

Seizures and status epilepticus in anti-NMDA receptor encephalitis

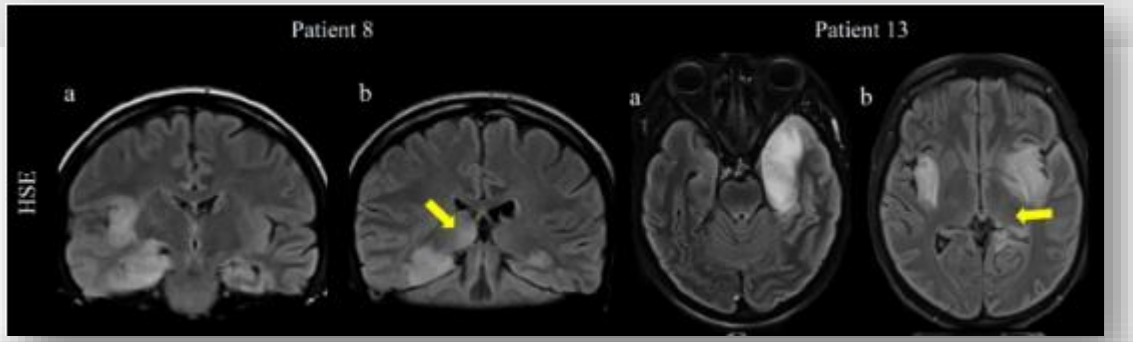
- N = 29
- Seizures were the first symptom
- Motor seizures in 87%
  - 53% bilateral tonic-clonic seizures
- Subclinical seizures occurred in 52%
- EEG abnormal in 90% (most temporal onset)
- Status epilepticus 41%
- Long-term outcomes:
  - 86% seizure-free by 6 months
  - 96% seizure-free by 2 years
  - Only 1 patient (3.4%)-> chronic epilepsy



Farooqi AM et al. J Neurol 2025

Specific clinical and radiological characteristics of anti-NMDA receptor autoimmune encephalitis following herpes encephalitis

- 13 patients with post-HSE anti-NMDAR encephalitis:
  - **69%** had seizures during the initial HSE
  - **62%** had seizures during the subsequent anti-NMDAR phase
- At the last follow-up, **12 of 13 patients (92%) remained on antiseizure treatment**
- Patients had more extensive and bilateral MRI abnormalities and worse 12-month functional outcomes than patients with non-HSV anti-NMDARE



N°, age, sex	NMDARE onset: time after HSE, symptoms	Peak mRS NMDARE	Immunotherapy	12-month mRS
N°1, 48, M	**Day 32.** Fever, behavior trouble (agitation), confusion, seizure, dyskinesia (orofacial), dysautonomia, vigilance disorders, ataxia	5	IvMP, IvIg, rituximab	Death at 3 months
N°2, 52, F	**Day 46.** Severe aphasia, depression, dyskinesia (orofacial), urinary retention, apraxia	5	IvMP, IvIg, cyclophosphamide, mycophenolate mofetil	2
N°3, 6 mo, M	**Day 26.** Behavior trouble (agitation), hypotonia, focal seizures	4	IvMP	1
N°4, 13, M	**Day 35.** Behavior trouble (agitation), anxiety, aggressiveness, depression	4	IvIg, rituximab	1
N°5, 11, F	**Unknown.** Fever, alertness disorder, behavior trouble (auto aggressive), psychiatric symptoms	5	IvIg	4
N°6, 49, M	**Day 24.** Dystonia (larynx and diaphragm), memory disorder, central ventilatory disorder, vigilance disorders, tremor	4	IvIg	2
N°7, 1, M	**Day 24.** Choreoathetosis, dyskinesia (orofacial), behavior trouble (agitation), seizures, swallowing disorders, hypotonia	5	IvMP, IvIg, rituximab	1
N°8, 30, F	**Day 33.** Confusion, delirium, memory disorder, alertness disorder, polydipsia, dyskinesia (orofacial), ataxia, seizures, limb paresis, urinary retention, apraxia	5	IvMP, IvIg, rituximab, cyclophosphamide	3
N°9, 3, F	**Day 25.** Fever, vigilance disorders, behavior trouble (agitation), dystonia (limbs and face), dyskinesia (limbs), hypotonia, hemiparesis, choreoathetosis, swallowing disorders, dysautonomia	5	IvMP, IvIg, PLEX, rituximab, ITh methotrexate	Unknown
N°10, 73, M	**Day 43.** Fever, vigilance disorders, swallowing disorders, memory disorders, hypotonia	5	IvMP, IvIg, rituximab	3
N°11, 10 mo, F	**Day 27.** Fever, dyskinesia (facial, limbs), sleep troubles (insomnia), hemiparesis, seizures, spasms	5	IvMP, IvIg, rituximab	4
N°12, 51, H	**Day 45.** Behavior trouble, seizures, confusion, depression, memory disorder	5	IvMP, IvIg, PLEX, rituximab	2
N°13, 19, F	**Day 21.** Behavior trouble (agitation), psychiatric symptoms (delirium, anxiety, depression), dysautonomia, confusion, memory disorder, aphasia	5	IvIg, cyclophosphamide, rituximab	1

Dumez, P. J Neurol 2024

EST 30th ANNIVERSARY ANNUAL MEETING

Practical Bedside Strategy for Suspecting Autoimmune Epilepsy

Step 1. Identify "Red Flags"

New-onset focal epilepsy (<12 months)
(esp. adult or adolescent onset)

Rapid progression (days–weeks)

High seizure frequency

Early drug resistance

New-onset status epilepticus/NORSE/FIRES

Faciobrachial dystonic seizures (LGI1)

Multifocal seizures

Associated Neurological Symptoms:
- Memory impairment/ Psychiatric symptoms/ Movement disorders

Step 2. Look for Autoimmune Clues

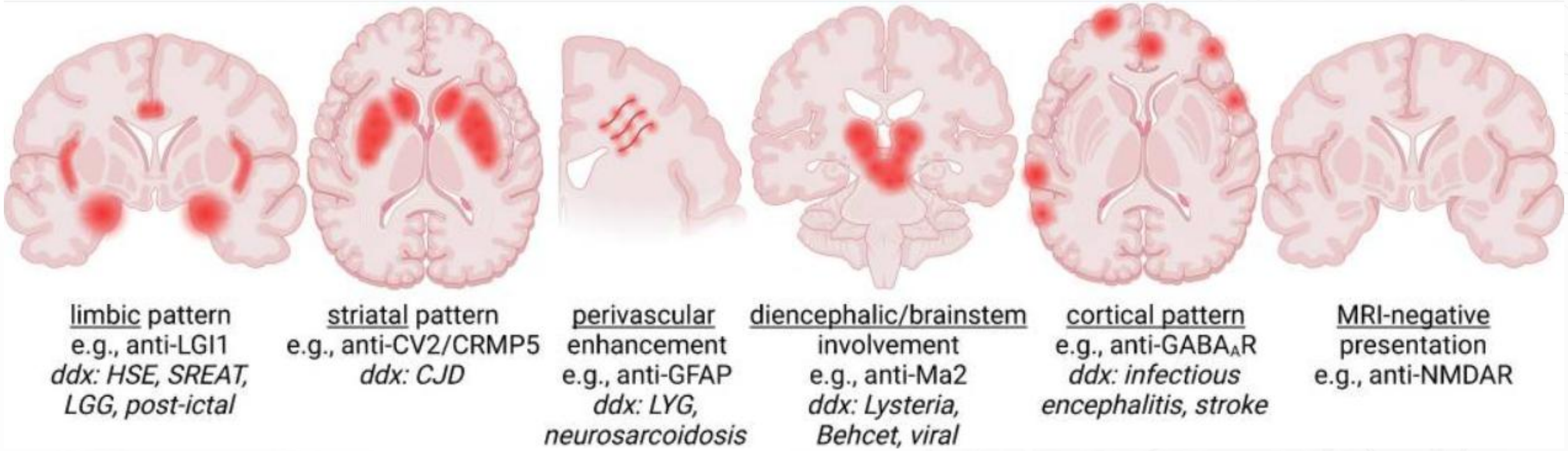
History

✓ Personal autoimmune disease
- Thyroiditis
- Type 1 diabetes
- Lupus
- Celiac disease

✓ Family history of autoimmune disease
✓ Recent viral illness
✓ History of cancer
✓ Hyponatremia (particularly LGI1)

Graus F et al. *Lancet Neurol.* 2016; Dubey et al. epilepsia 2019; De Bruijn et al. Ann Neurol 2021

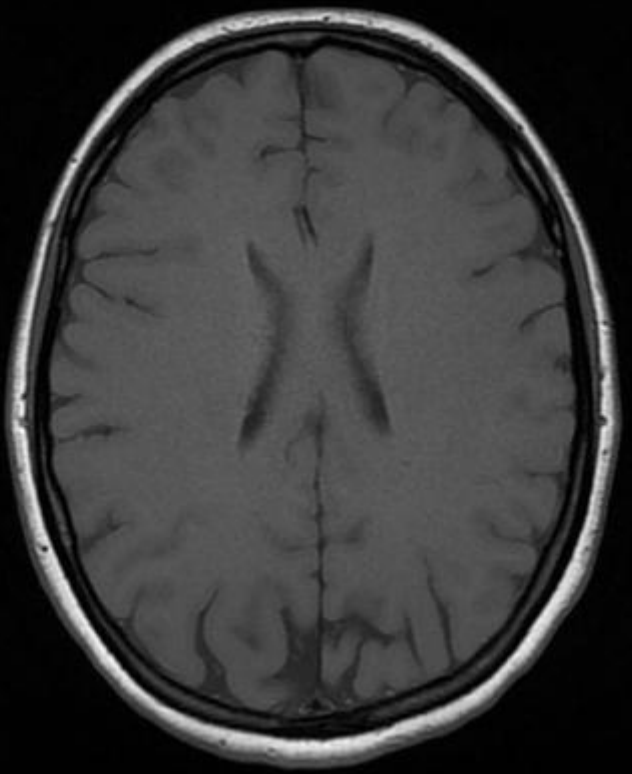
Step 3. MRI findings in Autoimmune epilepsy



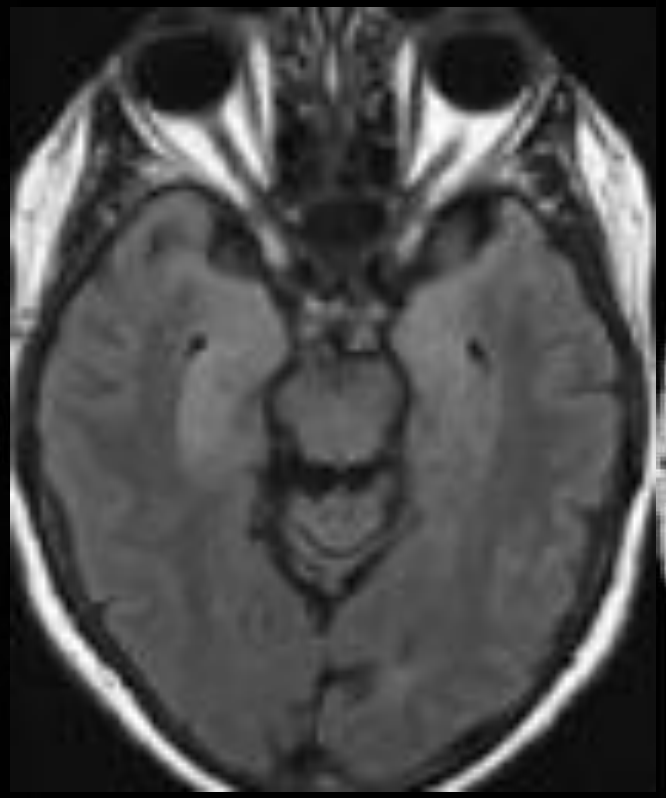
HSE = Herpes simplex encephalitis, SREAT = steroid-responsive encephalopathy associated with autoimmune thyroiditis, LGG = low-grade glioma, CJD = Creutzfeldt-Jakob Disease, LYG = lymphomatoid granulomatosis.

Sanvito F. Neuroradiology. 2024

MRI findings

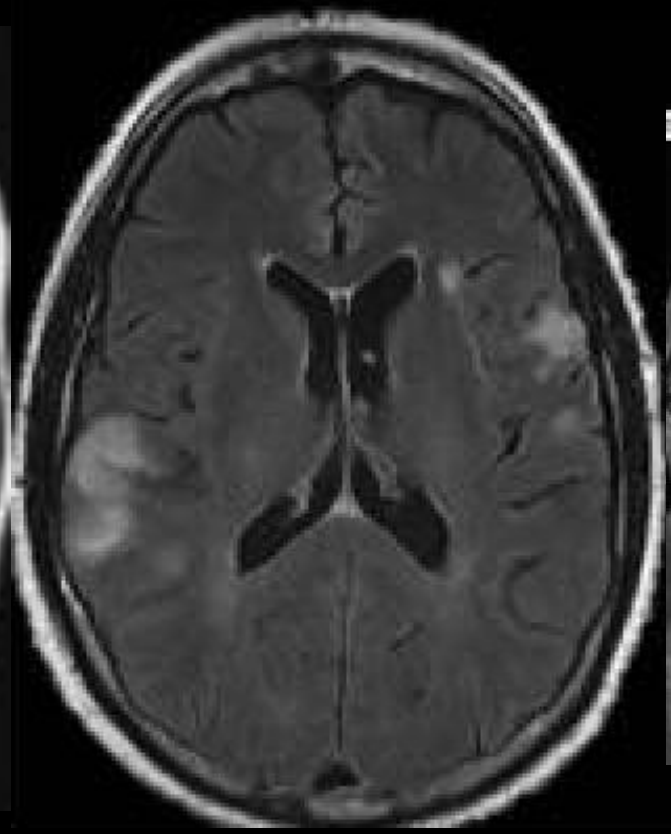


53% normal MRI in anti-NMDAR encephalitis

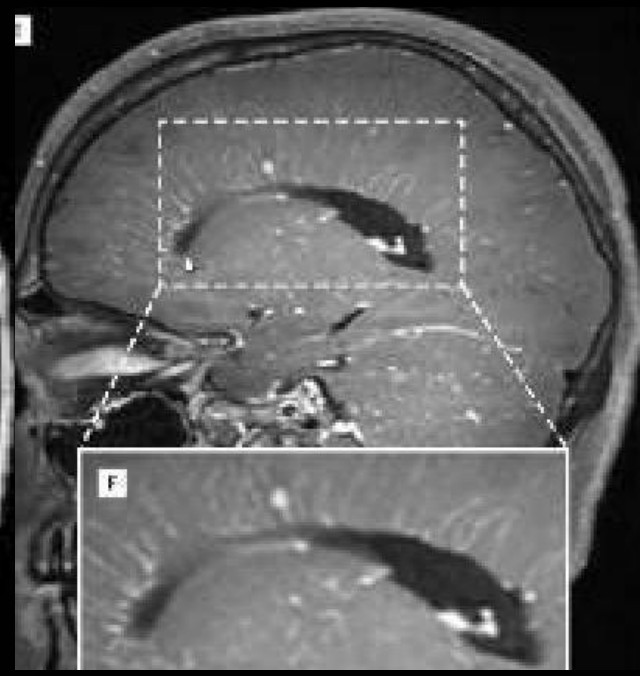


Ab associated Limbic encephalitis:

- **LGI1**
- **CASPR2**
- ANNA1
- $GABA_BR$
- AMPAR
- Ma2



Cortical lesions:
$GABA_AR$
MOG-IgG



Perivascular enhancement:
GFAP-IgG

Practical Bedside Strategy for Suspecting Autoimmune Epilepsy

Step 4. EEG

Focal slowing

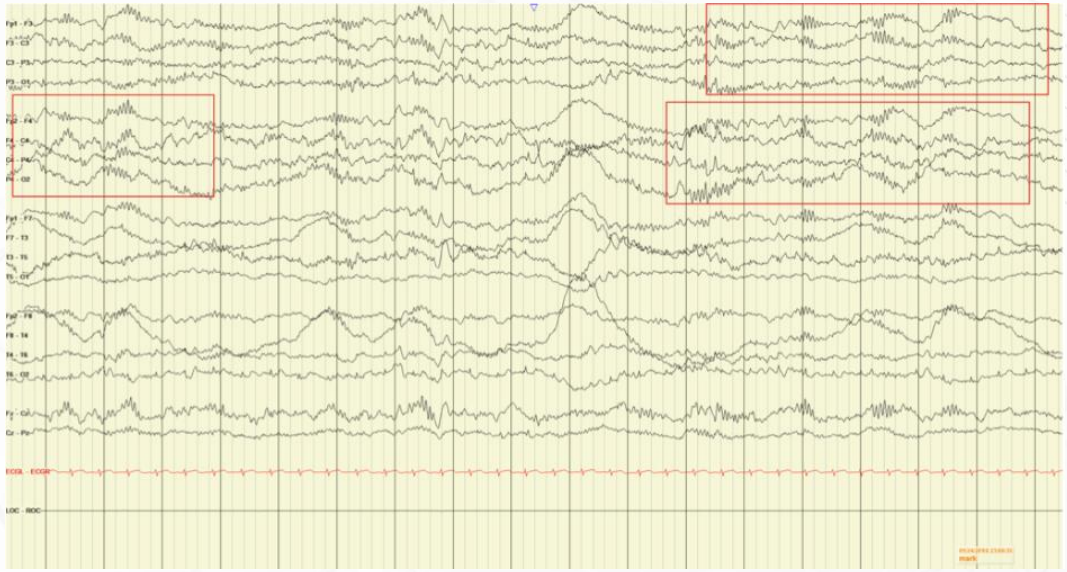
Temporal slowing

Multifocal epileptiform discharges

Frequent electrographic seizures

Extreme delta brush (anti-NMDAR)

Lateralized periodic discharges



extreme delta brush pattern in anti-NMDAR encephalitis

May guide some diseases but not specific

Antibody Prevalence in Epilepsy and Encephalopathy [APE2] score

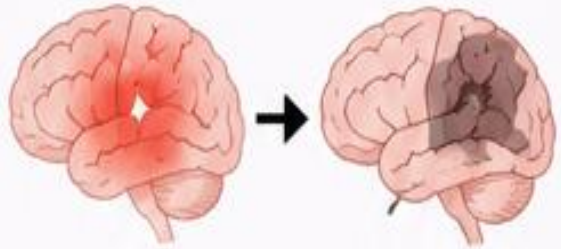
1A: Antibody Prevalence in Epilepsy and Encephalopathy (APE2) score	Value
New-onset, rapidly progressive mental status changes that developed over 1–6 weeks or new-onset seizure activity (within 1 year of evaluation)	(+1)
Neuropsychiatric changes: agitation, aggressiveness, emotional lability	(+1)
Autonomic dysfunction (sustained atrial tachycardia or bradycardia, orthostatic hypotension, hyperhidrosis, persistently labile blood pressure, ventricular tachycardia, cardiac asystole or gastrointestinal dysmotility)*	(+1)
Viral prodrome (rhinorrhea, sore throat, low-grade fever) to be scored in the absence of underlying systemic malignancy within 5 years of neurological symptom onset	(+2)
Faciobrachial dystonic seizures	(+3)
Facial dyskinesias, to be scored in the absence of faciobrachial dystonic seizures	(+2)
Seizure refractory to at least to two antiseizure medications	(+2)
CSF findings consistent with inflammation† (elevated CSF protein > 50 mg/dL and/or lymphocytic pleocytosis > 5 cells/mcL, if the total number of CSF RBC is < 1000 cells/mcL)	(+2)
Brain MRI suggesting encephalitis (T2/FLAIR hyperintensity restricted to one or both medial temporal lobes, or multifocal in gray matter, white matter, or both compatible with demyelination or inflammation)	(+2)
Systemic cancer diagnosed within 5 years of neurological symptom onset (excluding cutaneous squamous cell carcinoma, basal cell carcinoma, brain tumor, cancer with brain metastasis)	(+2)
	Total (max, 18)

- Predicts neural antibody positivity in the patient with seizures
- APE2 score ≥ 4 -> sensitivity 98% and specificity 85%
- An APE2 score ≥ 7 had specificity of 100% for an autoimmune etiology of epilepsy

Husari et al Neurotherapeutics 2019

Practical Bedside Strategy for Suspecting Autoimmune Epilepsy

Step 6. Treatment: Diagnosis and phase-dependent

1. ACUTE SYMPTOMATIC SEIZURES Secondary to Autoimmune Encephalitis	2. AUTOIMMUNE-ASSOCIATED EPILEPSY (AAE) Chronic epilepsy caused by autoimmune brain injury	3. AUTOIMMUNE ENCEPHALITIS-ASSOCIATED EPILEPSY (AEAE) Epilepsy that develops after a previous episode of autoimmune encephalitis
 Active inflammation → neuronal hyperexcitability	 Autoimmune-mediated injury → structural damage & gliosis → chronic epileptogenic network	 Past encephalitis → Residual injury → chronic epilepsy
THERAPY (PRINCIPLE) • Immunotherapy is the main treatment – IV methylprednisolone – IVIG – Plasma exchange – ± Rituximab/Cyclophosphamide • ASMs are supportive only	• Immunotherapy usually has limited benefit (unless active inflammation recurs) • Long-term antiseizure medications (ASMs) are the mainstay • Epilepsy surgery/neuromodulation in selected cases	• Immunotherapy only if there is evidence of active or relapsing inflammation • Long-term ASMs are the mainstay • Epilepsy surgery/neuromodulation for drug-resistant cases

Take-Home Messages

- Suspect autoimmunity in patients with new-onset focal epilepsy presents with red flags (clinical + paraclinical)
  - Rapid onset, early drug resistance, cognitive/psychiatric symptoms
  - MRI/CSF inflammation, or associated autoimmune disease
- Treat early when autoimmune encephalitis is highly suspected
- Long-term antiseizure medication is not required for every patient with AE
  - Acute symptomatic seizures: may become seizure-free after resolution of inflammation
- Identify classification guides prognosis and treatment
  - Acute symptomatic seizures may resolve after immunotherapy
  - AAE and AEAE often require long-term ASMs, with immunotherapy is less promising


สมาคมโรคลมชักแห่งประเทศไทย
EPILEPSY SOCIETY OF THAILAND
30 Years of Epilepsy Care in Thailand: From Foundations to Future Outcomes
INTEGRATING CLINICAL EXPERIENCE, INNOVATION, AND PRECISION CARE
6 - 8 AUG 2026
ห้องประชุมราชมณเฑียร แกรนด์บอลรูม
โรงแรมมณเฑียร สุรวงศ์ กรุงเทพฯ
Thursday
6 AUG 2026
DAY 1
Foundations & The Clinical Frontier
Focus: Honoring the past (1996–2026), current standard of care, and optimizing medical management.
08:00 - 08:30 Registration & Opening Ceremony Video Tribute: "3 Decades of the EST"
SPEAKER EST President & Organizing Committee
08:30 - 09:00 Epilepsy Society of Thailand (EST) & 30 Years of Epilepsy Care in Thailand
SPEAKER ศ.นพ.อนันต์นิตย์ วิสุทธิพันธ์
09:00 - 09:30 The Changing Landscape of Anti-Seizure Medications (ASMs)
SPEAKER พล.ท. ดร. นพ.โยธิน ชินวลัญช์
09:30 - 10:00 Genetic Testing in Epilepsy: Who, When, Interpretation, and Clinical Utility
SPEAKER Prof. Ingrid Scheffer
10:00 - 10:30 Coffee Break
10:30 - 11:10 Neonatal and Infantile Epilepsies: Early Intervention and Long-Term Outcomes
SPEAKER รศ.พญ.ณัฐรุจี วิวรรธนดิฐกุล
SPEAKER ผศ.พญ.กมรวรรณ กตัญญวงศ์
11:10 - 11:40 Neurostimulation Therapies: VNS, RNS, and DBS - Current Evidence and Practical Use in Thailand
SPEAKER รศ.นพ.อัตถพร บุญเกิด
11:40 - 12:40 Luncheon Symposiums sponsored by Eisai: Precision Medicine in Genetic Epilepsy from Childhood to Adulthood
SPEAKER Prof. Ingrid Scheffer
12:40 - 13:25 Epilepsy Society of Thailand Meeting
13:25 - 14:25 Luncheon Symposiums Sponsored by Zuellig Pharma: Early ASM Optimization: The Key for Acute Seizure
SPEAKER รศ.พญ.กนกวรรณ บุญญพิสิฏฐ์
SPEAKER รศ.นพ.สุรชัย ลิขสิทธิ์วัฒนกุล
14:25 - 15:05 The "First Seizure" Dilemma – To Treat or Not to Treat
SPEAKER อ.นพ.ศรัทธาวุธ วงษ์เวียงจันทร์
SPEAKER อ.นพ.พีรสิทธิ์ ตรีสุทธาชีพ
15:05 - 15:35 Afternoon Break
15:35 - 16:15 Autoimmune Epilepsy: Diagnostic Challenges, Treatment Strategies, and Case Presentation
SPEAKER อ.นพ.เมธา อภิวัฒนากุล
SPEAKER พ.อ.หญิง รศ.ภิรดี สุวรรณภักดี
16:15 - 16:45 Cannabinoids in Thailand: 5-Year Outcomes
SPEAKER อ.พญ.อาภาศรี ลุสวัสดิ์
16:45 End of Day 1 – Gala Dinner (Open 17:30-18:00)
Friday
7 AUG 2026
DAY 2
Innovation & Future Outcomes
Focus: Surgical advances, Technology (AI/Neurostimulation), and Improving quality of life.
08:30 - 09:00 Real-World Evidence in Epilepsy Care: Bridging Clinical Trials and Daily Practice
SPEAKER ศ.นพ.คณิตพงษ์ ปราบพาล
09:00 - 09:30 Advanced Neuroimaging in Epilepsy Beyond MRI: Post-processing, Functional and Network-Based Imaging
SPEAKER อ.พญ.กาญจนา อั๋นวงศ์
09:30 - 10:00 Continuous EEG Monitoring Detects More Seizures, but Does It Improve Outcomes?
SPEAKER Prof. Cecil Hahn
10:00 - 10:30 Coffee Break
10:30 - 11:00 Evolution of Epilepsy Surgery in Thailand
SPEAKER อ.นพ.ธีรเดช ศรีกิจวิไลกุล
11:00 - 11:30 Epilepsy in the Elderly: Diagnostic Complexity and Treatment Considerations
SPEAKER อ.พญ.ฐาปนี สมบูรณ์
11:30 - 12:00 Sleep and Epilepsy: The Bidirectional Relationship
SPEAKER รศ.นพ.ทายาท ดีสุดจิต
12:00 - 13:00 Management of Status Epilepticus: Current Evidence and Future Directions
SPEAKER Prof. Cecil Hahn
13:00 - 13:30 Cognitive and Behavioral Comorbidities in Epilepsy: Assessment and Management
SPEAKER รศ.นพ.พร กิลยากร
13:30 - 14:00 Integrating Neurotechnology and Artificial Intelligence(AI) in Epilepsy Care: Current Applications and Future Potential
SPEAKER รศ.ดร.นพ.ชูศักดิ์ ลิโมทัย
14:00 - 14:30 Afternoon Break
14:30 - 15:10 SUDEP: Risk Stratification, Prevention, and Communication with Families
SPEAKER รศ.นพ.อธิวัฒน์ สุนทรพันธ์
SPEAKER ผศ.(พิเศษ)นพ.กุลเสฏฐ ศักดิ์พิชัยสกุล
15:10 - 15:40 The Future of the Epilepsy Society of Thailand: Innovation, Collaboration, and Sustainability
SPEAKER รศ.พญ.กนกวรรณ บุญญพิสิฏฐ์ นายกสมาคมโรคลมชักแห่งประเทศไทย
15:40 Closing
Saturday
8 AUG 2026 (Advanced EEG Workshop)
DAY 3
EEG in Critical Care Course
Navigating Uncertainty: Advanced EEG, Prognostication & AI
08:30 - 08:45 Opening Remarks: Course overview & role of EEG in neurocritical care
08:45 - 09:15 Keynote: EEG in Critical Care – From Pattern to Decision: Translating EEG into clinical action
09:15 - 09:45 Ictal–Interictal Continuum & Pattern Interpretation: LPDs, GPDs, LRDA, GRDA; treatment thresholds
09:45 - 10:00 Coffee Break
10:00 - 11:00 Hands-on EEG Workshop 1 (Small Groups)
11:00 - 11:30 NCSE: Real-World Diagnostic Challenges: Salzburg criteria, borderline patterns, pitfalls
11:30 - 12:00 EEG-Guided Treatment Strategies: Treat vs observe, ASM escalation, anesthetic coma
12:00 - 13:00 Lunch Break
13:00 - 14:00 Hands-on EEG Workshop 2 (Small Groups)
14:00 - 14:30 Prognostication & Biomarkers Integration: EEG + Other markers
14:30 - 15:00 Quantitative EEG in Practice: Spectrograms, seizure trends, ICU pitfalls
15:00 - 15:45 Hands-on EEG Workshop 3 (Small Groups)
ค่าลงทะเบียน 2 วัน งานประชุมวิชาการประจำปี
สมาชิก 3,000 บาท | ไม่ใช่สมาชิก 3,500 บาท
ค่าลงทะเบียนทั้ง 3 วัน
สมาชิก 4,500 บาท | ไม่ใช่สมาชิก 5,000 บาท
ค่าลงทะเบียน EEG in Critical Care Course
สมาชิก 2,000 บาท | ไม่ใช่สมาชิก 2,500 บาท
"Advanced EEG Workshop รับจำนวนจำกัด เพียง 100 ท่านแรก"
วิธีการชำระเงิน ชำระเป็นเช็ค สั่งจ่าย "สมาคมโรคลมชักแห่งประเทศไทย"
โอนเงินเข้าบัญชี ชื่อ "สมาคมโรคลมชักแห่งประเทศไทย"
ธนาคารไทยพาณิชย์ สาขารามาธิบดี
เลขที่บัญชี 026-2-86057-1
ติดต่อสอบถามรายละเอียดเพิ่มเติมได้ที่
คุณพิมพ์ชญา ปักศรีสิงห์ สมาคมโรคลมชักแห่งประเทศไทย
อาคารเฉลิมพระบารมี ๕๐ ปี ชั้น 7 เลขที่ 2 ซอยศูนย์วิจัย ถนนเพชรบุรีตัดใหม่ แขวงบางกะปิ เขตห้วยขวาง กรุงเทพฯ 10310 โทรศัพท์. 02-716-5114
Email: epilepsy09@gmail.com
SCAN TO REGISTER
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