

# **Recognition of Anti-NMDA receptor encephalitis in children**

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# Disclosures

NA

# Case: Previously well 14-year-old girl

## 1 Presentation (Day 1)

- Previously healthy 14-year-old girl
- Slurred speech and lip biting
- Altered mental status
- Irritability and erratic behaviour

## 2 Examination

- Long tract signs
  - Hyperreflexia
  - Sustained clonus bilaterally

### Pause and consider

A teenager with new speech change, abnormal movements and behaviour change: primary psychiatric illness, epilepsy or encephalitis?

## Format



### Define

encephalitis,  
autoimmune  
encephalitis and anti-  
NMDAR encephalitis



### Diagnostic criteria

probable and definite  
diagnostic criteria,  
including paediatric  
criteria



### Recognise

the clinical presentation  
in younger children and  
adolescents



### Complications

acute and long-term  
complications



### Investigations

CSF, paired serum, EEG,  
MRI and tumour  
screening



### Manage

Early referral and  
immunotherapy,  
without waiting for  
antibodies

# What is anti-NMDAR encephalitis?

- **Immune-mediated encephalitis described in 2007**
  - IgG antibodies target the GluN1 subunit of the NMDA receptor
- **Antibodies cause receptor internalisation, not neuronal death**
  - The illness is therefore potentially reversible
- **Most common antibody-mediated encephalitis in children**

Antibody binds GluN1



Receptors cross-linked and internalised



Fewer synaptic NMDA receptors

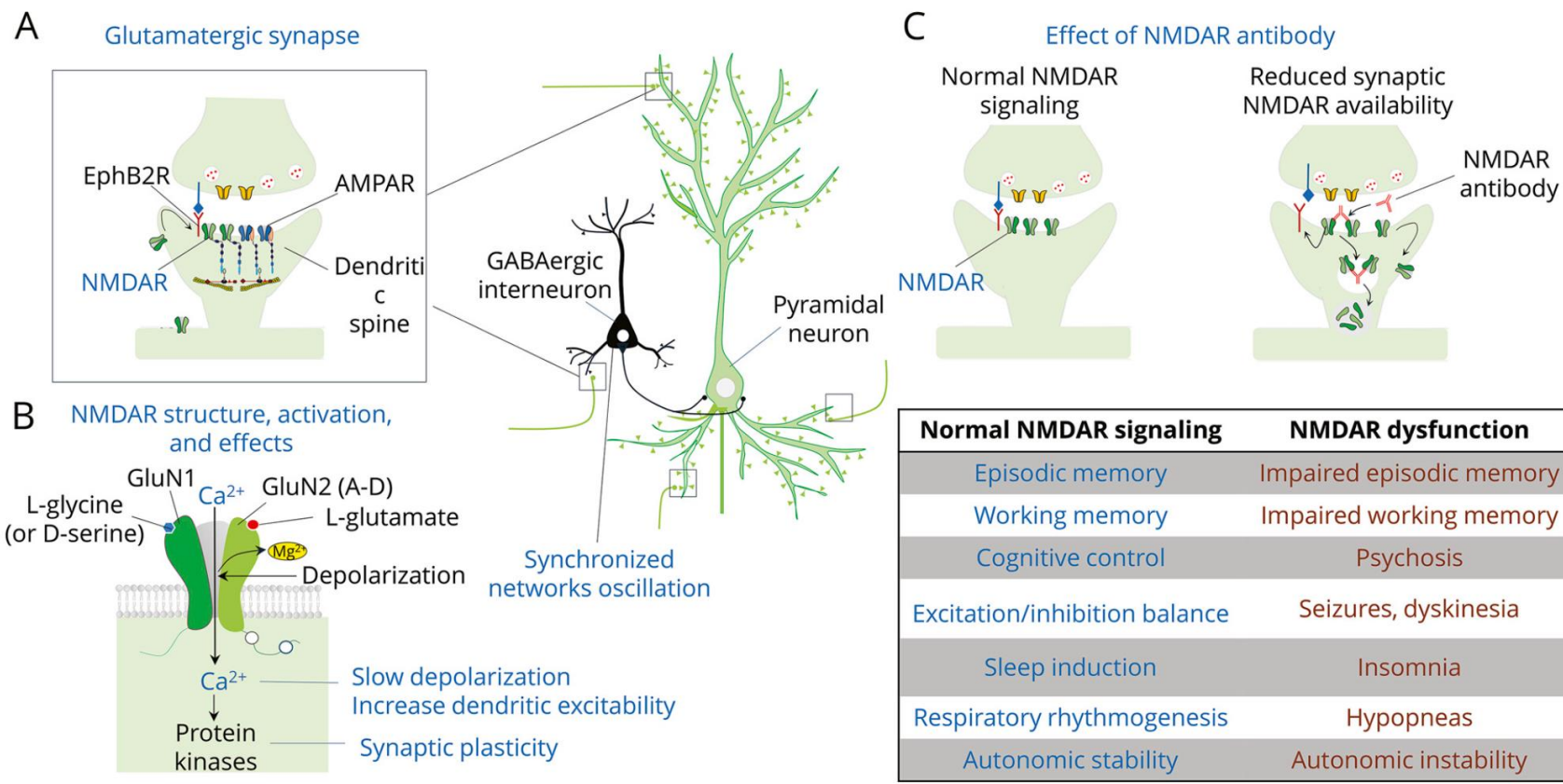


Psychosis, dyskinesia, seizures, dysautonomia

*Dalmau 2007; Dalmau 2008*

# Pathophysiology

**Figure 1** NMDAR-Mediated Neurotransmission and Proposed Mechanisms Underlying the Clinical Features of Anti-NMDAR Encephalitis



What Are the Proposed Mechanisms Contributing to the Manifestations of Anti-NMDAR Encephalitis? Andreu Vilaseca,<sup>1</sup> Josep O. Dalmau,<sup>2,3,4</sup> and Eduardo Benarroch<sup>1</sup> *Neurology*<sup>®</sup> 2025;105:e214232.

doi:10.1212/WNL.0000000000214232

# Definitions

## Encephalitis

**Brain inflammation** with altered mental status for 24 h or more, plus supportive features: fever, seizures, focal signs, CSF pleocytosis, or imaging or EEG change

## Autoimmune encephalitis

Non-infectious encephalitis caused by antibodies directed **against neuronal antigens**

## Anti-NMDAR encephalitis

IgG antibodies against the **GluN1 subunit of the NMDA receptor**

## NMDA receptor

Glutamate-gated ion channel (GluN1 + GluN2 subunits); essential for **synaptic plasticity, memory and behaviour**

## Paraneoplastic

Triggered by a tumour that **expresses the antigen**, typically an ovarian teratoma

## Probable vs definite

**Probable:** clinical criteria met, antibodies pending.

**Definite:** compatible illness plus IgG anti-GluN1 antibodies

*Venkatesan 2013; Dalmau 2008; Graus 2016*

# Three recognised triggers



## Idiopathic

- Most children
- No tumour or infection found



## Tumour (teratoma)

- Mainly adolescent girls
- rare under 12 years.
- Rarely testicular germ-cell tumours in boys

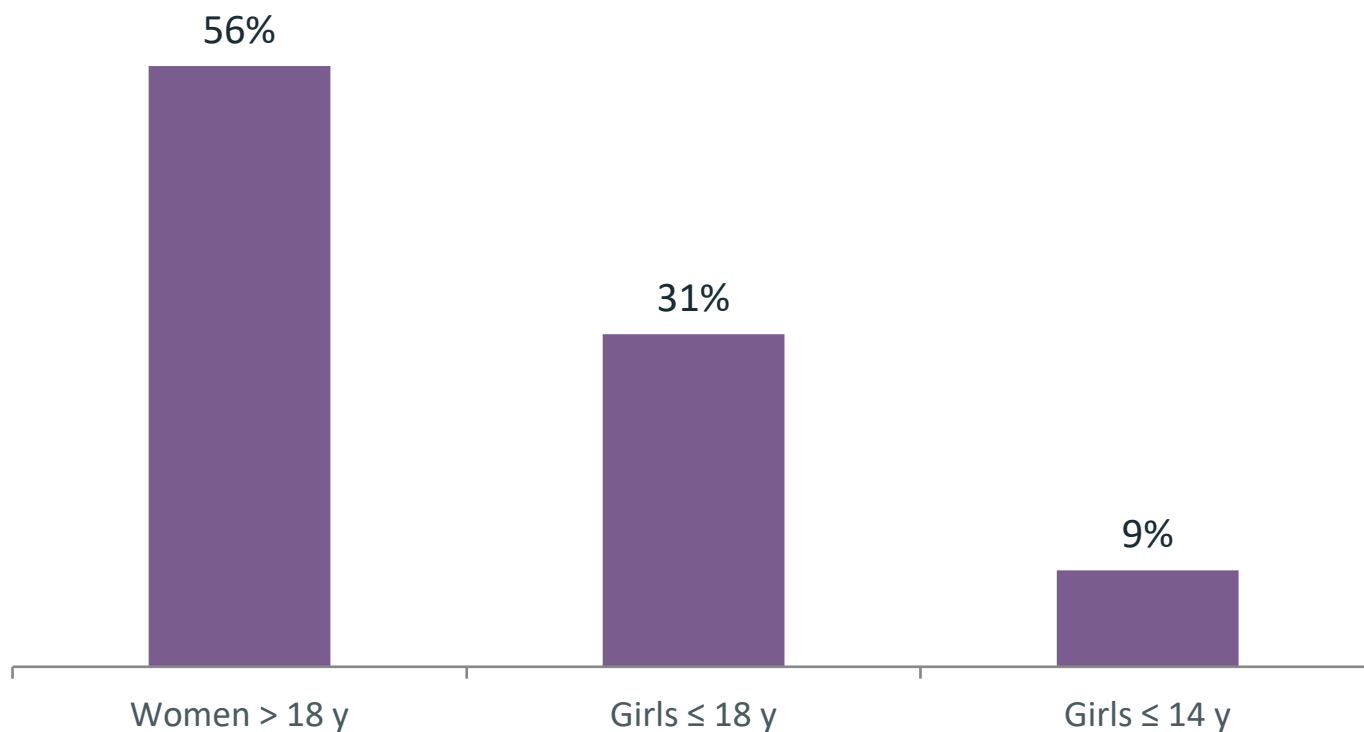


## After HSV encephalitis

- About 1 in 4 develop autoimmune encephalitis
- usually within 2 months

# Tumour risk falls sharply with younger age

Ovarian teratoma by age group (%)



## Children in the series

- 32 patients aged 23 months to 18 years
- 26 girls, 6 boys
- Behaviour or personality change was the first symptom in 28 of 32 (88%)
- Male predominance is weaker in young children

# Diagnostic Criteria: Probable anti-NMDAR encephalitis: the 2016 criteria

## 1. Rapid onset (< 3 months) of $\geq 4$ of 6:

- **Abnormal behaviour** or cognitive dysfunction
- **Speech dysfunction**
- **Seizures**
- Movement disorder, dyskinesia, rigidity or abnormal postures
- Decreased level of consciousness
- Autonomic dysfunction or central hypoventilation

MC-BASS

## 2. At least one of:

- **Abnormal EEG** (slowing, epileptic activity or extreme delta brush)
- CSF pleocytosis or **oligoclonal bands**

## 3. Other causes reasonably excluded

Or: **3 of the 6 groups plus a systemic teratoma**

# Definite anti-NMDAR encephalitis



- ≥ 1 of the 6 symptom groups
- + IgG anti-GluN1 antibodies
- + other disorders reasonably excluded

## Test CSF

CSF is the most reliable sample. Serum may be negative in a minority of true cases.

## If only serum is positive

Confirm with CSF or a second assay; low-titre serum results can be false positives.

## Paediatric autoimmune encephalitis criteria



### Compatible clinical picture

Acute or subacute onset of neurological and/or psychiatric symptoms in a previously well child



### Evidence of inflammation

CSF, MRI or EEG findings supporting an inflammatory process



### Alternatives excluded

Infection, metabolic, toxic and other causes reasonably excluded

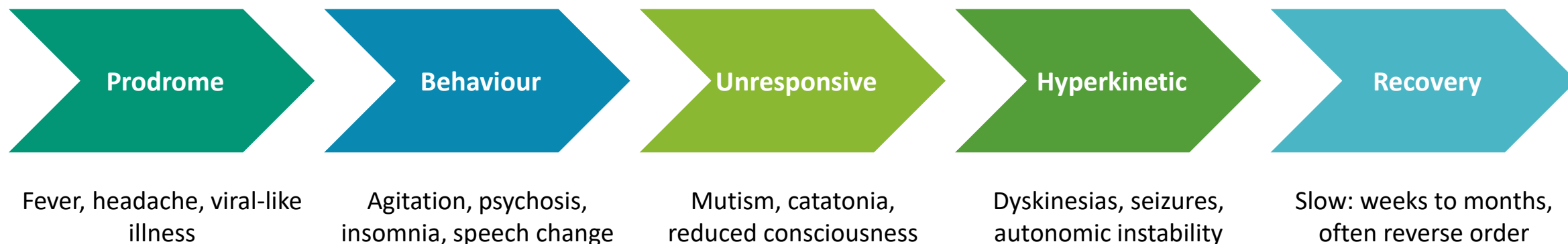


### Antibodies support

A positive antibody confirms subtype; a pending result should not delay treatment

*Cellucci 2020 (Neurol Neuroimmunol Neuroinflamm); provisional criteria*

# Clinical : The typical course unfolds in stages



## Children often present here

At the behaviour stage, when the illness looks psychiatric. Young children may instead present with seizures or abnormal movements first.

*Dalmau 2008; Florance 2009; Titulaer 2013*

# Clinical course

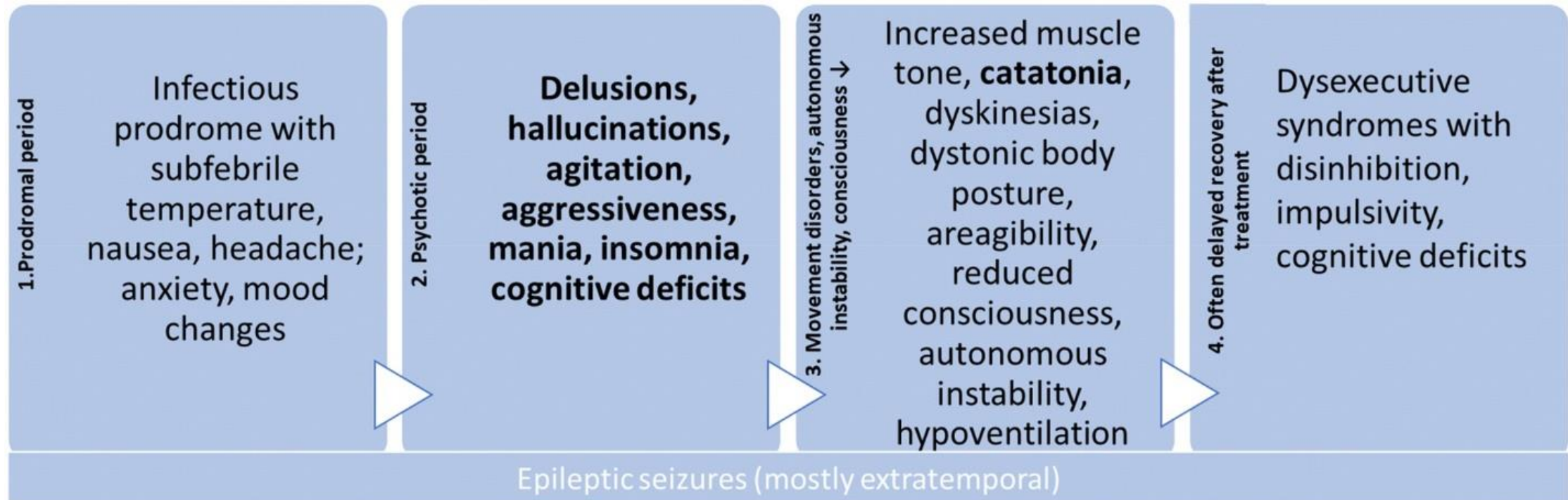


**Figure 1.** Typical course of anti-NMDA-R encephalitis Eur Arch Psychiatry Clin Neurosci. October 1, 2020.

# Behaviour change looks different in children



## Younger children

- Tantrums, irritability, aggression
- Hyperactivity or agitation
- Loss of toileting or other regression
- Fearfulness, inconsolable crying
- Rarely report hallucinations



## Adolescents

- Psychosis: hallucinations, delusions
- Anxiety, mood lability, mania
- Disinhibition, bizarre behaviour
- Catatonic features
- Often first seen by psychiatry

# Two subtle signs: speech and sleep



## Speech

- Reduced speech, progressing to mutism
- Echolalia, perseveration
- Pressured or incoherent speech
- A core criterion in diagnostic criteria



## Sleep

- Severe insomnia early in the illness
- Reversed sleep-wake cycle
- Hypersomnia during recovery
- Ask parents directly: it is rarely volunteered

# Seizures: often the first sign in young children

- Focal or generalised seizures; may progress to status epilepticus
- first symptom, particularly in the younger pt
- Two-thirds of patients initially present with convulsive seizures
  - Focal seizures- 47-79%
  - Status epilepticus in 30-40%
  - refractory in 21%
- Seizures plus new behaviour or sleep change, should prompt the question
- Dyskinesias are often mistaken for seizures, and vice versa
  - Video EEG helps separate the two and avoids escalating antiseizure drugs for movements



**Seizure + behaviour change =  
encephalitis work-up**

## Movement disorders: the most distinctive clue in children

### Orofacial dyskinesia

Lip smacking, chewing, grimacing, tongue thrusting

### Choreoathetosis

Writhing, flowing limb movements

### Dystonia & rigidity

Abnormal postures; may mimic neuroleptic reactions

### Stereotypies

Repetitive purposeless movements of limbs or trunk

### Catatonic signs

Posturing, waxy flexibility, staring

### Often misread

As seizures, functional disorder or drug reaction

*Florance 2009; Titulaer 2013; Graus 2016*

# Autonomic instability and central hypoventilation



## Heart rate & BP

Tachycardia  
Bradycardia  
labile blood pressure



## Temperature

Hyperthermia without infection



## Breathing

Central hypoventilation may need  
ventilatory support



## Other

Hypersalivation  
urinary retention  
sweating

*Dalmau 2008; Titulaer 2013*

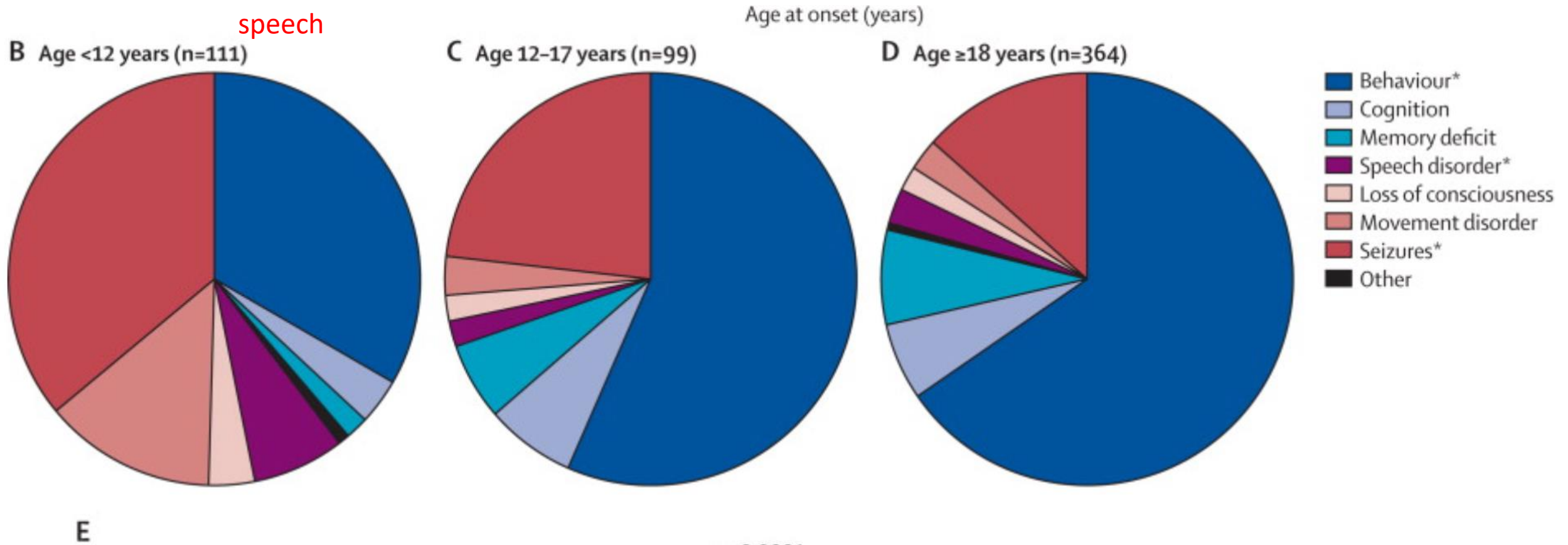
# Presentation differs with age

| Feature               | Young children (< 12 y)                           | Adolescents                             |
|-----------------------|---------------------------------------------------|-----------------------------------------|
| Usual first symptom   | Seizures, abnormal movements, behaviour change    | Psychiatric symptoms                    |
| Behaviour             | Tantrums, irritability, hyperactivity, regression | Psychosis, mania, catatonia             |
| Speech                | Reduced speech, mutism, regression                | Pressured speech, mutism                |
| Movement disorder     | Prominent: chorea, dystonia, dyskinesia           | Orofacial dyskinesia, catatonia         |
| Tumour                | Rare                                              | Ovarian teratoma in a minority of girls |
| Usually first seen by | Paediatrics, emergency, neurology                 | Psychiatry, emergency                   |

*Florance 2009; Titulaer 2013; Armangue 2018*

# Clinial presentation

Seizures  
behavior  
LOC  
speech



Titulaer M, McCracken L, Gabilondo I et al., **Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study**, The Lancet Neurology, 2013; 12, 157-165

# Red flags: when to suspect anti-NMDAR encephalitis



**Reduced speech or  
mutism**



**Abnormal movements,  
especially orofacial**



**Seizures with behaviour  
change**



**Severe insomnia**



**Autonomic instability or  
hypoventilation**



**Severe reaction to  
antipsychotics**



**Reduced consciousness  
or catatonia**



**Recent HSV  
encephalitis, or a  
prodromal fever**

# CSF: abnormal in most, but can be normal early

## Typical findings

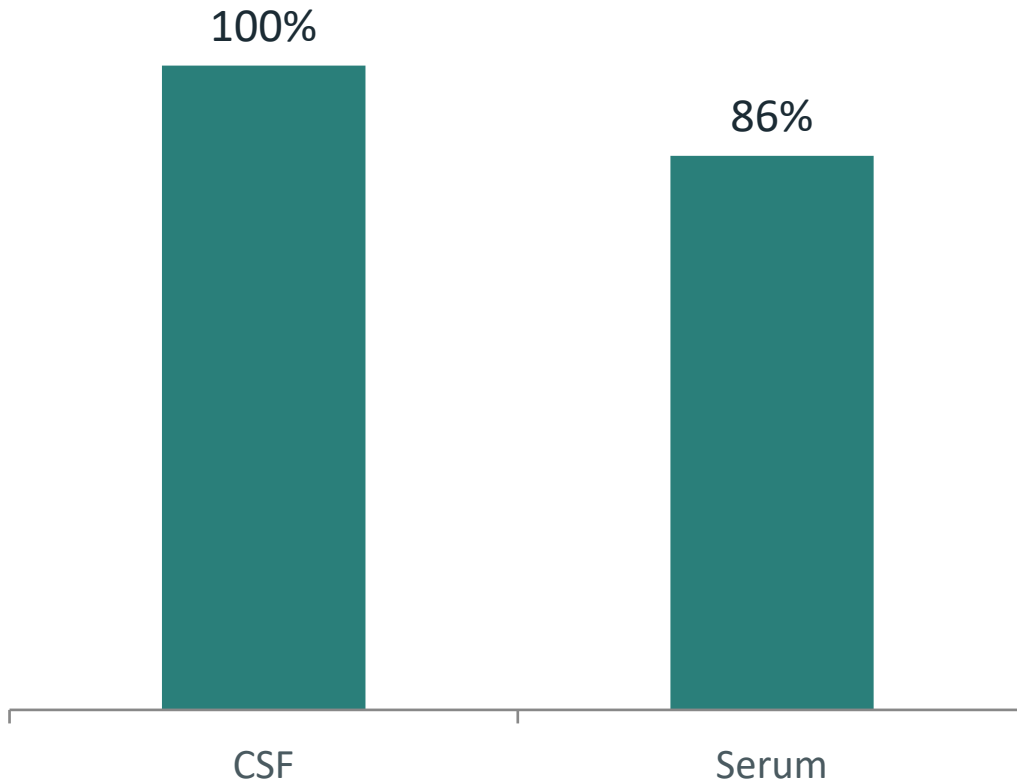
- Mild **lymphocytic pleocytosis**
- Normal or **mildly raised protein**
- normal glucose
- CSF-specific **oligoclonal bands** in some
- Can be normal, especially early

## Lab test

- Cell count, protein, glucose, culture
- HSV/enterovirus PCR (and others per context)
- **NMDAR antibodies (CSF + paired serum)**
- **Oligoclonal bands with paired serum**
- **Store extra CSF frozen**

# Antibody testing: CSF beats serum

Antibodies detected (%), paired samples

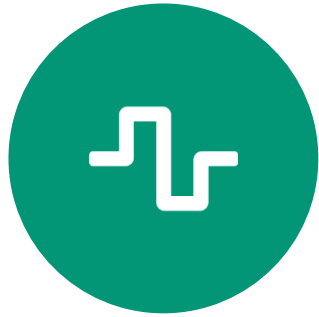


- **Always test CSF, with paired serum**
- **Cell-based assay for IgG anti-GluN1**
- **Results can take days to weeks**
  - Start treatment on clinical grounds
- **Titres do not reliably track recovery**

*Gresa-Arribas 2014 (Lancet Neurol)*

Anti-NMDAR encephalitis in children

# EEG: abnormal in most children



## Most common

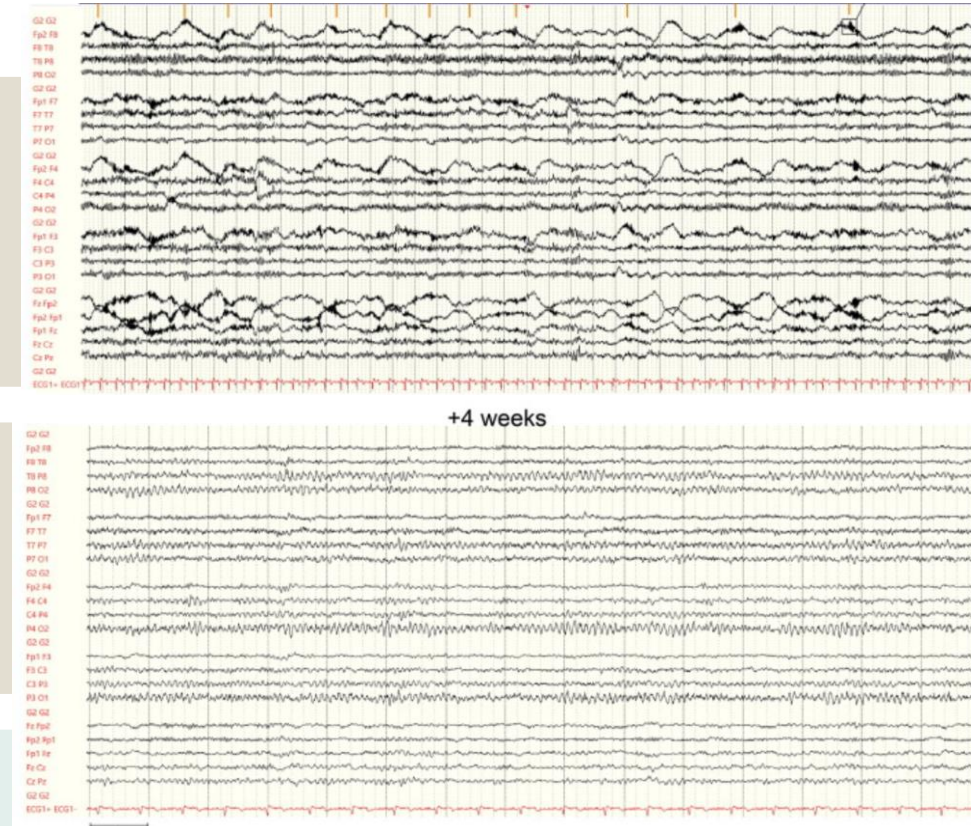
Diffuse or focal slowing  
disorganised background

## Supports diagnosis

Epileptiform discharges  
electrographic seizures

## Relatively specific, but uncommon

Extreme delta brush: delta activity with superimposed beta bursts



*Schmitt 2012 (Neurology); Graus 2016,*

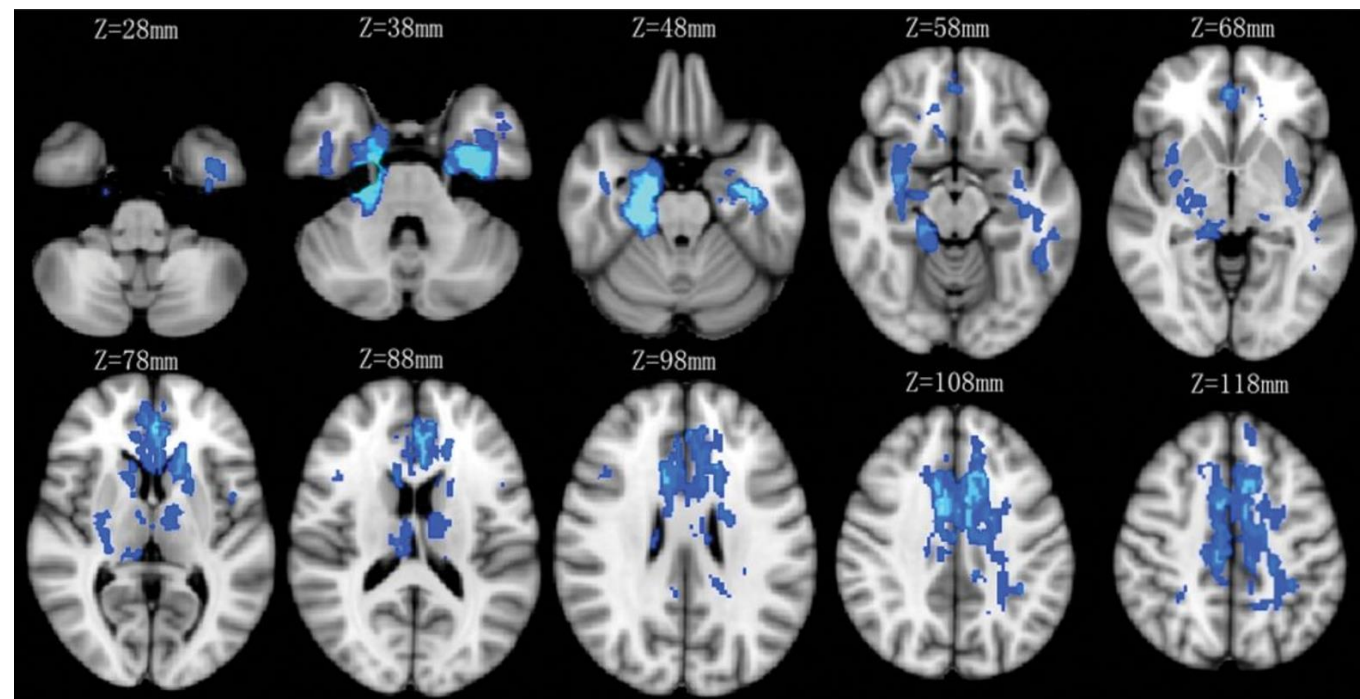
Anti-NMDAR encephalitis in children

*Christine Heuer Clinical Neurophysiology Practice, 2023,*

# MRI: often normal, and that is expected

>50% normal

- non-specific T2/FLAIR changes
  - Cortical, subcortical, **hippocampal**; often transient
- A normal MRI does not exclude the diagnosis
- MRI is still needed to exclude other causes
  - ADEM, MOG antibody disease, infection, tumour



T. Zhang et al. AJNR Am J Neuroradiol  
doi:10.3174/ajnr.A5593



*Dalmau 2008; Titulaer 2013*

*T Zhnag, 2018, AJNR*

# Tumour screening



## Females

- Pelvic ultrasound (transabdominal)
- MRI pelvis if available or if ultrasound is equivocal
- Repeat screening if poor response



## Males

- Testicular examination
- Testicular ultrasound
- Tumours are very rare

If a teratoma is found, treatment is surgical excision and improves outcome.

# Back to the case: Week 1 Ix: brain inflammation

## 1 CSF

- WBC 86, 96% lymphocytes
- 7 oligoclonal bands
- Raised kappa light chains

## 2 EEG

- Seizures of left temporal onset
- Later evolved to frontal onset

## 3 MRI brain

- Punctate T2 hyperintense foci
- Right basal ganglia
- Left cingulate white matter

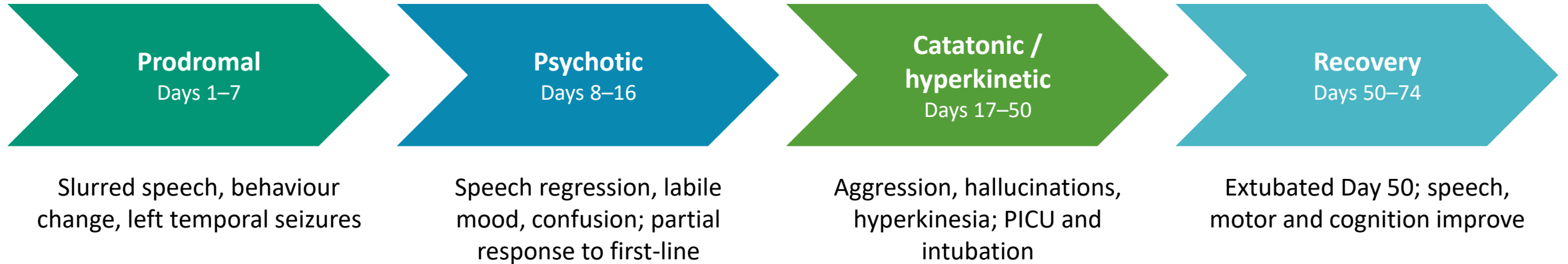
## 4 Pelvis

- 1.7 cm right adnexal cyst
- Tumour screen for teratoma

### Probable anti-NMDAR encephalitis before the antibody result

Rapid onset of behaviour change, speech dysfunction, seizures and reduced consciousness (4 of 6 groups), plus abnormal EEG and CSF pleocytosis with oligoclonal bands

# Course followed the classic stages



## Day 15: neuroleptic sensitivity

Risperidone caused dystonia and tongue thrusting, with acute deterioration. Seizure focus shifted from left temporal to frontal.

## Day 16: diagnosis confirmed

Anti-NMDAR antibodies positive. Transferred to PICU on Day 17 and ventilated for 33 days for refractory agitation and delirium.

# Immunotherapy escalated in stepwise

## 1 First-line

- **IVIG** over 4 days
- **IV methylprednisolone** × 5 days
- **Oral prednisone** taper
- Partial response, then deterioration

## 2 Second-line

- **Rituximab** Day 16; second dose delayed to Day 67 by B-cell depletion and infection
- **Plasma exchange** 15 sessions (usual 5–7)
- **IVIG replacement** × 2 for IgG < 109 mg/dL

## 3 Supportive

- **Seizures:** lacosamide from Day 1; oxcarbazepine added Day 15
- **Agitation:** > 10 sedative agents incl. ketamine, dexmedetomidine, phenobarbital
- **Sleep:** melatonin, sleep hygiene

# Complications and outcome

## ! Complications

- **Infection:** C. difficile colitis, Klebsiella UTI, S. aureus respiratory infection
- **Immune:** hypogammaglobulinaemia after PLEX; profound B-cell depletion
- **PLEX:** hypofibrinogenaemia (2-day pause), citrate hypocalcaemia, line pulled out
- **Other:** prolonged ventilation, sedative withdrawal risk, anaemia

74

days in hospital

33

days ventilated

## Outcome at discharge

- Alert, walking independently; home with outpatient rehabilitation
- Residual mild ataxia, cognitive impairment, dysgraphia
- EEG and MRI normal; IgG normalised
- Prednisone taper, monthly IVIG, TMP-SMX
- **Follow up: memory problems and insomnia**

# Differential diagnosis

| Group               | Consider                                                                                               |
|---------------------|--------------------------------------------------------------------------------------------------------|
| Infection           | HSV and other viral encephalitis; TB meningitis; HIV-related; neurosyphilis; cerebral malaria (travel) |
| Other immune        | ADEM; MOG antibody disease; other antibody encephalitis; lupus; Sydenham chorea                        |
| Psychiatric         | First-episode psychosis; catatonia of psychiatric origin                                               |
| Toxic / drug        | Neuroleptic malignant syndrome; serotonin syndrome; drug intoxication                                  |
| Metabolic / genetic | Inborn errors (e.g. urea cycle), Wilson disease, mitochondrial disease                                 |
| Other               | Non-convulsive status epilepticus; brain tumour; functional disorder                                   |

*Graus 2016; Cellucci 2020*

# First-line work-up checklist

## Lumbar puncture



Cell count, protein, glucose  
culture  
viral PCR  
oligoclonal bands  
NMDAR antibodies  
store extra CSF

## Blood



NMDAR antibodies (paired serum)  
FBC, UE, LFT, glucose  
CK  
ammonia, thyroid  
HIV, syphilis  
toxicology

## EEG



Prolonged or video EEG if available

## MRI brain



With contrast; exclude structural and demyelinating causes

## Tumour screen



Pelvic ultrasound (girls), testicular examination (boys)

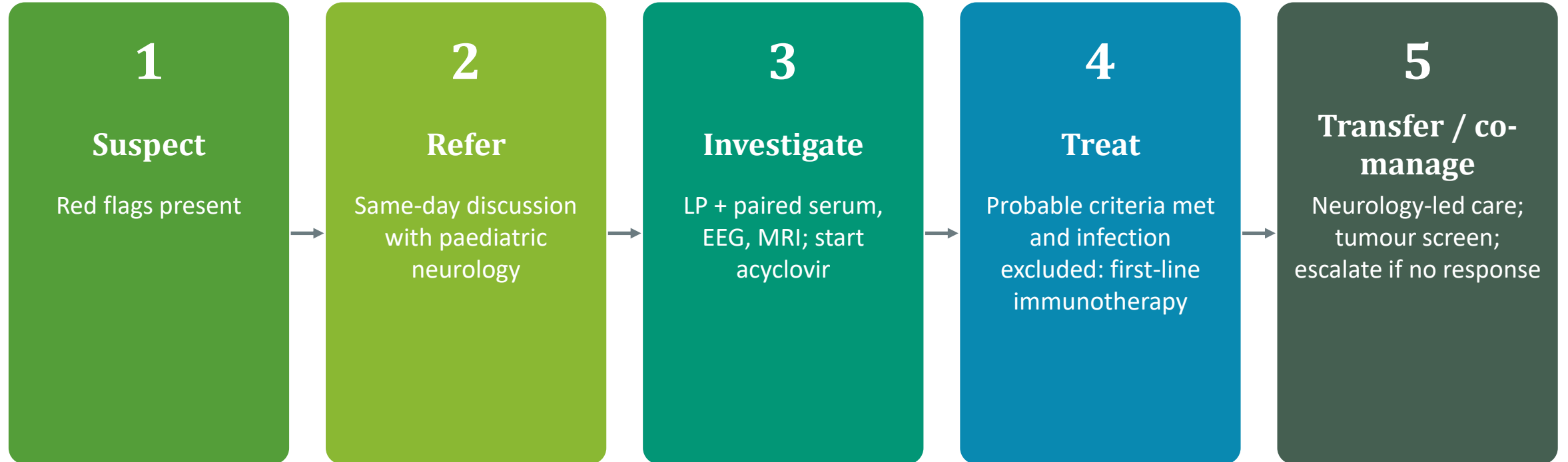
## Start acyclovir



Until HSV PCR is negative

# Referral pathway

**Call**



**Do not wait for antibody results to call or to treat**

*Adapted from Graus 2016; Cellucci 2020; Nosadini 2021*

# First-line immunotherapy

| Treatment             | Typical paediatric regimen          | Notes                                                                 |
|-----------------------|-------------------------------------|-----------------------------------------------------------------------|
| IV methylprednisolone | 30 mg/kg/day (max 1 g) for 3–5 days | Then oral taper, guided by neurology                                  |
| IV immunoglobulin     | 2 g/kg total, over 2–5 days         | Often combined with steroids                                          |
| Plasma exchange       | 5–7 exchanges                       | Needs central access; less tolerated in agitated or unstable children |
| Tumour removal        | If a teratoma is found              | Part of first-line treatment                                          |

*Doses are typical and for orientation only: follow local protocols and specialist advice*

*Titulaer 2013; Nosadini 2021 (international paediatric consensus)*

# Second-line immunotherapy

## When to escalate

- Inadequate response to first-line therapy
- Commonly reviewed at about 2 weeks
- Severe illness: ICU, status epilepticus, dysautonomia
- Neurology-led decision

## Rituximab

Most commonly used second-line agent in children

## Cyclophosphamide

Alternative or addition in refractory cases; other agents in specialist centres

# Third-Line immunotherapy and maintenance

## When to escalate

- refractory to first- and second-line approaches- approximately 10%
- third-line treatments
  - Tocilizumab: IL-6 receptor antagonist
  - Cyclophosphamide: If not used as second-line

## Maintenance immunosuppression beyond 6 months is generally not required

- more severe course or prolonged impairments and hospitalization
- For patients with relapsing disease
- treatment duration is dictated by severity and clinical course:
  - **median 3 months in best responders**
  - **9 months in average responders**
  - **18 months in poorest responders.**

# Acute complications



## Seizures

Status epilepticus,  
sometimes refractory;  
non-convulsive seizures



## Breathing

Central hypoventilation,  
aspiration;  
may need ventilation



## Cardiac and autonomic

Bradycardia,  
asystole;  
labile BP,  
hyperthermia



## Antipsychotic reactions

NMS-like picture:  
rigidity, fever, raised CK



## Injury

Self-injury,  
oral trauma,  
rhabdomyolysis from  
dyskinesia or agitation



## Treatment and ICU- related

Infection,  
VTE, pressure injury  
steroid, IVIG and rituximab  
side effects

# Supportive care and symptom management

| Problem                 | Approach                                                                                |
|-------------------------|-----------------------------------------------------------------------------------------|
| Breathing and airway    | Monitored bed; early ICU input for hypoventilation, secretions or reduced consciousness |
| Autonomic instability   | Cardiac monitoring; treat bradycardia and pauses; temperature control                   |
| Seizures                | Standard antiseizure medication; video EEG to separate seizures from dyskinesia         |
| Agitation and catatonia | Calm environment; benzodiazepines (lorazepam); avoid high-dose antipsychotics           |
| Insomnia                | Sleep hygiene; melatonin or sedatives as advised                                        |
| Movement disorder       | Often settles with immunotherapy; specific drugs per neurology                          |
| Nursing and nutrition   | Pressure care, feeding, VTE prevention, protection from injury                          |

*Nosadini 2021; Cellucci 2020*

# After discharge: long-term follow-up



## Neurology follow-up

Monitor recovery; watch for  
relapse; steroid taper



## Neuropsychology

Attention, memory, executive  
function



## School support

Graded return; individual  
education plan



## Rehabilitation

Physiotherapy, occupational  
and speech therapy



## Family support

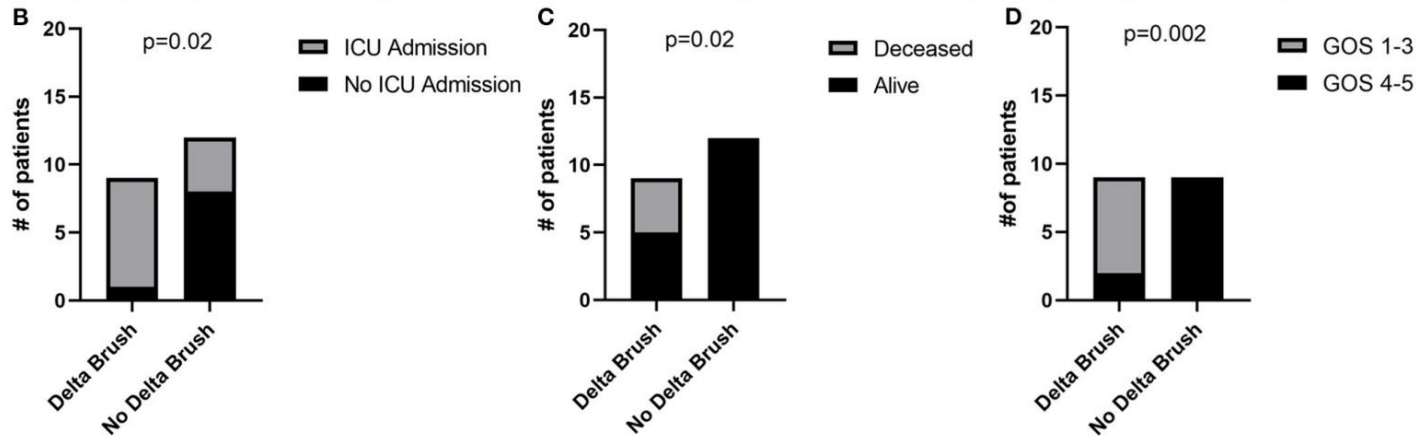
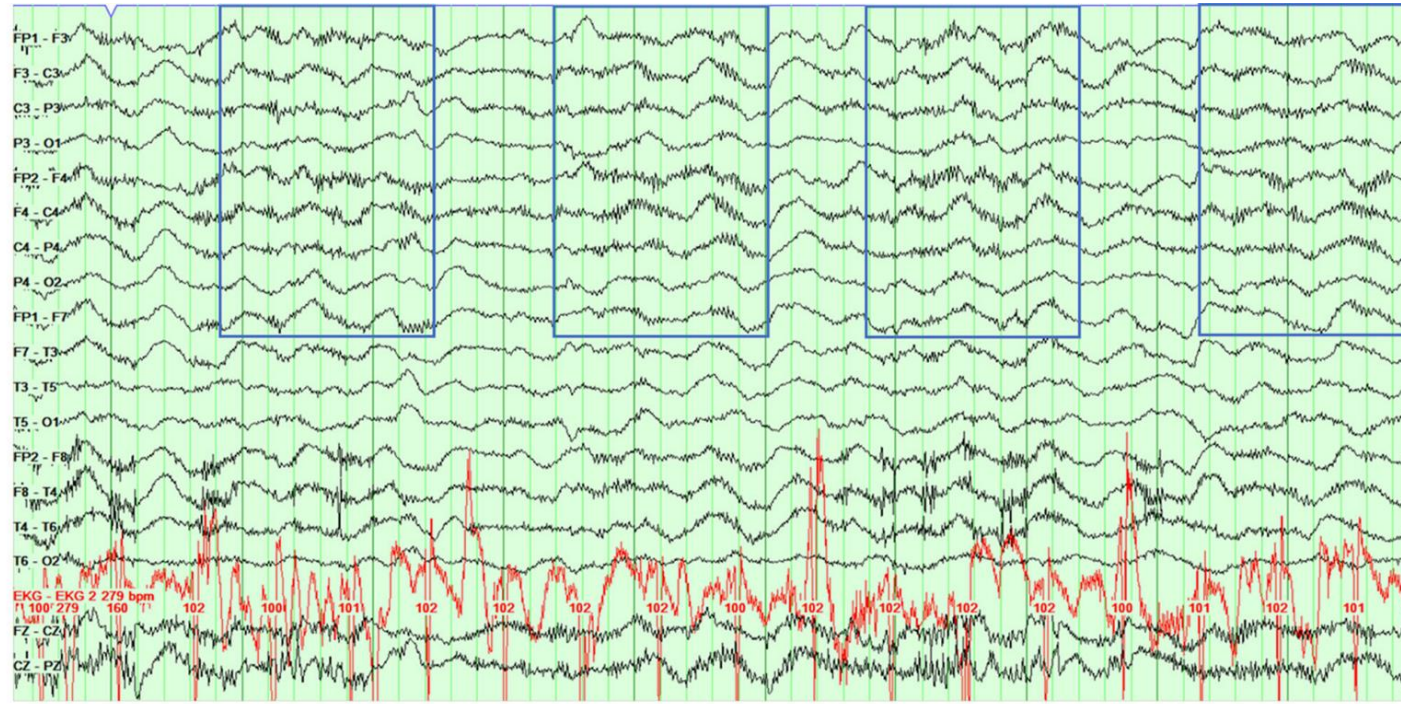
Psychological support;  
relapse warning signs



## Relapse vigilance

Return of sleep, behaviour or  
speech change

# prognostic outcomes



Nathoo N, Anderson D and Jirsch J (2021)  
 Extreme Delta Brush in Anti-NMDAR Encephalitis  
 Correlates With Poor Functional Outcome and Death.  
 Front. Neurol. 12:686521  
 doi:  
 10.3389/fneur.2021.686521

# Long-term complications and outcomes

81%

good outcome (mRS 0–2) at 24 months

12%

relapse risk within 2 years;  
usually milder

6%

died within 24 months (30 of 501)

## Long-term complications (recovery continues for 18 months or more)

Deficits in attention, memory and executive function; behaviour and mood problems; sleep disturbance; fatigue; school difficulties; relapse

*Titulaer 2013 (Lancet Neurol); Florance 2009*

# Prognosis

## At least 2 year follow up

- 45% cognitive impairment
- 70% academic difficulties
- 92% needed rehabilitation
- **88% achieved mRS  $\leq 2$  (apparent good functional outcome)**

Factors associated with **good outcome** include:

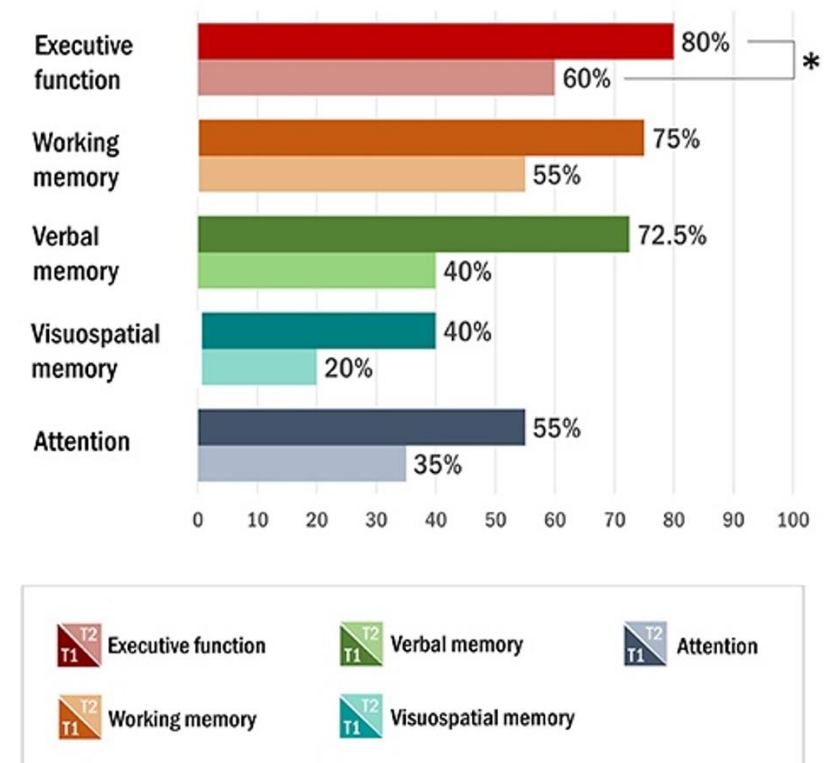
- Early immunotherapy initiation
- Milder symptoms not requiring ICU admission
- Tumor removal (if present)
- Use of second-line immunotherapy when indicated

Factors associated with **poor outcome** include: [15][18]

Younger age at illness onset

- Higher number of symptoms at diagnosis
- ICU admission
- Abnormal EEG at diagnosis
- Delayed immunotherapy

**B** Deficits on domain level (% of patients, N=40)



## encephalitis work-up

**Figure 2.** Cognitive impairment at first and second study visit at test and domain levels (n = 40).

[Long-Term Cognitive Outcome in Anti-N-Methyl-D-Aspartate Receptor Encephalitis.](#)

Ann Neurol. December 1, 2021.

# QUIZ

Which symptoms are red flags for NMDRE in a 5 yr old female? Choose the best option.

- A. incontinence
- B. Stereotypy
- C. Behavior, change, speech regression and seizure
- D. Meningism
- E. Catatonia



Choose the correct statement.

- A. Approximately 80% with NMDRE have a good outcome
- B. seizures occur in 90% of patients
- C. ICU stay was associated with a better outcome
- D. Delay in treatment is associated with a poorer prognosis
- E. A and D

Which criteria confirms the diagnosis of NMDRE ?

- A. CSF pleocytosis
- B. serum oligoclonal bands
- C. MRI hippocampal sclerosis
- D. Cell-based assay for IgG anti-GluN1
- E. MOG antibody positivity

# Take-home practice points

- 1 **Think of it:** New behaviour change + any neurological sign in a child (**MC-BASS**)
- 2 **Use the criteria:** Probable diagnosis is clinical; do not wait for antibodies
- 3 **Test CSF:** With paired serum; a normal MRI or CSF does not exclude it
- 4 **Anticipate complications:** Monitor breathing, heart rhythm and seizures; avoid high-dose antipsychotics
- 5 **Refer and treat early:** Same-day paediatric neurology; first-line immunotherapy once infection is excluded
- 6 **Follow up:** Cognition, behaviour, school and relapse

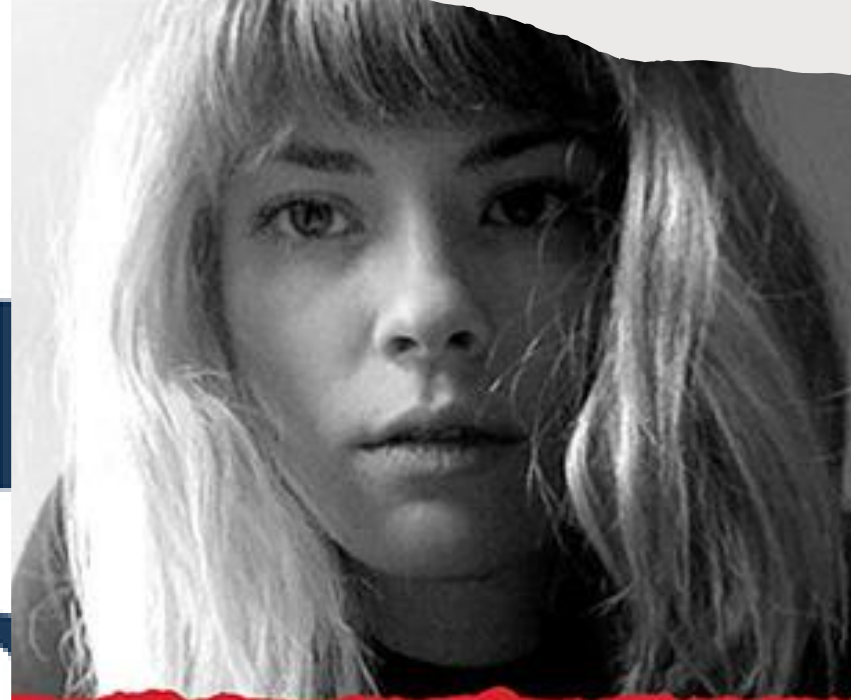


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# Ochs Health

Thank you  
Questions?



**BRAIN ON FIRE**  
MY MONTH OF MADNESS

