MOGAD in children - evolving phenotypes and treatments strategies

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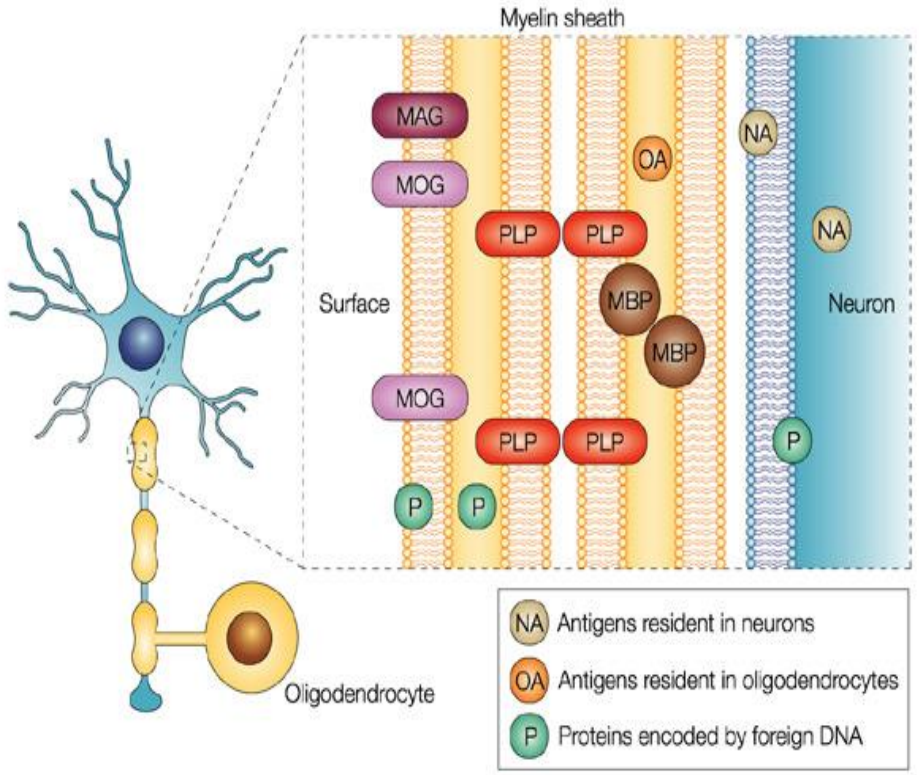
Educational grants to organize meetings by Novartis, Biogen Idec, Merck Serono and Bayer

Summary

- Clinical spectrum of MOGAD
- Applying the diagnostic criteria
- Beginnings of a management consensus

Myelin oligodendrocyte glycoprotein

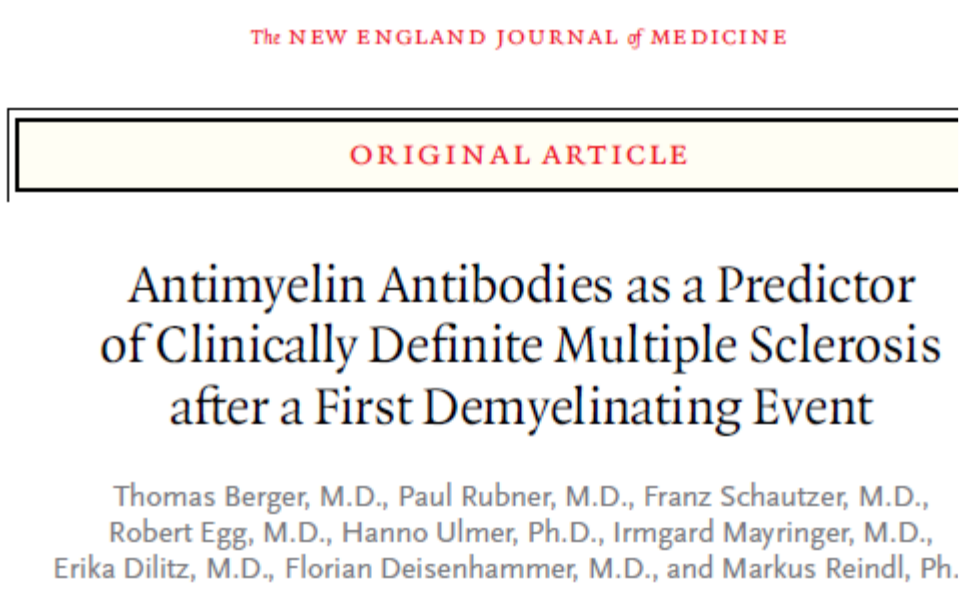
- "Exclusively" expressed in the CNS
  - Outermost lamellae of the myelin sheath
  - Plasma membrane of oligodendrocytes
- Minor component of myelin (0.05%)
- Surface marker of oligodendrocyte maturation
- Abs to MOG have been shown to induce or contribute to demyelination in various animal models



Reindl, M. *et al.* 2013 *Nat. Rev. Neurol.* **9(8)**:455-61

Hemmer B. *et al.* 2002. *Nature Reviews Neuroscience* **3(4)**, 291-301

It's all about the assay



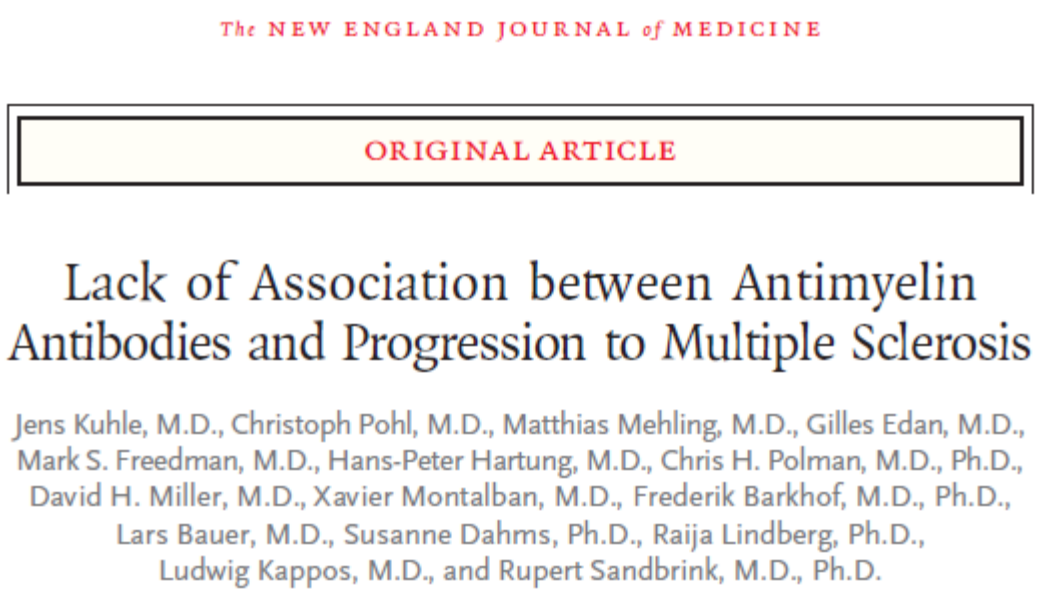
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Antimyelin Antibodies as a Predictor of Clinically Definite Multiple Sclerosis after a First Demyelinating Event

Thomas Berger, M.D., Paul Rubner, M.D., Franz Schautzer, M.D., Robert Egg, M.D., Hanno Ulmer, Ph.D., Irmgard Mayringer, M.D., Erika Dilitz, M.D., Florian Deisenhammer, M.D., and Markus Reindl, Ph.

N Engl J Med 2003 Jul 10;349(2):139-45



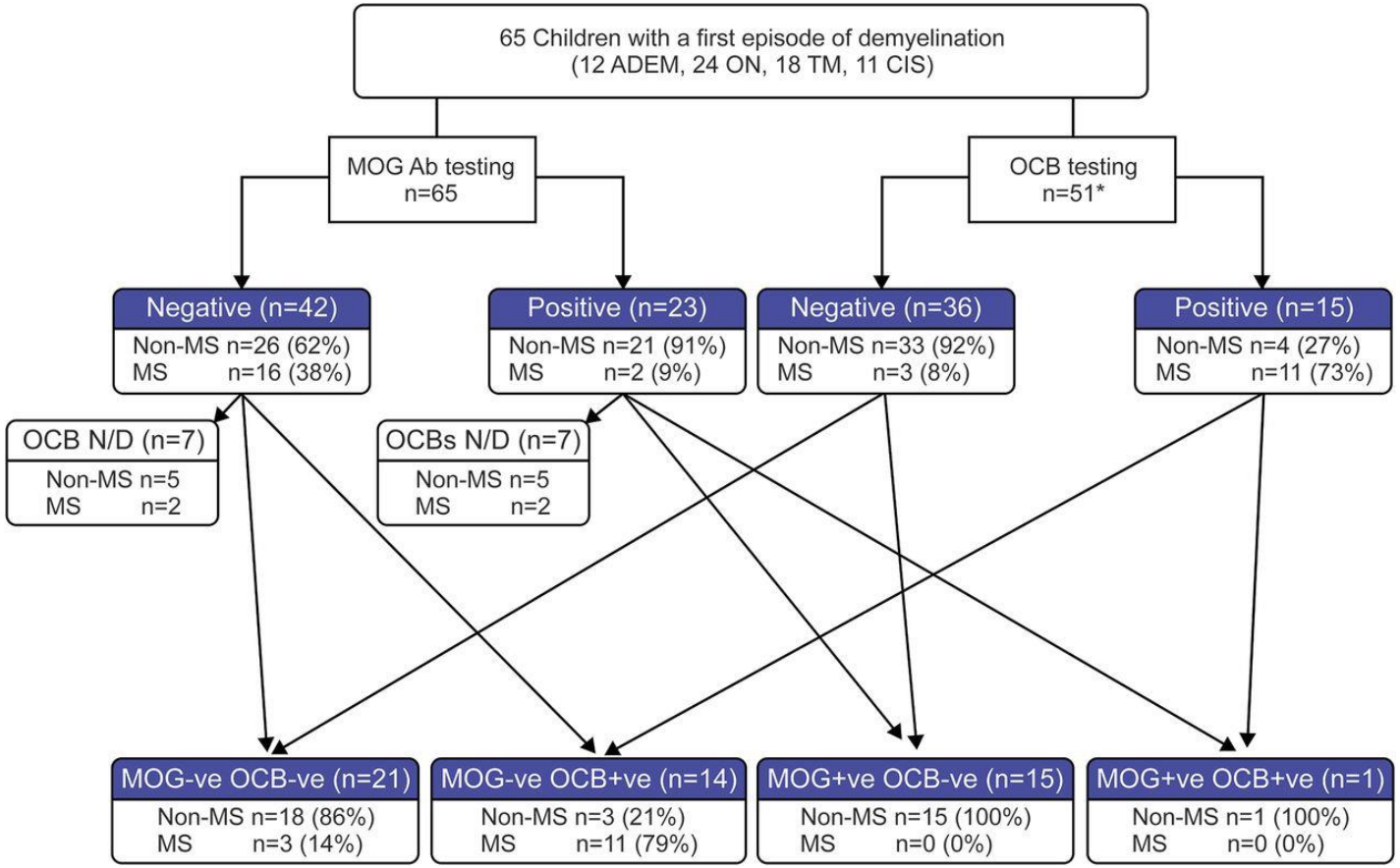
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Lack of Association between Antimyelin Antibodies and Progression to Multiple Sclerosis

Jens Kuhle, M.D., Christoph Pohl, M.D., Matthias Mehling, M.D., Gilles Edan, M.D., Mark S. Freedman, M.D., Hans-Peter Hartung, M.D., Chris H. Polman, M.D., Ph.D., David H. Miller, M.D., Xavier Montalban, M.D., Frederik Barkhof, M.D., Ph.D., Lars Bauer, M.D., Susanne Dahms, Ph.D., Raija Lindberg, Ph.D., Ludwig Kappos, M.D., and Rupert Sandbrink, M.D., Ph.D.

N Engl J Med 2007 Jan 25;356(4):371-8



MOG antibodies are associated with a non-MS course in children

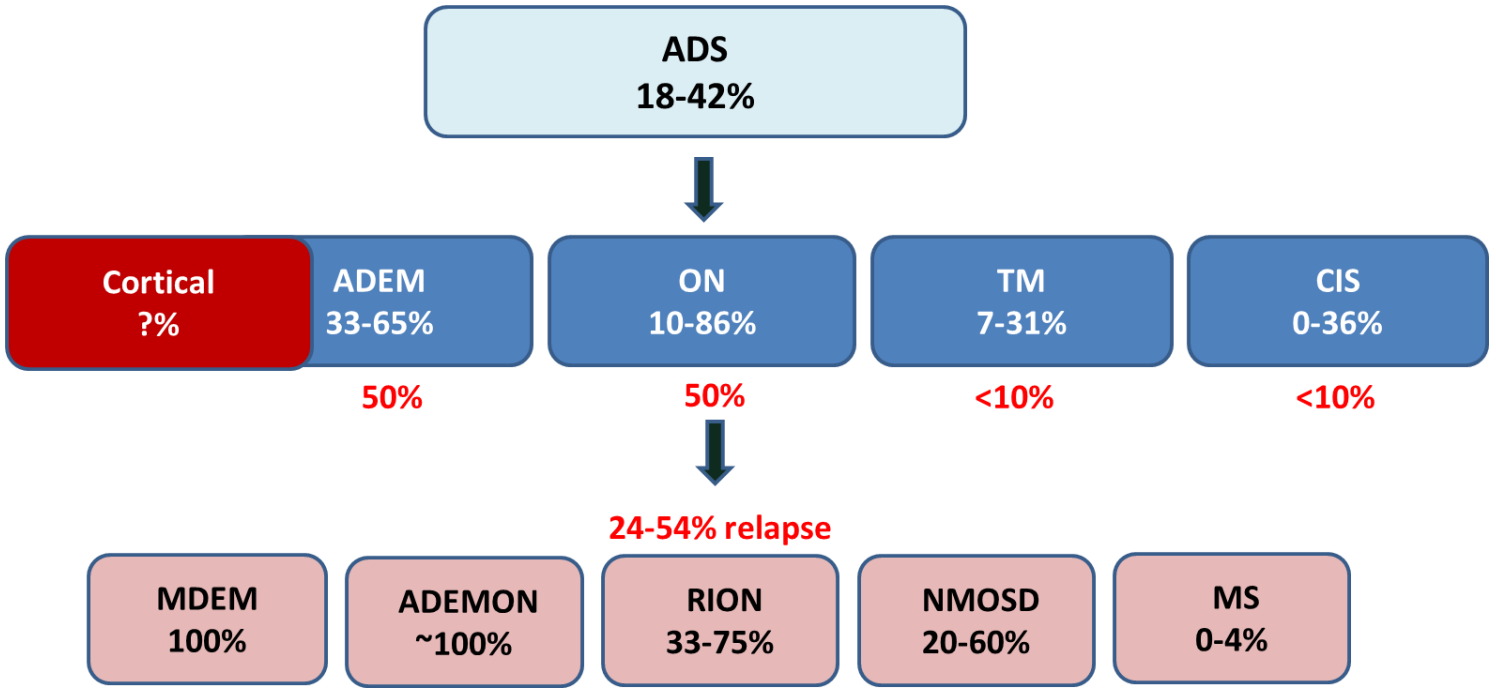
MOG-Abs by cell-based assay

Dale et al., 2014 ***N2*** 1(1):e12
Hacohen et al., 2015 ***N2*** 2(2):e81

Anti-MOG antibodies plead against MS diagnosis in an ADS

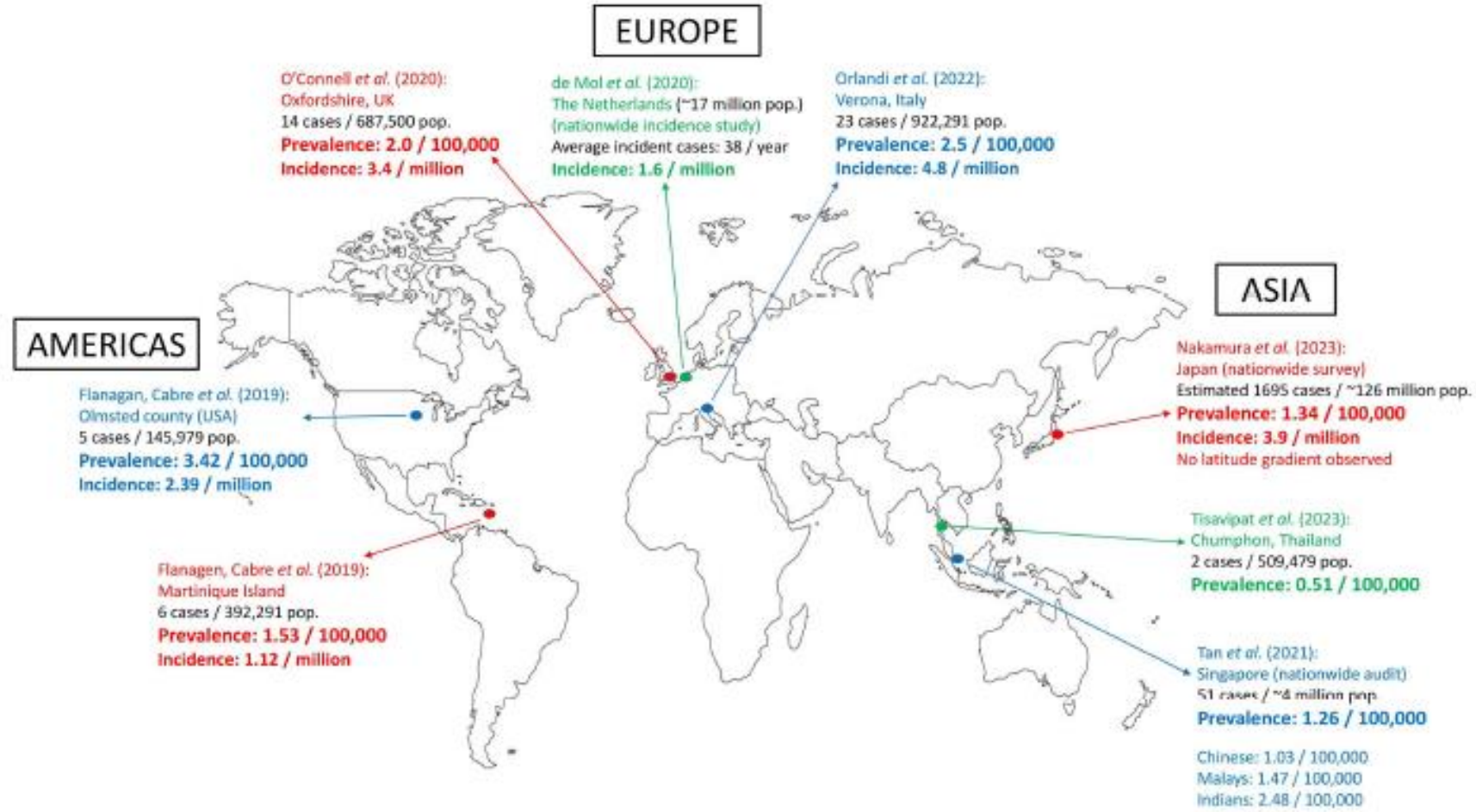
Ketelslegers et al., 2015 ***Mult Scler.*** 21(12):1513-2

MOG antibodies in relapsing ADS

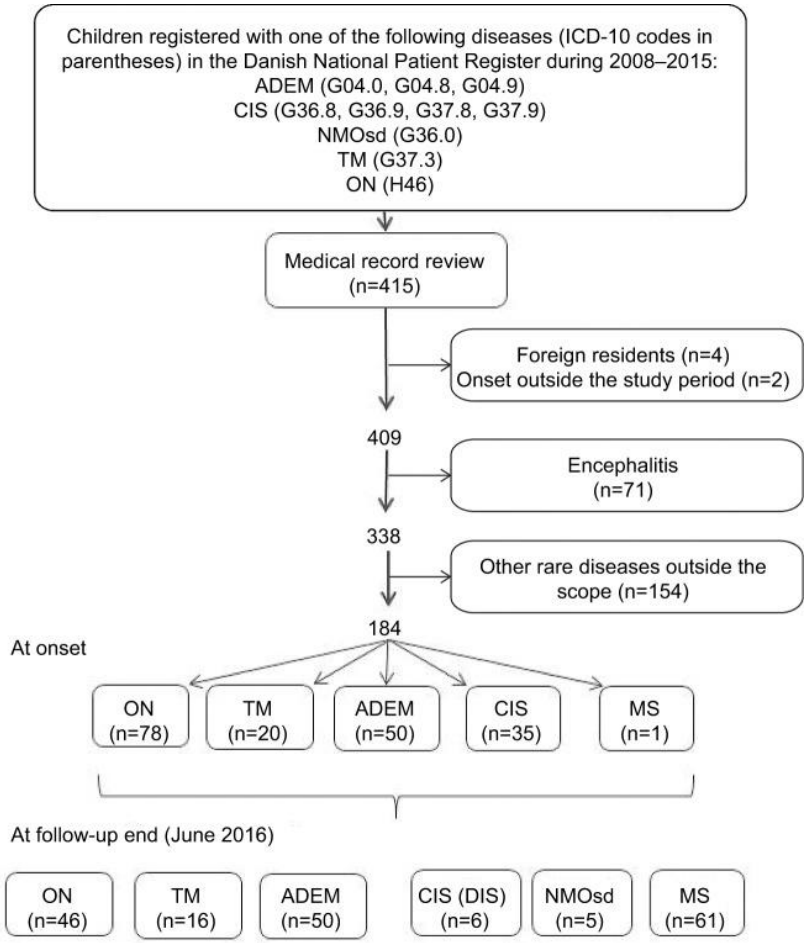


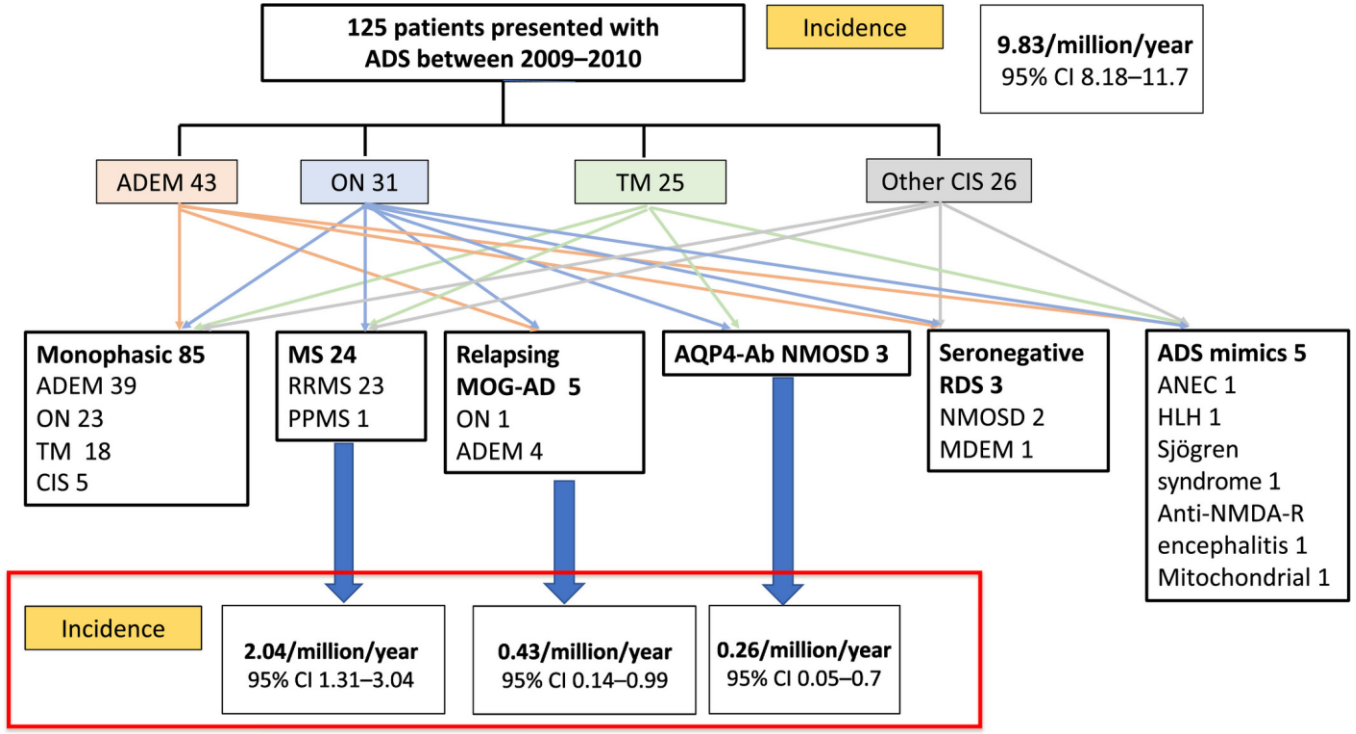
Selter et al., 2010 ***Neurology.*** 74(21):1711-5; Dale et al., 2014 ***N2*** 1(1):e12; Fernandez-Carbonell et-al., 2016 ***Mult Scler.*** 22(2): 174-84; Hacohen et al., 2015 ***N2*** 2(2):e81; Ketelslegers et al., 2015 ***Mult Scler.*** 21(12):1513-20; Hacohen et al., 2017 ***Neurology*** 89(3):269-278; Hennes et al., 2017 ***Neurology*** 89(9):900-908; Ramanathan et al., 2018 ***J Neurol Neurosurg Psychiatry.*** 89(2):127-137; de Mol et al., 2018 ***J Neurol*** 265(6):1310-1319; Duignan et al., 2018 ***Dev Med Child Neurol*** 60(9):958-962; Lopez-Chriboga et al., 2018 ***JAMA Neurol*** 75(11):1355-1363

Prevalence/Incidence of MOGAD



Hor & Fujihara ***Front Neurol***. 2023 14:1260358





Boesen et al., 2018 ***Clin Epidemiol*** 10:391-399

Abdel-Mannan et al., 2022 ***Dev Med Child Neurol.*** 64(4):502-508

58-73% Monophasic

MS still the most prevalent RDS phenotype

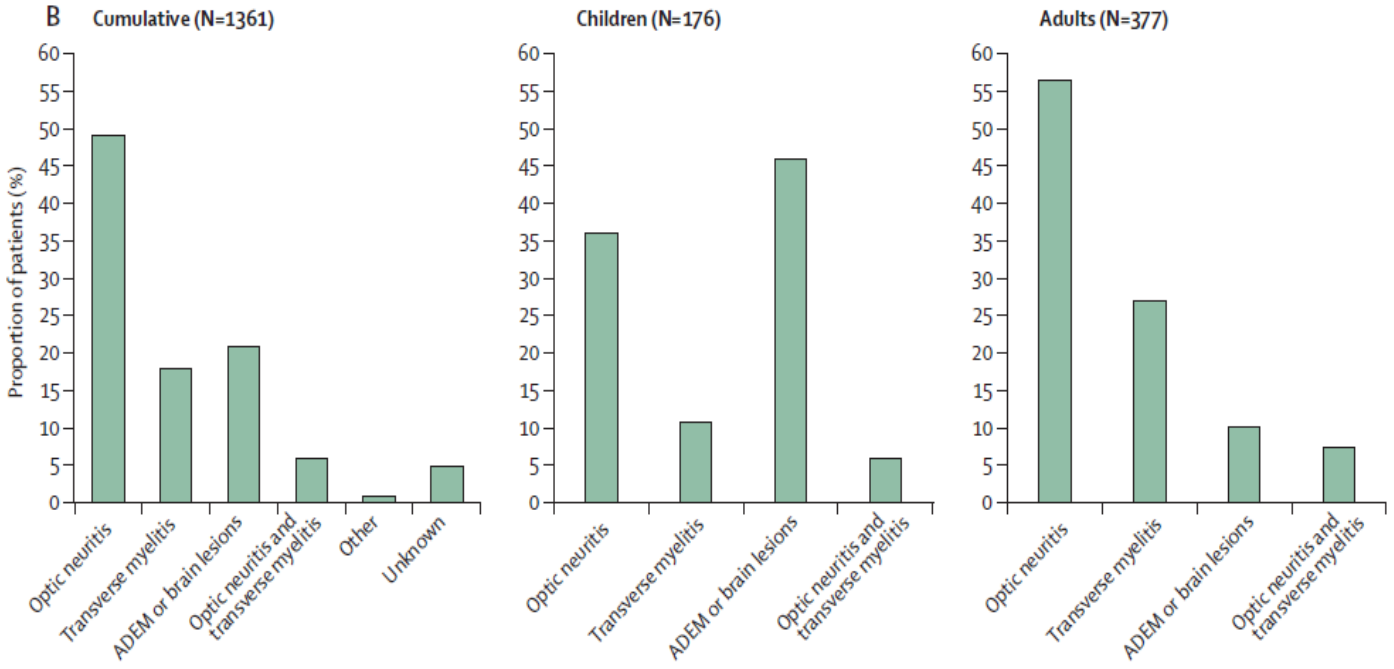
Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria

Brenda Banwell, Jeffrey L Bennett*, Romain Marignier*, Ho Jin Kim*, Fabienne Brilot, Eoin P Flanagan, Sudarshini Ramanathan, Patrick Waters, Silvia Tenembaum, Jennifer S Graves, Tanuja Chitnis, Alexander U Brandt, Cheryl Hemingway, Rinze Neuteboom, Lekha Pandit, Markus Reindl, Albert Saiz, Douglas Kazutoshi Sato, Kevin Rostasy*, Friedemann Paul*, Sean J Pittock*, Kazuo Fujihara*, Jacqueline Palace**

Recognised demyelinating (including cortical) phenotypes
Widely available cell based assays
Supportive radiological (and sometime clinical features)

Banwell et al., 2023 ***Lancet Neurol*** 22: 268-82

Presentation phenotype of patients with MOGAD



- Visual acuity 6/60 (and can be worse)
- Moderate to severe myelopathy (EDSS >4) in 50%
- ADEM or brainstem syndrome has significant morbidity

Banwell et al., 2023 ***Lancet Neurol*** 22: 268-82

UK, France, Japan, Sri Lanka, Netherlands, Australia cohorts

Evelina London

KING'S HEALTH PARTNERS

NHS Guy's and St Thomas' NHS Foundation Trust

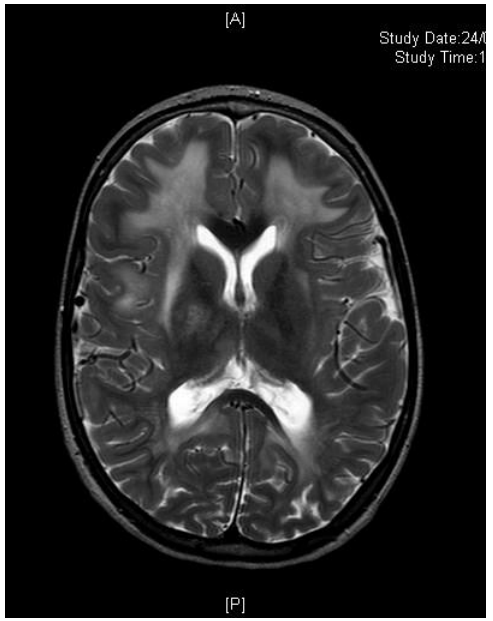
Features of MOG-ab associated demyelination

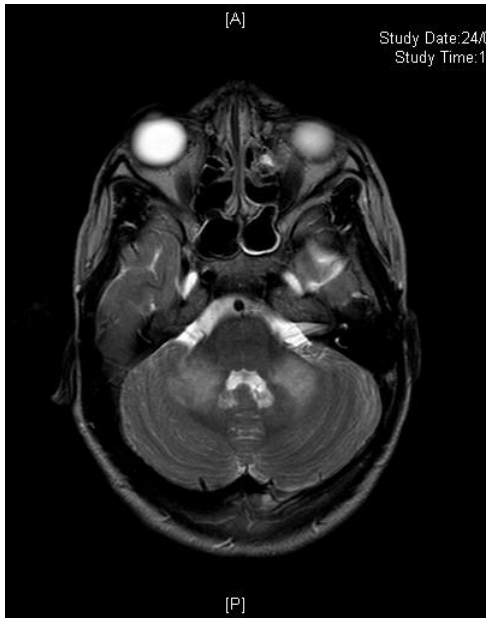
- *Younger patients*
  - *30% under 5*
- More features of systemic inflammation
  - CSF reactive
  - ESR elevated
  - Seizures
- Paraclinical feature different from MS
  - Frequency of EBV
  - Intrathecal oligoclonal bands

Hacohen et al., 2015 ***Neurol Neuroimmunol Neuroinflamm.*** 2015 2(2):e81; Ketelslegers et al., 2015 ***Mult Scler.*** 21(12):1513-2; Fernandez-Carbonell et-al., 2016 ***Mult Scler.*** 22(2): 174-84; Hennes et al., 2017 ***Neurology*** 89(9):900-908

Case 5Y M

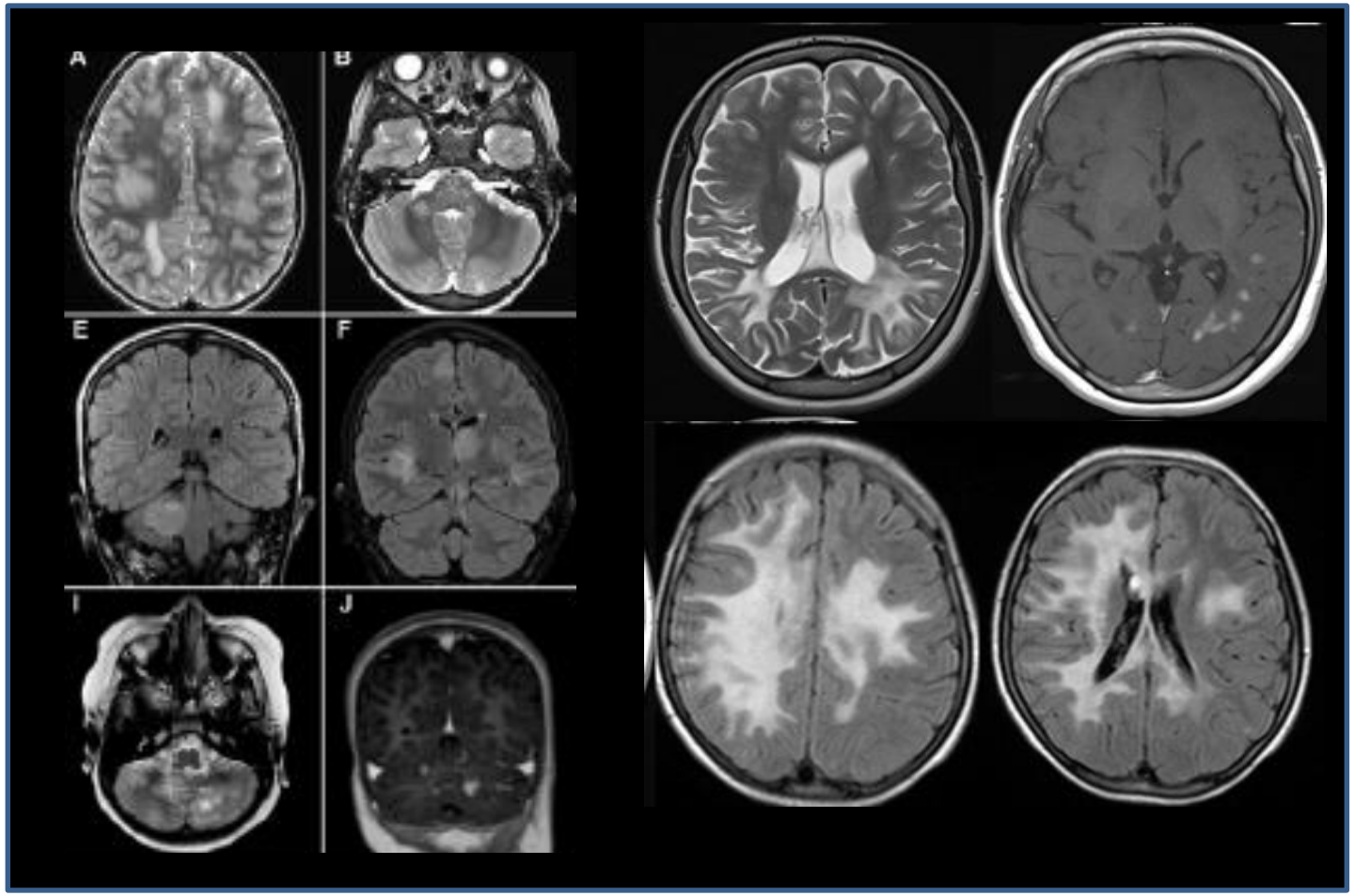
- Viral illness 3 prior
- Tired and lethargic
- Sleepy and unwilling to weight bear
- Clinically
    - Depressed level of consciousness
    - Disorientated
    - Irritable with left sided posturing
    - No meningism
    - Nystagmus and long tract signs





Clinical and neuroradiological differences of MOG + and – are not that easy to discern

Baumann et al., 2015 ***J Neurol Neurosurg Psychiatry*** 86(3):265-72



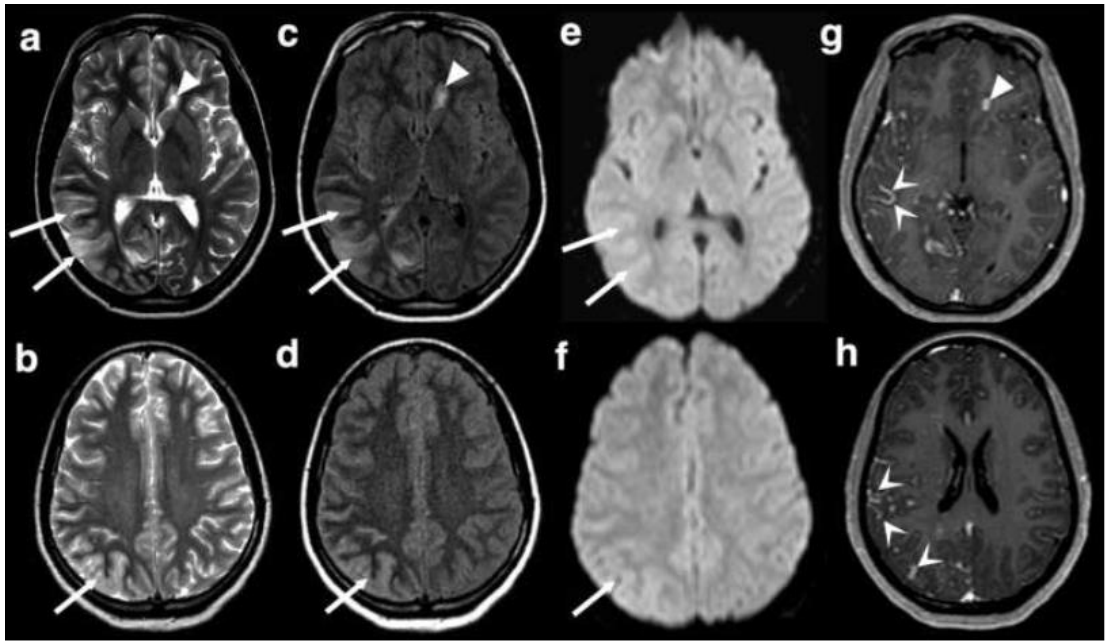
Imaging features of MOGAD CNS disease

- Multiple ill-defined T2 hyperintense lesions in supratentorial and often infratentorial white matter
- Deep grey matter involvement
- Ill-defined T2 hyperintensity involving pons, middle cerebellar peduncle, or medulla
- Cortical lesion with or without lesional and overlying meningeal enhancement

Hennes et al., 2020 ***Eur J Paediatr Neurol***. 29:14-21

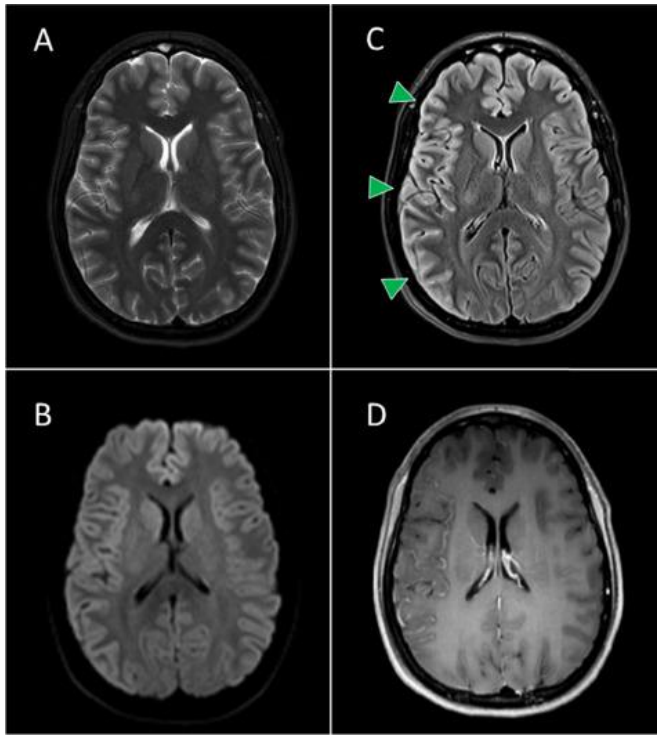
Hacohen et al., 2018 ***Dev Med Child Neurol***. 60(4):417-423

Cortical involvement in MOGAD



Cortical encephalitis

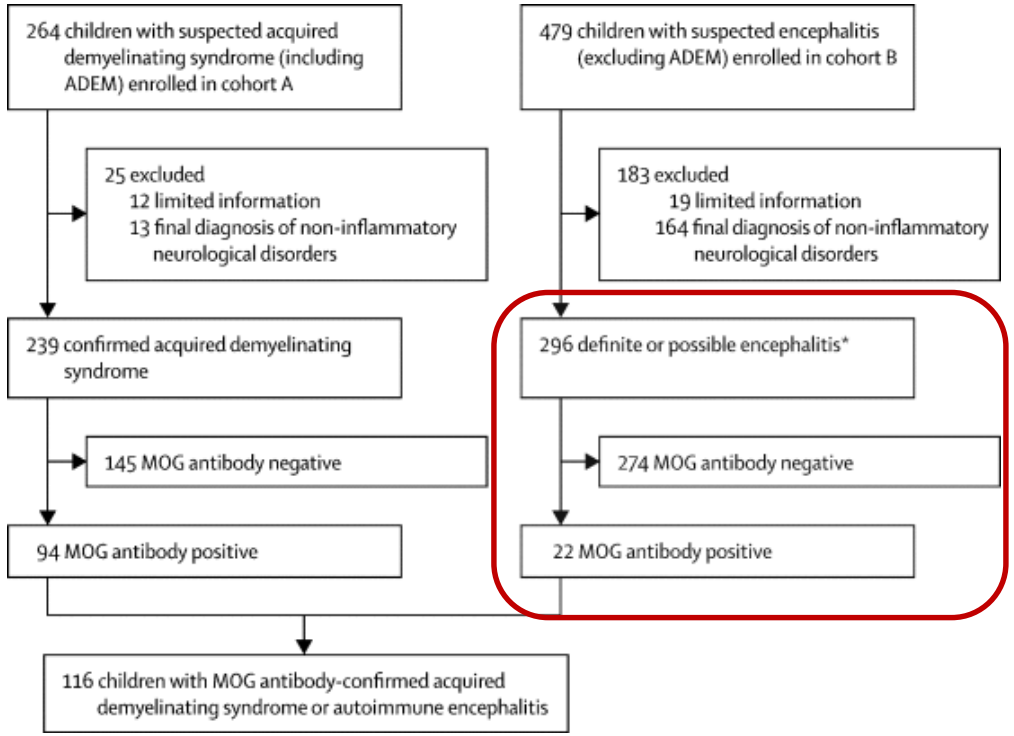
N2 2020; 7: e731; JAMA Neurol. 2018; 75: 65-71; N2 2017; 4: e322



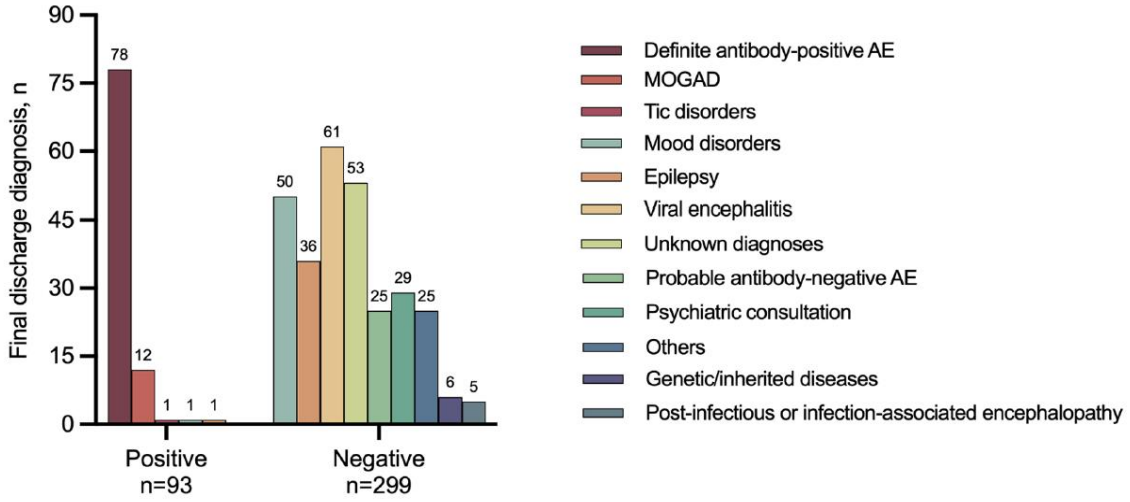
FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis With Seizures (FLAMES)

Mult Scler Relat Disord 2020 Sep;44:102283

MOG antibodies in autoimmune encephalitis



Armangue et al., 2020 ***Lancet Neurol.*** 19(3):234-246



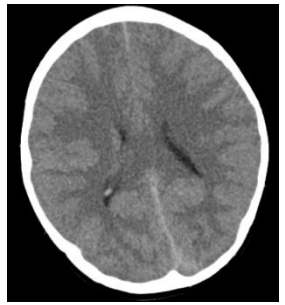
Yang et al., 2024 ***Dev Med Child Neurol*** 66(4):483-492

50%NMDAR 25% MOG vs 49% MOG 35% NMDAR

Olivé-Cirera et al. 2025 ***Lancet Neurol*** 24(1):54-64

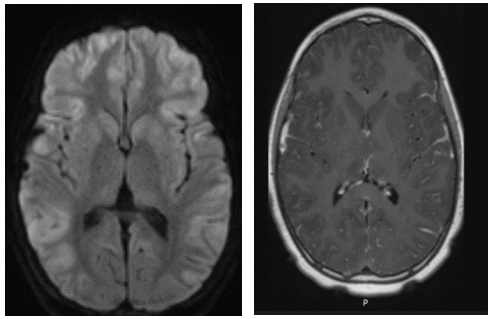
MOG is perhaps as common as NMDAR antibodies but may be population specific

Expanding cortical MOGAD phenotypes



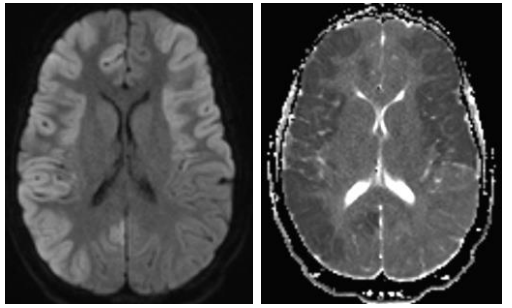
9yr M acute febrile encephalopathy following seizure

Fulminant cerebral oedema



10yr M meningoencephalitis and seizures

Meningoencephalitis



10yr F Febrile and refractory seizures

FIRES/NORSE

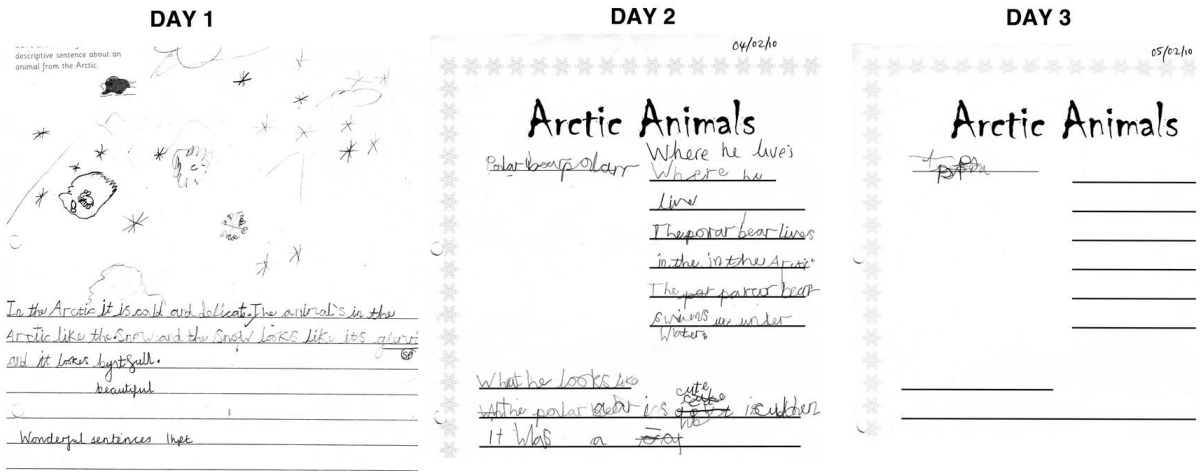
32/235 (14%) of MOGAD cases have cortical encephalitis phenotype

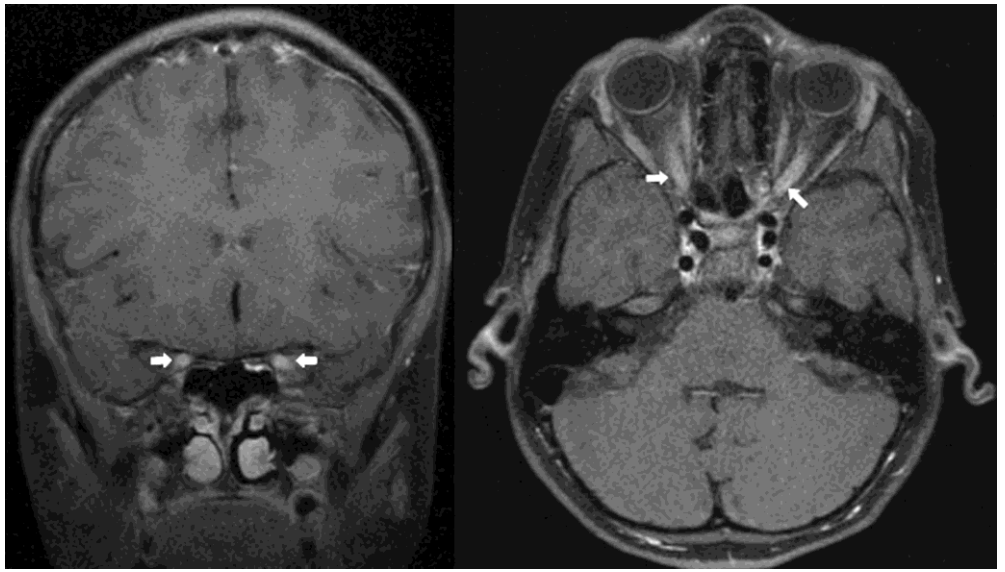
Kim et al., 2024 ***N2*** 11(6):e200323

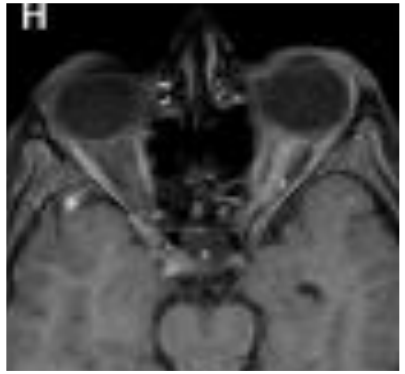
Case 6Y F

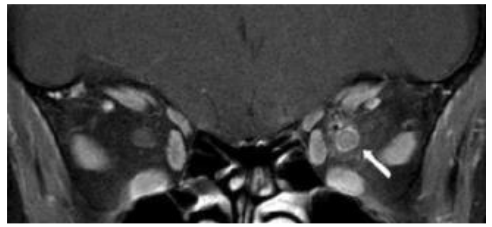
Bilateral visual failure

- 6/60 right and only to movement left
- No significant colour vision
- Hyperaemic fundus



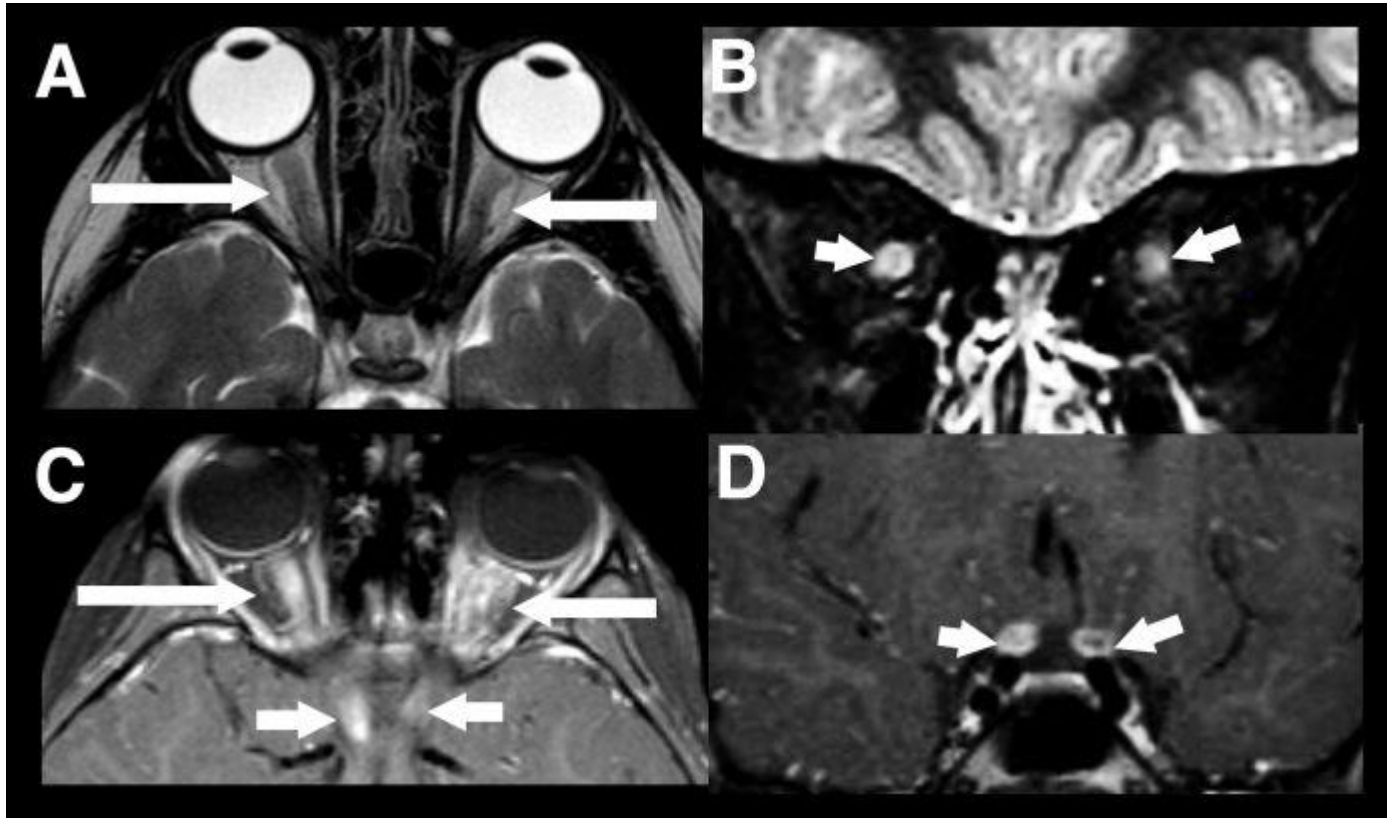






Gad enhancement+ Perineuritis

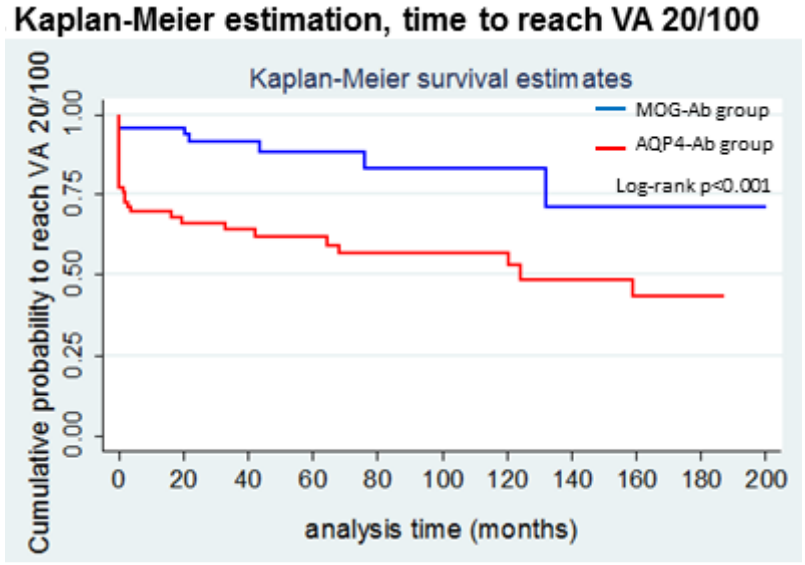
MOGAD Optic neuritis



- Bilateral simultaneous clinical involvement
- Longitudinal optic nerve involvement (>50% of the optic nerve length)
- Perineural optic sheath enhancement
- Optic disc oedema

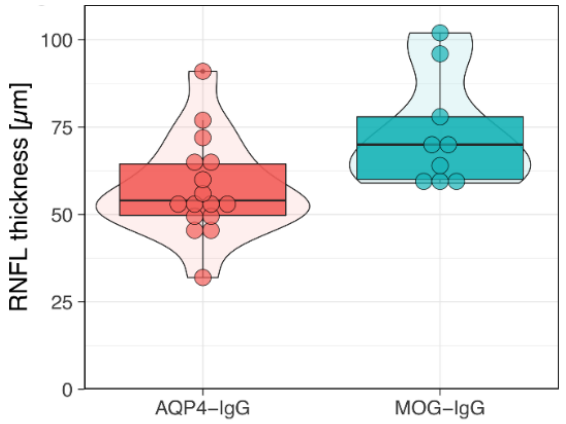
[Paediatric Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) - EyeWiki]

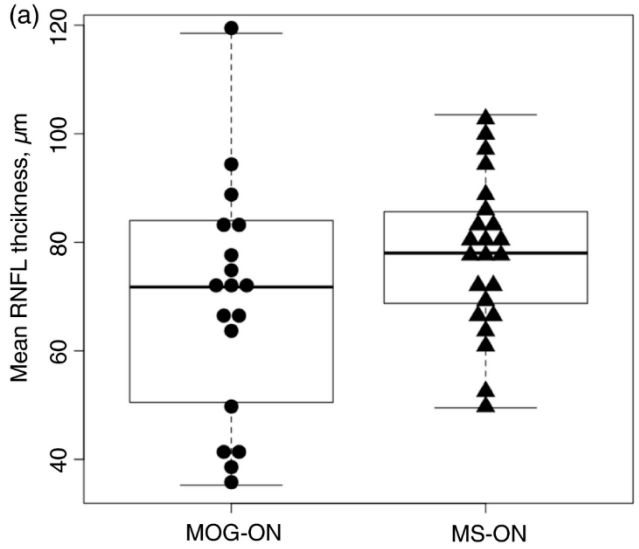
Good recovery



MOGADOR 2018 ***Neurology*** 90(21):e1858-e1869

OCT correlates less will with vision in MOGAD





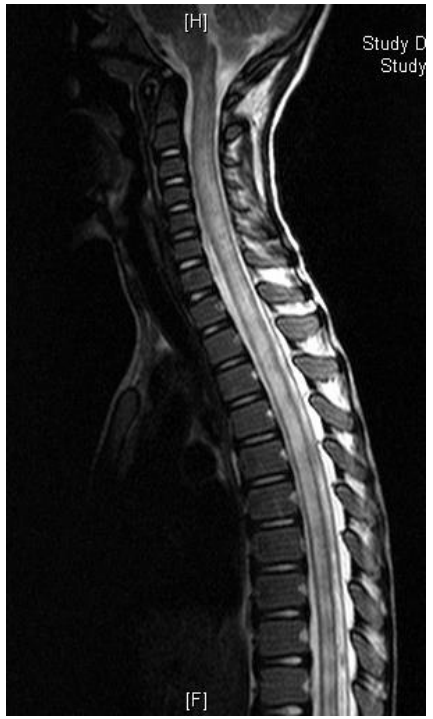
Stiebel-Kalish et al., 2017 **PLoS One.**12(1):e0170847

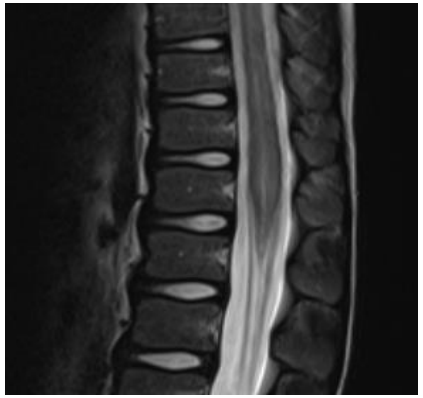
Eyre et al., 2018 ***Dev Med Child Neurol***. 60(12):1244-1250

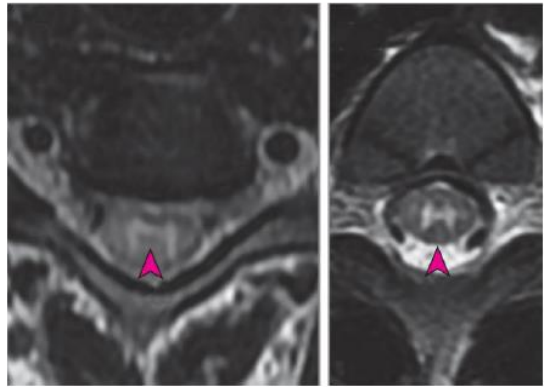
Narayan et al., 2019 ***Mult Scler Relat Disord.*** 28:86-90

Case 8Y F

- Transferred to our centre
  - Clinically dehydrated
  - Fretful
  - Meningism
  - Clinically
    - ➢ Fundoscopy normal
    - ➢ No antigravity power in lower limbs
    - ➢ No apparent positive sensory symptoms
    - ➢ Brisk reflexes and upgoing plantars
    - ➢ Palpable bladder

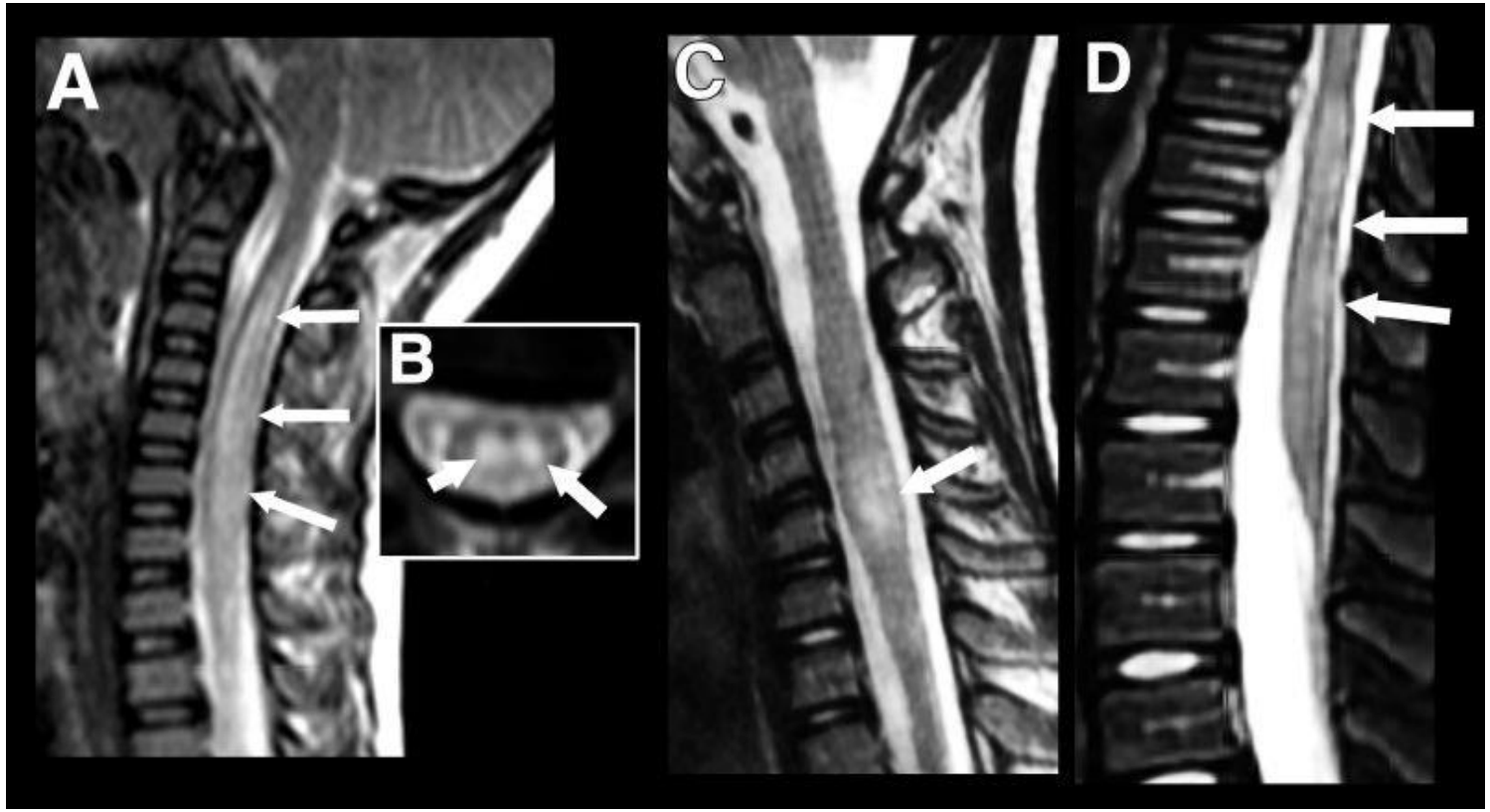






Striking recovery is seen

MOGAD Transverse Myelitis



- Longitudinal extensive myelitis
- Central cord lesion or H-sign
- Conus lesion

[Paediatric Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) - EyeWiki]

Diagnosis of MOGAD (requires fulfilment of A, B, and C)			
(A) Core clinical demyelinating event	Optic neuritis* Myelitis† ADEM‡ Cerebral monofocal or polyfocal deficits§ Brainstem or cerebellar deficits¶ Cerebral cortical encephalitis often with seizures‖		
(B) Positive MOG-IgG test	Cell-based assay: serum**	Clear positive††	No additional supporting features required
		Low positive‡‡	• AQP4-IgG seronegative AND • ≥1 supporting clinical or MRI feature
		Positive without reported titre	
		Negative but CSF positive§§	
Supporting clinical or MRI features	Optic neuritis		• Bilateral simultaneous clinical involvement • Longitudinal optic nerve involvement (> 50% length of the optic nerve) • Perineural optic sheath enhancement • Optic disc oedema
	Myelitis		• Longitudinally extensive myelitis • Central cord lesion or H-sign • Conus lesion
	Brain, brainstem, or cerebral syndrome		• Multiple ill-defined T2 hyperintense lesions in supratentorial and often infratentorial white matter • Deep grey matter involvement • Ill-defined T2-hyperintensity involving pons, middle cerebellar peduncle, or medulla • Cortical lesion with or without lesional and overlying meningeal enhancement
(C) Exclusion of better diagnoses including multiple sclerosis¶¶			

Banwell et al., 2023 ***Lancet Neurol*** 22: 268-82

MOGAD 2023 criteria have high specificity and sensitivity

Table 3 Performance of the MOG-Ab Seropositivity and the 2023 International MOGAD Criteria When Clinical MOGAD Diagnosis Was Used as Benchmarking Against Which the New Criteria Were Tested

	Sensitivity, % (95% CI)	Specificity, % (95% CI)	Accuracy, % (95% CIs)	PPV, % (95% CI)	NPV, % (95% CIs)
All patients					
MOG-Ab testing	100 (95.7–100)	96.3 (94.1–97.6)	96.9 (95.0–98.2)	83.5 (75.2–89.4)	100 (99.1–100)
2023 MOGAD diagnostic criteria	96.5 (90.2–99.1)	98.9 (97.4–99.5)	98.5 (97.1–99.4)	94.3 (87.4–97.6)	99.3 (98.1–99.8)
Children					
MOG-Ab testing	100 (92.7–100)	98.8 (93.7–99.9)	99.2 (95.9–100)	98.0 (89.5–99.9)	100 (95.7–100)
2023 MOGAD diagnostic criteria	100 (92.7–100)	98.8 (93.7–99.9)	99.2 (95.9–100)	98.0 (89.5–99.9)	100 (95.7–100)
Adults					
MOG-Ab testing	100 (90.69–100)	95.6 (93.0–97.3)	96.0 (93.7–97.2)	69.8 (56.5–80.5)	100 (98.9–100)
2023 MOGAD diagnostic criteria	91.9 (78.7–97.2)	98.9 (93.7–99.9)	98.3 (96.5–99.3)	89.4 (75.8–95.8)	99.2 (97.6–99.7)

Abbreviations: MOG-Ab = MOG antibody; MOGAD = MOG-Ab–associated disease; NPV = negative predictive value; PPV = positive predictive value.

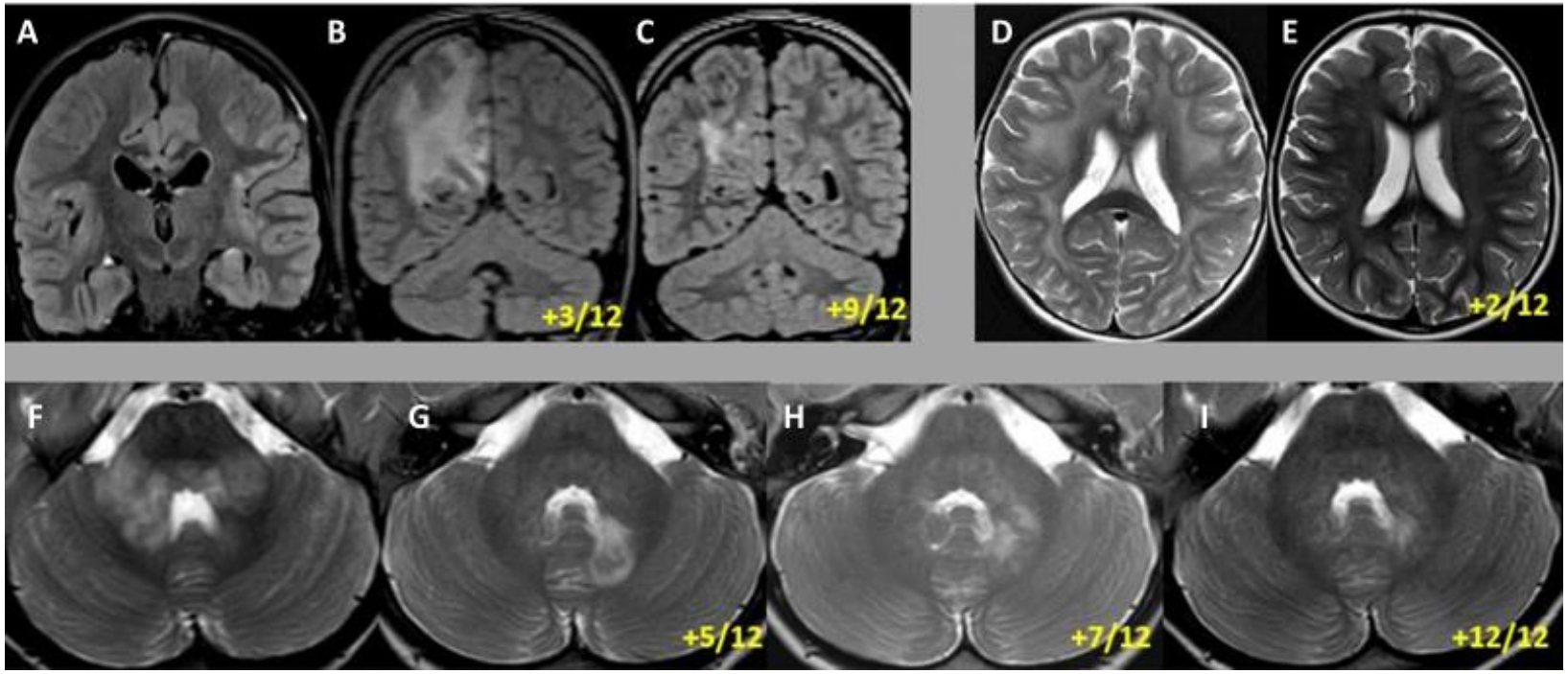
Risi et al., 2024 ***J Neurol.*** 271(5):2840-2843

Filippatou et al., 2024 ***J Neurol Neurosurg Psychiatry*** 95(9):870-873

Cai et al., 2025 ***BMC Med*** 23(1):40

Varley et al., 2024 ***Neurology*** 103(1):e20932

Can lesion dynamics help?



97 MOGAD; 103 MS

- Lesion resolution in 83% in MOGAD (only one MS case)
- Lesion resolution decreased with subsequent attack 40% vs 21% vs None

Abdel-Mannon et al., 2023 ***J Neurol Neurosurg Psychiatry*** 95(5):426-433

MRI T2-lesion evolution in pediatric MOGAD, NMOSD, and MS

T2-lesion resolution

MOGAD	AQP4 + NMOSD	MS
Brain 9 /15 [60%] spine 8 /12 [67%]	brain 1/4 [25%] spine 0/7 [0%])	Brain 0.18 [0%] Spine 1/13 [8%]

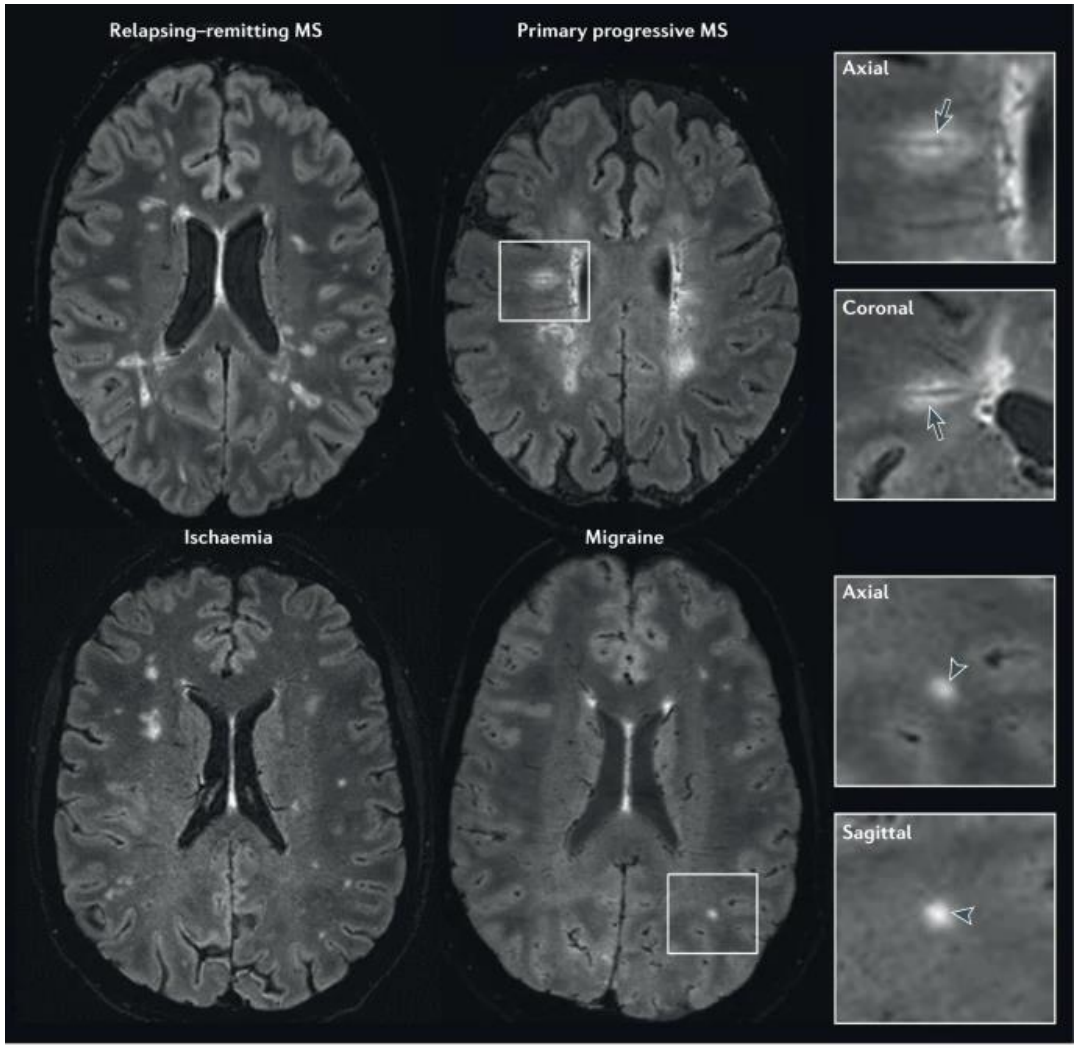
Total T2-lesion resolution

MOGAD	AQP4 + NMOSD	MS
Brain 6/15 [40%] spine 7 /12 [58%]	brain 1/4 [25%] spine 0/7 [0%])	Brain 0.18 [0%] Spine 1/13 [8%]

Median index T2 area

MOGAD Brain 305 mm Spine, 23 mm
AQP4 + NMOSD Brain 133 mm Spine, 19.5 mm
MS Brain 42 mm Spine 10 mm

Redenbaugh et al., 2023 ***Mult Scler .*** 29(7):799-808



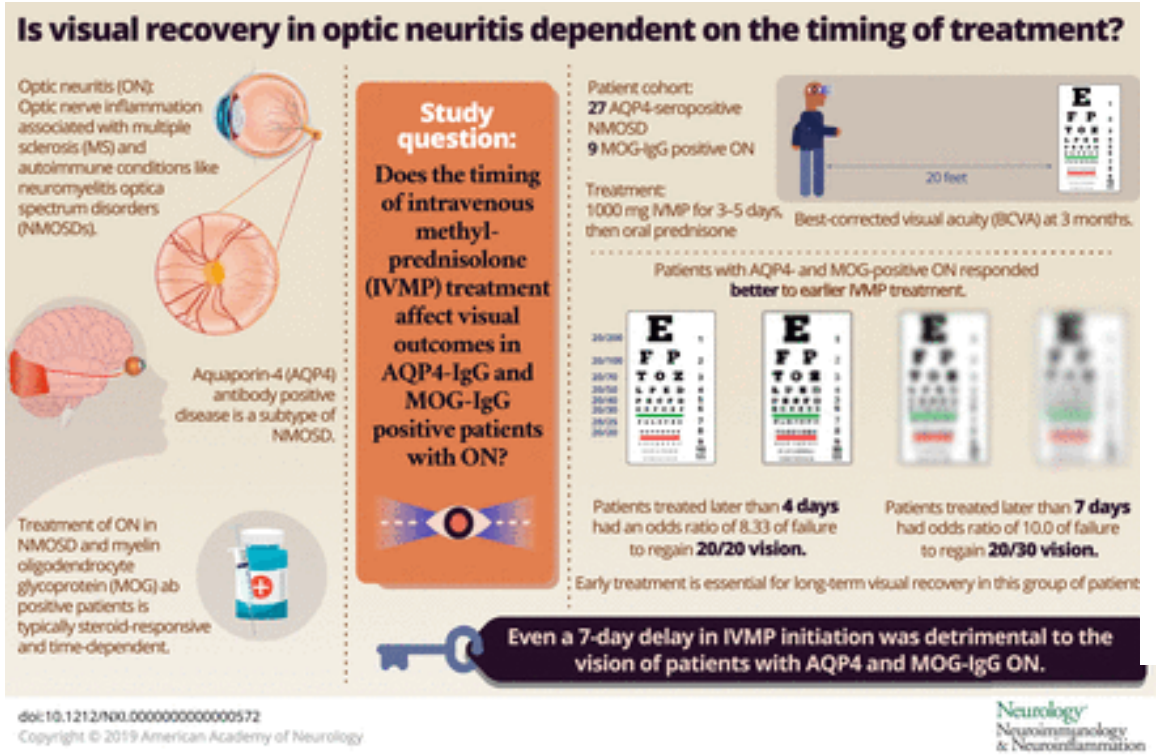
Can lesion characteristic help?

- 68.1% sensitivity and 82.9% specificity for distinguishing MS from not MS (35% threshold)
- 61.9% sensitivity and 89% specificity (3 CVS)

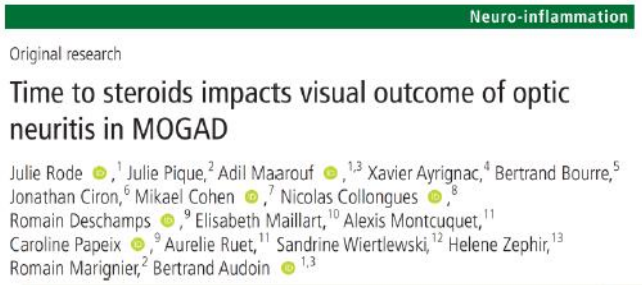
Sati et al.,. 2016 ***Nat Rev Neurol*** 12(12):714-722

Sinnecker et al. 2019 ***JAMA Neurol*** 76(12):1446-1456

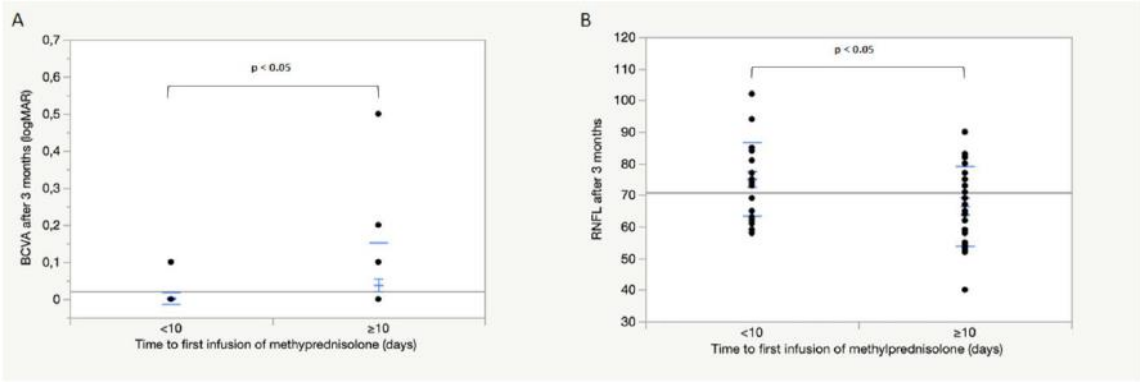
Time is vision



Stiebel-Kalish et al. 2019 ***N2*** 6:e572



First episode of ON
N=82
Mean VA at nadir= 1/10



Absence of full recovery at 3 months was associated delay in MP treatment ≥10 days (OR 16, 95% CI 1.14 to 213, p=0.01).
pRNFL thickness after 3 months was related to better BCVA at nadir and time to first MP treatment <10 days (r2=19%, p=0.004 and r2=11%, p=0.03, respectively).

Rode et al., 2023 ***J Neurol Neurosurg Psychiatry.*** 94(4):309-313

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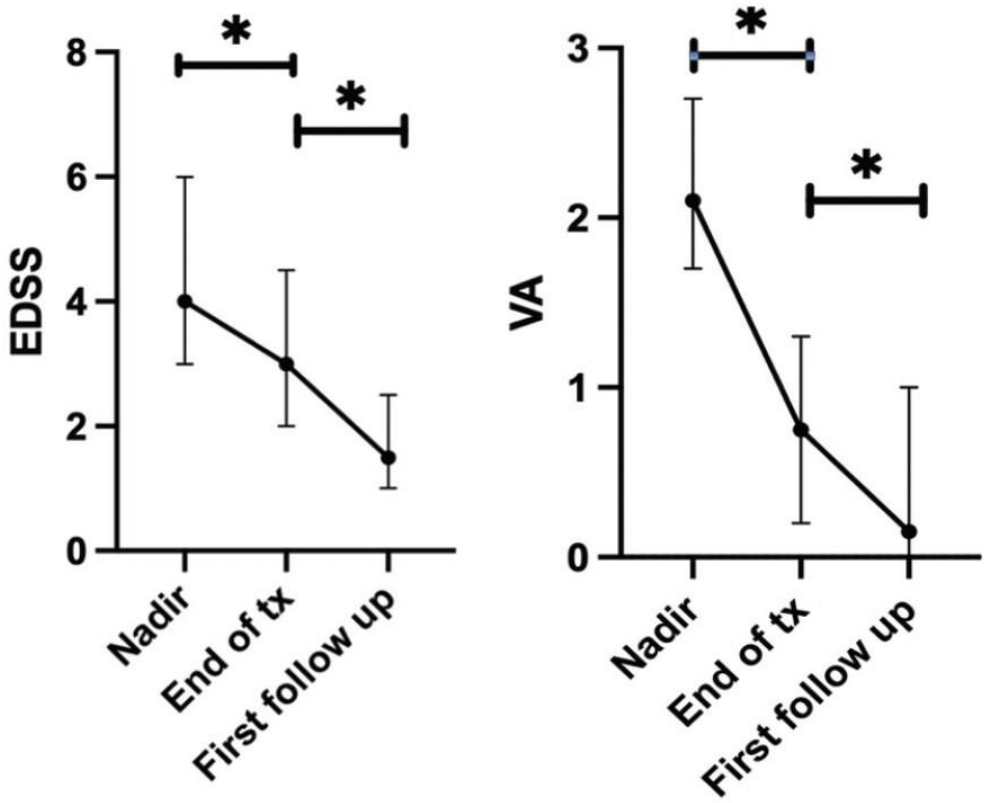


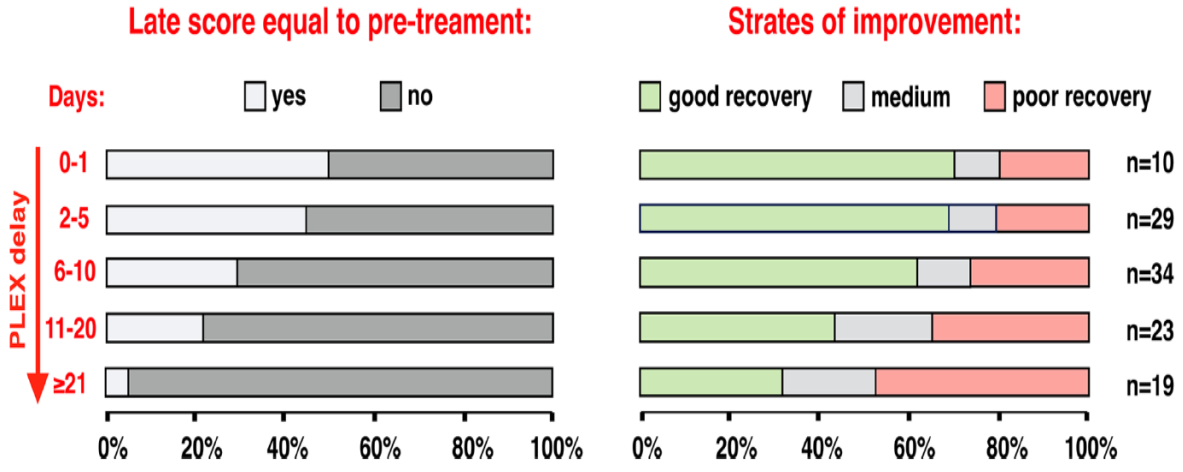
IVIG may be an effective treatment option for acute MOGAD attacks

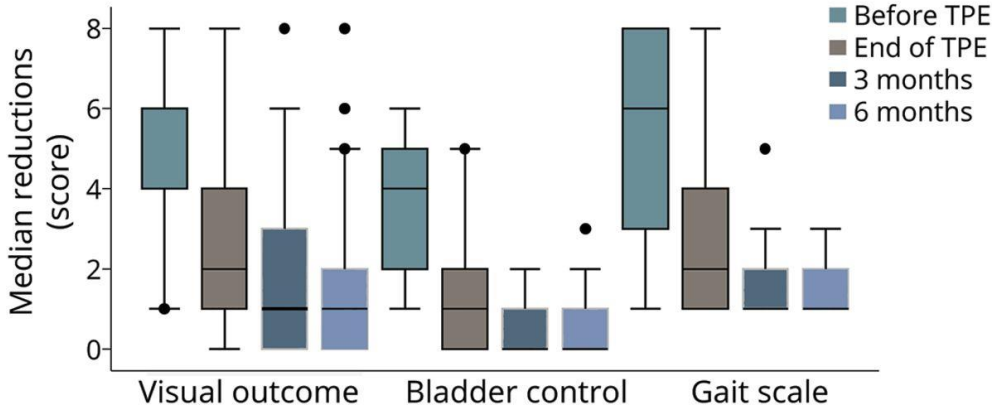
Lotan et al., ***Mult Scler.*** 2023 Aug;29(9):1080-1089

Short delay to initiate plasma exchange is the strongest predictor of outcome in severe attacks of NMO spectrum disorders

BUT window is probably bigger in children

Median time between the index attack onset and TPE initiation was 23 days (range 3–186 days)





Respond even up to 6 months

Bonnan et al., 2018 ***J Neurol Neurosurg Psychiatry.*** Apr;89(4):346-351

Savransky et al., 2019 ***Neurology*** 93(22):e2065-e2073

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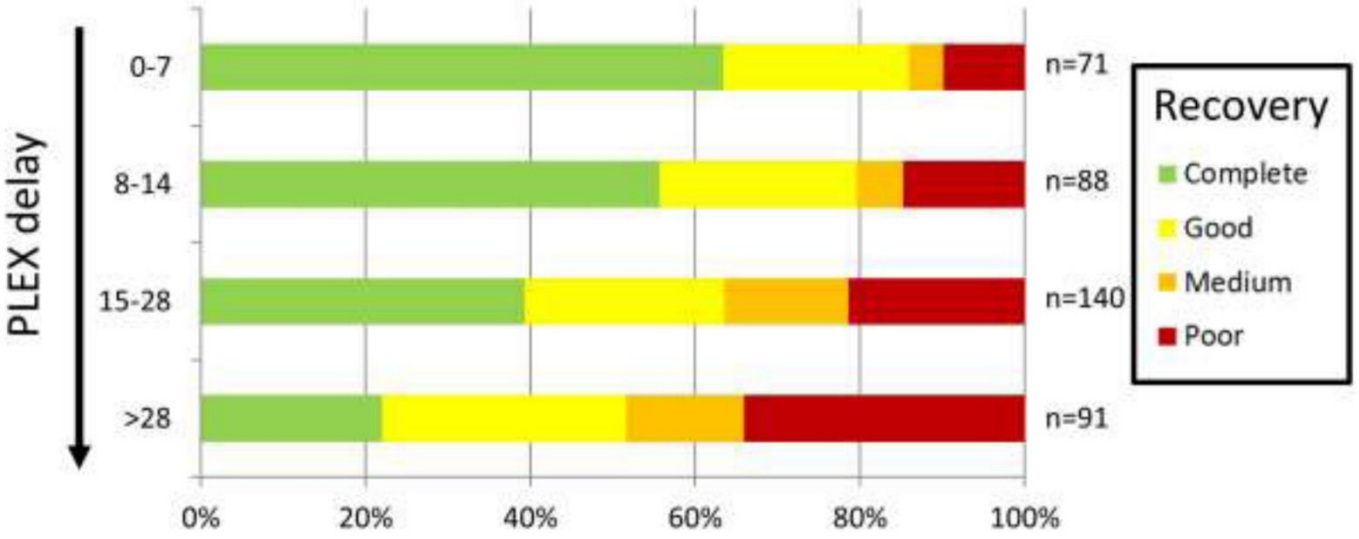
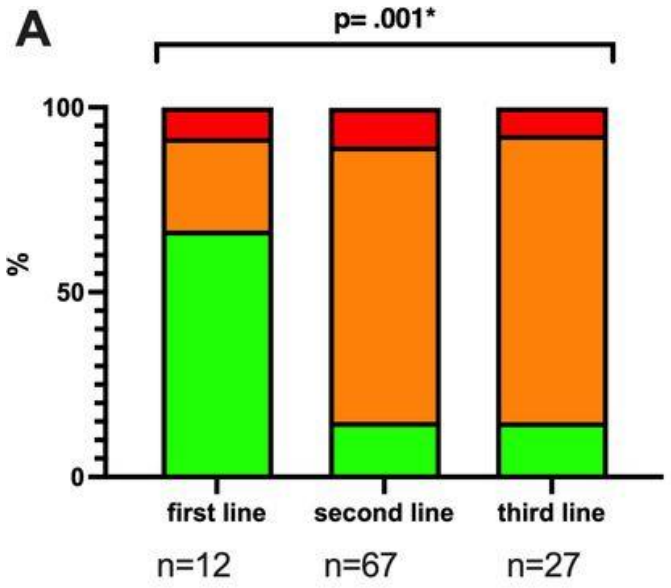
Plasma exchange delay is predictor of outcome in optic neuritis

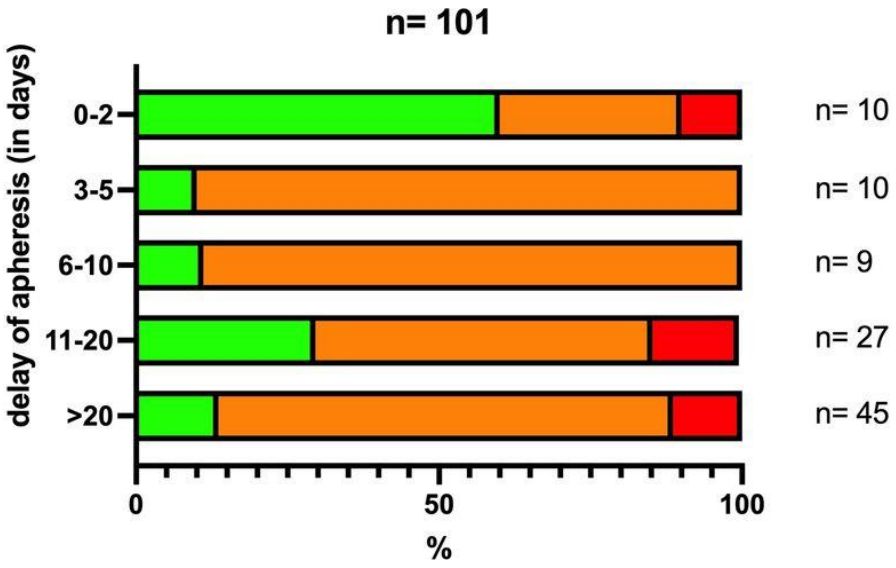


- 395 ON attack treated with PLEX from 317 patients were evaluated.
- Median age was 37 years (range 9 to 75) 71% were female
- 105 MS ; 92 MOGAD; 75 AQP4 NMOSD; 34 DN NMOSD; 83 idiopathic and 2 other

Chen et al., 2023 ***Am J Ophthalmol*** 252:213-224

Efficacy of plasma exchange in MOGAD

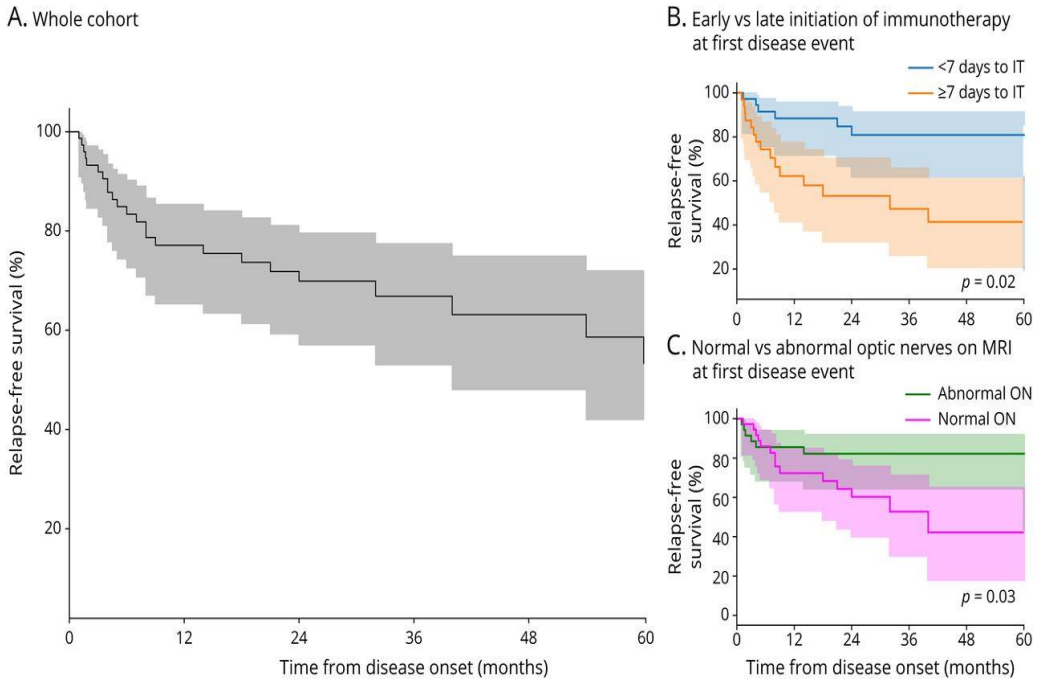




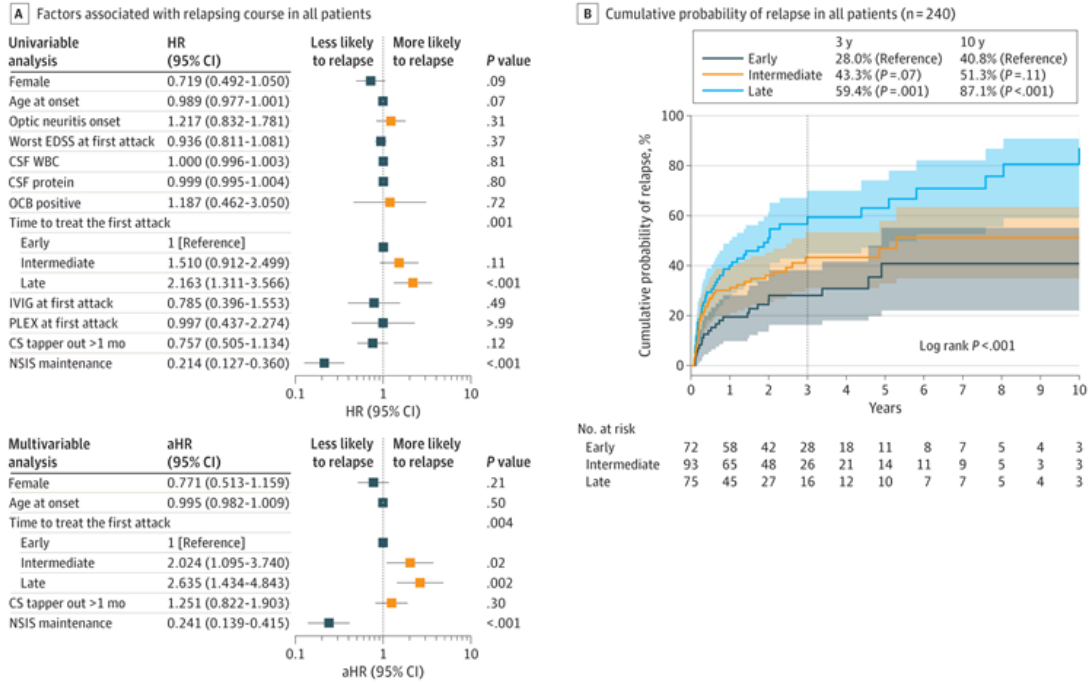
117/571 (20.5%) attacks in 85/209 (40.7%) patients

Schwakew et al., 2024 ***J Neurol Neurosurg Psychiatry***. Nov 4

Early immunotherapy and/or longer corticosteroid treatment influences relapsing disease course in adult and paediatric MOGAD



Nosadini et al., 2022 ***N2*** 10(1):e200065



Kwon et al., 2024 ***JAMA Neurol.*** 81(10):1073-1084

Reduced relapse risk for those dosed ≥12.5 mg/day for at least 3 months (HR 0.12, 95% CI 0.03 to 0.44; p=0.0012)

Trewin et al., 2024 ***J Neurol Neurosurg Psychiatry*** 95(11):1054-63

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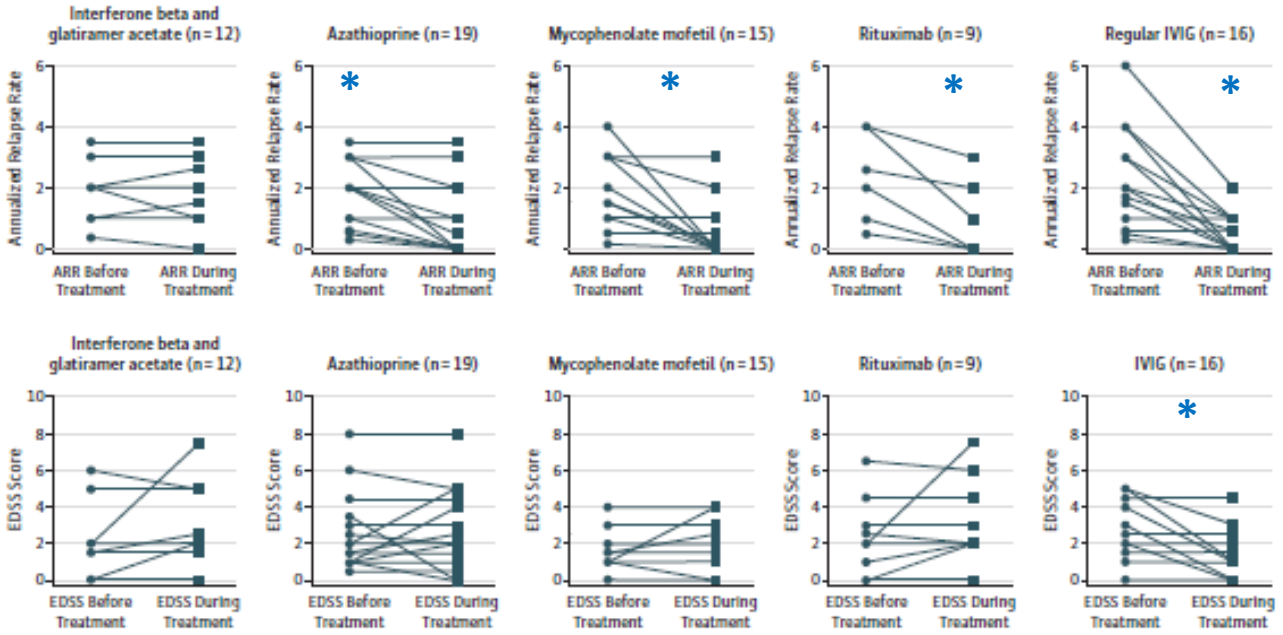
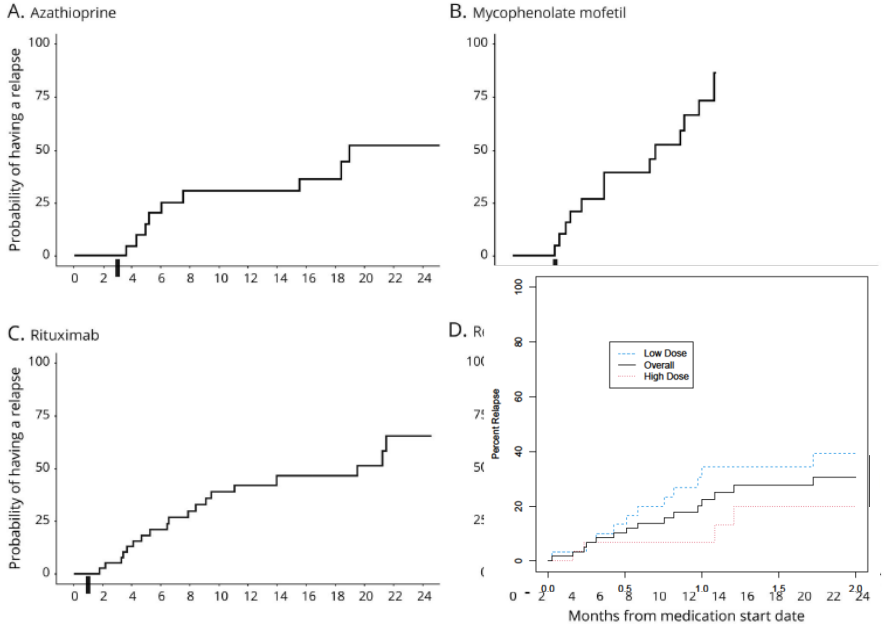
Efficacy of disease modifying therapies in MOGAD

Figure 3. Efficacy of Various Disease-Modifying Therapies in Patients With Myelin Oligodendrocyte Glycoprotein Antibody-Associated Relapsing Demyelination Patients



Hacohen et al., 2018 ***JAMA Neurol.*** 75(4):478-487

Figure 2 Kaplan–Meier estimates of time to relapse on different maintenance therapies



Chen et al., 2020 ***Neurology*** 95(2):e111-e120
Chen et al., 2022 ***JAMA Neurol***. 79(5):518-525

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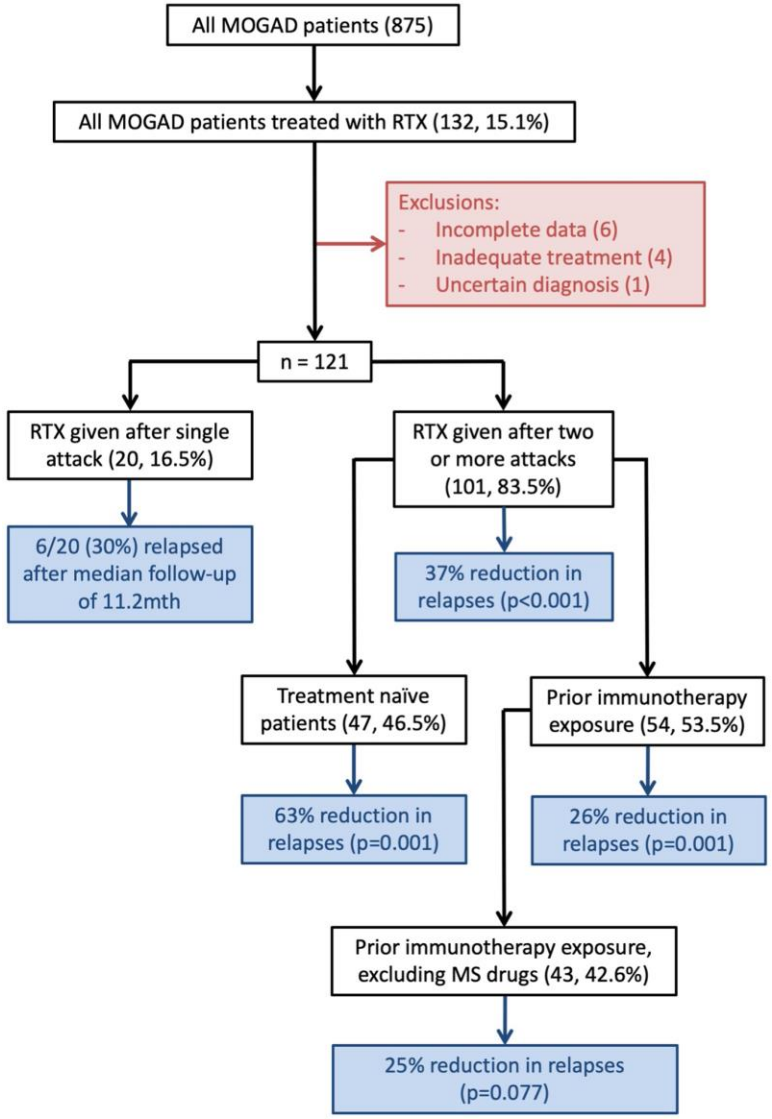
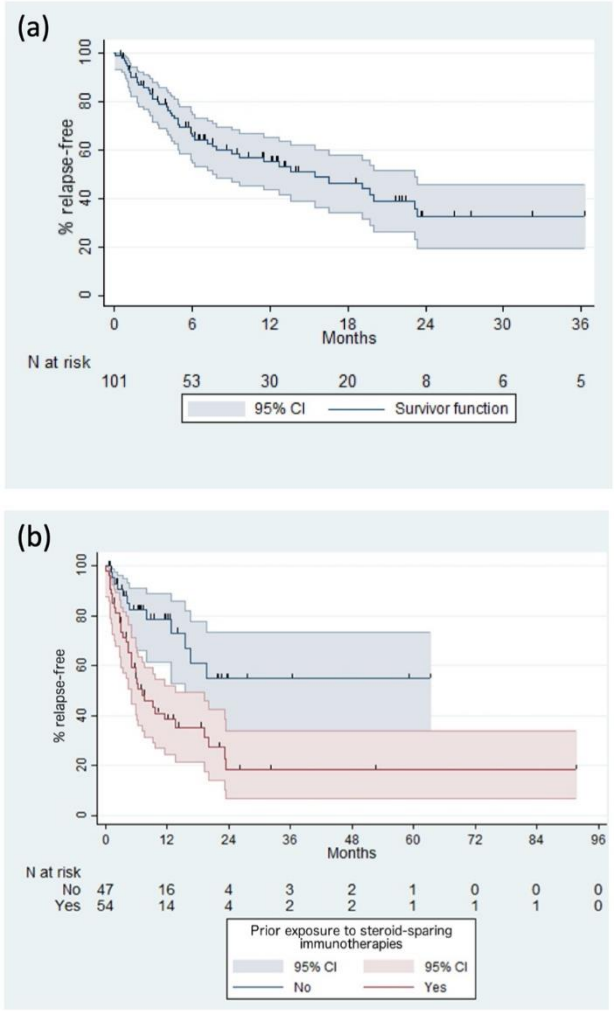


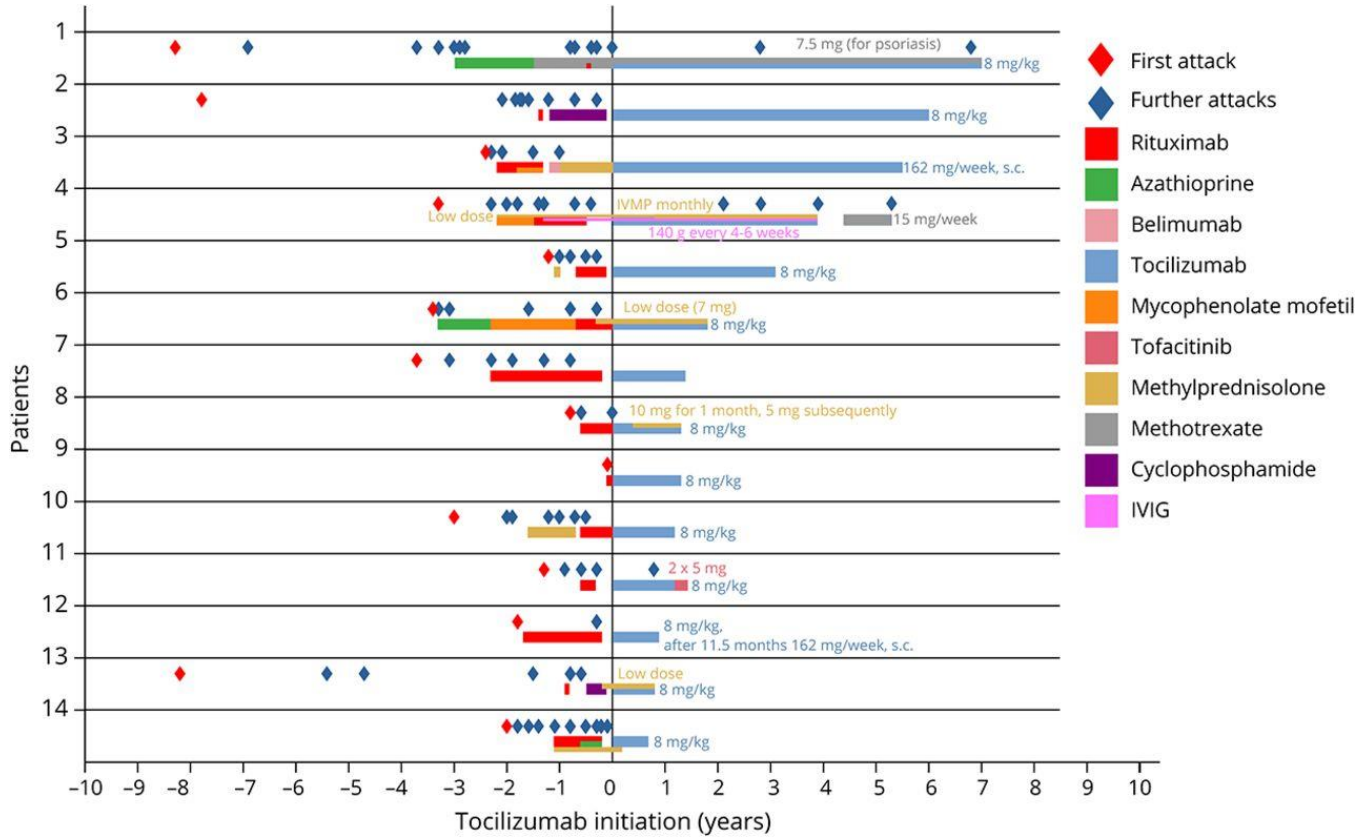


Very modest effect of Rituximab

Circulating CD19⁺*B*-cells were suppressed to <1% of total circulating lymphocyte population at the time of 45/57 (78.9%) relapses.

Whittam et al., 2020 ***Mult Scler Relat Disord***. 44:102251

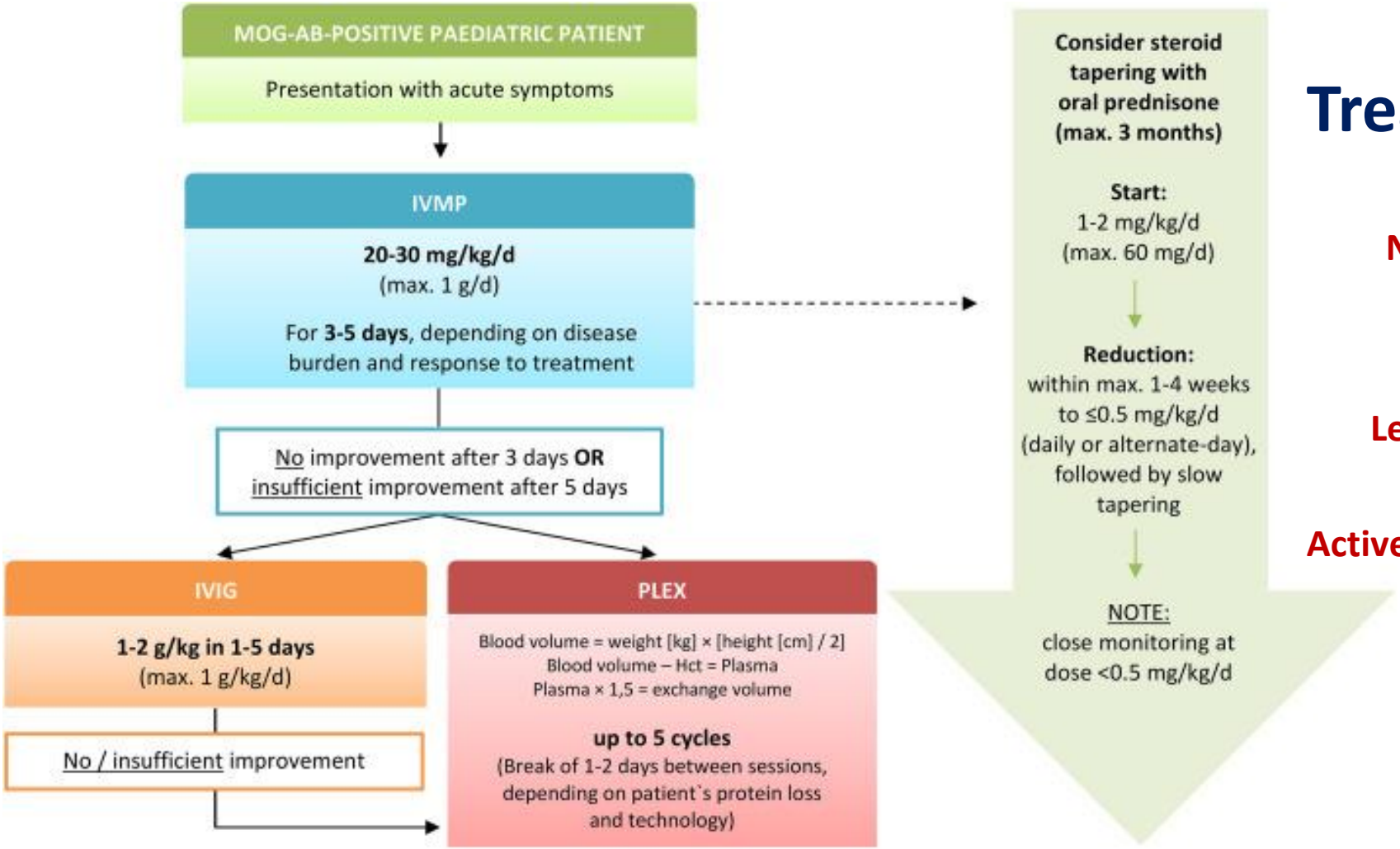
Efficacy of disease modifying therapies in MOGAD



Have we hit the sweet spot with IL6 blockade?

ARR 6 to 0
Relapse free in 11/14
Monotherapy 8/14

Ringelstein et al., 2021 ***N2*** 9(1):e1100



Treatment of MOGAD

Need to treat acute attack quickly and optimally

Less certain re choice of 2nd line

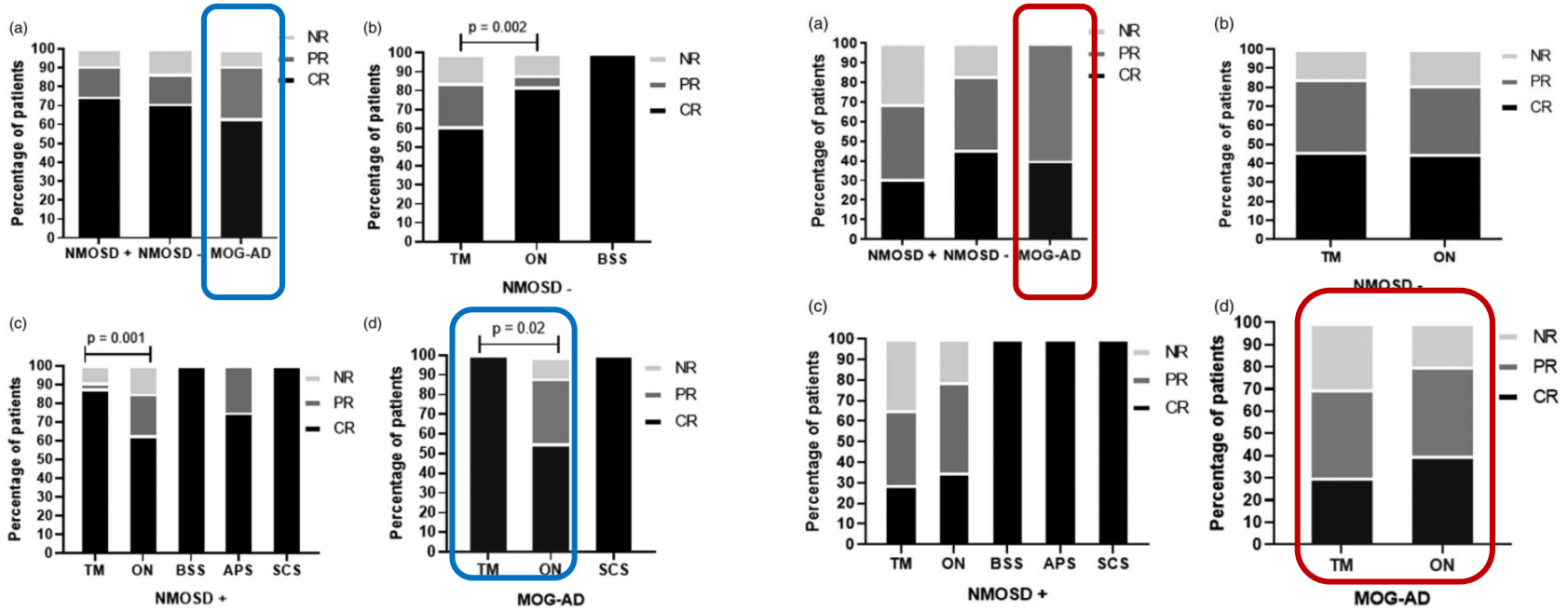
Active consideration of de-escalation after disease stability 3-5 years

Eur J Paediatr Neurol 2020 Nov:29:41-53

Lancet Neurol. 2021 Sep;20(9):762-77

E.U. paediatric MOG consortium consensus 2020

Complete recovery reduces at second attack in adults



Crucial to prevent relapse and disability accrual

Contentti et al., 2021 *Mult Scler J Exp Transl Clin* 7(3):20552173211032334

Impaired Brain Growth in Myelin Oligodendrocyte Glycoprotein Antibody–Associated Acute Disseminated Encephalomyelitis

Figure 2 Brain Volumes at Baseline in MOG-Negative vs MOG-Positive ADEM

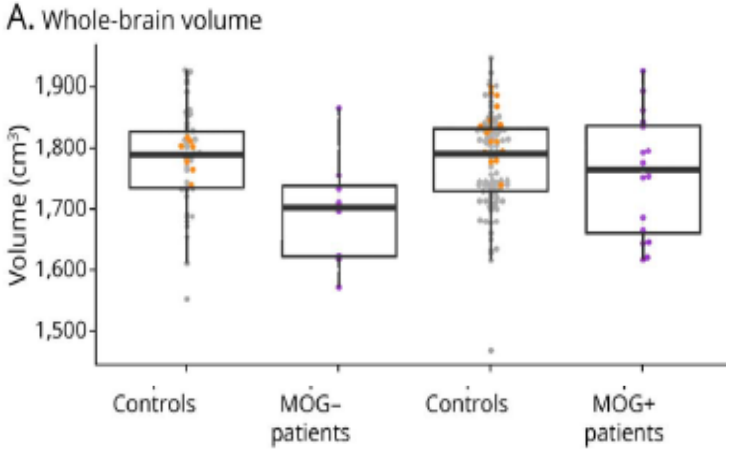
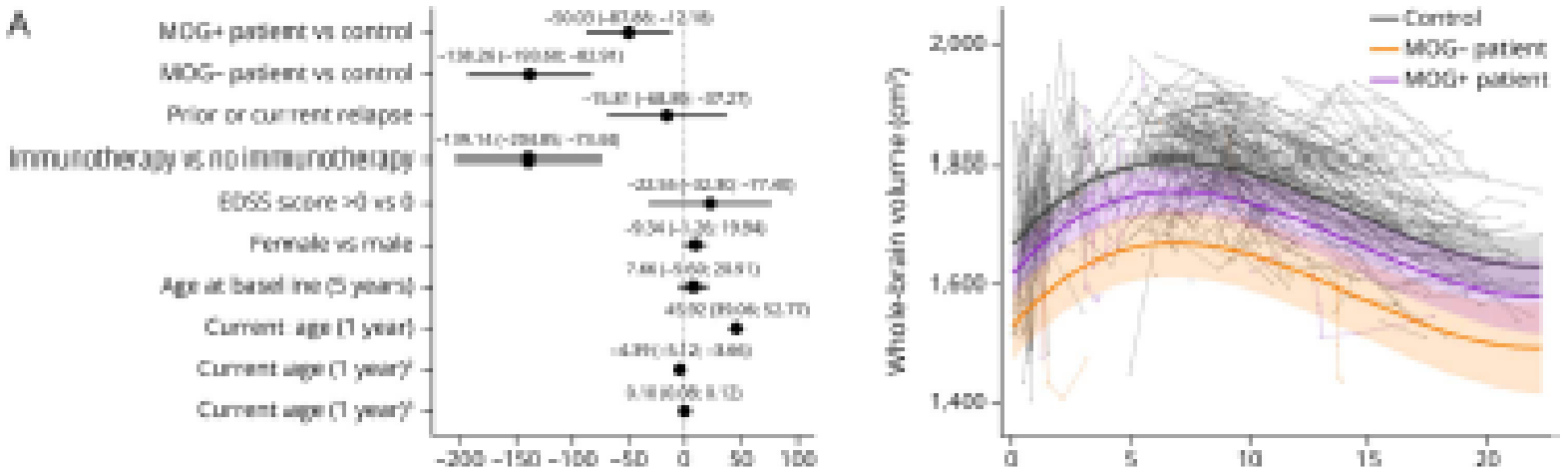


Figure 3 Longitudinal Brain Volume Development



Bartels et al., 2023 ***N2*** 10(2):e200066

Significant cognitive impact in children

Table 1. Demographic, Clinical, and Paraclinical Features of Children According to Their Original Relapsing Demyelination Syndrome Diagnosis[a]

Variable Outcome	MDEM (n = 20)	ADEM-ON (n = 20)	NMOSD (n = 44)	RON (n = 18)	All Patients (N = 102)
Follow-up duration, median (range), y	6.3 (2.0-10.2)	7.0 (3.6-9.2)	5.0 (3.1-7.6)	4.3 (3.0-6.7)	5.5 (3.1-9.0)
TTFR, median (range), mo	5.5 (3.5-28.2)	10.0 (3.0-28.0)	5.0 (2.0-19.0)	12.0 (4.0-27.0)	6.0 (3.0-22.0)
Total No. of relapses, median (IQR)	2.5 (1.0-5.0)	2.0 (2.0-4.0)	2.0 (1.0-4.5)	2.0 (1.0-4.0)	2.0 (1.0-4.0)
EDSS score, median (range)	1.5 (0-5.0)	1.0 (0-4.0)	1.2 (0-10.0)	1.0 (0-2.0)	1.0 (0-10.0)
Good recovery (EDSS score = 0 and no relapse >6 mo)	5.0 (25.0)	5.0 (25.0)	14.0 (31.8)	8.0 (44.4)	32.0 (31.4)
Cognitive problems	10.0 (50.0)	6.0 (30.0)	4.0 (9.1)	0	20.0 (19.6)

Hacohen et al., 2018 ***JAMA Neurol.*** 75(4); 478-487

Factors associated with relapses in MOGAD

TM First attack
OR 0.03

Negative seroconversion
OR 0.11

Male
OR 0.16

Early steroids
OR 0.28 (adults)
OR 0.15 (paediatrics)

Longer steroids
OR 0.15 (paediatrics)

ON First attack
HR 12.5

First remission titres > 1: 2560
HR 10.9

Younger Adult
HR 2.71
Adult vs Child
HR 1.4

New silent MRI lesion
HR 23.86

Huda et al., 2021 ***BMJ Open*** 11:e055392

Nosadini et al., 2022 ***N2*** 10(1):e200065

Kwon et al., 2024 ***JAMA Neurol.*** 81(10):1073-1084

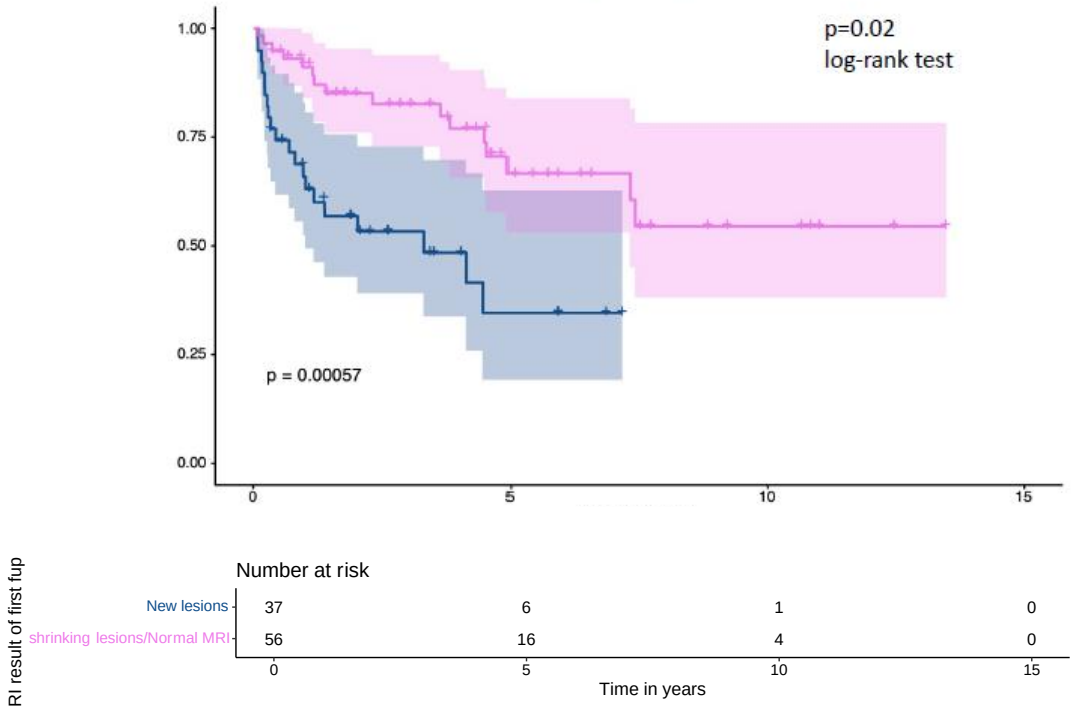
Gastaldi et al., 2023 ***JNNP*** 94; 201-10

Cobo-Calvo et al., 2020 ***Ann Neurol***. 89; 30-41

Camera et al., 2021 ***Jama Network Open*** 4(12) e2137833

Satukijchai et al., 2021 ***Jama Network Open 5***(1) e2142780

Can lesion dynamics predict relapsing disease in children?



38% had silent lesion

New interval lesions are also seen in MOGAD

14% had silent lesion

- 44% detected in first 3 months
- 66% within the year

20% clinical relapses

Fadda et al,. 2021 ***Ann Neurol*** 89 408-13

Abdel-Mannon et al., 2023 ***J Neurol Neurosurg Psychiatry*** 95(5):426-433

Maintenance treatment to prevent future attacks should be offered after the first episode of a demyelinating illness to patients, especially those with:-

- Poor recovery following 1st attack
- Significant risk of relapse (combined risk factors particularly)

?

Crucial to prevent relapse and disability accrual
Maintain NO rather than minimal disability

Conclusion

- MOGAD is recognisable clinically
- Wider differentials in children
- Treatment should be initiated as quickly as possible

Brain and Spine Inflammation

Hock Sin Heng
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Renata Paolilo
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Naomi De Souza
Christina Benetou

Children's Neurosciences

Sarosh Irani; J Palace;
Patrick Waters; M Leite

Evelina Paediatric Research

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European Network on Rare Primary Immunodeficiency, AuToinflammatory and Autoimmune diseases

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UK & Ireland Childhood Neuro-inflammatory Disorder Working Group (UK-CNID)