CLINICAL/SCIENTIFIC NOTE

MOG Antibodies Restricted to CSF in Children With Inflammatory CNS Disorders

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Neurology® 2024;102:e209199. doi:10.1212/WNL.0000000000209199

Abstract

Objectives

To assess the clinical significance of myelin oligodendrocyte glycoprotein antibodies (MOG-abs) restricted to CSF in children with inflammatory CNS disorders.

Methods

Patients included 760 children (younger than 18 years) from 3 multicenter prospective cohort studies: (A) acquired demyelinating syndromes, including acute disseminated encephalomyelitis (ADEM); (B) non-ADEM encephalitis; and (C) noninflammatory neurologic disorders. For all cases, paired serum/CSF samples were systematically examined using brain immunohistochemistry and live cell-based assays.

Results

A total of 109 patients (14%) had MOG-abs in serum or CSF: 79 from cohort A, 30 from B, and none from C. Of these, 63 (58%) had antibodies in both samples, 37 (34%) only in serum, and 9 (8%) only in CSF. Children with MOG-abs only in CSF were older than those with MOG-abs only in serum or in both samples (median 12 vs 6 vs 5 years, $p = 0.0002$) and were more likely to have CSF oligoclonal bands (86% vs 12% vs 7%, $p = 0.0001$) and be diagnosed with multiple sclerosis (6/9 [67%] vs 0/37 [0%] vs 1/63 [2%], $p < 0.0001$).

Discussion

Detection of MOG-abs in serum or CSF is associated with CNS inflammatory disorders. Children with MOG-abs restricted to CSF are more likely to have CSF oligoclonal bands and multiple sclerosis than those with MOG-abs detectable in serum.

Introduction

Autoantibodies to myelin oligodendrocyte glycoprotein (MOG-abs) have been described in children and adults with acquired demyelinating syndromes and less frequently with cortical encephalitis without evidence of demyelination.[1-3] MOG-abs occur more frequently in serum than in CSF, and therefore, serum is considered the sample of choice for MOG-abs testing.[4]

A few studies (mainly in adults) have reported that some patients may have MOG-abs only in CSF,[3,5-9] but the clinical significance of this finding is unclear. For these cases, current diagnostic criteria for MOG-abs–associated disease (MOGAD) require additional supporting clinical or

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Go to Neurology.org/N for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

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radiologic evidence, similar to when antibodies are detected at a low titer in serum.[2] Although MOGAD is more frequent in children than adults, the presence of CSF-restricted MOG-abs has been less frequently studied in children and its significance is yet more uncertain. We determined the frequency and clinical relevance of MOG-abs in serum and CSF of children with acute demyelinating syndromes, encephalitis, or non-inflammatory neurologic disorders.

Methods

Participants and Samples

This study was conducted with patients younger than 18 years who between June 1, 2013, and June 30, 2022, were recruited in 3 cohorts: (A) patients with suspected acute disseminated encephalomyelitis (ADEM) or other demyelinating syndromes, (B) patients with encephalitis other than ADEM,[3] and (C) patients excluded from cohorts A and B who had a non-inflammatory neurologic disorder. Overall, 40 collaborating pediatric centers in Spain contributed to patient recruitment. Clinical information was collected through structured questionnaires that were prospectively completed by the treating physicians at disease onset and every 6 months.

In all patients, comprehensive neuronal surface and glial antibody testing was performed in serum and CSF at IDIBAPS-Hospital Clinic, University of Barcelona, Spain, as previously reported (eMethods, links.lww.com/WNL/D416).[3,10] The clinical syndrome at onset of the disease and at final diagnosis was defined according to current international criteria.[2,11-13] Disease severity was assessed with the modified Rankin Scale adapted for children.[14]

Statistical Analysis, Standard Protocol Approvals, Registrations, and Patient Consents

This study was approved by the Ethical Board Committee of Hospital Clínic, Barcelona. Written consents were obtained from the proxies (eMethods, links.lww.com/WNL/D416).

Data Availability

Anonymized data are available by request from qualified investigators. The corresponding author takes full responsibility for the data, the analyses and interpretation, and the conduct of the research; the authors have full access to all the data and have the right to publish any and all data, separate and apart from the guidance of any sponsor.

Results

A total of 760 patients with paired serum/CSF samples were recruited: 309 with acquired demyelinating syndromes (cohort A), 287 with suspected encephalitis other than ADEM (cohort B), and 164 with noninflammatory neurologic disorders (cohort C). Diagnoses at the last follow-up in patients from Cohort C were epilepsy (79 patients), primary psychiatric disorder (48), confirmed genetic disorder (22), and isolated movement disorder (15).

Overall, 109 of 760 patients (14%) tested positive for MOG-abs in either serum or CSF including 79 of 309 (26%) from cohort A, 30 of 287 (10%) from cohort B, and 0 of 164 (0%) from cohort C. Of these 109 MOG-abs–positive patients, 63 (58%) were positive in both serum and CSF, 37 (34%) only in serum, and 9 (8%) only in CSF. None of the 9 patients with CSF-restricted MOG-abs underwent plasma exchange before obtaining the samples. Of the 109 MOG-abs–positive patients, 5 (4.5%) had concurrent GlyR-abs (3 only in serum, 1 in serum and CSF, and 1 only in CSF) and another 4 (4%) had concurrent NMDAR-abs in CSF (2 with MOG-abs in both serum and CSF and 2 with MOG-abs only in serum).

Clinical Features of Patients With MOG-Abs

At disease presentation, the syndromes of MOG-abs–positive patients included ADEM (n = 46, 42%), non-ADEM encephalitis or FLAIR-hyperintense lesions in anti-MOG–associated encephalitis with seizures (FLAMES) (n = 30, 28%), optic neuritis (ON) and/or transverse myelitis (TM) (n = 29, 27%), and other (n = 4, 4%) (Table 1). At the last follow-up, 74 patients (68%) had been diagnosed with monophasic MOGAD, 28 (26%) with relapsing MOGAD, and 7 (6%) with multiple sclerosis (MS) (Table 1). Two of the patients with concurrent NMDAR-abs, both with MOG-abs in serum and CSF, developed a typical clinical picture of anti-NMDAR encephalitis that was followed by a demyelinating episode 2 and 19 months later. The remaining 2 patients with NMDAR-abs (both with MOG-abs in serum only) had clinical manifestations of anti-NMDAR encephalitis without clinical or radiologic demyelinating features during the course of the disease. All 5 patients with concurrent GlyR-abs had typical clinicoradiologic features of MOGAD.

Patients' demographic and clinical information is presented in Table 1. The clinical features of patients with MOG-abs detected only in serum were similar to those of patients with MOG-abs in both serum and CSF, including the presenting symptoms and outcome, but different from patients who had MOG-abs only in CSF (Table 1). Children with MOG-abs only in CSF were older than those with MOG-abs only in serum or in both samples (median 12 vs 6 vs 5 years, respectively, $p = 0.0002$), had higher protein levels in CSF (median 40 vs 22 vs 36 mg/dL, $p = 0.0094$), were more likely to have CSF oligoclonal bands (86% vs 12% vs 7%, $p = 0.00013$), and were more often diagnosed with MS (6/9 [67%] vs 0/37 [0%] vs 1/63 [2%], $p < 0.0001$) (Table 1, Figure 1). The 3 patients with MOG-abs only in CSF who were not diagnosed with MS had a definite diagnosis of monophasic ADEM (n = 1), ADEM followed by ON (n = 1), and non-ADEM encephalitis (n = 1). Symptom severity at disease onset and at the last follow-up was not different between the groups (Table 1).

Discussion

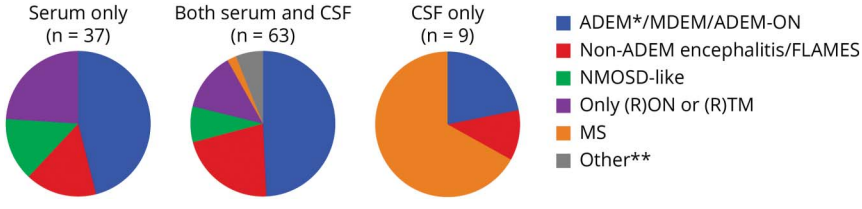
In this large cohort of pediatric patients with inflammatory and noninflammatory disorders, we found that (1) the presence of

Table Characteristics of Pediatric Patients With MOG-Abs Positive in Serum and/or CSF

	MOG-abs–positive				
	All patients (n = 109)	Serum only (n = 37)	Both serum and CSF (n = 63)	CSF only (n = 9)	*p* Value
Age at onset, y, median (IQR)	6 (3–10)	6 (3–10)	5 (3–7)	12 (11–14)	0.0002[f]
Female, n (%)	58 (53)	19 (51)	32 (50)	7 (78)	0.3
Syndrome or clinical presentations? At onset, n (%)					0.09
ADEM	46 (42)	17 (46)	27 (43)[b]	1 (11)	
Non-ADEM encephalitis/FLAMES[a]	30 (28)	8 (22)	20 (32)	2 (22)	
ON and/or TM	29 (27)	12 (32)	14 (22)	4 (45)	
Other[c,d]	4 (4)	0	2 (3)[c]	2 (22)[d]	
CSF analysis					
OCB, n (%)	12/52 (23)	2/17 (12)	4/28 (14)	6/7 (86)	0.0001[f]
WBC (cell/mm^3), median (IQR)	20 (5–60)	13 (3–34)	30 (8–77)	10 (10–23)	0.1
Protein (mg/dL), median (IQR)	32 (22–45)	24 (16–37)	36 (24–52)	40 (36–44)	0.009[f]
Disease course, n (%)					<0.0001
Monophasic MOGAD	74 (68)	23 (62)	49 (78)	2 (30)	
Relapsing MOGAD	28 (26)	14 (38)	13 (20)	1 (10)	
MS	7 (6)	0	1 (2)	6 (60)	
Outcome					
mRS onset, median (IQR; total n)	4 (3–4; n = 99)	4 (3–4; n = 32)	4 (3–4; n = 58)	3 (3–4; n = 9)	0.9
mRS final FU, median (IQR; total n)	0 (0–1; n = 88)	0 (0–1; n = 30)	0 (0–1; n = 49)	1 (0–1; n = 9)	0.5
FU time, mo, median (IQR)	18 (7–45)	18 (4–52)	18 (7–41)	43 (15–67)[e]	0.2

Abbreviations: ADEM = acute disseminated encephalomyelitis; CIS = clinically isolated syndrome; FLAMES = FLAIR-hyperintense lesions in MOG-associated encephalitis with seizures; FU = follow-up; IQR = interquartile range; MOG = myelin oligodendrocyte glycoprotein; mRS = modified Rankin Scale; MS = multiple sclerosis; OCB = oligoclonal band; ON = optic neuritis; TM = transverse myelitis; WBC = white blood cell.
p Values were obtained using Kruskal-Wallis test for numerical variables and Fisher exact test for categorical variables.
[a] Patients with confirmed encephalitis who did not fulfill criteria of ADEM[12] (e.g., cortical involvement or not predominant bilateral demyelinating involvement) were categorized as non-ADEM encephalitis.[3] Patients with FLAIR-hyperintense lesions with MOG-abs and seizures without encephalopathy were classified as FLAMES.[2]
[b] Includes 3 patients without clear encephalopathy but MRI with typical ADEM lesions.
[c] Includes 1 patient with a tumefactive lesion and 1 patient with ataxia only and normal MRI (monophasic).
[d] Includes 1 patient with a hemispheric CIS (finally diagnosed with MS) and 1 patient with a polyfocal CIS (finally diagnosed with MS).
[e] Although not statistically significant because of longer follow-up in the CSF-only group, we performed an additional Cox proportional hazards model analysis, which showed that the risk to convert to MS is related to serum/CSF MOG status ($p < 0.0001$), but unrelated to time of follow-up ($p = 0.6$).
[f] $p < 0.05$

Figure 1 Diagnoses in Patients With MOG-Abs Detected in Serum Only, Both Serum and CSF, and CSF Only



*Includes 2 patients without clear encephalopathy but MRI with typical ADEM lesions (both with MOG-abs in serum and CSF). **Includes 1 patient without encephalopathy and a single tumefactive lesion; 1 patient with isolated ataxia, without encephalopathy; 1 patient with non-ADEM encephalitis followed by ADEM; and 1 patient with a leukodystrophy-like disease (all with MOG-abs in serum and CSF). ADEM = acute disseminated encephalomyelitis; FLAMES = FLAIR-hyperintense lesions in MOG-associated encephalitis with seizures; ON = optic neuritis; MDEM = multiphasic disseminated encephalomyelitis; MOG = myelin oligodendrocyte glycoprotein; MS = multiple sclerosis; NMOSD = neuromyelitis optica spectrum disorder; (R) = recurrent; TM = transverse myelitis.

MOG-abs was highly specific for CNS inflammatory disorders; (2) most patients with MOG-abs had antibodies only in serum or in serum and CSF (92%); and (3) among the small subset (8%) of patients with MOG-abs restricted to CSF, approximately two-thirds developed MS and one-third MOGAD.

The low frequency of cases with CSF-restricted MOG-abs is in line with previous studies.[2,5,7-9] However, in contrast to other reports (mainly focused on adults) showing that CSF-restricted MOG-abs associated with MOGAD, we found that almost two-thirds of children with CSF-restricted MOG-abs developed MS. Moreover, none of the 37 patients with serum-restricted MOG-abs and only 1 of 63 (1.6%) with MOG-abs in serum and CSF developed MS. The importance of this observation is emphasized by the large sample size of our cohort and the extensive number of paired serum/CSF samples. These findings suggest a note of caution for the interpretation of CSF-restricted MOG-abs in children, given that in this setting, MOG-abs also occurs in MS.

It has been reported that some adult and pediatric patients with MS may have a low serum MOG-abs titer.[15] Because MOG-abs CSF status is usually not provided in those cases, it cannot be excluded that they may have higher MOG-abs in CSF along with intrathecal synthesis of MOG-abs, as occurs with those with CSF-restricted MOG-abs. A limitation of our study is that we were not able to determine the intrathecal synthesis of antibodies in patients who were positive in both samples (serum and CSF) because the limited amount of remaining CSF precluded the proper analysis. By contrast, strengths of our study include the large number of patients from nonbiased prospective nationwide cohort studies; inclusion of many participants with noninflammatory disorders; and centralized antibody testing in a single institution using live cell-based assays, which is considered the most sensitive and specific diagnostic test.[2,4]

The main clinical implication of this study is that in children, MOG-abs can be found restricted to CSF, but different from adults, this immunologic setting is often associated with MS.

Acknowledgment

The authors thank the patients and families for their participation.

Study Funding

This work was supported in part by grants from Plan Nacional de I+D+I and cofinanced by the ISCIII—Subdirección General de Evaluación y Fomento de la Investigación Sanitaria—and the Fondo Europeo de Desarrollo Regional (ISCIII-FEDER; PI21/00316 to T. Armangué); Fundació Marató de TV3 (37/C/2021, T. Armangué), Torrons Vicens Foundation (PFNR0144 to T. Armangué); 2021 Invest AEP Grant to T. Armangué (PI047351), and 2019 Invest-AEP Support to G. Olivé-Cirera from Pediatric Spanish Society. G. Olivé-Cirera is recipient of a Rio Hortega grant (CM22/00066) from the Instituto de Salud Carlos III (ISCIII), Spain, co-financed by Fondo Social Europeo Plus (FSE+). A.L. Bruijstens is recipient of the KNAW Ter Meulen Grant/KNAW Medical Sciences Fund, Royal Netherlands Academy of Arts & Sciences (2022), co-financed by the Dutch Society of Child Neurology and Guthy-Jackson Charitable Foundation. M. Guasp is recipient of a Rio Hortega grant (CM21/00016) from the Instituto de Salud Carlos III (ISCIII), Spain, co-financed by Fondo Social Europeo Plus (FSE+).

Disclosure

G. Olivé-Cirera, A.L. Bruijstens, E. Fonseca, L.W. Chen, and E. Caballero report no disclosures relevant to the manuscript. E. Martínez-Hernandez received speaking compensation from Biogen. M. Sepulveda received speaking honoraria from Roche, Biogen, and UCB Pharma and travel reimbursement from Biogen, Sanofi, Merck, and Roche for national and international meetings. M. Guasp, M. Sepulveda, L. Naranjo, and R. Ruiz-García report no disclosures relevant to the manuscript. Y. Blanco received speaker honoraria from Novartis, Roche, Sanofi, Merck, and Biogen. A. Saiz received personal compensation for consulting, serving on a scientific advisory board, speaking, or other activities with Merck, Sanofi, Biogen, Roche, Novartis, Janssen, and Horizon Therapeutics. J. Dalmau holds patents for the use of Ma2, NMDAR, GABABR, GABAAR, DPPX and IgLON5 as autoantibody tests. J. Dalmau receives royalties related to autoantibody tests from Athena Diagnostics and Euroimmun, Inc. The rest of the authors have no conflicts of interest related to the submitted work. T. Armangué received personal compensation for speaking fees from Sanofi and Roche. Go to Neurology.org/N for full disclosures.

Publication History

Received by *Neurology* September 19, 2023. Accepted in final form December 13, 2023. Submitted and externally peer reviewed. The handling editor was Associate Editor Courtney Wusthoff, MD, MS.

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Appendix 1 *(continued)*

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Appendix 2 Coinvestigators

Coinvestigators are listed at links.lww.com/WNL/D415.

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