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# Frequency and clinical relevance of MOG-antibodies in CSF in pediatric patients with MOG antibody-associated diseases

G Galati <sup>1</sup>, J Pique <sup>2</sup>, P Horellou <sup>3</sup>, C Leroy <sup>3</sup>, M Poinso <sup>2</sup>, R Marignier <sup>2</sup>, L Giorgi <sup>4</sup>, K Deiva <sup>5</sup>; Kidbiosep cohort and MIRCEM network

## Collaborators

- **Kidbiosep cohort and MIRCEM network:**

Elisabeth Maillart, Anne-Gaëlle LE Moing, Daniel Amsalem, Frédéric Villega, Sylviane Peudenier, Sylvie Nguyen-The Tich, Anne Lepine, Pierre Meyer, Hélène Vincent, Florence Renaldo, Melodie Aubart, Stéphane Auvin, Anne DE Saint-Martin, Emmanuel Cheuret, Pierre Castelnau, Stéphanie Robin, Aurélie Ruet, Hélène Zephir, Bertrand Audoin, Xavier Ayrignac, David Laplaud, Mickael Cohen, Caroline Papeix, Bertrand Bourre, Nicolas Collongues, Jonathan Ciron

<sup>1</sup> Pediatric Neurology Department, Assistance Publique-Hôpitaux de Paris, Paris-Saclay University Hospitals, Bicêtre Hospital, Le Kremlin Bicêtre, France; Child Neuropsychiatry Unit, Department of Surgery, Dentistry, Paediatrics and Gynaecology, University of Verona, Verona, Italy; Child Neuropsychiatry Unit, Department of Mental Health, Infermi Hospital, Rimini, Italy. Electronic address: giulia.galati@auslromagna.it.

<sup>2</sup> National Referral Center for Rare Inflammatory and Auto-Immune Brain and Spinal Diseases and Service de Neurologie, Sclérose en Plaques, Pathologies de La Myéline et Neuro-inflammation, Hôpital Neurologique Pierre Wertheimer, Hospices Civils de Lyon, Lyon, France.

<sup>3</sup> National Referral Center for Rare Inflammatory and Auto-Immune Brain and Spinal Diseases, Le Kremlin-Bicêtre, France.

<sup>4</sup> Pediatric Neurology Department, Assistance Publique-Hôpitaux de Paris, Paris-Saclay University Hospitals, Bicêtre Hospital, Le Kremlin Bicêtre, France; National Referral Center for Rare Inflammatory and Auto-Immune Brain and Spinal Diseases, Le Kremlin-Bicêtre, France.

<sup>5</sup> Pediatric Neurology Department, Assistance Publique-Hôpitaux de Paris, Paris-Saclay University Hospitals, Bicêtre Hospital, Le Kremlin Bicêtre, France; National Referral Center for Rare Inflammatory and Auto-Immune Brain and Spinal Diseases, Le Kremlin-Bicêtre, France; Institut Universitaire De France, France.

**\* Corresponding author.**

Pediatric Neurology Department, Assistance Publique-Hôpitaux de Paris, Paris-Saclay University Hospitals, Bicêtre Hospital, Le Kremlin Bicêtre, France. *E-mail address:* giulia.galati@auslromagna.it (G. Galati).

# Abstract

## Objective:

This retrospective study aimed to describe a cohort of 38 pediatric patients with MOGAD and to investigate the clinical differences between patients with CSF-negativity and CSF-positivity for MOG-abs.

## Methods:

The clinical and laboratory characteristics of pediatric patients with MOGAD were retrospectively studied. Demographics, clinical characteristics, CSF analysis, treatments and prognosis of patients were recorded. All patients' serums and CSF were tested for MOG-IgG by live cell-based assays (CBA). The data were statistically analysed.

## Results:

A total of 38 pediatric MOGAD patients were enrolled in the study, including 22 (57.9 %) females and 16 male (42.1 %) with a mean age of  $8.4 \pm 4.0$  years at disease onset. Twenty-seven (71.7 %) patients were CSF-positive for MOG-abs while 11 (28.9 %) patients were CSF-negative for MOG-abs. The median follow-up was 25.5 months (IQR 5.5-73.25). Seventeen (44.7 %) patients presented a relapsing disease course, and the majority of these patients was CSF positive with a significant difference between the two groups ( $p = 0.038$ ) in terms of recurrent diseases. CSF-positive patients presented more often an increased white cell count ( $p = 0.043$ ), and in this cohort clinical phenotypes with spinal involvement were more frequent while encephalitis-like phenotypes were more frequent in the CSF negative cohort ( $p = 0.019$ ).

## Conclusions:

CSF-status appears to identify two subgroups in this pediatric MOGAD population; thus, CSF-status requires further studies in pediatric patients with MOGAD.

# 1. Introduction

Myelin oligodendrocyte glycoprotein antibody-associated diseases (MOGAD) are a broad spectrum of acquired demyelinating disorders of the central nervous system affecting both adult and paediatric patients.

Serum is the preferred specimen type for MOG-IgG diagnostic testing [1], while the prevalence and clinical relevance of MOG-Abs positivity in CSF in children with MOGAD is unclear.

Therefore, we aim to address this through the analysis of clinical and paraclinical data of a cohort of children with MOGAD.

## 2. Patients and methods

### *2.1. Patients*

In this retrospective double-centre study (National Reference Centres for Rare Inflammatory Brain and Spinal Diseases of Paris and Lyon) of a French pediatric cohort, patients were recruited with the following inclusion criteria: [1] age <18 years, [2] positive MOG-IgG test at onset or at the time of a clinical relapse detected by live cell-based assays (CBA), [3] clinical phenotype compatible with MOGAD [2], [4] exclusion of better diagnoses, [5] availability of paired samples of serum and CSF obtained during an acute event.

We analysed demographic and clinical data including: sex, age at onset, clinical manifestations, neuroimaging exams, Expanded Disability Status Scale (EDSS) score at last observation, acute and chronic treatments, presence of impaired visual acuity at last observation, follow-up duration and relapse rate.

Clinical manifestations were defined as acute disseminated encephalomyelitis (ADEM), optic neuritis (ON), transverse myelitis (TM), transverse myelitis and concurrent optic neuritis (NMOSD-like phenotype), encephalitis and atypical clinical phenotypes (Leukodystrophy-like phenotype) [2].

The participants are members of the KidBioSEP cohort; this study was covered by a general approval and it did not require any additional procedure.

**Table 1**  
Demographics, clinical features and outcomes of pediatric patients with MOGAD.

	Age/ Sex	Clinical manifestation at onset	CSF-positivity MOG-IgG	CSF, analysis	Acute therapy	Relapses/ N	EDSS last FU/impaired visual acuity
Case 1	12/F	Neuromyelitis optica spectrum disorders (NMOSD)-like phenotype	yes	Protein >0.45 mg/dL, WCC >5/ $\mu$ l	IVMP, PLEX	yes, 1	0/yes
Case 2	9/F	Transverse Myelitis (TM) phenotype	yes	Protein >0.45 mg/dL, WCC >5/ $\mu$ l	IVMP, PLEX	yes, 2	0/no
Case 3	6/F	Transverse Myelitis (TM) phenotype	yes	WCC >5/ $\mu$ l	IVMP	yes, 2	0/no
Case 4	6/F	Acute Disseminated Encephalomyelitis (ADEM)	yes	Protein >0.45 mg/dL	IVMP, PLEX	yes, 5	0/no
Case 5	5/F	Neuromyelitis optica spectrum disorders (NMOSD)-like phenotype	no	no abnormality	IVMP	yes, 2	0/no
Case 6	5/M	Leukodystrophy-like phenotype	yes	no abnormality	IVMP, IVIG	yes, 3	0/no
Case 7	2/M	Optic Neuritis (ON) phenotype	yes	no abnormality	IVMP	yes, 1	0/no
Case 8	6/F	Neuromyelitis optica spectrum disorders (NMOSD)-like phenotype	yes	WCC >5/ $\mu$ l	IVMP	yes, 8	0/no
Case 9	14/F	Neuromyelitis optica spectrum disorders (NMOSD)-like phenotype	yes	WCC >5/ $\mu$ l	IVMP, IVIG	yes, 2	1/no
Case 10	9/F	Transverse Myelitis (TM) phenotype	yes	Protein >0.45 mg/dL, WCC >5/ $\mu$ l	IVMP, IVIG, PLEX	yes, 2	7/yes
Case 11	8/M	Optic Neuritis (ON) phenotype	yes	no abnormality	IVMP	yes, 2	0/no
Case 12	6/M	Optic Neuritis (ON) phenotype	yes	WCC >5/ $\mu$ l, OCB	IVMP	yes, 2	0/no
Case 13	13/F	Transverse Myelitis (TM) phenotype	yes	WCC >5/ $\mu$ l	IVMP	yes, 1	0/no
Case 14	13/F	Optic Neuritis (ON) phenotype	yes	no abnormality	IVMP	yes, 1	0/no
Case 15	13/M	Optic Neuritis (ON) phenotype	no	no abnormality	IVMP	yes, 1	0/no
Case 16	9/F	Transverse Myelitis (TM) phenotype	yes	no abnormality	IVMP, PLEX	yes, 4	./yes
Case 17	2/F	Acute Disseminated Encephalomyelitis (ADEM)	yes	Protein >0.45 mg/dL, WCC >5/ $\mu$ l	IVMP	yes, 6	2/no
Case 18	15/M	Transverse Myelitis (TM) phenotype	yes	Protein = 0.45 mg/dL, WCC >5/ $\mu$ l	IVMP	no	1/no
Case 19	15/M	Optic Neuritis (ON) phenotype	yes	no abnormality	IVMP, IVIG, PLEX	no	0/no
Case 20	14/M	Transverse Myelitis (TM) phenotype	yes	WCC >5/ $\mu$ l	IVMP	no	2/yes
Case 21	14/F	Acute Disseminated Encephalomyelitis (ADEM)	no	Protein >0.45 mg/dL	IVMP, PLEX	no	4/yes
Case 22	13/M	Transverse Myelitis (TM) phenotype	yes	WCC >5/ $\mu$ l	IVMP	no	0/no
Case 23	12/M	Optic Neuritis (ON) phenotype	yes	not available	IVMP	no	0/yes
Case 24	11/M	Encephalitis-like phenotype	no	no abnormality	IVMP	no	0/no
Case 25	11/M	Optic Neuritis (ON) phenotype	no	no abnormality	IVMP	no	0/no
Case 26	10/F	Optic Neuritis (ON) phenotype	yes	WCC >5/ $\mu$ l	IVMP	no	0/no
Case 27	9/F	Encephalitis-like phenotype	no	Protein >0.45 mg/dL	.	no	0/no
Case 28	9/F	Optic Neuritis (ON) phenotype	no	WCC >5/ $\mu$ l	IVMP	no	0/no
Case 29	8/F	Optic Neuritis (ON) phenotype	yes	no abnormality	IVMP	no	0/no
Case 30	7/F	Optic Neuritis (ON) phenotype	no	no abnormality	IVMP	no	0/no
Case 31	7/F	Acute Disseminated Encephalomyelitis (ADEM)	yes	Protein >0.45 mg/dL, OCB	IVMP, IVIG	no	1/no
Case 32	6/F	Encephalitis-like phenotype	no	WCC >5/ $\mu$ l	.	no	0/no
Case 33	5/M	Encephalitis-like phenotype	no	no abnormality	.	no	.
Case 34	5/F	Acute Disseminated Encephalomyelitis (ADEM)	no	not available	PLEX	no	10
Case 35	4/M	Encephalitis-like phenotype	yes	WCC >5/ $\mu$ l	.	no	0/no
Case 36	3/M	Acute Disseminated Encephalomyelitis (ADEM)	yes	Protein >0.45 mg/dL, WCC >5/ $\mu$ l	IVMP	no	1/no
Case 37	2/M	Acute Disseminated Encephalomyelitis (ADEM)	yes	WCC >5/ $\mu$ l	IVMP	no	1/no
Case 38	2/F	Acute Disseminated Encephalomyelitis (ADEM)	yes	not available	IVMP	no	0/no

In Table 1: F = female; M = male; WCC = white cell count; IVMP = intravenous methylprednisolone; PLEX = plasma exchange; IVIG=Intravenous immunoglobulin. NMOSD-like phenotype: transverse myelitis (TM) and concurrent optic neuritis (ON).

## ***2.2. Detection of MOG-antibodies***

All patients were tested for MOG-IgG by live cell-based assays (CBA). Briefly, HEK293A cells transfected with full-length human MOG were used as antigenic substrate to detect Ig by flow cytometry. Cells were incubated with FITC conjugated anti-human immunoglobulin secondary antibody. Analysis was performed using a FACSCanto II instrument and Flow Jo software. Binding was expressed as median fluorescence intensity (MFI); levels of specific antibody binding were expressed as  $\Delta$ MFI (MFI MOG-HEK293cells- MFI HEK293cells). For plasma analysis, a  $\Delta$ MFI greater than mean + 6 standards deviations (SDs) of the control patients' samples was considered positive as previously described [3].

For CSF there was no cut off value, as control patients' samples were all at background level. Quantitative results were expressed as MOG-ratio of  $\Delta$ MFI.

## ***2.3. Evaluation of the CSF parameters***

A total CSF protein level  $>0.45$  mg/dL was classified as increased, as well as a white cell count (WCC)  $> 5/\mu\text{l}$ . Oligoclonal IgG bands were assessed by isoelectric focusing.

## ***2.4. Statistical analysis***

To perform the comparisons and to calculate the odds, parametric and non-parametric tests were used (Student's t-test, Chi-squared test, Fisher's exact test and the Mann-Whitney *U* test).

The ARR was calculated as stated in previous studies by the number of total relapses divided by the total duration of follow-up. P-value was considered significant at 0.05. Statistical analyses were performed using the software IBM SPSS Statistics 25.

# **2. Results**

This study included 38 patients with MOG-Abs associated diseases (MOGAD).

Within the totality of patients with serum positivity for MOG-abs and MOGAD, the CSF was positive in 71.7 % (27/38).

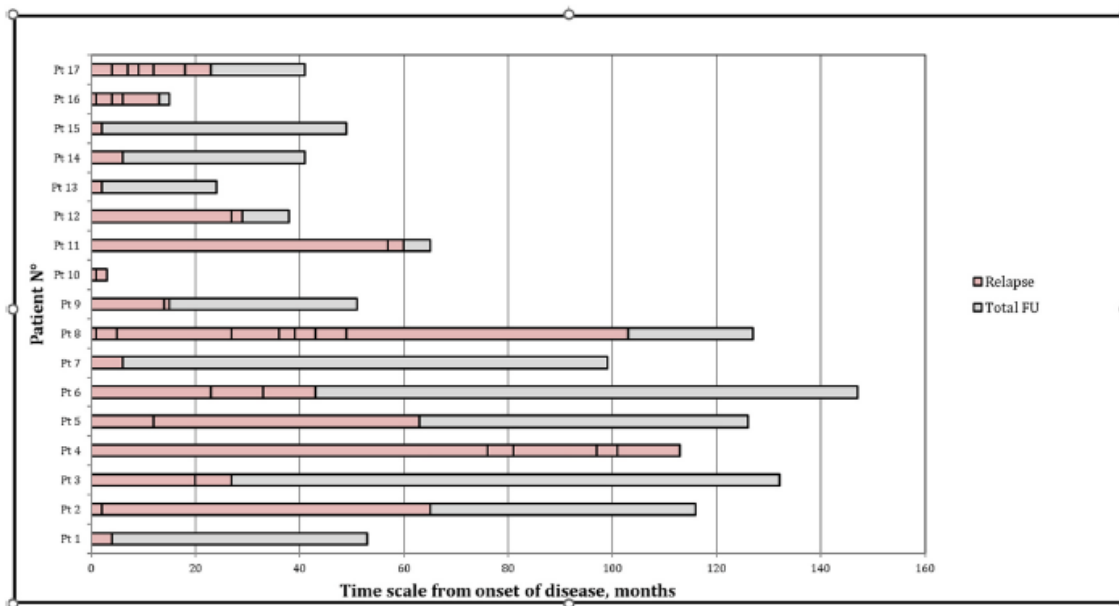


Fig. 1. Recurrence distribution among relapsing MOGAD children.

### 3.1. MOGAD cohort description

Among the 38 patients with MOGAD, 22 (57.9 %) were females and the mean (SD) age at disease onset was 8.4 ( $\pm 4$ ) years (63.2 % with 9 years or less at onset).

Eleven (28.9 %) patients were CSF-negative and 27 (71.7 %) patients were CSF-positive.

Isolated ON was the most frequent phenotype at onset (31.6 %); afterwards, 21.1 % patients with ADEM and 21.1 % patients with TM, 10.5 % with NMOSD-like phenotype and 15.7 % with encephalitis-like and other phenotypes.

Seventeen (44.7 %) patients presented a relapsing disease course.

The CSF evaluations found an increased white cell count in 47.4 % cases and increased protein concentrations in 26.3 % cases.

Oligoclonal bands were detected in 2 patients with CSF-positivity (data available in 26 cases).

The median follow-up was 25.5 months (IQR 5.5–73.25).

The follow-up duration was less than 12 months in 12 cases. Comprehensive clinical and paraclinical findings are summarized in Table 1.

### 3.2. CSF-positive versus CSF-negative patients

The comparison between CSF-positive and CSF-negative subgroup found no differences in male to female ratio, age at disease onset or outcome parameters at last follow-up.

No CSF-negative patient presented a TM-phenotype at onset, while CSF-positive patients with transverse myelitis at onset were frequent (8/ 27, 29.6 %). On the contrary, the majority of patients with an encephalitis-like phenotype at onset were CSF-negative (**p<sup>1/4</sup>0.019**).

Concerning the CSF findings, we found a significantly major increase in white cell count in the CSF-positive group (**p<sup>1/4</sup>0.043**), while the amount of total proteins was not significantly different.

The two groups were significantly different (**p<sup>1/4</sup>0.038**) in the rate of relapsing diseases, with 2 patients (18.2 %) with a relapsing phenotype in the CSF-negative population and 15 patients (55.6 %) in the CSF-positive one.

The distribution of recurrence among relapsing MOGAD children is displayed in Fig. 1.

The annualized relapse rate (ARR) was found significantly different among the two groups (**CSF negative: 0,04 ± 0,09 vs CSF positive: 0,5 ± 1,2, p<sup>1/4</sup>0.045, Mann-Whitney test**)

We couldn't find a significant difference of  $\Delta$ MFI MOG-Abs in serum between CSF-negative and CSF-positive patients, while  $\Delta$ MFI MOG-Ab in CSF and  $\Delta$ MFI ratio were obviously different.

An odds ratio (OR) was computed to assess the association between CSF-positivity for MOG-Abs and a relapsing disease course, and we found that patients with a double positivity for MOG-Abs were over five times as likely to have experienced one or more relapses (18.2 % versus 55.6 %; OR = 5.62, 95 % CI [1.017–31.097]).

Comparison between the CSF-positive and the CSF-negative subgroup in Table 2.

### 3. Discussion

This study presents a description of a pediatric cohort of patients with MOG antibody-associated diseases. Within this cohort we displayed a comparison between CSF-positive and CSF-negative patients in order to highlight clinical differences between the two groups.

According to our data, paired serum and CSF MOG-abs positivity occurred more often than previously described in other adult or mixed cohorts (71.7 % vs 40.7 % [4] or 56.8 % [5] or 61.3 % [6]).



**Table 2**

CSF-negative and CSF-positive patients

In Table 2: CSF = cerebrospinal fluid; ADEM = acute disseminated encephalomyelitis; ON = optic neuritis; TM = transverse myelitis, WCC = white cell count; EDSS = Expanded Disability Status Scale, FU = follow up.

TABLE: 2	CSF-negative n = 11	CSF-positive n = 27	CSF-n vs CSF-p n = 38
Female, n (%)	7 (63.3)	15 (55.6)	,729
Age at disease onset (yrs) mean ± SD	8.6 ± 3.2	8.3 ± 4.3	,837
<b>Clinical phenotype 1st attack:</b>			
ADEM, n (%)	2 (18.2)	6 (22.2)	,578
ON, n (%)	4 (36.4)	8 (29.6)	,685
TM, n (%)	0	8 (29.6)	,077
NMOSD-like phenotype, n (%)	1 (9.1)	3 (11.1)	,674
Encephalitis, n (%)	4 (36.4)	1 (3.7)	,019
Others, n (%)	0	1 (3.7)	,711
CSF at onset: WCC > 5/μL, n (%)	2 (18.2)	16 (59.3)	,043
CSF at onset: protein levels > 45 mg/dL, n (%)	2 (18.2)	8 (29.6)	,683
Relapsing disease course, n (%)	2 (18.2)	15 (55.6)	,038
<b>Clinical phenotype 2nd attack:</b>			
ADEM, n (%)	0	2 (7.4)	,499
ON, n (%)	1 (9.1)	13 (48.1)	,030
TM, n (%)	1 (9.1)	0	,289
other forms, n (%)	0	0	-
Age at last FU (yrs), mean ± SD	12 ± 3.6	12.4 ± 4.8	,811
Chronic treatments, n (%)	2 (18.2)	10 (37)	,444
Follow-up (months), median ± IQR	17.5 (2–61)	27.0 (12–99)	,515
<b>Acute treatments:</b>			
Corticosteroids, n (%)	6 (54.5)	17 (63.0)	,722
Corticosteroids + IVIG, n (%)	0	3 (11.1)	,542
Corticosteroids + PLEX, n (%)	2 (18.2)	3 (11.1)	,615
Corticosteroids + IVIG + PLEX, n (%)	0	3 (11.1)	,542
Maintenance therapy, n (%)	2 (18.2)	10 (37.0)	,444
Time to first relapse (months), mean ± sd	7 ± 7.07	16.27 ± 22.40	,391
	2 patients	15 patients	

In the entire cohort we found that almost half of the individuals presented a relapsing disease course; it is unclear whether the amount of recurrence is high due to a selection bias for the enrolment in a tertiary referral centre, or if this can be considered the rate of polyphasic diseases in the paediatric age [7]. This population also revealed that almost one third of the cohort presented a spinal involvement at onset. Among these, the vast majority was CSF-positive for MOG-abs, and the totality of patients with transverse myelitis at onset was CSF-positive. We emphasize that the majority of patients with transverse myelitis had a relapsing phenotype and that all the patients with NMOSD-like phenotype had a recurrent disease. In contrast to previous findings [8], taking into account patients with cerebral encephalitis we noticed that the vast majority of these patients were CSF-negative. CSF positive MOGAD children presented a significant trend to have a relapsing course and a higher ARR but the small number of patients and the possibility of multifactorial involvement in ARR require caution regarding the conclusion. In comparison with the larger mixed cohort of adult and pediatric patients with MOGAD recently described by Carta et al. [5], this study verified in

an entirely pediatric cohort that (1) CSF-positive patients displayed more frequently CSF pleocytosis [2]; (2) in CSF-positive patients myelitis represented a common clinical presentation at onset. This study did not confirm significantly increased protein levels in CSF-positive patients compared to CSF-negative patients.

Furthermore, pediatric serum and CSF MOG-abs positive patients presented an higher probability of subsequent recurrence and higher relapse rates, though no differences in terms of neurological impairment at last follow up were found.

The pathogenicity of MOG-Abs is largely unknown. Our main hypothesis is that increased permeability of the blood-brain barrier allows the transfer of MOG-antigens which in turn elicit an immune response in the periphery. Circulating anti-MOG IgG appear to mediate the immune response [9, 10] and CSF MOG-Abs are thought to be derived from passive transfer through the blood-brain barrier. However, lately it has been suggested a possible portion of intrathecal synthesis of MOG-IgG in patients with MOGAD [11, 12].

We acknowledge several limitations of this study; probably, the main limitations are its retrospective nature and the small sample size. This study was conducted in tertiary centres with a potential bias of inclusion of patients with more severe phenotypes. Moreover, the follow up duration is heterogeneous and, in some cases, limited. Therefore, these results need to be confirmed in larger populations.

In conclusion, in this study two subgroups with different clinical features and CSF-status are outlined. We speculate that the CSF-status could identify two subgroups in MOGAD population and that CSF-positivity might represent a risk factor for a relapsing disease course in the paediatric population. Thus we conclude that CSF-status should be the subject of further studies and that CSF-positive patients could deserve a closer monitoring.

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