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Validation of the MOG-AR Score

A Retrospective Multicenter Study

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Abstract

Objectives

A score evaluating age at onset, sex, clinical phenotype, and treatment received (MOG-AR) has been proposed to identify MOGAD patients at high relapse risk. The aim of this study was to validate the MOG-AR score in a multicenter cohort and to assess other variables potentially associated with relapses.

Methods

MOGAD patients were retrospectively enrolled from 24 centers. The MOG-AR score was applied and 4 categories of relapse risk were identified (grade I: lowest risk; grade IV: highest risk), accordingly. The association of MOG-AR score and additional variables with a relapsing course were then explored.

Results

Of 190 included patients, the median age at onset was 37 [IQR 23–51] years and 107 (56%) were female. A total of 78 patients (41%) experienced a relapse during a median of 43.6 months [24.8–75.4]. Using the proposed cutoff of 9, the MOG-AR score had a sensitivity of 53.9% [95% CI 55.6–73.9] and a specificity of 65.18% [95% CI 55.60–73.93]; area under the curve: 0.64 (95% CI 0.57–0.72). Among additional investigated factors, only immunosuppressive treatment after the presenting MOGAD attack was associated with a lower relapse risk.

Discussion

MOG-AR score failed to accurately predict a relapsing disease course. Only immunosuppressive treatment after the first event was significantly associated with a lower relapse risk.

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Supplementary Material

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Introduction

Myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD) is an antibody-mediated disorder characterized by a demyelinating event and serum/CSF MOG antibodies (MOG-Abs) positivity.[1] Relapses can occur in 40%–60% of cases,[2] increasing to 70% over a follow-up > 5 years.[3,4] Of note, disease course is highly unpredictable. Among factors potentially predicting relapses, MOG-Abs persistency, age, sex, ethnicity, onset phenotype, early relapses, early treatment with immunosuppression, and prolonged steroid have been identified.[4-10]

Recently, a simple score (the MOG-AR Score), including onset age, sex, onset attack phenotype, use of immunosuppressive therapy, and duration of oral glucocorticoids treatment, has been proposed to identify patients at high relapse risk since onset.[11]

The aim of this study was to provide the first validation of the MOG-AR Score in a national multicenter cohort and to assess other factors associated with a relapsing disease.

Methods

We retrospectively (January 2017–January 2025) identified patients with MOGAD[1] and at least 1-year follow-up recruited from 24 Italian centers.

Parameters of the MOG-AR Score (onset age ≥45 years, sex, attack phenotype, oral steroids use for at least 3 months, use of immunosuppressive treatment after the first event) were analyzed to assess association with disease course. Patients were stratified in 4 groups based on the MOG-AR score (grade 1: 0–4, grade 2: 5–8, grade 3: 9–12, grade 4: 13–16). Additional clinical and paraclinical data were collected and analyzed (eMethods).

Group comparisons were assessed using nonparametric tests (χ^2 and Mann–Whitney U tests). ROC curves were constructed to assess MOG-AR score performance.

True positive (TP), true negative (TN), false positive (FP), and false negative (FN) cases were identified considering that MOG-AR score ≥9 was reported as predictive of relapses.[11] Sensitivity, specificity, PPV, NPV, and accuracy with 95% CI were calculated.

Univariate binary logistic regression models, multivariate binary regression model, and Kaplan-Meier survival curves were performed to assess the risk of relapses (eMethods).

Standard Protocol Approvals, Registrations, and Patient Consents

The study was part of the research protocol approved by the Ethics Committees of the enrolling centers (eMethods).

Data Availability

Anonymized data not published within this article will be made available on request from any qualified investigator.

Results

Cohort Description

Among 260 patients, 34 were excluded for insufficient clinical information and 36 for insufficient follow-up. Of the 190 included patients, the median age at onset was 37 years [IQR 22.5–50.6], and 107 (56.4%) were female. The most frequent clinical presentation at onset were optic neuritis (n = 91, 47.9%), myelitis (n = 48, 25.3%), and acute disseminated encephalomyelitis (n = 24, 12.6%), Table 1. The median expanded disability status scale at onset was 3 [IQR 2–4.5]. High-dose steroids followed by at least 3 months oral steroid tapering were administered in 58 (30.7%) patients, while immunosuppressive treatment at first event was initiated in 54 (28.4%) patients. Of note, 18 (9.5%) patients received at least 3 months of steroid tapering and an immunosuppressive treatment after the first event. The median follow-up was 43.6 months [24.8–75.4], and 78 (41%) experienced at least 1 relapse, with an annualized relapse rate of 0.61 (SD 0.410).

MOG-AR Score Application

Overall, 23 (12.1%) patients were classified as grade I, 51 (26.8%) as grade 2, 88 (46.3%) as grade 3, and 28 (14.7%) as grade 4. Relapses were observed in 17.4% (n = 4) of grade 1 cases, 33.3% (n = 18) of grade 2, 46.6% (n = 41) of grade 3, and 53.6% (n = 15) of grade 4, $p = 0.030$. ROC curve analysis using the MOG-AR showed an area under the curve of 0.644 (95% CI 0.565–0.723), eFigure 1.

Using the proposed cutoff of 9,[11] that was similar to the optimal cut-off identified in our cohort (Youden index = 9.5), MOG-AR score had a sensitivity of 53.85% (95% CI 55.60–73.93), a specificity of 65.18 (95% CI 55.60–73.93%), a PPV of 51.85% (95% CI 43.73–59.88), and a NPV of 88.97% (95% CI 60.63–72.76) with an accuracy of 60.53% (95% CI 53.19–67.53).

When assessing time to relapse with the Kaplan-Meier analysis (n = 168) according to the MOG-AR score, the difference was not statistically significant across different groups (p = 0.120), Figure 1.

Analysis of Individual Factors Associated With Relapse Risk

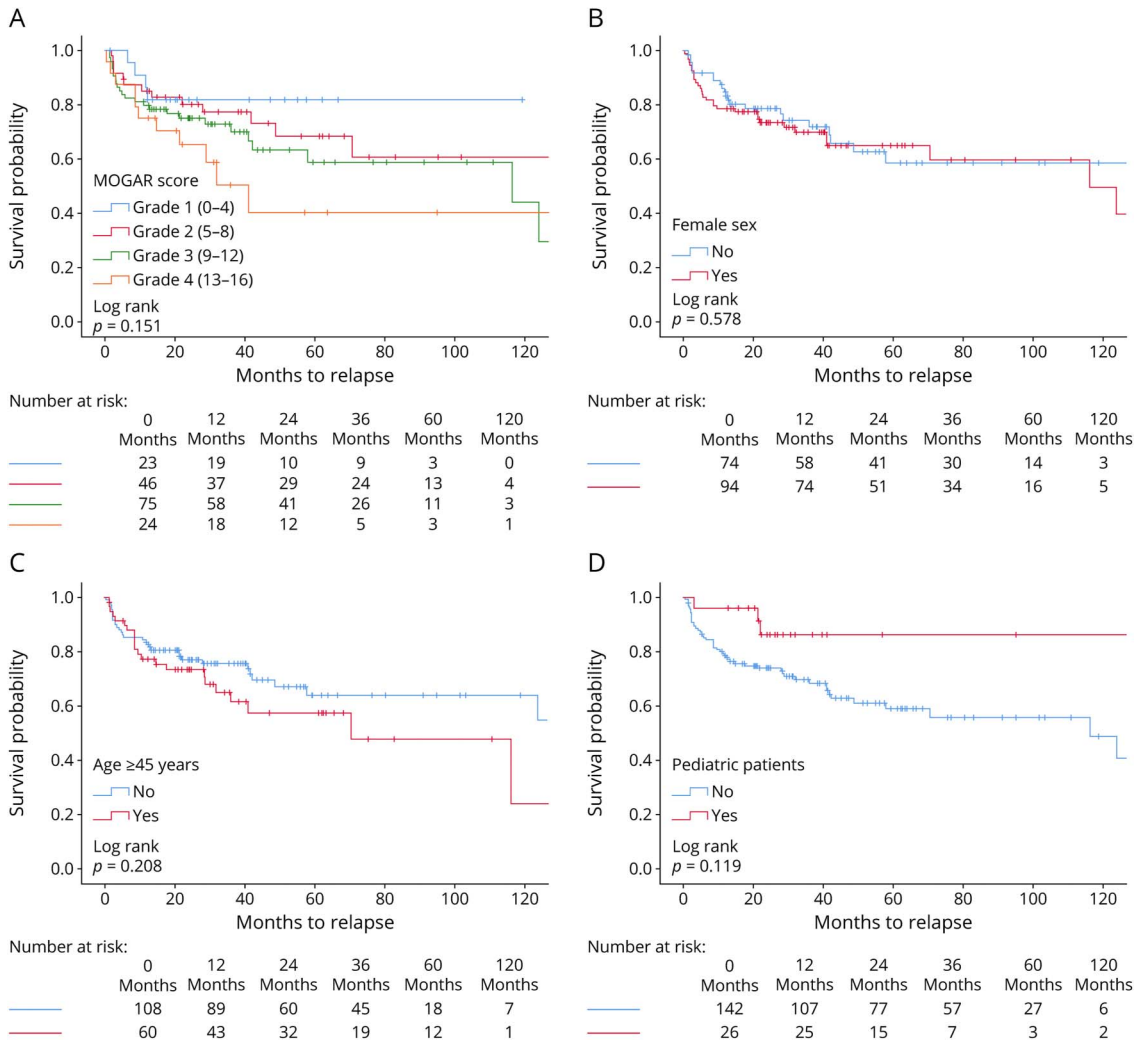
Oral steroids administration ≥3 months (0.51 95% CI 0.26–0.97, $p = 0.044$), receiving an additional acute treatment (OR 0.31 95% CI 0.12–0.80, $p = 0.016$), and starting immunosuppressive treatment after the first event (OR 0.37 95% CI 0.17–0.73, $p = 0.006$) were associated with lower relapse risk at univariate analysis (eTable 1). At multivariate analysis, only the initiation of immunosuppressive treatment after the first event was an independent factor associated with

Table 1 Cohort Description According to the Disease Course (Monophasic vs Relapsing)

	Total cohort (n = 190)	Monophasic (n = 112)	Relapsing (n = 78)	*p* Value
Age at onset, median [IQR]	37 [22.5–50.6]	35.5 [19.8–49.6]	39.5 [29.9–51.8]	0.070
Age<45 year-old at onset, n (%)	69 (36.6)	37 (33)	32 (41)	0.260
Pediatric patients, n (%)	30 (15.8)	22 (19.5)	8 (10.4)	0.092
Female n, (%)	107 (56.4)	63 (56.3)	44 (56.4)	0.983
Infectious/vaccinal trigger, n, (%)	50/145 (34.5)	35/86 (40.2)	15/59 (25.9)	0.075
Autoimmune comorbidities, n, (%)	35 (18.4)	20 (17.9)	15 (19.2)	0.756
Clinical phenotype at onset, n (%)				
Optic neuritis	91 (47.9)	50 (44.2)	41 (53.2)	0.223
Myelitis	48 (25.3)	34 (30.4)	14 (17.9)	0.059
ADEM	24 (12.6)	17 (15.2)	7 (9)	0.205
Cortical encephalitis	6 (3.2)	3 (2.7)	3 (3.8)	0.651
Brainstem syndrome	10 (5.3)	4 (3.6)	6 (7.7)	0.365
Polyfocal onset	8 (4.2)	5 (4.5)	3 (3.8)	0.835
Other	3 (1.6)	0	3 (3.8)	0.036
EDSS at onset (nadir)	3 [2–4.5]	3.5 [2–5.5]	3 [2–4]	0.160
Visual acuity at onset (in patients with optic neuritis n = 91)	0.35 [0.1–0.6]	0.35 [0.1–0.7]	0.35 [0.1–0.5]	0.452
Acute treatment strategies				
MP iv	175 (93.6)	105 (94.6)	70 (92.1)	0.495
IvIg	24 (12.6)	18 (16.1)	6 (7.7)	0.087
PLEX	12 (6.3)	11 (9.8)	1 (1.3)	0.017
Steroid tapering ≥3 months	58 (30.7)	40 (35.7)	18 (23.4)	0.071
Additional acute treatment (PLEX and/or IvIG)	30 (15.8)	24 (21.4)	6 (7.7)	0.011
Immunosuppressive treatment after the first event	54 (28.4)	41 (36.6)	13 (16.7)	0.004
Azathioprine	15 (27.8)	10 (24.4)	5 (38.46)	
MMF	4 (7.4)	4 (9.8)	0	
IvIG	9 (16.7)	8 (19.5)	1 (7.7)	
Rituximab	24 (44.4)	18 (43.9)	6 (46.2)	
Tocilizumab	2 (3.7)	1 (2.4)	1 (7.7)	
CSF analysis	n = 168	n = 97	n = 71	
Cell count (n/uL), median [IQR]	5.5 [1–26.5]	6 [1–37]	2 [1–12.5]	0.079
Protein levels (n/uL), median [IQR]	41.7 [28.8–57]	44 [31–59]	38.5 [23.8–54]	0.138
OCBs, n (%)	68 (40.5)	39 (40.2)	29 (40.8)	0.497
Median follow-up, mo [IQR]	43.6 [24.8–75.4]	34.4 [21.6–57.2]	61.1 [40.7–96.3]	<0.001
EDSS at the last follow-up	1 [0–2]	1 [0–1]	1.5 [1–2.5]	<0.001
Visual acuity at last follow-up (in patients with optic neuritis n = 91)	1 [0.9–1]	1 [1-1]	0.9 [0.8–1]	0.040

Abbreviations: ADEM = acute disseminated encephalomyelitis; EDSS = expanded disability status scale; IQR = interquartile range; IVIg = IV immunoglobulin; MMF = mycophenolate mofetil; MP IV = IV methylprednisolone; OCBs = oligoclonal bands; PLEX = plasma exchange.

Figure 1 Time to Reach the First Relapse According to the MOG-AR Score and Demographic Features



(A–D) Kaplan-Meier Analysis estimation of time to reach a first relapse according to MOG-AR score, sex, and age at onset ≥45 and pediatric onset.

a lower relapse risk (OR 0.42 95% CI 0.20–0.85, $p = 0.019$). When assessing time to relapse with Kaplan-Meier, female sex, age ≥45, clinical phenotype, receiving an additional acute treatment, and use of corticosteroids ≥3 months were not associated with the risk of having a relapse, Figure 1. Similarly, pediatric onset, MOG-Abs status (seropositive at high titer, seropositive at low titer, unknown titer, CSF only positive), an infectious or vaccinal trigger, the use of a second line treatment in the acute stage, and oligoclonal bands presence were not correlated with relapse risk (Figures 1 and 2). Only starting an immunosuppressive treatment after the first event was associated with a lower relapse risk ($p = 0.035$).

Discussion

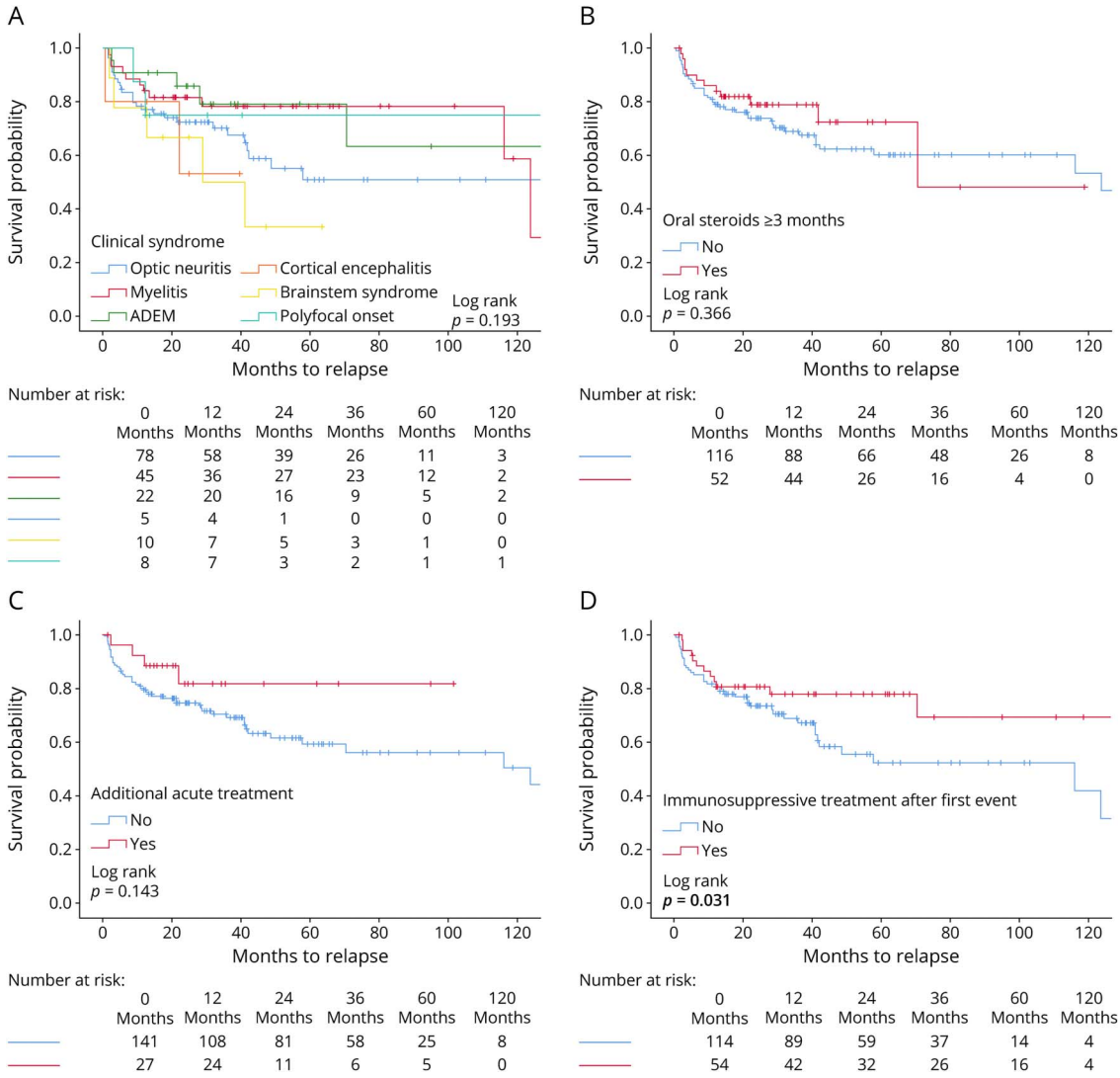
We herein observed that (1) the MOG-AR score cannot predict a relapsing course; (2) relapse risk is not influenced by age at onset, sex or clinical phenotype; and (3) prolonged steroid taper, use of additional acute treatment, and initiation of immunotherapy after the first event are associated with a lower relapse risk at univariate analysis. However, at multivariate and survival analyses, only initiation of immunotherapy after the first event remained significantly associated with a reduced relapse risk.

Other previous studies have emphasized that MOGAD is a complex and heterogenous disease with no reliable predictors of disease course.[3]

In our cohort, older age at onset was not associated with relapse risk. Previous studies reported contradictory results.[7]

Although some studies have reported a lower relapse risk in male patients,[11,12] sex was not associated with disease course in our cohort. This finding is consistent with other prospective and retrospective studies.[4,7]

Figure 2 Time to Reach the First Relapse According to Clinical Phenotype and Treatment Strategies



(A–D) Kaplan-Meier Analysis estimation of time to reach a first relapse according to clinical syndrome at onset, prolonged steroid taper, administration of an additional acute treatment, and use of an immunosuppressant after the first event.

Similarly to what already reported, patients presenting with myelitis had a trend toward a lower relapse rate.[7,13] However, in our cohort, the clinical phenotype at disease presentation did not have a significant impact on relapse risk.

Of note, our data show that treatment is the main factor affecting relapse risk in MOGAD.[7,13] Although a prolonged steroid course and the use of additional acute treatment decreased relapse risk at univariate analysis, this was not confirmed at survival/multivariate analyses. This may be related to the limited sample size. Conversely, the prompt initiation of immunosuppressive therapy was associated with a reduced relapse risk. However, the potential adverse effects of these treatments make controversial the initiation of immunosuppression after the first event. This is particularly relevant given that previous studies suggest that most of the long-term disability could be attributable to the initial attack.[2] Previous studies have shown the heterogeneity of treatment strategies and the relevance of establishing consensus on how to treat MOGAD patients after the first event.[14]

The primary limitations of this study are its retrospective design, which may introduce selection and information biases, and the relatively small sample sizes in certain subgroup analyses, which limit the statistical power and generalizability of our findings and could partially explain the limited predictive values of the explore items.

Nevertheless, our findings confirm that MOGAD is a highly heterogeneous disease and that further research is needed to detect reliable markers able to predict a relapsing disease course. Our data underline how immunosuppressive treatment administered after the onset event can influence disease course, which administration should take into consideration the risk of overtreatment and adverse events occurrence.

Author Contributions

S. Carta: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data. R. Tiberi: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data. N. De Rossi: major role in the acquisition of data. G.T. Maniscalco: major role in the acquisition of data. G. Greco: major role in the acquisition of data. A. Lotti: major role in the acquisition of data. A. Marziali: major role in the acquisition of data. A. Sartori: major role in the acquisition of data. A. Favero: major role in the acquisition of data. F. Rossi: major role in the acquisition of data. A. Dinoto: major role in the acquisition of data. M. Trentinaglia: major role in the acquisition of data. V. Chiodega: major role in the acquisition of data. F. Boso: major role in the acquisition of data. S. Miante: major role in the acquisition of data. S. de Biase: major role in the acquisition of data. F. Caleri: major role in the acquisition of data. R. Orlandi: major role in the acquisition of data. E. Guso: major role in the acquisition of data. I. Volonghi: major role in the acquisition of data. M. Nosadini: major role in the acquisition of data. S. Sartori: major role in the acquisition of data. P. Palmieri: major role in the acquisition of data. A. Cossu: major role in the acquisition of data. F. Calabria: major role in the acquisition of data. P. Zara: major role in the acquisition of data. M.P. Giannoccaro: major role in the acquisition of data. L. Zuliani: major role in the acquisition of data. M. Vianello: major role in the acquisition of data. G. De Luca: major role in the acquisition of data. M. Zoccarato: major role in the acquisition of data. A. de Mauro: major role in the acquisition of data. L. Massacesi: major role in the acquisition of data. R. Cortese: major role in the acquisition of data. A. Gajofatto: major role in the acquisition of data. P. Rossi: major role in the acquisition of data. E. Sechi: major role in the acquisition of data. A. Vogrig: major role in the acquisition of data. V. Damato: major role in the acquisition of data. M. Gastaldi: major role in the acquisition of data. S. Mariotto: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data.

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