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Review article

Latin American RAND/UCLA modified Delphi consensus recommendations for management and treatment of adult MOGAD patients in clinical practice

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ABSTRACT

Introduction: Over the last decade, myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) has seen significant advancements, with new diagnostic criteria and emerging biomarkers, increased recognition of more diverse clinical phenotypes, and new insights into disease prognosis and therapeutic strategies. Consequently, the management of MOGAD patients in Latin America (LATAM) has become more complex in clinical settings. This consensus was established to assess the best practices and treatment approaches for MOGAD in LATAM, with the goal of improving long-term outcomes for these populations. It encompasses both practical guidance and theoretical definitions to ensure a comprehensive and regionally relevant framework for diagnosis and management.

Methods: A panel of expert neurologists specializing in demyelinating diseases in LATAM met virtually from 2023 to 2024 to establish consensus recommendations for the management of MOGAD. A list of 59 statements and recommendations developed by the steering group was submitted to the rating group in the form of a questionnaire. Statements were organized into 5 categories as follows: 1-Diagnosis and serological tests; 2-Imaging and other complementary tests; 3- Prognostic factors; 4- Acute and 5- Long-term treatment.

The RAND/UCLA modified Delphi panel process was utilized to achieve a formal consensus.

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Results: All statements reached strong or relative agreement during the first round, and additional rounds of votes were not conducted. The panel deliberated on various aspects such as diagnosis, differential diagnoses, disease prognosis, personalized treatment strategies, and identification of inadequate treatment responses, incorporating published evidence and expert opinions.
Conclusions: These recommendations outlined in this consensus seek to enhance the management and specific treatment protocols tailored for MOGAD patients in LATAM, with the aim of optimizing patient outcomes over long term.

1. Introduction

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a rare and inflammatory disorder of the central nervous system characterized by immune-mediated demyelinating attacks that mainly target the optic nerves, brain, and spinal cord (Banwell et al., 2023). The identification of a disease-specific serum immunoglobulin G (IgG) antibody that binds myelin oligodendrocyte glycoprotein (MOG), based on the development of cell-based assays (CBA) using human MOG transfected cells (Sato et al., 2014; Reindl et al., 2020), has increased understanding of a wide range of clinical phenotypes, and the MOGAD nosologic entity has become clearer (Jarius et al., 2018; Cobo-Calvo et al., 2018, Sechi et al., 2022). Despite some overlapping features, there are significant differences clinically, radiologically, and in cerebrospinal fluid (CSF) analysis that distinguish MOGAD from multiple sclerosis (MS) and aquaporin-4-IgG-seropositive neuromyelitis optica spectrum disorder (AQP4-IgG NMOSD) (Marignier et al., 2021; Solomon et al., 2023, Wingerchuk et al., 2015; Carnero Contentti et al., 2023). As a result, MOGAD has been confirmed as a distinct CNS demyelinating disease (Banwell et al., 2023; Bruijstens et al., 2020).

Latin America (LATAM) is a vast region of the American continent that encompasses from Mexico (32° North latitude) to Argentina in South America (56° South latitude), including the Caribbean Islands. LATAM residents are a diverse and multiethnic population (Carnero Contentti et al., 2020). They exhibit a wide range of genetic variations and proportions, particularly among Mestizos, the most prominent ethnic group, who are themselves the result of centuries of intermixing between Native Americans (or Amerindians), White Caucasian Europeans, and Black Africans (Carnero Contentti et al., 2024; Rivera et al., 2021). While regional epidemiological differences may be observed in MOGAD patients, there are significant unmet needs in terms of diagnosis, broader symptom management, and decision-making regarding specific treatment in LATAM (Rojas et al., 2021; Tkachuk et al., 2023; Marignier et al., 2021).

In recent years, MOGAD has undergone significant changes, including new diagnostic markers and criteria, better recognition of clinical phenotypes, improved disease prognosis, and distinct off-label therapeutic approaches, which are variably used in clinical practice (Banwell et al., 2023, Marignier et al., 2021). Consequently, the management of MOGAD patients in LATAM has become more complex and challenging in clinical practice, particularly due to barriers in accessing healthcare for MOGAD patients, including MOG-IgG testing (Rojas et al., 2021; Tkachuk et al., 2023). Therefore, we aimed to develop a consensus recommendation for neurologists in LATAM that encompasses both practical guidance and theoretical definitions for the management and treatment of adult patients with MOGAD. These recommendations utilize a pre-defined and structured methodology, combining available evidence with the opinions and experiences of a group of experts to enhance long-term outcomes and optimize resources for this population.

2. Methodology

A modified Delphi study using the RAND/UCLA (University of California, Los Angeles) Appropriateness Method (RAM) was conducted to reach a formal consensus recommendation (https://www.hassante.fr/portail/upload/docs/application/pdf/201803/good_practice_guidelines_fc_method.pdf., Broder et al., 2022; https://apps.dtic.mil/sti/citations/ADA393235). An expert panel of LATAM neurologists specializing in MOGAD/NMOSD care (diagnosis, management, and treatment) convened virtually in 2023 and 2024 to develop a consensus recommendation guideline on management and treatment of adults MOGAD in clinical practice in the region.

The RAND/UCLA modified Delphi panel method is a formal group consensus process that systematically and quantitatively combines expert opinion and scientific evidence by asking panelists to rate, discuss, and then re-rate items (Jorm et al., 2015). This process involves a literature review, selection of panelists, creation of a rating questionnaire, a first-round rating form questionnaire, a virtual meeting where panelists discuss areas of disagreement (if necessary), final ratings and analysis of those ratings, and the development of a written summary of areas of agreement (https://www.hassante.fr/portail/upload/docs/application/pdf/201803/good_practice_guidelines_fc_method.pdf.; Broder et al., 2022; https://apps.dtic.mil/sti/citations/ADA393235).

As a recommendation consensus method, the aim is to formalize the level of agreement among experts by identifying and selecting, through iterative ratings with feedback, the proposals on which experts agree and those points on which they disagree or are undecided. The guideline methods are subsequently based on agreed proposals. As a practice recommendation guideline method, the goal is to draft several statements and recommendations that address questions of interest in clinical practice. This is a rigorous and explicit method based on the involvement of user representatives and professionals in the field to which the guidelines relate, and on the use of an external peer review phase, transparency, independence of development, and management of conflicts of interest. Thus, these consensus statements can contribute to clinical treatment recommendations in a therapeutic or disease area, particularly for rare diseases, where standard practice recommendations may not yet be established or regularly updated (Paul et al., 2023).

The first step in the process involved the inclusion of experts from a working group. Neurologists were identified through the Latin American Committee for Treatment and Research in MS (LACTRIMS) database, and they were selected by their experience in managing patients with MOGAD in various regions of LATAM. The working group was then divided as follows: a) a steering group, which consisted of three neurologists, including one chairperson (E.C.C), two international external neurologists (M.L and F.P) who have extensive expertise in MOGAD and a project manager; and b) a rating group of 16 neurologists who are directly involved in MOGAD care in their daily practice.

Once the working group had been formed, a comprehensive literature search in English and Spanish was performed by the chairperson and articles published between January 1, 2010, and December 31, 2023, were analyzed. An initial search with search terms “myelin oligodendrocyte glycoprotein”, “myelin oligodendrocyte glycoprotein associated disease”, “MOG” and “MOGAD” identified 754 articles and additional articles with focus on different clinical, paraclinical and treatment aspects were also reviewed. Relevant clinical papers were distributed to the working group for review and summarization so that they could respond to the statements and recommendations for discussion. We found several manuscripts discussing therapeutic approaches (without formal clinical trials), which were also included.

Statements were organized into 5 categories as follows: 1-Diagnosis and serological tests; 2-Imaging and other complementary tests; 3-Prognostic factors; 4- Acute and 5- Long-term treatment. A list of 59

statements developed by the steering group (initially developed by the chairperson and then reviewed by two international external experts) was submitted to the rating group in the form of a questionnaire. Panel members received an evidence summary from the targeted literature review. Responses were securely collected online, via Google forms, and then extracted anonymously into an Excel spreadsheet. At this stage, the statements complemented or contradicted each other insofar as they considered all opinions expressed by the group members during the work sessions.

The rating process began by identifying the statements and recommendations on which the panelists of the rating group agreed. Participation in the round of votes was mandatory for each panel member. In the first round, panel members anonymously voted their level of agreement with each statement and/or recommendation according to the median value and the distribution of the scores, as shown in Supplementary Table 1. Nonparametric outcome data were described as median and percentage. Statistical analysis was performed using Microsoft Excel. Consensus was defined as 70 % of the respondents agreeing, and lack of consensus as $\geq$ 30 % disagreeing. Each response on the 1–9 scale was recorded, and a median appropriateness rating was calculated for each statement. Median ratings of 7–9 were considered appropriate, 4–6 uncertain and 1–3 inappropriate. All statements reached strong or relative agreement during the first round, and additional rounds of votes were not conducted.

The rules for rating and analysis of the scores were defined at the outset and communicated to the rating group before the first round via email. Throughout the rating phase, panel members were able to comment on their response to each statement, and all comments were qualitatively analyzed to include in the next rating phase.

After these phases, the steering group drafted the initial version of the consensus recommendation to be submitted to the peer review group, based on the final consensus statements. Additionally, an analytical report was prepared, consolidating all scores and comments from the peer review group members and, if applicable, from the panel members in the public consultation. The final version of the evidence reports consensus recommendations, and a summary of the recommendations were finalized. The validated versions of these documents were then distributed.

This study did not require standard protocol approvals, registrations, or patient consents.

3. RAND/UCLA modified Delphi consensus recommendations

3.1. MOGAD diagnosis and serological tests

Recommendations for MOGAD diagnosis and serological tests are shown in Table 1. In 2007, conformation-sensitive MOG-IgG was identified using three-dimensional assays of MOG, instead of linear epitopes, in some patients with optic neuritis (ON) or acute disseminated encephalomyelitis (ADEM), and AQP4-seronegative NMOSD, but not in those with MS (O'Connor et al., 2007; Menge et al., 2011). MOG-IgG was initially detected using enzyme-linked immunosorbent assay (ELISA) or Western Blot, which were later abandoned in clinical practice primarily due to low specificity, resulting in false-positive results in both MS patients and healthy individuals (Kuhle et al., 2007; Lampasona et al., 2004). False-positive results can pose a significant challenge in the evaluation for MOGAD, particularly when results are positive at low titers in patients with non-suggestive clinical manifestations or MRI features for MOGAD. Importantly, the issue of FP at low titers may be observed with CBA, and the use of ELISA is susceptible to both FP and FN results. Since glycosylation and conformation of the MOG protein are important for MOG-IgG recognition, the surface expression of full-length human MOG (typically the α-1 isoform, 218 amino acids) on HEK293 cells is employed to accurately detect MOG-IgG, confirming the presence of MOG-IgG in non-MS demyelinating CNS disorders (Chan et al., 2010).

Approximately 40 % of patients suspected of NMOSD who test negative for AQP4-IgG in serum (Hamid et al., 2017) are positive for MOG-IgG. Notably, in a LATAM cohort, only 53.7 % of AQP4-IgG negative patients were tested for MOG-IgG, likely due to a lack of availability, and 27.7 % of those tested were positive (Carnero Contentti et al., 2021). Dual positivity (AQP4-IgG and MOG-IgG) is extremely rare in clinical practice and when it was found (0.07 %), all adult patients

Table 1
Recommendations for MOGAD diagnosis and serological tests.

	%	Median
• MOGAD should be considered as a different disease from AQP4-IgG-seropositive NMOSD and MS.	90	9
• In order to confidently diagnose MOGAD, clinical involvement in at least one of the following is necessary: optic neuritis, myelitis, ADEM, cerebral monofocal or polyfocal deficits, brainstem or cerebellar deficits, or cerebral cortical encephalitis often with seizures.	87	9
• To make a diagnosis, the 2023 International Panel for MOGAD diagnostic criteria should be applied to LATAM patients suspected of having MOGAD.	87	9
• It is recommended to rule out other regional diseases (local infections and nutritional diseases), as well as MS and AQP4-IgG NMOSD, that may mimic MOGAD in the LATAM population.	88	9
• Patients with suspected MOGAD should be evaluated in a center with experience in diagnosing of demyelinating diseases, to ensure an earlier and more precise diagnosis and adequate management.	87	9
• MOG-IgG testing is not recommended for screening of all patients with suspected demyelinating diseases.	80	9
• Patients with suspected MOGAD should be ideally tested for both serum AQP4-IgG and MOG-IgG	80	9
• Fixed or live cell-based assay (CBA) method must be used for evaluating serum MOG-IgG in clinical practice because of greater sensitivity and specificity.	90	9
• ELISA method is not recommended for evaluating serum MOG-IgG in clinical practice.	87	9
• Serum MOG-IgG testing should be ideally performed during an attack.	87	9
• Serum MOG-IgG testing should be ideally performed before the administration of high dose of IV steroids, immunoglobulins, or plasmapheresis. However, acute treatment should not be deferred until the results are available.	89	9
• Serum MOG-IgG reports should ideally include qualitative results (i.e., negative, low positive, and clear positive)	78	9
• Serum MOG-IgG reports should ideally include semi-quantitative results (i.e., titers), although the access to this report is difficult in LATAM clinical practice.	87	9
• Fixed cell-based assay result with a titer $\geq$1:100 is considered a clear positive assay.	89	9
• Fixed cell-based assay result with a titer $\geq$1:10 and <1:100 is considered a low positive assay.	83	9
• Low or clear positive live cell-based assay results are defined based on the individual assay cutoffs.	78	9
• Serum MOG-IgG testing should be repeated after 3-6 months if the initial results was negative and suspicion of MOGAD is high (based on clinical and MRI characteristics).	87	9
• Patients with at least one core clinical characteristic of NMOSD who are seronegative for AQP4-IgG should be tested for serum MOG-IgG.	87	9
• CSF MOG-IgG testing should be performed if the initial MOG-IgG result was negative and suspicion of MOGAD is high (based on clinical and MRI characteristics).	73	9
• In patients with clinically and radiologically suspected MOGAD with non-typical brain or spinal cord MRI lesions suggestive of MS, lumbar puncture is recommended to evaluate differential diagnoses.	83	9
• OCB in CSF (type II or III) can be observed in MOGAD patients, and their presence does not rule out the diagnosis of MOGAD.	87	9

Abbreviations: ADEM: Acute Disseminated Encephalomyelitis, AQP4-IgG: Aquaporin-4 Immunoglobulin G, CSF: Cerebrospinal Fluid, ELISA: Enzyme-Linked Immunosorbent Assay, MOG-IgG: Myelin Oligodendrocyte Glycoprotein Immunoglobulin G, MOGAD: Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease, MS: Multiple Sclerosis, NMOSD: Neuromyelitis Optica Spectrum Disorder, MRI: Magnetic Resonance Imaging, OCB: Oligoclonal Bands

had high-titer AQP4-IgG and low titer MOG-IgG, suggesting false-positive results for MOG-IgG (Kunchok et al., 2020).

Despite the high sensitivity and specificity of CBA compared to ELISA, testing a diverse and unselected population for MOG-IgG can result in a false-positive rate of up to 28 % (Held et al., 2021; Sechi et al., 2021). Additionally, positivity for MOG-IgG is uncommon in adults with a first demyelinating event suggestive of MS (Villacieros-Álvarez et al., 2023; Soelberg et al., 2017). Notably, MOG-IgG testing, as measured by CBA, is available in 42 % of countries in LATAM and MOG-IgG titers are not broadly available in LATAM (Rojas et al., 2021), it being a significant limitation in reaching an accurate diagnosis. A multicenter study found that MOG-IgG testing was available in 38 % of 60 countries evaluated worldwide. Low-income countries had lower availability of MOG-IgG testing compared with high-income countries (Holroyd et al., 2019), suggesting that barriers and disparities to MOGAD care are observed, especially in countries of LATAM (Carnero Contentti, 2023). Currently, serum live-CBA is the gold standard for detecting MOG-IgG with specificity ranging from 97.8 % to 100 % (Waters et al., 2019; Reindl M et al., 2020). However, two recent studies with 322 and 257 patient samples showed excellent agreement between live and fixed CBA for diagnosing MOGAD (Pandit et al., 2023; Smith et al., 2023). These results may have significant implications for the diagnosis of MOGAD in low- to middle-income and limited-resources countries, considering that very few laboratories in the region conduct live CBA with antibody titer measurement (in serum and CSF) to detect MOG-IgG.

MOG-IgG titers are important in clinical practice and the 2023 MOGAD diagnostic criteria have incorporated this concept. Additional supporting criteria have been added to enhance the specificity of diagnosing this condition in patients with low positive or unknown titers (Figure 1). Endpoint titers, representing the highest dilution with a signal above a cutoff, may potentially be valuable for confirming a diagnosis of MOGAD, unlike AQP4-IgG where even low positive titers are specific for NMOSD (Matsumoto et al., 2023). Low positive MOG-IgG titers in patients without supporting criteria should be interpreted with caution. In a study involving patients from the U.S. with demyelinating syndromes who were consecutively tested for MOG-IgG by live CBA during their diagnostic workup, 1260 clinical samples were examined and 92 tested positive for MOG-IgG. The positive predictive value (PPV) of MOG-IgG was reported to be 10 % (95 % CI, 2 %-40 %) for atypical phenotypes with titers below 1:100, and 46 % (95 % CI, 33 %-60 %) for those with either atypical phenotypes or titers below 1:100. In contrast, at moderate titers (1:100), 18 % were false positives, while at high titers (≥1:1000), no false positives were observed (Sechi et al., 2021). Another study of 2107 adult inpatients at a German hospital found MOG-IgG positivity in 1.2 % of cases, predominantly at low titers, with only 0.2 % having true MOGAD (Held et al., 2021). Many patients with low-positive titers tested negative in follow-up. Multicenter comparisons indicate that high titers are consistently reliable, whereas low titers show poor agreement (Waters et al., 2019; Reindl M et al., 2020).

Fig. 2.

MOGAD can be monophasic or relapsing, with approximately equal chance at onset (Cobo-Calvo et al., 2018). MOG-IgG titers at onset do not predict the risk of developing a relapsing disease so far (Flanagan and Waters, 2023), but there is some data that persistent MOG-IgG positivity over time, and high remission titers, might stratify relapse risk (Wendel et al., 2022; Gastaldi et al., 2023). MOG-IgG titers are typically higher during an acute attack in young children compared to adolescents or adults but are more likely to become negative during follow-up (Jarius et al., 2016). Timing of testing is crucial (Lui et al., 2021; Forcadela et al., 2023), as antibody titers fluctuate and can decrease over months from presentation, with some patients subsequently testing negative (Jurynczyk et al., 2017; Waters et al., 2020; Lopez-Chiriboga et al., 2018).

Recent observational studies suggest that MOG-IgG in CSF may have diagnostic and prognostic utility (Hacohen et al., 2021; Kwon et al., 2021; Mariotto et al., 2019, Akaishi et al., 2021; Pace et al., 2021; Carta et al., 2023). CSF MOG-IgG testing was incorporated in the 2023 MOGAD criteria, but additional supporting characteristics are required, similar to those with low or unknown serum MOG-IgG titers. CSF testing can be used in cases highly suspicious for MOGAD with MOG-IgG negative serum, as false positive results are rarely found (Mariotto et al., 2019; Jarius et al., 2018). A study involving 133 MOGAD patients revealed that 70 % experienced MOG-IgG present in both CSF and serum, while 13 % exhibited MOG-IgG solely in serum and 17 % only in

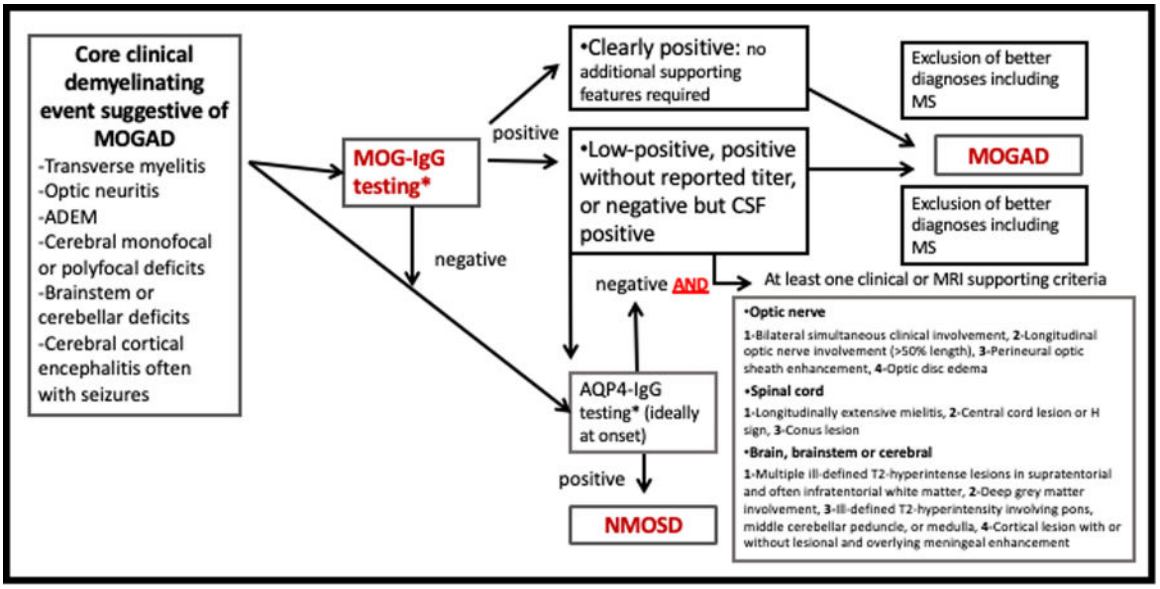

Fig 1. Although not pathognomonic, there are clinical and/or radiological symptoms and signs that are suggestive of MOGAD. Given that positivity for both serum MOG-IgG and AQP4-IgG is extremely rare using CBA, if core clinical demyelinating events are observed both antibodies should ideally be tested (if available). CSF testing may be valuable in patients with features suggestive of MOGAD but in whom serum testing was negative, in particular if confounded by plasmapheresis or other therapeutic interventions. Fixed cell-based assay result with a titer ≥1:100 is considered a clear positive assay and a titer ≥1:10 and <1:100 is considered a low positive assay. Exclusion of no better explanation requires the expertise of the physician and to rule out highly prevalent infectious and nutritional diseases in this region, as well as NMOSD and MS. Relevant features of MOGAD, AQP4-IgG-positive NMOSD and multiple sclerosis were compared in Table 2.

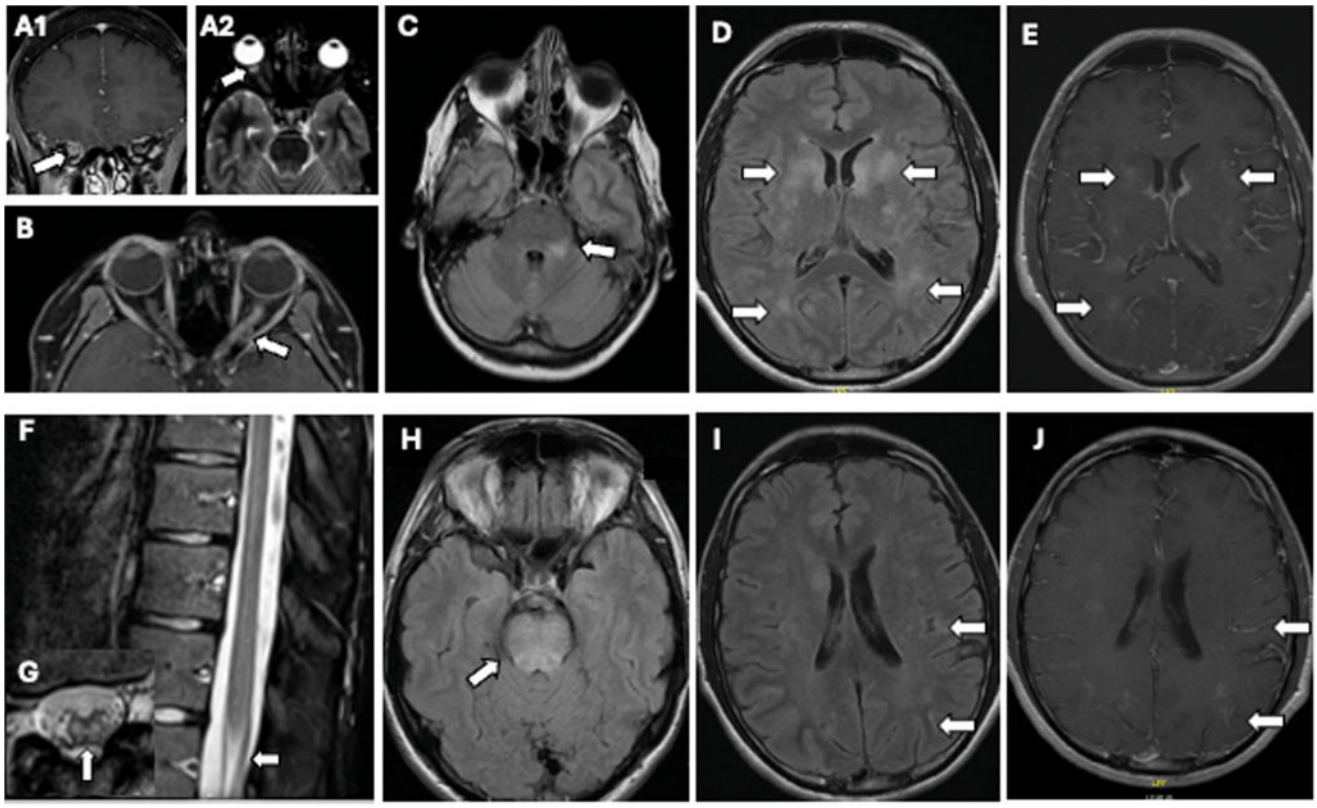

Fig 2. MRI scans highlighting features identified as either typical or suggestive of MOGAD. Examples of lesions (white arrows) associated with adult MOGAD patients. A1) Right perineural optic sheath enhancement (white arrows). A2) Radiologically visible right optic disc swelling. B) Left longitudinally extensive optic nerve hyperintensity. C) fluffy infratentorial lesion (left middle cerebellar peduncle). D) Large ill-defined T2-hyperintense lesions involving supratentorial white matter and deep grey matter with E) Gadolinium-enhancing lesions. F) Conus lesion. G) Central spinal cord involvement with H sign. H) Diffuse T2-hyperintense pontine lesion I) Cortical FLAIR-hyperintense lesions with J) Diffuse leptomeningeal gadolinium-enhancing lesions.

CSF (Matsumoto et al., 2023). CSF-restricted MOG-IgG was associated with ADEM and cortical encephalitis, whereas serum MOG-IgG was associated with ON. Serum negative but CSF positive MOG-IgG, in the appropriate clinical setting, was found in 9 of 83 (11 %) MOGAD patients, all of whom met the 2023 MOGAD diagnostic criteria (Redenbaugh et al., 2024). Nevertheless, the significance of CSF testing for MOG-IgG requires further investigation. Additionally, performing lumbar puncture including oligoclonal bands (OCB) in CSF and serum to evaluate differential diagnoses is useful in clinical practice. Approximately half of the patients exhibit CSF pleocytosis (lymphocytes and monocytes) and elevated CSF protein levels (Jarius et al., 2020). The white cell count is typically higher than that observed in MS. OCB are rare in MOGAD, occurring in fewer than 15 % of cases (Flanagan et al., 2016). When OCBs are present, a thorough clinical and neuroradiological review should be conducted to ensure diagnostic certainty.

ON is the most common presentation in adult MOGAD patients followed by myelitis and less commonly ADEM, brainstem and other cerebral manifestations (Marignier et al., 2021), as also observed in a large LATAM cohort (Carnero Contentti et al., 2024). ON can be isolated or recurrent, often bilateral, with severe papillitis on fundoscopic examination associated with frontotemporal headache before the visual impairment in up to 50 % of cases (Asseyer et al., 2020; Carnero Contentti et al., 2023). Myelitis is commonly severe with paraparesis requiring a gait aid, and/or bladder dysfunction requiring catheterization at myelitis nadir (Perez-Giraldo et al., 2023). Notably, recovery from relapses is generally good; however, some patients present neurological sequalae as a result of the first attack (Jurynczyk et al 2017) or subsequent relapses, with age at presentation being an important risk factor for disability (Chen et al., 2023; Carnero Contentti et al., 2021).

Referring patients to a neurologist experienced in demyelinating disorders increases the chance of an accurate MOGAD diagnosis (Carnero Contentti et al., 2024). Misdiagnosis of MS and NMOSD has been reported worldwide (Carnero Contentti et al., 2023; Gaitan et al., 2022; Kaisey et al., 2019), including LATAM, and such errors can significantly impede an appropriate management and increase healthcare costs for patients, leading to accumulated disability and delayed diagnosis, especially in this region (Papp et al., 2024; Carnero Contentti et al., 2024). Although limited data have been reported for MOGAD, this risk may be reduced if experienced neurologists manage these patients. Excluding regional diseases that may mimic MOGAD clinically and radiologically is important (Amezcua et al., 2024; Correale et al., 2024). In LATAM populations, higher prevalence of certain infectious and nutritional diseases can resemble MOGAD as previously reported in other MS and NMOSD LATAM consensus publications. Details of these differential diagnoses were previously published elsewhere (Cristiano et al., 2020; Silva et al., 2023; Carnero Contentti et al., 2020). An integrated approach combining clinical, laboratory, and imaging studies enhances diagnostic accuracy for suspected MOGAD cases; therefore, continuing medical education in the region should be a key point to consider.

Although MOGAD does not have entirely unique clinical features that are exclusive to the disease, some characteristics are highly suggestive and can help differentiate it from other neuroinflammatory disorders (Table 2). The 2023 MOGAD criteria outlined three key aspects for the diagnosis (Banwell et al., 2023): 1) Core clinical demyelinating events and supporting clinical or MRI features, 2) MOG-IgG and their titers using pre-fixed commercial or in-house live CBA and 3) Exclusion of alternative diagnoses, including MS (Manzano et al., 2024). The panel emphasized the importance of MOG-IgG serostatus and

Table 2
Comparison of the most relevant demographic, clinical and paraclinical characteristics in adults MS, AQP4-ab-positive NMOSD and MOGAD patients.

	MS	AQP4-ab-posivite NMOSD	MOGAD
Age at onset (median years)	Around 30	Around 40	Pediatric to 40
Sex	More common in women	More common in women	Similar proportion
F:M	3:1	9:1	1:1
Ethnicity predominance	Caucasian	Afro-descendant and Asian (higher risk)	No clear racial predominance
Disease course	Usually relapsing, or progressive (~ 15%)	Usually relapsing (~ 90%)	Monophasic (~ 50%) or relapsing (~ 50%)
Typical or cardinal attacks	ON, TM or Brainstem syndromes	Usually ON and/or TM followed by area postrema syndrome	Usually ON followed by TM, encephalopathy/ cortical is rare
Presence of oligoclonal bands (type II)	Common (~ 90%) Persistent	Uncommon (~ 30%) Usually, transient	Uncommon (~ 15%) Usually, transient
Brain MRI lesions			
Suggestive or typical	Ovoid (>3 mm), periventricular (perpendicular to LV), corpus callosum (Dawson's fingers), inferior temporal lobe, S-shaped U-fiber (juxtacortical)	Often dot-like lesions, peri 3rd and 4th ventricle, corpus callosum (posterior), long corticospinal tract lesions and deep white matter or subcortical lesions (typically >3 cm)	May be normal initially, fluffy subcortical white matter (typically tumefactive) and the internal capsule, deep gray matter, thalamic and basal ganglia.
Cortical	Usually, present	Usually, absent.	May be present.
CVS	Common	Very uncommon	Uncommon
New asymptomatic T2-hyperintensity	Common	Uncommon	Uncommon
Residual T1-hypointensity	Common	Common	Uncommon
Contrast enhancement	Ring and/or open ring, ovoid.	Pencil thin ependymal or cloud-like with poorly marginated enhancement	Nodular (typically not as open ring) or perivascular
Posterior fossa lesions			
Suggestive or typical	Brainstem peripheral lesions and cerebellar peduncles	Area postrema lesions (dorsal medulla), can be transient	Often poorly demarcated lesions affecting the pons, medulla oblongata and middle cerebellar peduncles (generally adjacent to the fourth ventricle).
Spinal Cord MRI lesions			
	Usually, cervical lesions (may extend rostrally to the dorsal medulla), short (typically, ≤1 segment), lateral or posterior.	Usually, cervical and thoracic, longitudinally extensive, bright spotty lesion (highly specific), and central	Usually, thoracic and/or lumbar involving the conus, longitudinally extensive, central (H-sign)
	Often Gd-enhancement	Often Gd-enhancement	Often Gd-enhancement
Orbital MRI lesions	Typically, unilateral, and short lesion, involving anterior, variable Gd-enhancement	Typically, bilateral, and longitudinally extensive lesions, involving posterior and optic chiasma, common Gd-enhancement	Typically, bilateral and longitudinally extensive lesions, involving anterior and head optic disc, common Gd-enhancement.
Acute treatment			
Strategies	IVMP, uncommon PLEX use.	IVMP alone or combined with PLEX or PLEX alone initially.	IVMP alone or combined with PLEX in severe cases, IgGIV can be used in pediatric.
Recovery	Usually, good	Usually, incomplete	Usually, good
Long-term treatment	RTCs have demonstrated efficacy and safety to using distinct DMTs	RTCs have demonstrated efficacy and safety to using eculizumab, inebilizumab or satralizumab	There is no RTCs. Debate on when, how and which drug to use is ongoing

Abbreviations: CVS, central vein sign; DMTs, drug-modifying treatments; F, female; Gd, gadolinium; IgGIV, intravenous immunoglobulin; IVMP, intravenous methylprednisolone; LV, lateral ventricles; M, male; ON, optic neuritis; PLEX, plasmapheresis; RCT, randomized control trial; TM, transverse myelitis.
The table is reproduced from a previous publication (Carnero Contentti et al., 2023)

typical or suggestive MRI lesions, reflecting a broader range of MOGAD phenotypes to enable earlier and more accurate diagnosis. The 2023 MOGAD criteria demonstrated diagnostic utility in a LATAM cohort of 171 patients who met the 2018 Mayo Clinic criteria (López-Chiriboga et al., 2018), with 84.2 % satisfying the 2023 criteria despite limited access to MOG-IgG titration (60.2 % lacked titers) (Carnero Contentti et al., 2024). Sensitivity was 86 % and specificity 100 % compared to the 2018 criteria (Jarius et al., 2018). Diagnosis rates aligned with those reported in the USA (81.5 % and 90 %), Korea (93 %) and Europe (97 %), supporting their clinical relevance (Kim et al., 2023; Alaboudi et al., 2024; Filippatou et al., 2024; Forcadela et al., 2023).

3.2. MRI and other complementary tests

Recommendations for MRI and other complementary tests are shown in Table 3. To ensure an accurate diagnosis of MOGAD, a standardized MRI protocol should be used, as recommended in a recent revision and LATAM consensus on NMOSD management (Carnero Contentti al., 2020; Geraldes et al., 2024). Standardizing MRI protocols for CNS inflammatory diseases is important for ensuring consistent execution and accurate interpretation of studies as recently published by the MAGNIMS Study Group (Bartels et al., 2021; Geraldes et al., 2024).

Recent diagnostic criteria for MOGAD highlight the following brain, brainstem and cortical MRI features as supporting criteria: 1. Multiple ill-defined T2 hyperintense lesions in supratentorial and infratentorial white matter, 2. Deep grey matter involvement, 3. Ill-defined T2 hyperintensities in the pons, middle cerebellar peduncles, or medulla, 4 Cortical lesions with or without meningeal enhancement (Banwell et al., 2023).

Brain lesions are present in approximately 45 % of MOGAD patients at disease onset, with a higher prevalence in children, regardless of the initial clinical presentation (Bartels et al., 2021; Clarke et al., 2021). The MOGADOR study on adult MOGAD identified the thalamus and pons as the most frequently affected regions, although other studies report a greater prevalence of lesions in subcortical white matter and the internal capsule (Cobo-Calvo et al., 2018). Recent studies indicate that diffuse middle cerebellar peduncle lesions are present in 46 % of patients with brainstem and cerebellar involvement (Banks et al., 2020; Trewin et al.,

Table 3
Recommendations for MRI and other complementary tests at diagnosis and follow-up.

	%	Median
• In patients with suspected MOGAD, a standardized MRI protocol should be applied at diagnosis and follow-up.	86	9
• In patients with suspected MOGAD, a whole spinal cord MRI (whenever possible), including STIR sequences and the conus medullaris, is recommended.	87	9
• MRI scanner with a minimum field strength of 1.5-T is strongly recommended in clinical practice.	89	9
• Patients presenting with short-segment myelitis (STM) on MRI and normal or non-typical brain lesions for MS, serum MOG-IgG testing should be performed.	82	9
• Patients presenting with ON and suspected MOGAD, orbital MRI (including FAT-SAT sequences) is recommended to facilitate differential diagnosis and to assess typical MOGAD lesions.	87	9
• In MOGAD patients on long-term immunosuppressant therapy, annual brain MRI is recommended to detect ongoing inflammatory disease activity, lesion evolution or treatments side effects (e.g., opportunistic infections).	73	8
• Patients presenting with ON and suspected MOGAD, OCT at onset and follow-up (at least once a year) is recommended.	76	8
• Patients presenting with ON and suspected MOGAD, visual EP at onset is recommended.	70	7
• Patients presenting with ON and suspected MOGAD, a neuroophthalmological evaluation, including fundus at onset and follow-up (at least once a year) is recommended.	87	9

Abbreviations: 1.5-T: 1.5 Tesla, EP: Evoked Potentials, FAT-SAT: Fat Saturation, MRI: Magnetic Resonance Imaging, OCT: Optical Coherence Tomography, ON: Optic Neuritis, STIR: Short Tau Inversion Recovery, STM: Short-Segment Myelitis

2025).

MRI findings in adults may be classified into three categories: white matter (focal or diffuse), brainstem, and cortical lesions (Shor et al., 2021; Geraldes et al., 2024). T2-hyperintense lesions are observed in 32–53 % of MOGAD patients, particularly those with ON or TM. These lesions are typically sparse (three or fewer), poorly demarcated, and involve deep grey or subcortical white matter (Jurynczyk et al., 2017). Persistent T1-hypointense lesions are rare compared to MS (Duan et al., 2021). Tumefactive lesions defined as lesions $>$ 2 cm in maximum diameter, present in 22 % of cases, have generally good outcomes after treatment but in rare cases can lead to life-threatening herniation (Cacciaguerra et al., 2023).

Gadolinium-enhancing lesions are either nodular or perivascular, often faint, predominantly involving white matter, and are observed in 44–65 % of patients during brain or myelitis attacks (Cobo-Calvo et al., 2018; Perez-Giraldo et al., 2023). Leptomeningeal enhancement around the brainstem and cortical enhancement (associated with encephalitis) are also reported and suggestive of MOGAD in a compatible clinical setting (Ogawa et al., 2017). Additionally, deep grey matter (GM) involvement is a common finding in MOGAD (Kitley et al., 2014; Xie et al., 2022; Salama et al., 2020; Cagol et al., 2024). Higher periventricular and cortical/juxtacortical lesions were associated with reduced temporal cortex, deep GM, and insula volumes (Cortese et al., 2023; Cortese et al., 2024). T2 brain lesions in MOGAD demonstrate significant resolution over time, with extra-callosal lesions and T2 lesion resolution noted in 56 % of cases (Sechi et al., 2021; Cortese et al., 2023). In this context, the panel have recommended annual brain MRI as additional support in the management of these patient. Although this remains a matter of debate, evidence supporting routine annual MRI in MOGAD is limited, and such imaging may not be useful in clinical practice. The initial brain MRI can be normal in 10 % of attacks despite patients being symptomatic. The dynamic nature of MRI lesions, characterized by the frequent appearance and occasional resolution of lesions within a single attack, supports a diagnosis of MOGAD over MS or AQP4-IgG-positive NMOSD (Cacciaguerra et al., 2024). Periventricular Dawson finger-like lesions are rare (Clarke et al., 2021). Diffuse brainstem involvement in the pons, medulla, and middle cerebellar peduncles is common, with infratentorial lesions appearing fluffy and poorly defined (Jurynczyk et al., 2017; Carnero Contentti et al., 2022; Geraldes et al., 2024).

MOGAD patients experiencing seizures may display a distinctive cortical lesion pattern on FLAIR sequences, termed FLAIR hyperintense lesions in anti-MOG encephalitis with seizures (FLAMES). (Jain et al., 2021; Budhram et al., 2019; Budhram et al., 2022). Thalamic and basal ganglia lesions are more common in MOGAD compared to AQP4-IgG-positive NMOSD or MS (Chen et al., 2019; Shahriari et al., 2021; Cobo-Calvo et al., 2018)

Multiple spinal lesions in MOGAD are commonly LETM or STM (Mariano et al., 2019), with LETM comprising 60–80 % of cases (Dubey et al., 2019), typically affecting the conus medullaris. During the acute phase, parenchymal cord enhancement is less frequent than in MS or AQP4-seropositive NMOSD and, when present, is usually faint (Cobo-Calvo et al., 2020). Spinal cord leptomeningeal enhancement, however, appears more frequent (7.4 %). (Fadda et al., 2021). GM involvement is typical, producing the "H sign" on axial views (29–38 %) and a central linear T2-hyperintensity on sagittal views. (Marignier et al., 2021; Carnero Contentti et al., 2024; Dubey et al., 2019; Trewin et al., 2025). Acute lesions may also show pseudo-dilatation of the central canal, mimicking physiological dilatation (Denève et al., 2019). Atrophy is rare, and imaging abnormalities often resolve after clinical recovery (Al-Ani A et al., 2023).

MOGAD commonly affects the optic nerve sheath or extends into orbital fat, with gadolinium-enhancing lesions involving more than 50 % of the optic nerve in 80 % of cases. (Chen et al., 2018; Akaishi et al., 2016; Ramanathan et al., 2016). Lesions are often edematous and typically localized in the anterior segment, sparing the optic chiasm and retrochiasmatic tracts, with the affected optic nerves being enlarged and tortuous (Akaishi et al., 2016). While optic chiasm involvement is significantly more common in AQP4-IgG-positive NMOSD (31.7 %) than in MOGAD (13.1 %) or MS-ON, optic nerve head swelling is seen in over 50 % of MOGAD cases (Carnero Contentti et al., 2022). In contrast, this feature is rare in AQP4-IgG-positive NMOSD and absent in MS-ON, making it a key distinguishing characteristic, as observed in a large LATAM cohort (Carnero Contentti et al., 2023; Trewin et al., 2025).

Some set of criteria to differentiate MS form NMOSD and MOGAD have been reported with good performance in clinical practice, including LATAM population with distinct AQP4-IgG serostatus (Bensi et al., 2018; Carnero Contentti et al., 2020). It may be relevant as an aid to achieve a correct diagnosis, particularly in countries where the availability of diagnostic tests is low or in patients where overlapping syndromes or borderline cases are found (Carnero Contentti et al., 2020).

Optical coherence tomography (OCT) may aid in diagnosing ON in MOGAD and differentiating it from ON in conditions like MS and NMOSD (Bartels et al., 2021; Oertel et al., 2017; Oertel et al., 2019; Filippatou et al., 2020). In acute ON, the peripapillary retinal nerve fibre layer (pRNFL) is significantly thickened in MOGAD compared to MS (Chen et al., 2022; Oertel et al., 2019). Over 3–6 months, thinning occurs in both the pRNFL and the macular ganglion cell-inner plexiform layer (mGCIPL) (Costello et al., 2015). While mGCIPL thinning begins within weeks, pRNFL thinning develops more gradually as optic nerve head edema resolves. Recurrent ON in MOGAD often causes severe pRNFL and mGCIPL thinning, whereas AQP4-IgG-positive NMOSD can lead to significant thinning after a single episode (Oertel et al., 2021; Pache et al., 2016).

OCT studies show that pRNFL thinning below 75 μm correlates with poorer visual outcomes (Costello et al., 2006) though some MOGAD patients maintain good vision despite substantial thinning. In contrast, ON in NMOSD is typically linked to more severe vision loss, despite similar thinning patterns (Carnero Contentti et al., 2023, Sotirchos et al., 2020). The discrepancy between structural damage and functional impairment may be due to astrocytopathy in AQP4-IgG-positive NMOSD

or OCT limitations in detecting damage below 50–60 μm, potentially underestimating optic nerve damage compared to MOGAD (Sechi et al., 2022).

VEPs are widely available in LATAM and can detect subclinical alterations, in some cases, severity and monitoring of progression. VEP abnormalities are often correlated with clinical symptoms (Yano et al., 2024). However, their interpretation should be made in conjunction with other diagnostic tests and the clinical evaluation of the patient.

3.3. Prognostic factors

The risk of relapse in adult MOGAD is variable and long-term relapses depend on factors such as age, follow-up time, very early relapses (within 30 to 90 days from onset), MOG-IgG titers and early initiation of maintenance treatment (Chen et al., 2023; Deschamps R, et al. 2024; Trewin et al., 2025; Satukijchai et al., 2022; Cobo-Calvo et al., 2019) (Table 4).

Studies confirm that persistent MOG-IgG positivity is associated with a higher likelihood of relapsing disease. Patients whose antibody titers decrease or become undetectable after the initial attack are more likely to follow a monophasic course (Jurynczyk et al., 2017; Gastaldi et al., 2023; Wang et al., 2023). This underscores the importance of serial antibody testing in predicting disease trajectory and guiding decisions on maintenance therapy. Another study showed that high MOG-IgG titers at onset were associated with a more severe presentation, but did not predict the future disease course (Cobo-Calvo et al., 2019). However, the utility of MOG-IgG titers for treatment planning is still a topic of debate (Carnero Contentti et al., 2021). As previously mentioned, the limited availability of tests and antibody titers, considering their high cost and the centralization of testing, is a significant limitation in LATAM (Rojas et al., 2021; Tkachuk et al., 2023). Additionally, elevated CSF protein and leukocytes (above 50/mm3) predicted a relapsing disease course in one study (Molazadeh et al., 2024; Rempe et al., 2024).

The severity and type of the initial CNS demyelinating event significantly influenced long-term outcomes. Severe ON, TM with LETM in MRI and polyphasic manifestation at presentation is often associated with a higher relapse risk and greater likelihood of residual disability (Molazadeh et al., 2024; Jeyakumar et al., 2024). Early aggressive treatment of such presentations can mitigate severity and recurrence risks (Deschamps R, et al. 2024).

Ethnicity may impact MOGAD relapse risk. Hispanic patients and non-Hispanic non-white patients with MOGAD appear to have a higher risk of relapse (Martin et al., 2024). However, no differences were found after evaluating distinct ethnic groups (Mixed vs. Caucasian vs. Afro-descendant) in a LATAM cohort (Carnero Contentti et al., 2023). Studies with longer follow-up are needed as the risk of relapse increases with longer follow-up.

3.4. Acute treatment

Recommendations for acute treatment are shown in Table 5. A considerable number of MOGAD patients may experience permanent disability due to incomplete recovery from attacks, with the likelihood of such disability being higher in adults, depending on the age and type at which symptoms first appear (Marignier et al., 2021; Carnero Contentti et al., 2023). Up to 60 % of patients may develop disability from the initial attack, while in the remaining cases, the accumulation of disability often results from subsequent relapses (Molazadeh et al., 2023; Jarius et al., 2016, Carnero Contentti et al., 2021). These observations underscore the necessity of prompt acute management, as timely intervention may be crucial in preventing permanent impairment (Schwake et al., 2024; Stiebel-Kalish et al., 2019; Rode et al., 2023;).

At present, there are no evidence-based protocols for the acute management of MOGAD patients. Typically, intravenous methylprednisolone and plasma exchange have been employed to address acute episodes (Galetta et al., 2022); however, some studies have also utilized intravenous immunoglobulins (Lotan et al., 2023). Intravenous methylprednisolone is administered at a dose of 1 *g* daily for 3 to 5 days, with approximately 50 % of patients achieving good or complete recovery (Liu et al. 2017). Other research has reported recovery rates from attacks reaching nearly 90 % (Liu et al. 2017; Rode et al., 2023; Soelberg et al., 2018); however, these results were often derived from a combination of sequential treatments rather than from the effects of a single therapeutic approach. Intravenous methylprednisolone is important for optimal visual recovery, as it has been reported that early steroid treatment is linked to better visual outcomes and less thinning of the peri-papillary retinal nerve fiber layer (Soelberg et al.2018). Another option might be the use of oral steroids, as demonstrated by studies showing that oral high-dose methylprednisolone for 3 days was as effective as IV administration in improving disability scores in MS patients, with a similar safety profile (Le Page et al., 2015; Liu et al., 2017). Following this, slow tapering of oral corticosteroids, e.g., weaning daily prednisone dose by 5 mg every 2 weeks until $\geq$ 20 mg, may be considered for up to 2–6 months to prevent early symptom recurrence, as reported in Australian and UK cohorts (Ramanathan et al., 2018; Trewin et al., 2024; Jurynczyk et al., 2017). While generally well tolerated, side effects can appear, and its long-term use is still controversial (Whittam et al., 2020). This intervention is widely available in LATAM; therefore, it is the treatment most used (Carnero Contentti et al., 2021).

In cases where steroid treatment did not lead to symptom relief, plasma exchange (usually consisting of 5–7 cycles) is used as a secondary treatment, resulting in substantial improvement in up to 40 % of those who did not respond to steroids (Schwake et al., 2024). Evidence

Table 4
Recommendations for disease prognosis.

	%	Median
• The accumulation of neurological disability in MOGAD patients is primarily relapse related.	87	9
• At disease onset, the presence of a severe attack with insufficient recovery predicts worse medium/long-term disability.	83	9
• Persistence of MOG-IgG seropositivity over time is predictive of a relapsing course.	75	9
• Persistence of higher titers of MOG-IgG over time is predictive of a relapsing course.	75	9

Abbreviations: IgG: Immunoglobulin G, MOG: Myelin Oligodendrocyte Glycoprotein, MOGAD: Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease

Table 5
Acute Treatment.

	%	Median
• The initial diagnosis of these patients should be considered uncertain until the serological status results are known.	70	8
• Early high-dose intravenous methylprednisolone (1000 mg daily for 3–5 consecutive days) in acute relapses is recommended.	89	9
• Oral prednisone at the equivalent dose of 1250 mg once daily for five days may be considered as an alternative to the 1000 mg of IV methylprednisolone infusion, when infusion center is not available.	79	9
• After IVMP treatment, it is recommended to gradually reduce the dosage of oral steroids until the serological status results are known.	71	8
• MOGAD patients who do not respond adequately to high IV steroids should be treated with PLEX.	88	9
• The clinical benefit of PLEX may decreases after day 20, regardless of whether IVMP has been given; therefore, initiating PLEX early is recommended.	85	9
• MOGAD patients who do not respond adequately to high IVMP should be treated with intravenous immunoglobulin (IVIG), if PLEX is not available.	80	8

Abbreviations: IV: Intravenous, IVIG: Intravenous Immunoglobulin, IVMP: Intravenous Methylprednisolone, MOGAD: Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease, PLEX: Plasmapheresis

supports early initiation within days of an acute attack (Stiebel-Kalish et al., 2019). No response or neurological worsening post-treatment are concerning and might indicate alternative diagnoses. Plasma exchange is often safe and well tolerated. However, PLEX was only used in 15.5 % of an Argentine cohort, likely due to limited access to this intervention (Carnero Contentti et al., 2021). Intravenous immunoglobulins may be useful for incomplete recovery after corticosteroid plasma exchange and as long-term therapy (Chen et al., 2020; Chen et al., 2022). Typical dosing is 0.4 g/kg/day for five days, especially in acute pediatric cases, with common side effects including headaches and elevated blood pressure during infusion. Serum IgA levels should be evaluated before the first infusion to prevent anaphylactic reactions related to IgA deficiency (Katz et al., 2007).

Table 6
Long-term or maintenance treatment.

	%	Median
• MS treatments (i.e., disease-modifying treatments) must not be used in MOGAD patients.	82	9
• After a first attack associated with MOG-IgG seropositivity (i.e., MOGAD), use of oral steroids 1 mg/kg per day for 3 months and then progressive tapering over the next 3 months (at least 20 mg/day) is recommended to reduce the risk of early relapse.	83	9
• After first attack with no history of other relapses, if MOG-IgG become negative at 6 months, treatment should be slowly discontinued.	85	9
• If MOG-IgG persist positive at 6 months, oral steroids should be maintained for 12 months, when they should be retested.	73	7
• If MOG-IgG become negative at 12 months and there are no relapses, initial oral steroids should be slowly discontinued.	82	9
• Long-term steroid-sparing immunosuppressant therapies should be used in MOGAD patients if new relapses (recurrent MOGAD) are observed.	87	9
• Long-term steroid-sparing immunosuppressant therapies should be used in MOGAD patients if MOG-IgG persist at 12-months of follow-up after initiating attack.	79	8
• Long-term steroid-sparing immunosuppressant therapies should be used in MOGAD patients if adverse effects and/or intolerance to oral steroids are observed after initiating attack.	82	9
• Azathioprine (AZA, 2-3 mg/kg/day divided into 2-3 doses per day) has been shown to be effective and safe in preventing relapses of MOGAD, making it a suitable first-line treatment for MOGAD patients.	75	8
• Mycophenolate mofetil (MMF, 2-3 g/day divided into two doses per day) has been shown to be effective and safe in preventing relapses of MOGAD, making it a suitable first-line treatment for MOGAD patients.	77	8
• A single IVIG infusion of 1 g/kg every three to four weeks can be used as first-line of long-term treatment for MOGAD patients.	73	8
• Induction with higher IVIG dose (2 g/kg) followed by 1 g/kg every three to four weeks can be used as first-line of long-term treatment for MOGAD patients.	72	8
• The induction protocol for rituximab should involve infusing doses of 375 mg/m2 of body surface area, given as an IV infusion once a week for four weeks, or 1000 mg IV with re-infusion at 14 days.	83	9
• Maintenance protocols using 1000 mg of rituximab with re-infusion at 14 days, or one infusion of 1000 mg, or one infusion of 375 mg/m2 repeated every six months, have been shown to be safe and effective in preventing relapses of MOGAD, making it a suitable first-line treatment for MOGAD patients.	75	9
• Tocilizumab can be used in MOGAD patients showing no response to other immunosuppressants in clinical practice.	78	9
• Regardless of the frequency and severity of relapses in MOGAD patients after starting treatment, the occurrence of relapses following the proper use and full biological effectiveness of the specific treatment is deemed to indicate a "suboptimal treatment response".	83	9
• Low doses of oral steroids should be maintained (4-6 months) while other long-term immunosuppressant medications (AZA or MMF) take full effect	85	9
• Considering the variable course of MOGAD, prolonged immunosuppressive strategies should be reconsidered at follow-up (at least 5 years of treatment) balancing risk/benefit.	80	8

AZA: Azathioprine, IVIG: Intravenous Immunoglobulin, MOGAD: Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease, MMF: Mycophenolate Mofetil, MS: Multiple Sclerosis

3.5. Long-term treatment

Recommendations for long-term treatment are shown in Table 6. There is no currently approved treatment for MOGAD, but off-label long-term therapies such as azathioprine (AZA), mycophenolate mofetil (MMF), rituximab, maintenance intravenous immunoglobulin (IVIG), and tocilizumab have been shown in observational retrospective studies and systematic revisions to reduce the incidence of relapses, whereas some MS-targeted disease-modifying therapies appear to lack efficacy or increase the risk of relapses for this condition (Lu et al., 2021; Chang et al., 2023; Wang et al., 2022; Jarius et al., 2016; Chen et al., 2020; Chen et al., 2022). This evidence is important for LATAM region, considering that these therapies are low-cost and widely available.

Given that up to 50–80 % of adult patients experience relapsing disease within the first two to five years of onset (Cobo-Calvo et al., 2019; Jurynczyk et al., 2017), a tailored approach to long-term management is needed, despite advancements, significant gaps remain in defining optimal long-term treatment strategies (Marignier et al., 2021; Carnero Contentti et al., 2021). Notably, there is no data from randomized controlled trials, although Phase II/III trials evaluating satralizumab (NCT05271409), tocilizumab (NCT06452537), rozanolixizumab (NCT05063162) and AZA (NCT05349006) in adults relapsing or after the first attack of MOGAD are ongoing with a very few centers participating from LATAM and will be completed in 2026. Debate on when, how and which medication to use is also ongoing in clinical practice (Whittam et al., 2020). Some neurologists may choose to initiate long-term therapy in individuals who have experienced a particularly severe presentation with significant disability; however, there is no high-quality evidence to inform these decisions (Carnero Contentti et al., 2021). Despite the availability of multiple options, no consensus exists regarding the optimal first-line agent and therapeutic failure (Whittam et al., 2020). In this consensus, regardless of the frequency and severity of relapses after starting treatment, the occurrence of at least one attack following the proper use and full biological effectiveness of the specific treatment was defined as “suboptimal treatment response".

Rituximab reduces annual relapse rates (ARR) by up to 63 % when used as first-line treatment (Cobo-Calvo et al., 2019; Ramanathan et al., 2018; Barreras et al., 2022; Whittam et al., 2020) and EDSS (Durozard et al., 2020) in MOGAD patients. However, breakthrough relapses have been frequently reported, indicating variability in response. The treatment regimens vary widely across studies.

Both MMF and AZA are widely used oral immunosuppressants with moderate efficacy. MMF ($\geq$ 2000 mg/day) has demonstrated a significant reduction in ARR to 0.32 in adult cohorts (Wang et al., 2021), but no positive changes in the EDSS was observed (Lu et al., 2021). The doses of AZA and MMF differ across patients and studies. These agents are particularly valuable in resource-limited settings where IVIG or rituximab therapies may be unavailable. IVIG has shown promise as a maintenance therapy, particularly in patients with frequent relapses or intolerance to other immunosuppressants (Ramanathan et al., 2018; Barreras et al., 2022; Chen et al., 2020). Studies indicate that IVIG was associated with ARRs as low as 0.13 and a relapse-free probability of 72 % after 6 months of therapy (Bilodeau et al., 2024). Additionally, the use of IVIG 2 g/kg monthly (optimal IVIG) was associated with an even lower ARR (0.00) and higher relapse-freedom probability (85 %) (Bilodeau et al., 2024). The optimal duration of maintenance therapy remains unclear. Tocilizumab, an IL-6 receptor inhibitor, is a promising option for refractory MOGAD, though robust data in adult cohorts are limited (Ringelstein et al., 2021; Kang et al., 2024). Factors such as high MOG-IgG titers and early relapses may predict a chronic disease course (Chen et al., 2022), but further research is needed to refine risk stratification models.

Long-term immunosuppression carries risks of infections, malignancy, and treatment-related toxicity; therefore, balancing efficacy and safety remains a clinical challenge (Marignier et al., 2021; Carnero Contentti et al., 2021).

While immunotherapies have demonstrated efficacy in reducing relapse rates, significant gaps remain in understanding optimal therapy duration, sequencing, and monitoring strategies. Continued research, real-world evidence and international collaboration will be pivotal in improving treatment approaches and in consequence outcomes for this rare but impactful disease.

4. Conclusions

These recommendations address the unique challenges in LATAM, including limited access to MOG-IgG testing, healthcare inequities, and socioeconomic disparities that delay MOGAD diagnosis and management, as discussed throughout this manuscript. Unlike high-income regions, LATAM experiences restricted availability of live or fixed CBA, further impeding timely diagnostic confirmation. Notably, certain infectious and nutritional diseases in LATAM can mimic MOGAD, complicating differential diagnoses (Carnero Contentti et al., 2019). These regional pathologies should be carefully ruled out, as their overlap is less common in other regions. Another distinction is the access to therapeutic resources, such as plasmapheresis and immunoglobulin therapies, which are less accessible in some LATAM countries compared to higher-resource regions. Finally, these recommendations emphasize practical and resource-sensitive approaches, such as relying on clinical suspicion and MRI patterns when serological confirmation may be delayed or unavailable. Additionally, the need for cost-effective, long-term immunosuppressants like AZA or MMF aligns with LATAM's economic realities, unlike the widespread use of IVIG or biologics in high-income settings. By addressing these challenges, these recommendations offer a much-needed framework for neurologists across LATAM to optimize outcomes despite regional limitations.

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Supplementary materials

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