REVIEW ARTICLE

Journal of Pre-Clinical and Clinical Research
www.jpccr.eu

Myelin Oligodendrocyte Glycoprotein antibody-associated disease as a novel diagnostic entity – clinical and radiological presentations and diagnostic caveats

Zuzanna Mularczyk[1,B-D], Piotr Tadeusz Oleksy[1,A-D]✉, Maciej Tenderenda[1,A,C-D], Aleksandra Śmigiel[1,B-D], Michalina Rzepka[2,A-B,E-F], Joanna Siuda[2,A-B,E-F]

[1] *Students' Scientific Association, Department of Neurology, Faculty of Medical Sciences, Medical University of Silesia, Katowice, Poland*
[2] *Department of Neurology, Faculty of Medical Sciences, Medical University of Silesia, Katowice, Poland*
A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article

Mularczyk Z, Oleksy PT, Tenderenda M, Śmigiel A, Rzepka M, Siuda J. Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease as a Novel Diagnostic Entity- Clinical and Radiological Presentations and Diagnostic Caveats. J Pre-Clin Clin Res. doi:10.26444/jpccr/219393

Abstract

Introduction and Objective. Myelin oligodendrocyte glycoprotein-associated disease (MOGAD) has emerged as a distinct entity among demyelinating diseases of the central nervous system (CNS). It most commonly manifests around the age of 30 and affects both sexes genders equally. MOGAD includes a broad range of clinical presentations, such as optic neuritis, transverse myelitis, acute disseminated encephalomyelitis, and cerebral cortical encephalitis. The aim of the review is to summarize recent advances in the clinical and radiological characterization of MOGAD, with a focus on diagnostic criteria.
Review Methods. A narrative review was conducted using PubMed, Scopus, and Web of Science. The search included MeSH-based key words: CNS demyelinating autoimmune diseases, myelin oligodendrocyte glycoprotein, myelin oligodendrocyte glycoprotein antibody–associated disease, neuromyelitis optica spectrum disorders, and multiple sclerosis, supplemented by free-text terms. Eligible studies were selected based on predefined criteria and methodological quality.
Brief description of the state of knowledge. MOGAD shares partial clinical and radiological overlap with other CNS demyelinating diseases. Optic neuritis and transverse myelitis are the most frequent manifestations, while and magnetic resonance imaging (MRI) often shows transient, poorly demarcated lesions that resolve over time. The 2023 International MOGAD Panel criteria unified diagnostic standards by integrating core clinical syndromes, MOG-IgG detection, and supportive MRI findings.
Summary. Although substantial progress has been made in characterizing the clinical spectrum, radiological features, and diagnostic approaches of MOGAD, significant gaps persist due to the lack of long-term data on disease trajectory. Future research should focus on disability outcomes and progression, as well as on elucidating epidemiological characteristics.

Key words

multiple sclerosis, CNS demyelinating autoimmune diseases, Myelin Oligodendrocyte Glycoprotein, Myelin Oligodendrocyte Glycoprotein Antibody-associated disease, Neuromyelitis Optica (NMO) Spectrum Disorders

INTRODUCTION AND OBJECTIVE

Myelin Oligodendrocyte Glycoprotein-Associated Disease (MOGAD) is a rare, antibody-mediated inflammatory demyelinating disorder of the central nervous system (CNS), which has recently been recognized as a distinct disease entity from both neuromyelitis optica spectrum disorders (NMOSD) and multiple sclerosis (MS) [1]. Although MOGAD shares some clinical and radiological features with NMOSD and MS, it is considered a distinct entity with different underlying immunopathology, patient profile, clinical manifestations, radiological features and treatment management [2]. MOGAD presents a wide range of clinical presentations, including optic neuritis (ON), transverse myelitis (TM), acute disseminated encephalomyelitis (ADEM), and cerebral cortical encephalitis [1]. The disease course can be either monophasic, usually linked to a favourable long-term outcome, or relapsing. Even in the relapsing form, recovery after attacks is often better than in NMOSD [3]. Unlike MS, where disability may gradually accumulate due to progression, long-term impairment in MOGAD typically results from incomplete relapse recovery [4, 5].

Because of the overlap with NMOSD and MS, and the lack of previously validated internationally recognized diagnostic criteria, in 2023, the International MOGAD Panel proposed consensus diagnostic criteria [5]. These criteria integrate well-defined clinical manifestations with MOG-IgG antibody testing and supportive imaging findings, while also emphasizing the importance of differential diagnosis, exclusion of alternative autoimmune demyelinating CNS disorders, and cautious interpretation of antibody testing results. Since then, the proposed criteria have been validated in multiple studies, demonstrating excellent performance

✉ Address for correspondence: Piotr Tadeusz Oleksy, Students' Scientific Association, Department of Neurology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Medyków 14, 40-752 Katowice, Poland
E-mail: poleksy99@gmail.com

Received: 22.12.2025; accepted:; first published: 19.03.2026

and high diagnostic utility in both paediatric and adult populations [6–9].

OBJECTIVE

The aim of this narrative review is to summarize the current state of knowledge regarding the clinical manifestations, radiological features, diagnostic work-up, and therapeutic strategies in MOGAD. The review provides an up-to-date and concise overview of this rare but clinically significant demyelinating disease of the CNS, characterized by considerable clinical overlap with MS and NMOSD. It summarizes current knowledge on clinical and radiological presentations, diagnostic criteria and their caveats, as well as the limitations and nuances involved in their interpretation, and further provides an overview of current strategies for the treatment of acute demyelinating episodes and maintenance therapy. Additionally, it highlights the need for heightened diagnostic vigilance, greater clinical awareness, and careful consideration of differential diagnoses.

MATERIALS AND METHOD

The review was designed to synthesize and critically evaluate the current state of knowledge on the clinical and radiological features, diagnostic approaches, and laboratory findings in MOGAD. A comprehensive literature search was carried out in July 2025 using three major electronic databases: PubMed, Scopus and Web of Science. These databases were selected for their broad biomedical coverage, methodological rigour in indexing, and recognized international standards, ensuring retrieval of high-quality peer-reviewed publications. The search strategy combined Medical Subject Headings (MeSH) with supplementary free-text terms, applied whenever necessary to broaden the scope of the search. The key words included: CNS demyelinating autoimmune diseases, myelin oligodendrocyte glycoprotein, myelin oligodendrocyte glycoprotein antibody–associated disease, neuromyelitis optica spectrum disorders, and multiple sclerosis. Additional free-text terms related to MOGAD comprised optic neuritis, transverse myelitis, acute disseminated encephalomyelitis, cerebral cortical encephalitis, MRI features, diagnostic criteria, serological testing, disease course, long-term outcomes, acute attack treatment, immunotherapy, maintenance therapy, and relapse prevention.. The literature selection process followed a two-stage approach. First, the titles and abstracts were screened for relevance to the subject, methodological transparency, and overall, the quality of the evidence. In the second step, the full texts of potentially eligible articles were assessed with particular emphasis on the precision of clinical manifestation description, the adequacy and detail of radiological and serological assessments, the consistency and transparency of applied diagnostic criteria and treatment methods, and the length and reliability of follow-up in reporting disease course and disability outcomes. The quality of the included evidence was assessed based on study design and scale, with particular attention to multicentre cohort studies, prospective and retrospective longitudinal studies, and case-control studies. Due to the scarcity of systematic reviews and meta-analyses, reflecting the relative novelty of MOGAD as a distinct disease entity, the limited duration of observational follow-up, and the heterogeneity of diagnostic standards across earlier studies, high-quality narrative reviews authored by experts with recognized experience in the field, were also considered, provided they demonstrated methodological transparency and critical appraisal of the literature. Ultimately, a total of 49 publications were included in the review.

Inclusion criteria. 1) *Time frame:* the search was restricted to articles published between 2016 – 2025, as this period encompasses the years in which most clinically relevant and methodologically robust studies on MOGAD were published, while also allowing inclusion of earlier works that were fundamental for understanding the disease in the years preceding the 2023 International MOGAD Panel diagnostic criteria. 2) *Eligible publications:* included prospective and retrospective cohort studies, case–control studies, and validation studies of diagnostic criteria. In view of the limited availability of systematic reviews and meta-analyses, due to the recent recognition of MOGAD as a distinct clinical entity, the broad scope of the present review, and text length restrictions, information from selected high-quality narrative reviews was referenced where necessary. 3) *Scope of content:* studies were included if they addressed clinical presentations, epidemiological characteristics, disease course and disability progression, radiological features, and the currently applied diagnostic criteria with their recognized limitations. 4) *Diagnostic criteria:* for studies published after 2023, only those employing the International MOGAD Panel diagnostic criteria [5] were included, while for earlier studies predating the current diagnostic consensus, inclusion required confirmed serum MOG-IgG seropositivity in combination with compatible clinical manifestations. This ensured diagnostic uniformity while allowing continuity with studies and publications predating 2023. 5) *The peer review process:* only publications appearing in journals that apply a double-blind peer review system were considered eligible. This ensured the methodological rigour and scientific reliability of the included studies.

Exclusion criteria. 1) *Language restrictions:* publications written in languages other than English were excluded to ensure consistency of interpretation and comparability of findings. 2) *Methodological shortcomings:* studies with inadequate or unclear methodology, insufficient follow-up periods, lack of control groups when required, or low transparency in outcome reporting were excluded to minimize the risk of bias and unreliable conclusions. 3) *Lack of clinical applicability:* articles primarily focused on immunopathology, and purely experimental or molecular neuroscience without direct clinical correlation to purely clinical aspects of the disease, diagnostic processes, or patient outcomes were excluded, to maintain the clinical focus of this review. 4) *Low-quality narrative reviews:* narrative reviews that lacked critical appraisal of literature, were based primarily on isolated case observations and lacked a systematic scientific approach or failed to provide a transparent description of their methodology and inclusion process were excluded.

Limitations of the study. Relocated to usual place before Conclusions.

STATE OF KNOWLEDGE

Clinical presentation. MOGAD is a recent addition to the group of CNS inflammatory demyelinating diseases, which also include MS and NMOSD. While patients with these disorders may present with some overlapping clinical manifestations, the diagnostic work-up is important to distinguish between them based on patient profile, clinical phenotype, neurological examination, serological and cerebrospinal fluid testing, and imaging features [3]. Table 1 summarizes the key demographic, clinical, serological, and radiological distinctions between MOGAD, NMOSD, and MS, highlighting the principal features that aid in differential diagnosis.

Due to the brief period since MOGAD was established as a distinct disease entity and the absence of international and regional patient registries, epidemiological data remain scarce compared with other CNS demyelinating disorders. Consequently, the incidence and prevalence in Poland and other Central European countries remains to be established. To date, the most comprehensive review on the epidemiological background of MOGAD was provided in 2023 by Hor and Fujihara, who analyzed seven large population-based studies from around the world [14]. According to their findings, the onset of MOGAD can occur across all age groups, with a median age at onset of 28–30 years, and there appears to be no clear female predominance in the overall gender distribution [14]. The estimated global prevalence of MOGAD was approximately 1.3–2.5 per 100,000 individuals, with the annual incidence ranging from approximately 3.4–4.8 per 1,000,000 individuals [14]. In the paediatric population below 18 years, MOGAD accounts for up to 35–40% of cases of CNS demyelinating attacks [14, 15]. While the disease onset can also manifest in individuals after the age of 50 years, being termed late-onset MOGAD, similarly to other CNS demyelinating disorders, older-onset groups have not yet received appropriate research focus. The epidemiological data on this subgroup remains limited, with only isolated cohort studies and case series reporting its prevalence and distinct clinical characteristics [16]. However, in all cases, it should be noted that the overall prevalence and incidence of MOGAD are expected to increase with rising disease recognition and availability of laboratory testing, including identification of patients with mildly symptomatic disease, monophasic disease course, and atypical clinical manifestations [3, 14]. Preliminary findings suggest that in 20–40% of cases, the onset of MOGAD is preceded by an infection or vaccination, and in contrast to MS and NMOSD, no definitive HLA associations have yet been consistently established [14, 17].

Table 1. Key distinguishing demographic, clinical, and MRI features of MOGAD, NMOSD, and MS [1,2,10–13]

	MOGAD	NMOSD	MS
Typical age of onset	Around 30	Around 40	Around 30
Paediatric onset	Common	Extremely rare	Rare
Gender distribution	Equal distribution in both genders	AQP4 –NMOSD: equal distribution in both genders AQP4 + NMOSD: female predominance	Female predominance
Precedent infection/ immunization	Common	Rare	Rare
Disease course	Relapsing or monophasic	Generally relapsing; a progressive course is extremely rare	Generally relapsing, progressive from onset, relapsing patient may develop secondary progression
Type of relapses	Commonly optic neuritis	Optic neuritis, longitudinally extensive transverse myelitis	Any, with relapse phenotype predicted by previous relapse phenotypes
Optic Neuritis	+++	+++	++
Transverse myelitis	++	+++	+++
Area postrema syndrome	Rare	++	Rare
Encephalopathy	++	Rare	Rare
Seizures	+	Rare	Rare
Focal neurological deficits	++	+/-	+++
Autoantibodies	MOG-IgG +; AQP4-IgG –	AQP4-IgG + in majority; AQP4-IgG – in seronegative NMOSD; MOG-IgG –	MOG-IgG –; AQP4-IgG –
CSF	Pleocytosis variable; OCBs rare	Commonly ≥ 50 WCC/mm^3; glial fibrillary acidic protein at relapse; OCBs in 10–25%	Commonly < 50 WCC/mm^3; OCBs in up to 95% of MS patients
MRI brain	'Fluffy-like' T2 hyperintense lesions in both white and deep grey matter; bilateral lesions at onset; periventricular infratentorial, juxtacortical lesions rare; common involvement of cerebellar peduncles	T2 hyperintense lesions around the 3rd and 4th ventricle and the aqueduct of Sylvius; hyperintense area postrema lesions; corticospinal tracts lesions, sometimes extensive white matter lesions	Presence of 'Dawson's fingers', ovoid periventricular, infratentorial, juxtacortical lesions
MRI spinal cord	Single or multiple longitudinally extensive lesions, occasionally shorter lesions; spinal conus often involved; central grey matter involvement	Single, longitudinally extensive lesion; common involvement of the entire transverse diameter of the spinal cord; conus rarely involved	Multiple short focal lesions; common peripheral involvement; spinal conus rarely involved

Abbreviations: AQP4 – aquaporin-4; **CSF** – cerebrospinal fluid; **MOG-IgG** – myelin oligodendrocyte glycoprotein immunoglobulin G; **MOGAD** – myelin oligodendrocyte glycoprotein antibody-associated disease; **MRI** – magnetic resonance imaging; **MS** – multiple sclerosis; **NMOSD** – neuromyelitis optica spectrum disorder; **OCBs** – oligoclonal bands, WCC white cell count

Table 2. The most common clinical manifestations of MOGAD, defined by the International MOGAD Panel, included in the 2023 proposed MOGAD diagnostic criteria [3–5,10,12,15,18–21]

Core clinical manifestation	Clinical presentation and distinguishing features
Optic neuritis	• The most common clinical presentation of MOGAD • Up to 50% of cases preceded by periorbital or frontotemporal headache • Uni- or bilateral involvement, with bilateral ON chracteristic of MOGAD • Relapses in 30-50% of patients, often unilateral • Severe vision loss with retroorbital pain • Optic disc oedema frequently visible on fundoscopic examination • Rapid improvement to full or near normal visual acuity following corticosteroid therapy
Transverse myelitis	• Moderate to severe deficits: sensory, motor, and sphincter involvement • Occurs as isolated clinical manifestation or in combination with ADEM or ON • Up to 10% of patients have no detectable spinal abnormalities on MRI at onset • EDSS score >4 in over 50% of patients during the acute phase • Quick motor recovery after treatment • Residual sphincter or erectile dysfunction may persist
ADEM	• Most frequent manifestation in pediatric population and rarely encountered in adults • Frequently preceded by an infectious prodrome (mostly respiratory) and fever • Symptoms include polyfocal neurological deficits with encephalopathy • Complete or almost complete recovery in most patients • Disease course monophasic in the majority of cases, with relapses in some patients presenting as MDEM
Brainstem or cerebellar deficits	• Rarely seen in isolation, mainly in combination with optico-spinal manifestations • Large MCP lesions with pons involvement characteristic of MOGAD • Symptoms may include ataxia, cranial nerve deficits, altered consciousness, hypoventilation, or weakness • Brainstem involvement may be a risk factor for higher disability in future
Cerebral cortical encephalitis	• Focal encephalitis with fever, headache, reduced consciousness, focal or generalized seizures • Seizures may be the sole clinical manifestation at onset • Signs of raised ICP may occur • Lesions localized mainly in cortical regions, often with FLAMES phenotype • In pediatric population may occur as dual seropositive autoimmune encephalitis, with concurrent NMDA receptor encephalitis
Cerebral mono-focal or multifocal deficits*	• Neurological deficits usually follow a subacute course, progressing from onset to peak over several hours to a few days • Initial MRI scans may be unremarkable in a subset of patients despite clear neurological deficits • Various presentation depending on the affected regions • Multifocal deficits without encephalopathy much more common in pediatric MOGAD, than in adults

Abbreviations: ADEM – Acute Disseminated Encephalomyelitis; **EDSS** – Expanded Disability Status Scale; **FLAMES** – Fluid-attenuated inversion recovery-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures; **ICP** – intracranial pressure; **MDEM** – multiphasic ADEM; **MCP** – middle cerebellar peduncle; **MOGAD** – myelin oligodendrocyte glycoprotein antibody-associated disease; **MRI** – magnetic resonance imaging; **MS** – multiple sclerosis; **NMDA** – N-methyl-D-aspartic acid; **NMOSD** – neuromyelitis optica spectrum disorder, **TM** – transverse myelitis.
*This broad category of deficits has been included in the 2023 proposed diagnostic criteria for MOGAD; includes all neurological deficits, with lesions characteristic of MOGAD confirmed on MRI scans

The clinical manifestations of MOGAD are diverse, but there are some combinations of symptoms, universal to most cases. MOGAD diagnostic criteria published by International MOGAD Panel in 2023 included optic neuritis, transverse myelitis, acute disseminated encephalomyelitis, cerebral monofocal or multifocal deficits, brainstem or cerebellar deficits, and cerebral cortical encephalitis, as core clinical features of MOGAD [5] (Tab. 2).

However, the symptomatic presentation of MOGAD varies greatly and may manifest as a combination of the above-mentioned syndromes or as other rare entities, including autoimmune or atypical encephalitis, aseptic meningitis, progressive leukodystrophies, peripheral nervous system syndromes, cranial neuropathies, and opsoclonus–myoclonus syndrome [20, 22, 23]. Importantly, clinical manifestation of MOGAD differs with age, while ON is the most common manifestation encountered in the adult population, whereas in the paediatric population, the most common first manifestation is an ADEM-like presentation, with or without accompanying ON [12]. Patients with late-onset MOGAD more often present with isolated optic neuritis, whereas other clinical manifestations, like ADEM and TM are less common in this group [24, 25]. Notably, while TM in younger-onset patients is typically characterized by a longitudinally extensive phenotype, older individuals are more likely to present with shorter lesions. In addition, elderly patients with MOGAD may more frequently exhibit white matter and periventricular abnormalities, which are thought to reflect the influence of comorbidities and age-related changes rather than the disease itself [24, 25].

Beyond the overview of radiological presentations, several imaging aspects deserve particular attention. The dynamic evolution of lesions on MRI during a single demyelinating attack is more frequently observed in MOGAD than in other CNS demyelinating diseases [28, 29]. The characteristic appearance and subsequent resolution of lesions within the same demyelinating attack on repeated MRI scans should prompt consideration of MOGAD in the differential diagnosis [29, 30]. Radiological lag, defined as a delay between the onset of symptom and the appearance of radiological abnormalities, appears to be more characteristic of MOGAD than of both MS and NMOSD. In a recent multicentre study, radiological lag was reported in approximately 10% of MOGAD patients, where the initial MRI, obtained a median of five days after symptoms onset, appeared normal despite clear neurological deficits on examination [29]. This feature was observed in both paediatric and adult patients, and intra-attack MRI changes were significantly more frequent in MOGAD, affecting 59% of patients, compared with 26% in MS and 21% in NMOSD [29]. Importantly, complete resolution of T2 abnormalities on MRI within weeks after a demyelinating attack has been described as a distinctive

Table 3. MRI characteristics of core clinical manifestations of MOGAD included in the 2023 International MOGAD Panel diagnostic criteria [4,12,15,18,20,26,27]

Core clinical manifestation	MRI imaging features
Optic neuritis	• T2 hyperintense lesions with optic nerve swelling • Gadolinium enhancement of the affected nerve on T1-weighted imaging • Lesions frequently bilateral, longitudinally extensive • Predominant involvement of the anterior part of the optic pathway • Optic nerve lesions accompanied by perineuritis with perineural or periorbital gadolinium enhancement
Transverse myelitis	• May present as LETM involving 3 or more vertebral segments, or as short-segment myelitis • In LETM cervical and/ or thoracic spinal cord mainly affected • Conus medullaris involvement frequent in MOGAD • Lesion visible in the axial plane as a T2 hyperintense 'H-sign' • Gadolinium enhancement poorly accumulated in the spinal lesions, detected in only 25% of cases
ADEM	• In paediatric ADEM multiple, diffuse, asymmetrical supra- and infratentorial T2 hyperintense lesions • Lesions in paediatric ADEM located in white matter, cortex or deep grey nuclei • Central vein sign rare • 'Open-ring' gadolinium enhancement uncommon • In adult MOGAD associated ADEM very few infratentorial or cortical T2 hyperintense lesions
Brainstem or cerebellar deficits	• Poorly demarcated, diffuse, bilateral, large T2 hyperintense lesions in brainstem or cerebellum • MS-like phenotype brainstem lesions uncommon in MOGAD • Characteristic bilateral pons and MCP involvement
Cerebral cortical encephalitis	• Isolated T2 hyperintense cortical lesions visible on FLAIR sequences • Lesions often present as FLAMES phenotype • Lesions mainly unilateral, rarely bilateral with leptomeningeal enhancement • Cortical hyperintensity may be the only MRI abnormality
Cerebral mono-focal or multifocal deficits*	• Isolated or multiple, poorly demarcated T2 hyperintense supra- and infratentorial lesions • Often located in the MCP, in supratentorial white matter or in cortical grey matter, or in deep grey nuclei

Abbreviations: MOGAD – myelin oligodendrocyte glycoprotein antibody-associated disease; **ADEM** – acute disseminated encephalomyelitis; **MS** – multiple sclerosis; **NMOSD** – neuromyelitis optica spectrum disorder; **MRI** – magnetic resonance imaging; **EDSS** – Expanded Disability Status Scale; **LETM** – Longitudinally extensive transverse myelitis; **MCP** – middle cerebellar peduncle; **FLAMES** – Fluid-attenuated inversion recovery-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures; **FLAIR** – fluid-attenuated inversion recovery.

*This broad category of deficits has been included in the 2023 proposed diagnostic criteria for MOGAD; includes all neurological deficits, with lesions characteristic of MOGAD confirmed on MRI scans.

hallmark of MOGAD, which is typically accompanied by substantial or even complete clinical recovery following immunotherapy [29–31].

Another notable observation involves optic nerve abnormalities on MRI. While Table 3 highlights the typical anterior, bilateral, and longitudinally extensive pattern of ON, it should be emphasized that optic perineuritis, defined by circumferential enhancement of the optic nerve sheath and adjacent orbital fat, constitutes a particularly suggestive radiological marker of MOGAD. This feature, documented in recent imaging studies [20, 26], is uncommon in other CNS demyelinating diseases.

On MRI, spinal cord lesions in MOGAD typically involve the central grey matter, producing the characteristic 'H-sign', although this feature may be overlooked on sagittal projections [20, 26]. Lesions may be short or multifocal and can occasionally mimic the patchy, peripheral distribution seen in MS [12]. Moreover, patients with late-onset MOGAD often display shorter and less extensive spinal cord involvement than younger individuals, a pattern likely shaped by age-related immune changes and comorbidities [24]. Cortical and subcortical brain lesions in MOGAD, observed during acute demyelinating episodes, are transient and do not leave chronic atrophic sequelae [27, 28]. In the supratentorial region, cortical and subcortical lesions predominate within the frontal and temporal lobes and may extend into juxtacortical white matter or deep grey nuclei, particularly the thalamus and basal ganglia. Infratentorial involvement most frequently affects the pons and middle cerebellar peduncles, occasionally extending to the cerebellar hemispheres or brainstem tegmentum [12, 20, 26].

In contrast, MS is characterized by numerous, sharply demarcated periventricular and juxtacortical plaques, frequently aligned along medullary veins in the typical 'Dawson's fingers' pattern, which may evolve into chronic T1-hypointense 'black holes' [28]. In NMOSD, by contrast, brain lesions preferentially localize around the third and fourth ventricles, the periaqueductal region, and the area postrema [32]. In MOGAD, leptomeningeal enhancement is often visible adjacent to the affected cortical regions, a radiological feature rarely encountered in either MS or NMOSD [26, 27]. A distinct cortical phenotype known as FLAMES (FLAIR-hyperintense Lesions in Anti-MOG-Associated Encephalitis with Seizures) represents a manifestation of cerebral cortical encephalitis associated with MOGAD. It is characterized by unilateral cortical hyperintensity, often with overlying leptomeningeal enhancement and seizures, and is considered highly suggestive of MOGAD [5, 33]. Although variants with bilateral or multifocal cortical lesions have been described, FLAMES remains relatively specific for MOGAD and may co-exist with other brain lesions or ON [20].

Relapse risk and disease course. MOGAD patients can experience either a monophasic or relapsing disease course [3]. The 2025 study by Trewin et al. analyzed data from 4,699 MOGAD patients compiled from studies conducted worldwide [4]. The authors noted that 47% of patients followed for 6–24 months experienced disease relapses, while this proportion increased to 72% among those observed for more than 5 five years [4]. Other prospective and retrospective studies have similarly reported relapse rates within the range of 40 – 83% of patients [3, 10]. In previous studies, several clinical and demographic risk factors for the disease relapsing course have been identified. One of the most consistent and widely established predictors for the future recurrence is the persistence of MOG-IgG seropositivity after initial treatment [34]. While seroconversion to seronegative status after initial treatment was shown to be heavily associated with monophasic disease course lower risk for future relapses [4, 35]. Early relapses within the first year of disease have

also been shown to predict a relapsing course in the long term [36]. In adults and children, bilateral ON has been repeatedly associated with an increased relapse risk compared to transverse myelitis, which as single manifestation or in combination with other systems involvement, more often has been associated with a monophasic disease course [4, 37]. In the paediatric population, ADEM-like phenotype was also shown to be largely associated with monophasic disease course [4].

Age at onset has been shown to influence the disease course on its own, with younger children, below 12 years, more often experiencing a monophasic trajectory and older children or adults being at higher risk of relapses [4]. In contrast to younger adults, individuals with late-onset MOGAD were shown to experience more disease course with fewer relapses [24]. The impact of age on disease course most likely stems from factors related both to immune system maturation in early life and to immune senescence later in adulthood.

Demographic factors are also becoming increasingly recognized. In the adult population, female gender and Hispanic ethnicity have emerged as risk factors for relapse, whereas children of white ethnicity seem comparatively less susceptible to recurrent attacks [38, 39]. Treatment-related factors also appear to play a significant role in shaping the course of the disease. Prolonged corticosteroid tapering after the demyelinating episode may delay early relapses, but long-term benefits are yet less consistent [4]. Studies suggest also that early initiation of immunotherapies, particularly with rituximab or intravenous immunoglobulin, is associated with reduced risk of relapses in both the paediatric and adult population [4, 12]. In contrast, while some studies found no clear protective effect of maintenance of oral corticosteroid, others reported a potential benefit [4, 37, 39]. However, given methodological variability, differences in management strategies between centres, and limited follow-up in most studies, conclusions regarding the impact of treatment on relapse risk and disability progression in MOGAD should be interpreted with caution.

Current studies suggest that at the time of disease onset, MOGAD patients generally face a lower long-term risk of accruing severe disability compared with NMOSD and MS, although disability still accumulates in a stepwise manner following relapses [40]. Progression independent of relapse activity (PIRA), a measure indicating disability accumulation between attacks and a hallmark of MS, is not observed in either MOGAD or NMOSD [41, 42]. Instead, disability accrual in MOGAD occurs only at the time of clinical attacks, leading to a stepwise worsening as measured by the Expanded Disability Status Scale (EDSS) [42]. Clinical and radiological data indicate that recovery after relapses is often substantial and may be complete or nearly complete, although persistent visual pathway damage could be detected even in some clinically asymptomatic patients [4]. In contrast, in NMOSD and MS relapses are typically more disabling, recovery is frequently incomplete, and disability milestones, such as EDSS 4 or 6, are reached earlier and after fewer relapses than in MOGAD [40, 43]. Several clinical and demographic factors have been associated with an increased risk of faster disability accrual in MOGAD. Higher age at onset, particularly above 50 years, markedly increases the probability of reaching disability milestones, such as EDSS 6 or 7, in comparison with younger onset populations [40, 44]. Furthermore, a higher EDSS score during the first attack has been linked with an elevated risk of sustained disability [43]. Finally, recurrent relapses remain the strongest predictor of long-term disability accumulation, as progression occurs more frequently after subsequent relapses than after the onset episode [42]. However, it should be emphasized that in most studies published to date, disability in MOGAD has been assessed using the EDSS, mainly because no other tools have yet been validated for measuring disability progression in either NMOSD or MOGAD [45]. The EDSS, although originally developed for MS, focuses mainly on motor manifestations and may underestimate disability progression in other functional systems that are more commonly affected in MOGAD. Consequently, its usefulness in MOGAD patients has recently increasingly been questioned [45]. To address this limitation, novel measures such as visual acuity testing, optical coherence tomography with peripapillary retinal nerve fibre layer thickness, as well as radiological markers including lesion burden and atrophy patterns on MRI, and patient-reported outcomes, better suited to capture the distinct disability profile of MOGAD, have been proposed for use in clinical studies [4]. Therefore, the interpretation of studies focusing on disability progression in CNS demyelinating diseases other than MS should, at present, be approached with considerable caution.

Treatment. The treatment of an acute demyelinating episode in MOGAD patients primarily relies on rapid commencement of immunosuppressive treatment aimed at limiting inflammatory tissue injury and preventing long-term disability progression [1, 3]. Current studies emphasize that immunosuppressive treatment should be initiated early on, at hospital admission, when a demyelinating attack is clinically and radiologically suspected, after appropriate serum and cerebrospinal fluid samples have been obtained for MOG-IgG testing [4, 12]. High-dose intravenous glucocorticoids currently remain the standard first-line treatment for acute attacks in MOGAD across most published studies, with intravenous methylprednisolone (IVMP) being the most widely-used and best-characterized among them [4, 46, 47]. The standard dosing regimen reported in most studies is 1,000 mg of IVMP daily for 3–5 days in adults, whereas in paediatric patients the dosage is typically weight-based at approximately 30 mg/kg/day for a similar period [3, 48]. Recent studies suggest that oral glucocorticoids can achieve outcomes comparable to intravenous therapy in some cases, especially in patients presenting with isolated ON [46, 48].

Post-attack oral glucocorticoid tapering is commonly reported across studies [46–48]. Studies suggest that maintaining oral glucocorticoid taper for at least 3 months may prolong time to first relapse and reduce the risk of early relapse [3, 12, 46]. In this regard, the management of MOGAD differs from that of MS, in which prolonged glucocorticoid tapering is generally avoided because of limited long-term efficacy, and concerns related to cumulative adverse effects [3, 12]. In patients presenting with severe demyelinating attacks or in cases with an unsatisfactory response to IVMP treatment, escalation to apheresis therapy is strongly recommended as a second-line therapeutic option [47]. Both plasmapheresis and immunoadsorption have demonstrated high effectiveness in patients with MOGAD, with plasmapheresis being more commonly used across studies. [46, 47]. Typical treatment protocols involve approximately 5–7 apheresis cycles administered within 7–10

Table 4. The 2023 International MOGAD Panel proposed diagnostic criteria [5]

For a diagnosis of MOGAD, criteria 1, 2, and 3 must be fulfilled			
1. Core clinical demyelinating event	Optic neuritis Myelitis ADEM Cerebral monofocal or polyfocal deficits Brainstem or cerebellar deficits Cerebral cortical encephalitis		
2. Positive MOG-IgG test	Serum cell-based assay	Clear positive [titer ≥ 1:100]	No additional supporting features required
		Low positive [titer ≥ 1:10 and < 1:100]	A and B must be true: A) AQP4-IgG seronegative B) one or more supporting clinical or MRI features ψ
		Positive without reported titer	
		Serum negative but CSF positive	
Ψ Supporting clinical and MRI features	Optic neuritis	• Simultaneous bilateral optic nerve involvement • Longitudinal optic nerve involvement [> 50% of the optic nerve length] • Perineural optic sheath enhancement • Optic disc oedema	
	Myelitis	• Longitudinally extensive myelitis • 'H-sign' or central cord lesion • Conus lesion	
	Brain, brainstem, or cerebral syndrome	• Multiple ill-defined T2 hyper-intense lesions in the supra-tentorial and infra-tentorial white matter • Cortical lesion with or without lesional and overlying meningeal enhancement • Ill-defined T2-hyperintensity involving medulla, pons, or middle cerebellar peduncle • Involvement of deep grey matter	
3. Exclusion of better diagnoses, including MS			

Abbreviations: MOGAD – Myelin oligodendrocyte glycoprotein-associated disease; **MOG** – antibody-associated disease; **ADEM** – acute disseminated encephalomyelitis; **MRI** – magnetic resonance imaging; **AQP4** – aquaporin 4; **CSF** – cerebral spinal fluid; **MS** – multiple sclerosis

days following glucocorticoid failure, and some studies have explored the use of concomitant apheresis therapy combined with non-glucocorticoid immunosuppressive agents [47].

Intravenous immunoglobulins (IVIG) have also been used in the acute management of MOGAD; however, as with apheresis therapy, they are generally considered a second-line or adjunctive option for patients in whom glucocorticoids are contraindicated, poorly tolerated, or insufficiently effective [3, 47]. IVIG use has been reported more commonly in the paediatric population, where safety considerations, ease of administration, and situations in which escalation to apheresis, is not readily feasible or clinically appropriate and often favour an IVIG-based approach [4, 12].

At present, maintenance management in patients with MOGAD remains substantially limited by several factors, most notably the absence of large-scale, long-term randomized controlled trials. Consequently, current evidence is derived mainly from retrospective observational cohorts, and treatment decisions are often influenced by local drug availability, as well as centre-specific experience and therapeutic preferences. Since maintenance treatment currently relies mainly on the off-label use of immunosuppressive agents, treatment regimens and dosing strategies are frequently adapted from other autoimmune and antibody-mediated CNS demyelinating diseases, which substantially affect both accessibility and standardization across centres [46]. Furthermore, there is a severe lack of clear biomarkers to guide the duration of treatment. While persistent serum MOG-IgG seropositivity has been used in previous studies as a rationale for prolonging maintenance therapy, the optimal timing and safety of treatment cessation, particularly in patients who undergo seroconversion, remain poorly defined [3]. Because of these limitations, and due to the scope constraints of the present article, a summary of the currently utilized therapeutic strategies in MOGAD maintenance is shown in Table 5.

Diagnostic criteria and caveats. Since MOGAD is a relatively newly characterized clinical entity, with varied clinical presentations and some overlap with other CNS inflammatory demyelinating diseases, the accurate diagnosis of MOGAD has posed a significant challenge. To address this challenge, in 2023, the International MOGAD Panel, based on an international consensus of experts and an extensive review of the available clinical, radiological, and serological data, proposed diagnostic criteria aimed at facilitating the diagnosis and management of MOGAD [5]. To date, the 2023 international diagnostic criteria for MOGAD have been validated in numerous studies in both paediatric and adult cohorts, consistently demonstrating high sensitivity and specificity, with particularly robust performance in children. They are now regarded as the methodological basis for case definition in both clinical and research contexts [6–9, 49] (Tab. 4).

A few important caveats regarding the interpretation of the 2023 diagnostic criteria should be noted. The 2023 International MOGAD Panel emphasized that diagnosis requires a combination of 3 factors: (1) a core clinical demyelinating event, (2) a positive MOG-IgG test result, and (3) the exclusion of better alternative diagnoses. Firstly, laboratory examinations play a crucial role in establishing a diagnosis of MOGAD, but they cannot stand alone. In this context, MOG-IgG testing should be performed only in patients whose clinical presentation is consistent with MOGAD, rather than used as a universal screening tool for all individuals with initial demyelinating attacks [5, 50]. Serum MOG-IgG testing remains the cornerstone, with serum being the preferred due to its higher sensitivity and specificity. CSF testing is not recommended for routine use due to substantially lower sensitivity and lack of internationally validated standards. Nevertheless, in selected cases, particularly when serum MOG-IgG is negative but clinical and radiological features strongly suggest MOGAD,

Table 5. Comparative overview of maintenance therapies currently used, described in the literature, or under clinical investigation for MOGAD [1,3,4,12,46–48]

Maintenance Therapy	Therapeutic Class	Mechanism of action	Clinical Use in MOGAD	Relative Effectiveness*	Main Disadvantages
Intravenous Immunoglobulin	Polyclonal immunoglobulin therapy	Modulation of Fc receptors, autoantibody neutralization, B/T-cell regulation	Relapsing course, refractory cases, or preferred first-line in children	High	High cost, requires frequent infusions, risk of thrombosis
Rituximab	Anti-CD20 monoclonal antibody	B-cell depletion	Standard maintenance therapy	Low to Moderate	Increased infection risk, infusion-related reactions, hypogammaglobulinemia, risk of PML
Azathioprine	Purine analogue	Inhibition of T- and B-cell proliferation	Standard oral maintenance therapy	Moderate	Hepatotoxicity, gastrointestinal intolerance, myelosuppression
Mycophenolate Mofetil	Inosine monophosphate dehydrogenase inhibitor	Inhibition of B- and T-cell proliferation	Standard oral maintenance therapy	Moderate	Increased infection risk, gastrointestinal intolerance, teratogenic
Methotrexate	Antimetabolite immunosuppressant	Folate pathway inhibition reducing B- and T-cell proliferation	Alternative oral maintenance therapy for mild-to-moderate relapsing MOGAD	Low to Moderate	Hepatotoxicity, pulmonary toxicity, gastrointestinal intolerance
Oral Glucocorticoids	Glucocorticoid therapy	Glucocorticoid receptor-mediated immunosuppression	Long-term low-dose maintenance therapy for steroid-dependent cases	High	Weight gain, osteoporosis, diabetes, iatrogenic Cushing's syndrome
IL-6R Inhibitors	Interleukin-6 receptor antagonist	IL-6 signaling inhibition	Refractory or relapsing MOGAD	Limited data, currently under clinical investigation	High cost, increased infection risk, neutropenia
Inebilizumab	Anti-CD19 monoclonal antibody	B-cell depletion	Refractory or relapsing MOGAD	Limited data, currently under clinical investigation	Increased infection risk, infusion-related reactions, hypogammaglobulinemia
Subcutaneous Ig	Polyclonal immunoglobulin therapy	Modulation of Fc receptors, autoantibody neutralization, B/T-cell regulation	Maintenance therapy in patients stable on IVIG or with poor intravenous access	Limited data, currently under clinical investigation	Injection-site reactions, requires frequent injections
Rozanolixizumab	Neonatal Fc receptor inhibitor	FcRn blockade reducing circulating IgG autoantibodies	Refractory or relapsing MOGAD	Limited data, currently under clinical investigation	Injection-site reactions, requires frequent injections, headache risk
Cyclophosphamide	Alkylating immunosuppressant	DNA alkylation causing T- and B-cell depletion	Rescue therapy for aggressive, life-threatening, or highly refractory MOGAD	Very high in severe refractory cases	Multiorgan toxicity, infertility risk, malignancy risk, severe myelosuppression

Abbreviations: FcRn – neonatal Fc receptor; **IgG** – immunoglobulin G; **IL-6R** – interleukin-6 receptor; **IVIG** – intravenous immunoglobulin; **MOGAD** – myelin oligodendrocyte glycoprotein antibody-associated disease; **PML** – progressive multifocal leukoencephalopathy.
*Due to the lack of large, long-term randomized controlled studies in MOGAD, the relative effectiveness of maintenance therapies cannot be definitively established. The estimates provided are based on extrapolation from observational data and expert consensus in the current literature and should be interpreted cautiously.

CSF positivity can provide supportive diagnostic evidence [5]. Secondly, serum cell-based assays using full-length human MOG are recommended as the gold laboratory standard [5, 50]. Interpretation must consider both MOG-IgG titers and the clinical probability of MOGAD, meaning the likelihood that a patient's clinical and radiological features are consistent with the disease. This consideration is crucial to minimize the risk of false-positive results, which are more likely to occur in individuals with atypical clinical presentations or low MOG-IgG titers [5]. In particular, low-positive MOG-IgG titers or results reported without a specified antibody titer are not sufficient on their own to establish a MOGAD diagnosis. In such cases, the criteria require the presence of at least one supporting clinical or MRI feature that is characteristic of MOGAD. These supporting features strengthen diagnostic certainty and help distinguish MOGAD from MS and NMOSD, particularly given that low serum titers are less reproducible across laboratories and more prone to represent false positives [5]. Finally, the timing of serum testing for MOG-IgG is crucial. Detection is most reliable when performed close to the first clinical manifestation and before the initiation of corticosteroids, plasma exchange, or other immunotherapies, as these can markedly reduce antibody detectability [51]. It should be noted that in many centres, because of the overlap between MOGAD and NMOSD, both MOG-IgG and AQP4-IgG are tested simultaneously to assist in differential diagnosis. A double-positive result is very rare, and when it does occur, AQP4-IgG titers are almost always at a high level, while MOG-IgG titers are low, and such patients manifest with the clinical presentation characteristic of NMOSD [5].

Limitations of the study. Several limitations should be acknowledged. First, although the review aimed to include the most comprehensive and up-to-date evidence, older studies published prior to 2023 were included only when they applied sufficiently transparent diagnostic criteria, and remained consistent with the currently employed International MOGAD Panel diagnostic criteria [5]. This could lead to potential omissions and authors' bias but was inherently necessary

to ensure diagnostic and methodological consistency, as inclusion of studies based on outdated or heterogeneous diagnostic standards would risk misclassification of patient populations and reduce the comparability and validity of the findings. Second, in some earlier studies, MOGAD may have been categorized as MOG-IgG–seropositive NMOSD, which may have confounded the interpretation of earlier data and warrants particular caution when comparing such findings. Additionally, differences in serological testing often limit comparability of findings across studies. When reported, the type of assay used to detect MOG-IgG varied across studies, with differences in sensitivity, specificity, and diagnostic cut-off thresholds. Furthermore, the timing of MOG-IgG testing in relation to demyelinating episodes or the initiation of immunotherapy was often inconsistently reported. As these factors substantially affect MOG-IgG detectability, particular caution is warranted when interpreting results across studies. Management standards also remain heterogeneous, which may affect the comparability of clinical outcomes across studies. In contrast to MS, comprehensive epidemiological data on MOGAD remains limited. Moreover, the limited number of randomized controlled trials evaluating treatment efficacy, together with variability in therapeutic approaches and outcome assessment methods across studies, complicates the interpretation of treatment effectiveness. Consequently, current therapeutic strategies and related conclusions are derived predominantly from observational and cohort studies, and should therefore be interpreted with caution.

CONCLUSIONS

Since the first reports of the disease in the early 2010s, substantial progress has been achieved in defining the clinical, radiological, and disability-related features of MOGAD. The publication of the 2023 International MOGAD Panel diagnostic criteria, together with the broadened access to more accurate diagnostic instruments in recent years, marked a major milestone in standardizing diagnostic approaches and enabling more consistent identification of MOGAD across diverse clinical settings and age groups. This review summarizes the most common clinical presentations, radiological features, disease courses, treatment methods and disability progression observed in MOGAD patients, with the aim of providing readers with an updated overview of the most recent findings and emerging data on this disease. It also compared MOGAD with MS and NMOSD, highlighting the key clinical and radiological distinctions among these entities, despite the evident clinical overlap. Future studies should strive to bridge the existing knowledge gaps by elucidating long-term disability outcomes, patterns of disease progression, and robust epidemiological indicators, including incidence, prevalence, and relapse rates.

REFERENCES

1. Ambrosius W, Michalak S, Kozubski W, et al. Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: Current Insights into the Disease Pathophysiology, Diagnosis and Management. Int J Mol Sci. 2020 Dec 24;22(1):100. https://doi.org/10.3390/ijms22010100
2. Rosenthal JF, Hoffman BM, Tyor WR. CNS inflammatory demyelinating disorders: MS, NMOSD and MOG antibody associated disease. J Investig Med. 2020 Feb;68(2):321–330. https://doi.org/10.1136/jim-2019-001126
3. Al-Ani A, Chen JJ, Costello F. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): current understanding and challenges. J Neurol. 2023 Aug;270(8):4132–4150. doi: 10.1007/s00415-023-11737-8. https://doi.org/10.1007/s00415-023-11737-8
4. Trewin BP, Brilot F, Reddel SW, et al. MOGAD: A comprehensive review of clinicoradiological features, therapy and outcomes in 4699 patients globally. Autoimmun Rev. 2025 Jan 3;24(1):103693. https://doi.org/10.1016/j.autrev.2024.103693
5. Banwell B, Bennett JL, Marignier R, et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. Lancet Neurol. 2023;22(3):268–282. https://doi.org/10.1016/S1474-4422(22)00431-8
6. Filippatou AG, Said Y, Chen H, et al. Validation of the international MOGAD panel proposed criteria: a single-centre US study. J Neurol Neurosurg Psychiatry. 2024 Aug 16;95(9):870–873. https://doi.org/10.1136/jnnp-2023-333227
7. Sondhi V, Desai N, Lingappa L, et al. Validation and Modified Application of the 2023 International Expert Consensus Criteria for Diagnosing Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease Among Children With Acquired Demyelination in Resource-Limited Regions. Pediatr Neurol. 2025 Aug 19;172:138–145. https://doi.org/10.1016/j.pediatrneurol.2025.08.010
8. Cai MT, Hua Y, Lai QL, et al. Performance of the 2023 diagnostic criteria for MOGAD: real-world application in a Chinese multicenter cohort of pediatric and adult patients. BMC Med. 2025 Jan 23;23(1):40. https://doi.org/10.1186/s12916-025-03875-9
9. Carta S, Sechi E, Dinoto A, et al. Real-Life Evaluation of the MOGAD Diagnostic Criteria: Application Challenges and Discrepancies. Neurol Neuroimmunol Neuroinflamm. 2025 Sep;12(5):e200456. https://doi.org/10.1212/NXI.0000000000200456
10. Wynford-Thomas R, Jacob A, Tomassini V. Neurological update: MOG antibody disease. J Neurol. 2019 May;266(5):1280–1286. https://doi.org/10.1007/s00415-018-9122-2
11. Fadda G, Flanagan EP, Cacciaguerra L, et al. Myelitis features and outcomes in CNS demyelinating disorders: Comparison between multiple sclerosis, MOGAD, and AQP4-IgG-positive NMOSD. Front Neurol. 2022 Nov 7;13:1011579. https://doi.org/10.3389/fneur.2022.1011579
12. Sechi E, Cacciaguerra L, Chen JJ, et al. Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD): A Review of Clinical and MRI Features, Diagnosis, and Management. Front Neurol. 2022 Jun 17;13:885218. https://doi.org/10.3389/fneur.2022.885218
13. Diem L, Hammer H, Hoepner R, et al. Sex and gender differences in autoimmune demyelinating CNS disorders: Multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD) and myelin-oligodendrocyte-glycoprotein antibody associated disorder (MOGAD). Int Rev Neurobiol. 2022;164:129–178. https://doi.org/10.1016/bs.irn.2022.06.011
14. Hor JY, Fujihara K. Epidemiology of myelin oligodendrocyte glycoprotein antibody-associated disease: a review of prevalence and incidence worldwide. Front Neurol. 2023 Sep 15;14:1260358. https://doi.org/10.3389/fneur.2023.1260358
15. Marignier R, Hacohen Y, Cobo-Calvo A, et al. Myelin-oligodendrocyte glycoprotein antibody-associated disease. Lancet Neurol. 2021 Sep;20(9):762–772. https://doi.org/10.1016/S1474-4422(21)00218-0
16. Dinoto A, Cacciaguerra L, Vorasoot N, et al. Clinical Features and Factors Associated With Outcome in Late Adult-Onset Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease. Neurology. 2025 May 27;104(10):e213557. https://doi.org/10.1212/WNL.0000000000213557
17. Grant-Peters M, Passos GRD, Yeung HY, et al. No strong HLA association with MOG antibody disease in the UK population. Ann Clin Transl Neurol. 2021 Jul;8(7):1502–1507. https://doi.org/10.1002/acn3.51378
18. Bartels F, Lu A, Oertel FC, Finke C, Paul F, Chien C. Clinical and neuroimaging findings in MOGAD-MRI and OCT. Clin Exp Immunol. 2021 Dec;206(3):266–281. https://doi.org/10.1111/cei.13641
19. Cobo-Calvo A, Ruiz A, Maillart E, et al. OFSEP and NOMADMUS Study Group. Clinical spectrum and prognostic value of CNS MOG autoimmunity in adults: The MOGADOR study. Neurology. 2018 May 22;90(21):e1858-e1869. https://doi.org/10.1212/WNL.0000000000005560
20. Li Y, Liu X, Wang J, et al. Clinical Features and Imaging Findings of Myelin Oligodendrocyte Glycoprotein-IgG-Associated Disorder (MOGAD). Front Aging Neurosci. 2022 Mar 15;14:850743. https://doi.org/10.3389/fnagi.2022.850743
21. Jurynczyk M, Messina S, Woodhall MR, et al. Clinical presentation and prognosis in MOG-antibody disease: a UK study. Brain. 2017 Dec

1;140(12):3128–3138. doi:10.1093/brain/awx276. Erratum in: Brain. 2018 Apr 1;141(4):e31. https://doi.org/10.1093/brain/awx276
22. Marignier R. Unusual presentations of MOG antibody-associated central nervous system demyelination: Expanding the spectrum. Mult Scler. 2019 Jan;25(1):128–129. https://doi.org/10.1177/1352458518804127
23. Du Y, Xiao L, Ding Z, et al. MOGAD Involving Cranial Neuropathies: A Case Report and Review of Literature. Brain Sci. 2022 Nov 11;12(11):1529. https://doi.org/10.3390/brainsci12111529
24. Huang Y, Luo W, Cheng X, et al. Clinical and imaging features of patients with late-onset myelin oligodendrocyte glycoprotein antibody-associated disease. Mult Scler Relat Disord. 2024 Feb;82:105405. https://doi.org/10.1016/j.msard.2023.105405
25. Mishan Y, Schwartz D, Elefant D, et al. Late-Onset MOGAD: A Case Series and Literature Review. Neuroimmunol Rep. 2025 Oct;100268. https://doi.org/10.1016/j.nerep.2025.100268
26. Shor N, Deschamps R, Cobo Calvo A, et al.; NOMADMUS study group. MRI characteristics of MOG-Ab associated disease in adults: An update. Rev Neurol (Paris). 2021 Jan-Feb;177(1–2):39–50. https://doi.org/10.1016/j.neurol.2020.06.016
27. Denève M, Biotti D, Patsoura S, et al. MRI features of demyelinating disease associated with anti-MOG antibodies in adults. J Neuroradiol. 2019;46(5):312–318. https://doi.org/10.1016/j.neurad.2019.06.001
28. Sechi E, Krecke KN, Messina SA, et al. Comparison of MRI Lesion Evolution in Different Central Nervous System Demyelinating Disorders. Neurology. 2021 Sep 14;97(11):e1097-e1109. https://doi.org/10.1212/WNL.0000000000012467
29. Cacciaguerra L, Abdel-Mannan O, Champsas D, et al. Radiologic Lag and Brain MRI Lesion Dynamics During Attacks in MOG Antibody-Associated Disease. Neurology. 2024 May 28;102(10):e209303. https://doi.org/10.1212/WNL.0000000000209303
30. Abdel-Mannan O, Champsas D, Tur C, et al.; UK-Childhood Neuroinflammatory Disease Network. Evolution of brain MRI lesions in paediatric myelin-oligodendrocyte glycoprotein antibody-associated disease (MOGAD) and its relevance to disease course. J Neurol Neurosurg Psychiatry. 2024 Apr 12;95(5):426–433. https://doi.org/10.1136/jnnp-2023-332542
31. Cacciaguerra L, Redenbaugh V, Chen JJ, et al. Timing and Predictors of T2-Lesion Resolution in Patients With Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease. Neurology. 2023 Sep 26;101(13):e1376-e1381. https://doi.org/10.1212/WNL.0000000000207478
32. Jarius S, Paul F, Weinshenker BG, et al. Neuromyelitis optica. Nat Rev Dis Primers. 2020 Oct 22;6(1):85. https://doi.org/10.1038/s41572-020-0214-9
33. Budhram A, Mirian A, Le C, et al. Unilateral cortical FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures (FLAMES): characterization of a distinct clinico-radiographic syndrome. J Neurol. 2019 Oct;266(10):2481–2487. https://doi.org/10.1007/s00415-019-09440-8
34. Oliveira LM, Apóstolos-Pereira SL, Pitombeira MS, et al. Persistent MOG-IgG positivity is a predictor of recurrence in MOG-IgG-associated optic neuritis, encephalitis and myelitis. Mult Scler. 2019 Dec;25(14):1907–1914. https://doi.org/10.1177/1352458518811597
35. Huda S, Whittam D, Jackson R, et al. Predictors of relapse in MOG antibody associated disease: a cohort study. BMJ Open. 2021 Nov 30;11(11):e055392. https://doi.org/10.1136/bmjopen-2021-055392
36. Chen B, Gomez-Figueroa E, Redenbaugh V, et al. Do Early Relapses Predict the Risk of Long-Term Relapsing Disease in an Adult and Paediatric Cohort with MOGAD? Ann Neurol. 2023 Sep;94(3):508–517. https://doi.org/10.1002/ana.26731
37. Satukijchai C, Mariano R, Messina S, et al. Factors Associated With Relapse and Treatment of Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease in the United Kingdom. JAMA Netw Open. 2022 Jan 4;5(1):e2142780. https://doi.org/10.1001/jamanetworkopen.2021.42780
38. Martin K, Srikanth P, Kanwar A, et al. Clinical and radiographic features of a cohort of adult and pediatric subjects in the Pacific Northwest with myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD). Mult Scler Relat Disord. 2024 Jan;81:105130. https://doi.org/10.1016/j.msard.2023.105130
39. Virupakshaiah A, Schoeps VA, Race J, et al. Predictors of a relapsing course in myelin oligodendrocyte glycoprotein antibody-associated disease. J Neurol Neurosurg Psychiatry. 2024 Dec 16;96(1):68–75. https://doi.org/10.1136/jnnp-2024-333464
40. Duchow A, Bellmann-Strobl J, Friede T, et al.; Neuromyelitis Optica Study Group (NEMOS). Time to Disability Milestones and Annualized Relapse Rates in NMOSD and MOGAD. Ann Neurol. 2024 Apr;95(4):720–732. https://doi.org/10.1002/ana.26858
41. Molazadeh N, Akaishi T, Bose G, et al. Progression independent of relapses in aquaporin4-IgG-seropositive neuromyelitis optica spectrum disorder, myelin oligodendrocyte glycoprotein antibody-associated disease, and multiple sclerosis. Mult Scler Relat Disord. 2023 Dec;80:105093. https://doi.org/10.1016/j.msard.2023.105093
42. Akaishi T, Misu T, Takahashi T, et al. Progression pattern of neurological disability with respect to clinical attacks in anti-MOG antibody-associated disorders. J Neuroimmunol. 2021 Feb 15;351:577467. https://doi.org/10.1016/j.jneuroim.2020.577467
43. Rojas JI, Pappolla A, Patrucco L, et al. Disability outcomes in NMOSD and MOGAD patients: data from a nationwide registry in Argentina. Neurol Sci. 2023 Jan;44(1):281–286. https://doi.org/10.1007/s10072-022-06409-w
44. Fan Y, Wang Z, Wu Y, et al. Fewer relapses and worse outcomes of patients with late-onset myelin oligodendrocyte glycoprotein antibody-associated disease. J Neurol Neurosurg Psychiatry. 2025 Jun 12;96(7):655–664. https://doi.org/10.1136/jnnp-2024-334613
45. Cortese R, Bianchi A, De Stefano N. It is time to see MOGAD from a different perspective than multiple sclerosis. Neurol Open Access. 2025;1(2). https://doi.org/10.1212/WN9.000000000000002
46. Schirò G, Iacono S, Salemi G, et al. The pharmacological management of myelin oligodendrocyte glycoprotein-immunoglobulin G associated disease (MOGAD): an update of the literature. Expert Rev Neurother. 024 Oct;24(10):985–996. https://doi.org/10.1080/14737175.2024.2385941
47. Misu T. Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: Pathophysiology, Clinical Patterns, and Therapeutic Challenges of Intractable and Severe Forms. Int J Mol Sci. 2025 Sep 2;26(17):8538. https://doi.org/10.3390/ijms26178538
48. Cacciaguerra L, Flanagan EP. Updates in NMOSD and MOGAD Diagnosis and Treatment: A Tale of Two Central Nervous System Autoimmune Inflammatory Disorders. Neurol Clin. 2024 Feb;42(1):77–114. https://doi.org/10.1016/j.ncl.2023.06.009
49. Varley JA, Champsas D, Prossor T, et al. Validation of the 2023 International Diagnostic Criteria for MOGAD in a Selected Cohort of Adults and Children. Neurology. 2024 Jul;103(1):e209321. https://doi.org/10.1212/WNL.0000000000209321
50. Budhram A, Flanagan EP. Testing for myelin oligodendrocyte glycoprotein antibodies: Who, what, where, when, why, and how. Mult Scler. 2025 Apr;31(5):505–511. https://doi.org/10.1177/13524585251313744
51. Forcadela M, Rocchi C, San Martin D, et al. Timing of MOG-IgG Testing Is Key to 2023 MOGAD Diagnostic Criteria. Neurol Neuroimmunol Neuroinflamm. 2023 Nov 17;11(1):e200183. https://doi.org/10.1212/NXI.0000000000200183