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MOGAD AS AN EMERGING CHALLENGE IN MODERN HEALTHCARE: FROM IMMUNOLOGY TO FUNCTIONAL RECOVERY

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ABSTRACT

Background. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a recently defined autoimmune demyelinating disorder of the central nervous system, characterized by antibodies against myelin oligodendrocyte glycoprotein (MOG).
Aim. The aim of this review is to systematize the most recent evidence regarding the MOGAD
Material and Methods. A systematic literature review was conducted through a comprehensive search of databases (via the PubMed and Google Scholar platforms)
Results. MOGAD may occur at any age, most frequently between 20 and 40 years, with variable epidemiological patterns. The clinical spectrum includes optic neuritis, transverse myelitis, acute disseminated encephalomyelitis (ADEM), cortical encephalitis, and brainstem or cerebellar involvement. Age-related phenotypic differences are observed, with ADEM predominating in children and optic neuritis being more common in adults. Biomarkers such as serum MOG-IgG titers, cerebrospinal fluid (CSF) leukocytosis, and oligoclonal band status may reflect disease activity and help predict relapse risk. Persistent seropositivity is associated with a higher likelihood of recurrence, whereas transient positivity correlates with a more favorable course. Acute management primarily includes high-dose corticosteroids, intravenous immunoglobulins (IVIG), and plasma exchange (PLEX). Long-term treatment involves immunomodulatory or immunosuppressive therapies, with oral corticosteroids and IVIG demonstrating the best balance of efficacy and tolerability.
Conclusions. MOGAD represents a distinct immunological entity with specific clinical, radiological, and serological features. Ongoing research continues to refine diagnostic criteria, biomarkers, and therapeutic strategies, improving disease recognition and management.

KEYWORDS

MOGAD, Anti- Mog, Mog Antibody Disease, Myelin Oligodendrocyte Glycoprotein Antibody

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Introduction

Myelin oligodendrocyte glycoprotein-associated disease (MOGAD) is an autoimmune disorder associated with antibodies against a glycoprotein found on the surface of myelinated oligodendrocytes. Myelin oligodendrocyte glycoprotein (MOG) is found exclusively in the central nervous system(Wynford-Thomas et al., 2019). Recently, MOGAD has been distinguished from multiple sclerosis (MS) and neuromyelitis optica spectrum disorders (NMOSD) with positive IgG antibodies against aquaporin 4 (AQP4)(Sechi et al., 2022). Due to the similarity of clinical symptoms and imaging findings, NMOSD and MOGAD were, until recently, diagnosed as MS. Research into MOG antibodies has been ongoing for several decades, suggesting that they play a role in inflammatory diseases of the central nervous system (CNS). This was confirmed when their presence was detected in serum and cerebrospinal fluid (CSF) in patients with multiple sclerosis (MS). Highly sensitive and specific methods for detecting MOG antibodies have recently been developed using cell-based assays, enabling the identification of patients with MOG antibodies (Wynford-Thomas et al., 2019). The course of the disease may be monophasic or relapsing, but recent studies indicate that in individuals under clinical observation for over 5 years, a relapsing course occurs in 72% of patients(Trewin et al., 2025). The disease is debilitating and may result in neurological disability—motor impairments, loss of visual acuity, and cognitive dysfunction (Andersen et al., 2025; Huda et al., 2021). Permanent disability develops in 50–80% of patients(Wynford-Thomas et al., 2019). Both MOGAD and NMOSD can present with optic neuritis and myelitis, but they differ in terms of pathophysiology, clinical features, biomarkers, and neuroimaging findings (Uzawa et al., 2024).

Materials and Methods

A systematic literature review was conducted through a comprehensive search of databases (via the PubMed and Google Scholar platforms). The following keywords were used:: „MOGAD", „MOG antibody", „Myelin oligodendrocyte glycoprotein antibody associated disease", „Myelin oligodendrocyte glycoprotein antibody"

History

In 1894, the French neurologist Eugène Devic described the case of a female patient with acute paralysis of the lower limbs and bilateral optic neuritis. During the post-mortem examination, demyelination of the optic nerves and spinal cord was observed, but no changes were noted in the brain. In the same year, Fernand Gault, a student of Devic's, published an analysis of this case in his doctoral thesis. The year 1894 is recognised as the first time the term NMO- inflammation of the optic nerve and spinal cord (Jarius & Wildemann, 2013; Sechi et al., 2022). It is now recognised that the case described by Devic may have been a case of MOGAD rather than NMO (neuro-optic myelitis). Both conditions share common features such as optic neuritis and myelitis, but the patient described had bilateral optic neuritis. Furthermore, it occurred almost simultaneously with inflammation of the spinal cord in the lumbar region, which suggests a diagnosis of MOGAD (Asseyer et al., 2020; Sechi et al., 2022; Uzawa et al., 2024).

In 2004, a study by Lennon et al. reported the presence of antibodies in patients with NMO that were absent in individuals with multiple sclerosis. It was later established that these antibodies target aquaporin-4 and were named AQP4-IgG. (Lennon et al., 2005). Over the years, it became evident that patients with AQP4-IgG may not meet the previous diagnostic criteria for NMO, which led to the introduction of a new definition—NMOSD. In 2015, experts published diagnostic criteria that also included seronegative NMOSD (Sechi et al., 2022; Wingerchuk et al., 2015). Research on the MOG protein began in the 1980s, when it was associated with autoimmune inflammation of the brain and spinal cord. In 2003, MOG-IgM was detected using the Western blot method and was considered a potential marker for predicting progression to multiple sclerosis. However, later studies showed that MOG antibodies are present not only in multiple sclerosis and other demyelinating diseases of the central nervous system, but also in healthy control groups.(Sechi et al., 2022; Wingerchuk et al., 2015). In 2007, it was demonstrated that MOG-IgG is present in patients with acute disseminated encephalomyelitis or optic neuritis, but not in those with multiple sclerosis. These antibodies were found in 30–70% of individuals with seronegative NMOSD, as well as in patients with demyelinating diseases of the central nervous system other than multiple sclerosis.(Sechi et al., 2022). In 2023, ECTRIMS recognized that the pathologies of multiple sclerosis, NMOSD, and MOGAD are distinct, and separate diagnostic criteria for MOGAD were established (Cacciaguerra & Flanagan, 2024).

Pathophysiology

As in many autoimmune diseases, the exact pathophysiology of MOGAD remains incompletely understood. The underlying mechanism involves an immune attack on oligodendrocytes mediated by MOG-IgG antibodies, leading to damage of the myelin sheath. Importantly, neurons and astrocytes are relatively spared, which may explain why patients with MOGAD generally have a more favorable prognosis compared to those with NMOSD or multiple sclerosis. Pathogenic antibodies—AQP4-IgG in NMOSD and MOG-IgG in MOGAD—are produced by peripheral B cells and predominantly circulate outside the central nervous system. The cytokine IL-6 is thought to facilitate their entry into the CNS by increasing the permeability of the blood–brain barrier.

MOG-IgG belongs to the IgG1 subclass and has the ability to activate the complement system; however, its exact role in MOGAD pathogenesis has not been clearly established. Some reports describe complement deposition around blood vessels and elevated complement levels within MOGAD lesions, but the evidence remains inconclusive.

Animal studies investigating CNS injury in MOGAD have yielded inconsistent results, partly because human MOG-IgG shows limited cross-reactivity with MOG proteins found in rodents (Höftberger et al., 2020; Takai et al., 2020).

In NMOSD, a central role is played by AQP4-IgG, which targets the aquaporin-4 water channel. This leads to activation of the complement cascade and, consequently, astrocyte damage. This process occurs because the anaphylatoxin C5a—a byproduct of complement activation—acts as a chemoattractant for granulocytes, which secondarily contributes to axonal loss and demyelination of surrounding tissues (Cacciaguerra & Flanagan, 2024; Sechi et al., 2022; Uzawa et al., 2024).

Epidemiology

The incidence and prevalence of MOGAD are difficult to determine, as clear diagnostic criteria for the disease were only established in 2023. According to available studies conducted in different countries, prevalence ranges from 0.51 to 3.42 per 100,000 individuals. Individual reports have estimated an incidence of 0.112 per 100,000 person-years in a cohort from Martinique and 0.48 per 100,000 person-years in Italy (Hor & Fujihara, 2023; Uzawa et al., 2024). Globally, the incidence of the disease is estimated at 6–34 new cases per million people per year, while prevalence is approximately 20 cases per million individuals (Banwell et al., 2023). To date, no significant association has been observed between geographic region or ethnic group and the incidence of MOGAD (Hor & Fujihara, 2023; Huda et al., 2021; Jurynczyk et al., 2017; Uzawa et al., 2024). MOGAD can occur at any age, but it is most commonly diagnosed between 20 and 40 years of age, in contrast to seropositive NMOSD, which typically presents in older patients (Cortese et al., 2023a; Duchow et al., 2024; Nakamura et al., 2023; Uzawa et al., 2024). Most studies report a higher prevalence of MOGAD in men. However, some reports suggest that the incidence may be similar between females and males (Banwell et al., 2023; Cobo-Calvo et al., 2018; Nagireddy et al., 2021). There are also studies indicating a higher incidence of the disease among women (Cortese et al., 2023b; Hor & Fujihara, 2023; Huda et al., 2021; Jurynczyk et al., 2017; Nakamura et al., 2023). Depending on the patient's age, different disease phenotypes predominate. For example, transverse myelitis and optic neuritis are more common in adults than in children. In contrast, children more frequently present with cortical encephalitis or acute disseminated encephalomyelitis (ADEM) (Banwell et al., 2023; Uzawa et al., 2024). MOGAD is typically associated with clinical phenotypes such as acute disseminated encephalomyelitis (ADEM), optic neuritis (ON), and transverse myelitis (TM), and less commonly with cortical encephalitis, or brainstem and cerebellar involvement (Andersen et al., 2025; Banwell et al., 2023). Among children, the most common phenotype is acute disseminated encephalomyelitis (ADEM), whereas in adults optic neuritis is the most frequently observed presentation (Andersen et al., 2025; Banwell et al., 2023; Spatola et al., 2023). Recent studies indicate that in patients followed for more than 5 years, a relapsing disease course occurs in 72% of cases (Trewin et al., 2025). It has been shown that in 20–57% of patients, an infection preceded the onset of MOGAD (Cobo-Calvo et al., 2018; Dubey et al., 2019; Ramanathan et al., 2018).

Disease Biomarkers

Among biomarkers of disease course, several are recognized: serial serum MOG-IgG status, serum MOG-IgG titer, cerebrospinal fluid (CSF) oligoclonal band status, white blood cell count in CSF, and total protein concentration in CSF.

In a 2025 review by Andersen et al., it was found that persistent MOG-IgG seropositivity across multiple serum measurements taken more than three months apart is associated with an increased risk of disease relapse. In contrast, transient seropositivity is linked to a lower risk of recurrence (Andersen et al., 2025).

The ability of biomarkers to distinguish between disease remission and relapse has also been evaluated. Biomarkers of disease activity include serum MOG-IgG titer, cerebrospinal fluid (CSF) white blood cell count, CSF protein concentration, and CSF oligoclonal band status.

Low or clearly positive serum MOG-IgG titers have been associated with disease relapse, whereas negative titers are linked to remission. The presence of leukocytosis in the CSF has been significantly associated with acute attacks, while CSF oligoclonal bands are more often associated with remission. At present, there are no validated reference ranges established for these biomarkers (Andersen et al., 2025).

Clinical Manifestations

The most common initial symptom in adults is optic neuritis, whereas in children—particularly those under 11 years of age—acute disseminated encephalomyelitis (ADEM) is most frequently observed, with or without associated optic nerve involvement (Banwell et al., 2023). In a study conducted in the United Kingdom, optic neuritis was reported in 55% of patients, with bilateral involvement in 24%. Isolated transverse myelitis occurred in 18% of cases, while longitudinally extensive myelitis was observed in 14% and short-segment involvement in 4%. Concurrent optic neuritis and transverse myelitis were found in 9% of patients, and acute disseminated encephalomyelitis (ADEM) or ADEM-like features were reported in 18% (Jurynczyk et al., 2017). Optic disc swelling is observed in 45–86% of patients with optic neuritis in the course of MOGAD, and in about half of these cases there is a reduction in visual acuity, which typically improves after acute treatment (Chen et al., 2018; Rempe et al., 2021). Symptoms such as nausea, vomiting, and hiccups have also been observed in 91% of patients during an attack, and in some cases they may occur prior to its onset (Jurynczyk

et al., 2017). Less common manifestations include cortical encephalitis, demyelinating attacks affecting the cerebellum and brainstem, cranial neuropathies, progressive white matter damage, tumefactive brain lesions, and central nervous system dysfunction associated with demyelinating changes (Banwell et al., 2023). Transverse myelitis is a common symptom in MOGAD, and it is estimated that more than half of patients experience moderate to even severe symptoms at the peak of an attack (Wegener-Panzer et al., 2020).

Disease Relapses

MOGAD can follow either a monophasic or a relapsing disease course (Armangue et al., 2020; Jurynczyk et al., 2017; Nakamura et al., 2023; Waters et al., 2020). In patients with long-term follow-up, the median time between disease onset and relapse is 6 months, and relapses occur in 50–70% of patients (Jurynczyk et al., 2017; Reindl & Waters, 2019). Among patients followed for more than 5 years, a relapsing disease course is observed in 72% of cases.

Most patients recover well; however, it is estimated that up to 45% of patients may be left with permanent disability (Ciron et al., 2020; Uzawa et al., 2024). In one study, severe visual impairment persisted in 16% of patients (Reindl & Waters, 2019; Uzawa et al., 2024). To date, the long-term prognosis has not been thoroughly investigated; however, residual disability in MOGAD is considered to be milder than in seropositive NMOSD.

The risk of recurrence is highest during the first year following the onset of the attack (Huda et al., 2021; Jurynczyk et al., 2017). It is believed that a reduced risk of relapse is associated with the TM attack phenotype, discontinuation of corticosteroids for more than one month, male gender, and onset of the disease after the age of 16 (Dubey et al., 2019; Jurynczyk et al., 2017; Trewin et al., 2025).

Diagnostic Criteria for MOGAD

The diagnostic criteria were published in 2023 by the International MOGAD Panel. Diagnosis primarily requires the exclusion of alternative conditions, including multiple sclerosis (MS). In addition, at least one core clinical demyelinating event is required together with a clearly positive serum MOG-IgG result.

If MOG-IgG is low, unknown, or negative in serum but positive in cerebrospinal fluid (CSF), diagnosis can still be made provided there is at least one core demyelinating event plus one characteristic clinical feature or MRI finding, along with a negative AQP4-IgG test result (Banwell et al., 2023; Uzawa et al., 2024).

Core demyelinating clinical events include optic neuritis, myelitis, acute disseminated encephalomyelitis (ADEM), cerebral deficits, brainstem or cerebellar deficits, and cortical encephalitis (often accompanied by seizures)

Clinical or MRI findings:

Optic neuritis, which may present as bilateral simultaneous involvement or longitudinal optic nerve lesions, with optic nerve sheath enhancement and swelling of the optic disc.

Myelitis, which may include central spinal cord lesions, the characteristic H-sign in the conus medullaris, or longitudinally extensive spinal cord involvement.

Brain involvement (including multiple ill-defined T2-hyperintense lesions in the supratentorial and often infratentorial white matter, deep grey matter involvement, cortical lesions with or without enhancement of the lesion and meninges, as well as poorly defined T2 signal abnormalities affecting the middle cerebellar peduncle, pons, or medulla oblongata) (Banwell et al., 2023).

Treatment

A survey conducted among practicing neurologists found that high-dose corticosteroids are the primary treatment for acute attacks. Intravenous administration of these drugs leads to a significant reduction in symptoms (in cooperation with the Neuromyelitis Optica Study Group (NEMOS) et al., 2016; Jurynczyk et al., 2017; Ramanathan et al., 2018; Trewin et al., 2024; Wong et al., 2018). One of the regimens recommended for treatment is 30 mg/kg per day or 1 g daily for 3–5 days (Andersen & Brilot, 2025). In the case of an acute attack, administration of intravenous immunoglobulins (IVIG) and/or plasma exchange (PLEX) is recommended (Hacohen & Banwell, 2019; Ramanathan et al., 2016). In a recent study, it was shown that early use of plasma exchange (PLEX) combined with disease-modifying therapy can help achieve remission and reduce the risk of permanent disability (Schwake et al., 2025). Stopping or reducing corticosteroid doses too quickly increases the risk of relapse (Andersen & Brilot, 2025). The proposed oral treatment and dosing regimen is 12.5 mg of prednisone daily for adults and 0.16 mg/kg/day for children for at least 3 months, or 60

mg daily for adults with a gradual taper of 5 mg per week over a total of 12 weeks (Andersen & Brilot, 2025; Trewin et al., 2024; Wolf et al., 2023).

In a meta-analysis, the effectiveness of several treatments was compared, including rituximab (RTX), mycophenolate mofetil (MMF), azathioprine (AZA), intravenous immunoglobulins (IVIG), oral corticosteroids (OC), cyclophosphamide (CTX), methotrexate (MTX), and disease-modifying therapies (DMT) (Wang et al., 2022). Immunoglobulin therapy was identified as the most effective option, followed by oral corticosteroids (OC). It was shown that azathioprine (AZA), mycophenolate mofetil (MMF), and rituximab (RTX) reduce the relapse rate and may therefore be used as alternative treatment options. Oral corticosteroids were among the most effective treatments and were associated with fewer adverse effects compared with other agents, except for immunoglobulin therapy.(Wang et al., 2022).

Conclusions

Over the past 20 years, demyelinating diseases of the central nervous system have been extensively studied, leading to the identification and characterization of disorders associated with antibodies other than those seen in multiple sclerosis (Flanagan, 2019; Lennon et al., 2004; O'Connor et al., 2007; Reindl & Waters, 2019; Sechi et al., 2022).

MOG-IgG is a key diagnostic criterion, together with the presence of compatible clinical manifestations (Andersen et al., 2025).

Despite their similarities, MOGAD and NMOSD differ significantly in terms of disease course, relapse prognosis, level of disability, and, most importantly, their clinical and radiological characteristics.

Serological status and MOG-IgG titers, as well as CSF leukocytosis, are biomarkers of disease activity, suggesting the value of regular serum monitoring. High MOG-IgG titers have been clearly associated with disease attacks, whereas low titers have been linked to remission (Andersen et al., 2025).

In the treatment of MOGAD, oral corticosteroids and intravenous immunoglobulins have shown the highest efficacy and the best tolerability (Wang et al., 2022).

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REFERENCES

1. Andersen, J., & Brilot, F. (2025). Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): Insights into pathogenesis and biomarkers of prognosis. *Seminars in Immunology, 78*, 101944. https://doi.org/10.1016/j.smim.2025.101944
2. Andersen, J., Trewin, B. P., Dale, R. C., Ramanathan, S., & Brilot, F. (2025). Biomarkers to predict relapse in myelin oligodendrocyte glycoprotein antibody-associated disease: A systematic review and meta-analysis. *Journal of Neurology, Neurosurgery & Psychiatry*. Advance online publication. https://doi.org/10.1136/jnnp-2025-337039
3. Armangue, T., Olivé-Cirera, G., Martínez-Hernandez, E., Sepulveda, M., Ruiz-Garcia, R., Muñoz-Batista, M., Ariño, H., González-Álvarez, V., Felipe-Rucián, A., Jesús Martínez-González, M., Cantarín-Extremera, V., Concepción Miranda-Herrero, M., Monge-Galindo, L., Tomás-Vila, M., Miravet, E., Málaga, I., Arrambide, G., Auger, C., Tintoré, M., ... Villar-Vera, C. (2020). Associations of paediatric demyelinating and encephalitic syndromes with myelin oligodendrocyte glycoprotein antibodies: A multicentre observational study. *The Lancet Neurology, 19*(3), 234–246. https://doi.org/10.1016/S1474-4422(19)30488-0
4. Asseyer, S., Cooper, G., & Paul, F. (2020). Pain in NMOSD and MOGAD: A systematic literature review of pathophysiology, symptoms, and current treatment strategies. *Frontiers in Neurology, 11*, 778. https://doi.org/10.3389/fneur.2020.00778
5. Banwell, B., Bennett, J. L., Marignier, R., Kim, H. J., Brilot, F., Flanagan, E. P., Ramanathan, S., Waters, P., Tenembaum, S., Graves, J. S., Chitnis, T., Brandt, A. U., Hemingway, C., Neuteboom, R., Pandit, L., Reindl, M., Saiz, A., Sato, D. K., Rostasy, K., ... Palace, J. (2023). Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. *The Lancet Neurology, 22*(3), 268–282. https://doi.org/10.1016/S1474-4422(22)00431-8
6. Cacciaguerra, L., & Flanagan, E. P. (2024). Updates in NMOSD and MOGAD diagnosis and treatment. *Neurologic Clinics, 42*(1), 77–114. https://doi.org/10.1016/j.ncl.2023.06.009
7. Chen, J. J., Flanagan, E. P., Jitprapaikulsan, J., López-Chiriboga, A. S. S., Fryer, J. P., Leavitt, J. A., Weinshenker, B. G., McKeon, A., Tillema, J.-M., Lennon, V. A., Tobin, W. O., Keegan, B. M., Lucchinetti, C. F., Kantarci, O. H., McClelland, C. M., Lee, M. S., Bennett, J. L., Pelak, V. S., Chen, Y., ... Pittock, S. J. (2018). Myelin oligodendrocyte glycoprotein antibody–positive optic neuritis: Clinical characteristics, radiologic clues, and outcome. *American Journal of Ophthalmology, 195*, 8–15. https://doi.org/10.1016/j.ajo.2018.07.020
8. Ciron, J., Cobo-Calvo, A., Audoin, B., Bourre, B., Brassat, D., Cohen, M., Collongues, N., Deschamps, R., Durand-Dubief, F., Laplaud, D., Maillart, E., Papeix, C., Zephir, H., Bereau, M., Brochet, B., Carra-Dallière, C., Derache, N., Gagou-Scherer, C., Henry, C., ... Marignier, R. (2020). Frequency and characteristics of short versus longitudinally extensive myelitis in adults with MOG antibodies: A retrospective multicentric study. *Multiple Sclerosis Journal, 26*(8), 936–944. https://doi.org/10.1177/1352458519849511
9. Cobo-Calvo, A., Ruiz, A., Maillart, E., Audoin, B., Zephir, H., Bourre, B., Ciron, J., Collongues, N., Brassat, D., Cotton, F., Papeix, C., Durand-Dubief, F., Laplaud, D., Deschamps, R., Cohen, M., Biotti, D., Ayrignac, X., Tilikete, C., Thouvenot, E., ... Desille-Dugast, M. (2018). Clinical spectrum and prognostic value of CNS MOG autoimmunity in adults: The MOGADOR study. *Neurology, 90*(21), e1858–e1869. https://doi.org/10.1212/WNL.0000000000005560
10. Cortese, R., Battaglini, M., Prados, F., Bianchi, A., Haider, L., Jacob, A., Palace, J., Messina, S., Paul, F., Wuerfel, J., Marignier, R., Durand-Dubief, F., De Medeiros Rimkus, C., Callegaro, D., Sato, D. K., Filippi, M., Rocca, M. A., Cacciaguerra, L., Rovira, A., ... Yousry, T. (2023a). Clinical and MRI measures to identify non-acute MOG-antibody disease in adults. *Brain, 146*(6), 2489–2501. https://doi.org/10.1093/brain/awac480
11. Cortese, R., Battaglini, M., Prados, F., Bianchi, A., Haider, L., Jacob, A., Palace, J., Messina, S., Paul, F., Wuerfel, J., Marignier, R., Durand-Dubief, F., De Medeiros Rimkus, C., Callegaro, D., Sato, D. K., Filippi, M., Rocca, M. A., Cacciaguerra, L., Rovira, A., ... Yousry, T. (2023b). Clinical and MRI measures to identify non-acute MOG-antibody disease in adults. *Brain, 146*(6), 2489–2501. https://doi.org/10.1093/brain/awac480
12. Dubey, D., Pittock, S. J., Krecke, K. N., Morris, P. P., Sechi, E., Zalewski, N. L., Weinshenker, B. G., Shosha, E., Lucchinetti, C. F., Fryer, J. P., Lopez-Chiriboga, A. S., Chen, J. C., Jitprapaikulsan, J., McKeon, A., Gadoth, A., Keegan, B. M., Tillema, J.-M., Naddaf, E., Patterson, M. C., ... Flanagan, E. P. (2019). Clinical, radiologic, and prognostic features of myelitis associated with myelin oligodendrocyte glycoprotein autoantibody. *JAMA Neurology, 76*(3), 301–309. https://doi.org/10.1001/jamaneurol.2018.4053
13. Duchow, A., Bellmann-Strobl, J., Friede, T., Aktas, O., Angstwurm, K., Ayzenberg, I., Berthele, A., Dawin, E., Engels, D., Fischer, K., Flaskamp, M., Giglhuber, K., Grothe, M., Havla, J., Hümmert, M. W., Jarius, S., Kaste, M., Kern, P., Kleiter, I., ... Neuromyelitis Optica Study Group. (2024). Time to disability milestones and annualized relapse rates in NMOSD and MOGAD. *Annals of Neurology, 95*(4), 720–732. https://doi.org/10.1002/ana.26858
14. Flanagan, E. P. (2019). Neuromyelitis optica spectrum disorder and other non–multiple sclerosis central nervous system inflammatory diseases. *Continuum, 25*(3), 815–844. https://doi.org/10.1212/CON.0000000000000742
15. Hacohen, Y., & Banwell, B. (2019). Treatment approaches for MOG-Ab-associated demyelination in children. *Current Treatment Options in Neurology, 21*(1), 2. https://doi.org/10.1007/s11940-019-0541-x

16. Höftberger, R., Guo, Y., Flanagan, E. P., Lopez-Chiriboga, A. S., Endmayr, V., Hochmeister, S., Joldic, D., Pittock, S. J., Tillema, J. M., Gorman, M., Lassmann, H., & Lucchinetti, C. F. (2020). The pathology of central nervous system inflammatory demyelinating disease accompanying myelin oligodendrocyte glycoprotein autoantibody. *Acta Neuropathologica, 139*(5), 875–892. https://doi.org/10.1007/s00401-020-02132-y
17. Hor, J. Y., & Fujihara, K. (2023). Epidemiology of myelin oligodendrocyte glycoprotein antibody-associated disease: A review of prevalence and incidence worldwide. *Frontiers in Neurology, 14*, 1260358. https://doi.org/10.3389/fneur.2023.1260358
18. Huda, S., Whittam, D., Jackson, R., Karthikeayan, V., Kelly, P., Linaker, S., Mutch, K., Kneen, R., Woodhall, M., Murray, K., Hunt, D., Waters, P., & Jacob, A. (2021). Predictors of relapse in MOG antibody associated disease: A cohort study. *BMJ Open, 11*(11), e055392. https://doi.org/10.1136/bmjopen-2021-055392
19. Jarius, S., Ruprecht, K., Kleiter, I., Borisow, N., Asgari, N., Pitarokoili, K., Pache, F., Stich, O., Beume, L.-A., Hümmert, M. W., Ringelstein, M., Trebst, C., Winkelmann, A., Schwarz, A., Buttmann, M., Zimmermann, H., Kuchling, J., Franciotta, D., ... Wildemann, B., in cooperation with the Neuromyelitis Optica Study Group. (2016). MOG-IgG in NMO and related disorders: A multicenter study of 50 patients. Part 2: Epidemiology, clinical presentation, radiological and laboratory features, treatment responses, and long-term outcome. *Journal of Neuroinflammation, 13*(1), 280. https://doi.org/10.1186/s12974-016-0718-0
20. Jarius, S., & Wildemann, B. (2013). The history of neuromyelitis optica. *Journal of Neuroinflammation, 10*(1), 8. https://doi.org/10.1186/1742-2094-10-8
21. Jurynczyk, M., Messina, S., Woodhall, M. R., Raza, N., Everett, R., Roca-Fernandez, A., Tackley, G., Hamid, S., Sheard, A., Reynolds, G., Chandratre, S., Hemingway, C., Jacob, A., Vincent, A., Leite, M. I., Waters, P., & Palace, J. (2017). Clinical presentation and prognosis in MOG-antibody disease: A UK study. *Brain, 140*(12), 3128–3138. https://doi.org/10.1093/brain/awx276
22. Lennon, V. A., Kryzer, T. J., Pittock, S. J., Verkman, A. S., & Hinson, S. R. (2005). IgG marker of optic-spinal multiple sclerosis binds to the aquaporin-4 water channel. *The Journal of Experimental Medicine, 202*(4), 473–477. https://doi.org/10.1084/jem.20050304
23. Lennon, V. A., Wingerchuk, D. M., Kryzer, T. J., Pittock, S. J., Lucchinetti, C. F., Fujihara, K., Nakashima, I., & Weinshenker, B. G. (2004). A serum autoantibody marker of neuromyelitis optica: Distinction from multiple sclerosis. *The Lancet, 364*(9451), 2106–2112. https://doi.org/10.1016/S0140-6736(04)17551-X
24. Nagireddy, R. B. R., Kumar, A., Singh, V. K., Prasad, R., Pathak, A., Chaurasia, R. N., Mishra, V. N., & Joshi, D. (2021). Clinicoradiological comparative study of aquaporin-4-IgG seropositive neuromyelitis optica spectrum disorder (NMOSD) and MOG antibody associated disease (MOGAD): A prospective observational study and review of literature. *Journal of Neuroimmunology, 361*, 577742. https://doi.org/10.1016/j.jneuroim.2021.577742
25. Nakamura, M., Ogawa, R., Fujimori, J., Uzawa, A., Sato, Y., Nagashima, K., Kuriyama, N., Kuwabara, S., & Nakashima, I. (2023). Epidemiological and clinical characteristics of myelin oligodendrocyte glycoprotein antibody-associated disease in a nationwide survey. *Multiple Sclerosis Journal, 29*(4–5), 530–539. https://doi.org/10.1177/13524585231156736
26. O'Connor, K. C., McLaughlin, K. A., De Jager, P. L., Chitnis, T., Bettelli, E., Xu, C., Robinson, W. H., Cherry, S. V., Bar-Or, A., Banwell, B., Fukaura, H., Fukazawa, T., Tenembaum, S., Wong, S. J., Tavakoli, N. P., Idrissova, Z., Viglietta, V., Rostasy, K., Pohl, D., ... Wucherpfennig, K. W. (2007). Self-antigen tetramers discriminate between myelin autoantibodies to native or denatured protein. *Nature Medicine, 13*(2), 211–217. https://doi.org/10.1038/nm1488
27. Ramanathan, S., Dale, R. C., & Brilot, F. (2016). Anti-MOG antibody: The history, clinical phenotype, and pathogenicity of a serum biomarker for demyelination. *Autoimmunity Reviews, 15*(4), 307–324. https://doi.org/10.1016/j.autrev.2015.12.004
28. Ramanathan, S., Mohammad, S., Tantsis, E., Nguyen, T. K., Merheb, V., Fung, V. S. C., White, O. B., Broadley, S., Lechner-Scott, J., Vucic, S., Henderson, A. P. D., Barnett, M. H., Reddel, S. W., Brilot, F., & Dale, R. C. (2018). Clinical course, therapeutic responses and outcomes in relapsing MOG antibody-associated demyelination. *Journal of Neurology, Neurosurgery & Psychiatry, 89*(2), 127–137. https://doi.org/10.1136/jnnp-2017-316880
29. Reindl, M., & Waters, P. (2019). Myelin oligodendrocyte glycoprotein antibodies in neurological disease. *Nature Reviews Neurology, 15*(2), 89–102. https://doi.org/10.1038/s41582-018-0112-x
30. Rempe, T., Tarhan, B., Rodriguez, E., Viswanathan, V. T., Gyang, T. V., Carlson, A., Tuna, I. S., & Rees, J. (2021). Anti-MOG associated disorder–clinical and radiological characteristics compared to AQP4-IgG+ NMOSD–a single-center experience. *Multiple Sclerosis and Related Disorders, 48*, 102718. https://doi.org/10.1016/j.msard.2020.102718
31. Schwake, C., Ladopoulos, T., Häußler, V., Kleiter, I., Ringelstein, M., Aktas, O., Kümpfel, T., Engels, D., Havla, J., Hümmert, M. W., Kretschmer, J. R., Tkachenko, D., Trebst, C., Ayroza Galvão Ribeiro Gomes, A. B., Pröbstel, A.-K., Korporal-Kuhnke, M., Wildemann, B., Jarius, S., Pul, R., ... Ayzenberg, I. (2025). Apheresis therapies in MOGAD: A retrospective study of 117 therapeutic interventions in 571 attacks. *Journal of Neurology, Neurosurgery & Psychiatry, 96*(7), 639–646. https://doi.org/10.1136/jnnp-2024-334863

32. Sechi, E., Cacciaguerra, L., Chen, J. J., Mariotto, S., Fadda, G., Dinoto, A., Lopez-Chiriboga, A. S., Pittock, S. J., & Flanagan, E. P. (2022). Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): A review of clinical and MRI features, diagnosis, and management. *Frontiers in Neurology, 13*, 885218. https://doi.org/10.3389/fneur.2022.885218
33. Spatola, M., Chuquisana, O., Jung, W., Lopez, J. A., Wendel, E.-M., Ramanathan, S., Keller, C. W., Hahn, T., Meinl, E., Reindl, M., Dale, R. C., Wiendl, H., Lauffenburger, D. A., Rostásy, K., Brilot, F., Alter, G., & Lünemann, J. D. (2023). Humoral signatures of MOG-antibody-associated disease track with age and disease activity. *Cell Reports Medicine, 4*(2), 100913. https://doi.org/10.1016/j.xcrm.2022.100913
34. Takai, Y., Misu, T., Kaneko, K., Chihara, N., Narikawa, K., Tsuchida, S., Nishida, H., Komori, T., Seki, M., Komatsu, T., Nakamagoe, K., Ikeda, T., Yoshida, M., Takahashi, T., Ono, H., Nishiyama, S., Kuroda, H., Nakashima, I., Suzuki, H., ... Fujimori, J. (2020). Myelin oligodendrocyte glycoprotein antibody-associated disease: An immunopathological study. *Brain, 143*(5), 1431–1446. https://doi.org/10.1093/brain/awaa102
35. Trewin, B. P., Brilot, F., Reddel, S. W., Dale, R. C., & Ramanathan, S. (2025). MOGAD: A comprehensive review of clinicoradiological features, therapy and outcomes in 4699 patients globally. *Autoimmunity Reviews, 24*(1), 103693. https://doi.org/10.1016/j.autrev.2024.103693
36. Trewin, B. P., Dale, R. C., Qiu, J., Chu, M., Jeyakumar, N., Dela Cruz, F., Andersen, J., Siriratnam, P., Ma, K. K. M., Hardy, T. A., Van Der Walt, A., Lechner-Scott, J., Butzkueven, H., Broadley, S. A., Barnett, M. H., Reddel, S. W., Brilot, F., Kalincik, T., & Ramanathan, S. (2024). Oral corticosteroid dosage and taper duration at onset in myelin oligodendrocyte glycoprotein antibody-associated disease influences time to first relapse. *Journal of Neurology, Neurosurgery & Psychiatry, 95*(11), 1054–1063. https://doi.org/10.1136/jnnp-2024-333463
37. Uzawa, A., Oertel, F. C., Mori, M., Paul, F., & Kuwabara, S. (2024). NMOSD and MOGAD: An evolving disease spectrum. *Nature Reviews Neurology, 20*(10), 602–619. https://doi.org/10.1038/s41582-024-01014-1
38. Wang, X., Kong, L., Zhao, Z., Shi, Z., Chen, H., Lang, Y., Lin, X., Du, Q., & Zhou, H. (2022). Effectiveness and tolerability of different therapies in preventive treatment of MOG-IgG-associated disorder: A network meta-analysis. *Frontiers in Immunology, 13*, 953993. https://doi.org/10.3389/fimmu.2022.953993
39. Waters, P., Fadda, G., Woodhall, M., O'Mahony, J., Brown, R. A., Castro, D. A., Longoni, G., Irani, S. R., Sun, B., Yeh, E. A., Marrie, R. A., Arnold, D. L., Banwell, B., Bar-Or, A., & Canadian Pediatric Demyelinating Disease Network. (2020). Serial anti–myelin oligodendrocyte glycoprotein antibody analyses and outcomes in children with demyelinating syndromes. *JAMA Neurology, 77*(1), 82–93. https://doi.org/10.1001/jamaneurol.2019.2940
40. Wegener-Panzer, A., Cleaveland, R., Wendel, E.-M., Baumann, M., Bertolini, A., Häusler, M., Knierim, E., Reiter-Fink, E., Breu, M., Sönmez, Ö., Della Marina, A., Peters, R., Lechner, C., Piepkorn, M., Roll, C., Höftberger, R., Leypoldt, F., Reindl, M., & Rostásy, K. (2020). Clinical and imaging features of children with autoimmune encephalitis and MOG antibodies. *Neurology: Neuroimmunology & Neuroinflammation, 7*(4), e731. https://doi.org/10.1212/NXI.0000000000000731
41. Wingerchuk, D. M., Banwell, B., Bennett, J. L., Cabre, P., Carroll, W., Chitnis, T., De Seze, J., Fujihara, K., Greenberg, B., Jacob, A., Jarius, S., Lana-Peixoto, M., Levy, M., Simon, J. H., Tenembaum, S., Traboulsee, A. L., Waters, P., Wellik, K. E., & Weinshenker, B. G. (2015). International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurology, 85*(2), 177–189. https://doi.org/10.1212/WNL.0000000000001729
42. Wolf, A. B., Palace, J., & Bennett, J. L. (2023). Emerging principles for treating myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD). *Current Treatment Options in Neurology, 25*(11), 437–453. https://doi.org/10.1007/s11940-023-00776-1
43. Wong, Y. Y. M., Hacohen, Y., Armangue, T., Wassmer, E., Verhelst, H., Hemingway, C., Van Pelt, E. D., Catsman-Berrevoets, C. E., Hintzen, R. Q., Deiva, K., Lim, M. J., Rostásy, K., & Neuteboom, R. F. (2018). Paediatric acute disseminated encephalomyelitis followed by optic neuritis: Disease course, treatment response and outcome. *European Journal of Neurology, 25*(5), 782–786. https://doi.org/10.1111/ene.13602
44. Wynford-Thomas, R., Jacob, A., & Tomassini, V. (2019). Neurological update: MOG antibody disease. *Journal of Neurology, 266*(5), 1280–1286. https://doi.org/10.1007/s00415-018-9122-2