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Systematic Review

Autoimmune diseases and cancers overlapping with myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): A systematic review

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Abstract

Background: Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) has various similarities with AQP4-IgG-seropositive Neuromyelitis Optica Spectrum Disorder (AQP4-IgG + NMOSD) in terms of clinical presentations, magnetic resonance imaging (MRI) findings, and response to treatment. But unlike AQP4-IgG + NMOSD, which is known to coexist with various autoimmune diseases and cancers, an association of MOGAD with these conditions is less clear.

Methods: We conducted a systematic search in PubMed, Scopus, Web of Science, and Embase based on the preferred reporting items for systematic reviews and meta-analysis (PRISMA). Duplicates were removed using Mendeley 1.19.8 (USA production) and the citations were uploaded into Covidence systematic review platform for screening.

Results: The most common autoimmune disease overlapping with MOGAD was anti-N-Methyl-D-Aspartate receptor encephalitis (anti-NMDAR-EN), followed by autoimmune thyroid disorders, and the most common autoantibody was antinuclear antibody (ANA), followed by AQP4-IgG (double-positive MOG-IgG and AQP4-IgG). A few sporadic cases of cancers and MOG-IgG-associated paraneoplastic encephalomyelitis were found.

Conclusion: Unlike AQP4-IgG + NMOSD, MOGAD lacks clustering of autoimmune diseases and autoantibodies associated with systemic and organ-specific autoimmunity. Other than anti-NMDAR-EN and perhaps AQP4-IgG + NMOSD, the evidence thus far does not support the need for routine screening of overlapping autoimmunity and neoplasms in patients with MOGAD.

Keywords: Myelin oligodendrocyte glycoprotein, autoimmune disease, autoantibody, neoplasm, paraneoplastic, systematic review

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Introduction

Myelin oligodendrocyte glycoprotein antibody disease (MOGAD) refers to a range of central nervous system (CNS) inflammatory conditions associated with the presence of autoantibodies against MOG. These conditions include acute disseminated encephalomyelitis (ADEM), optic neuritis (ON), or transverse myelitis (TM), as well as some cases of aquaporin-4 (AQP4)-IgG seronegative Neuromyelitis Optica Spectrum Disorder (NMOSD).[1] Despite the diversity of clinical and pathologic manifestations and disease outcomes, seropositive MOGAD is now regarded as a distinct entity among CNS inflammatory disorders linked together by immunity to MOG. Preliminary studies indicate that MOGAD has a more benign course and relatively better recovery from relapses in most patients compared to AQP4-IgG + NMOSD.[2]

Identifying associations among autoimmune diseases is important in patients with CNS demyelinating disorders because it may change the therapeutic strategies, monitoring approaches and intervals, and prognosis of both CNS inflammatory diseases and the co-existing autoimmune entity.[3] The underlying

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mechanisms of autoimmunity and autoantibody production in autoantibody-associated neurological syndromes are still unclear. One may postulate that a common immunologic defect may drive autoimmunity or autoantibody production in various organs, or that an existing autoimmune condition or malignancy may trigger additional defects in the immune system resulting in the development of new autoantibody or autoimmunity.[4]

One of the recognizable features of AQP4-IgG + NMOSD is the large overlap with other autoimmune conditions such as systemic lupus erythematosus (SLE), Sjogren's disease, autoimmune thyroid disorders (ATDs), and myasthenia gravis (MG).[5,6] In contrast, there is less known about the association of immunological comorbidities with MOGAD.

Published reports of co-existing autoimmunity in a large cohort of pediatric and adult patients with MOGAD and AQP4-IgG + NMOSD highlight the point that concomitant autoimmunity is less common in MOGAD when compared to AQP4-IgG + NMOSD.[3] Other comparative studies showed that antinuclear antibody (ANA) was positive in 43% of AQP4 + IgG NMOSD but only 7% of MOGAD patients.[7] Similarly, concomitant autoimmune disorders were observed in 45% and 11% of AQP4-IgG + NMOSD, and MOGAD patients, respectively.[8]

Herein, we systematically reviewed the literature for cases of autoimmune diseases, autoantibodies, neoplasms, and paraneoplastic syndromes that have been reported to overlap with MOGAD.

Methods

We conducted this systematic review to investigate any overlapping of MOGAD/positive MOG-IgG and other autoimmune diseases, autoantibodies, neoplasms, and paraneoplastic syndromes, based on the preferred reporting items for systematic reviews and meta-analysis (PRISMA).

Search strategy

We designed our systematic search syntax based on PRISMA from inception to July 2022, which included all mesh terms of "MOGAD," all autoimmune diseases, autoantibodies, neoplasms, and paraneoplastic syndromes (Supp. 1.) Our syntax was customized to match the four main systematic review databases including PubMed, Scopus, Web of Science, and Embase. The following results were imported to the desktop version of Mendeley 1.19.8 (USA production) to merge duplications and prepare for the screening. Two independent investigators (N.M. and G.B.) screened all publications to find probable appropriate studies by title and abstracts through Covidence systematic review software.[9] Disagreements in screening were resolved by debating. In addition, the gray literature and reference lists of all articles included in the search were reviewed to ensure no information was left out.

Study selection and data extraction

The data extraction table advanced by the variables including author, year, the number of cases, sex, age, clinical presentation, antibody testing assay and source, and notable clinical features and MRI findings. The inclusion criteria consisted of all studies (case reports, case series, observational studies, etc.) which reported at least one autoimmune disease, positive autoantibody (with or without disease presentation), neoplasm, or paraneoplastic syndrome, overlapping with MOGAD or positive MOG-IgG serostatus (either simultaneously or with an interval between them). On the other hand, exclusion criteria comprised papers without English abstracts, and all types of reviews, letters, and commentaries. N.M and G.B. separately extracted the included articles and disagreements resolved by the expert opinion (M.L).

Results

The PRISMA flow chart of this study is shown in Figure 1. Using the search strategy, 10,601 studies were identified however, 5664 studies remained after duplicate removes. After eliminating these duplicates, 296 and 343 studies assessed for full text eligibility by N.M. and I.L. independent screening. Our literature review showed that the most common autoimmune disease reported to overlap with MOGAD or positive MOG-IgG serostatus, is anti-NMDAR encephalitis (Anti-NMDAR-EN) (200 cases) (Table 1), followed by ATD (38cases), and SLE (35 cases) (Table 3). The most frequently reported autoantibodies overlapping with MOGAD were ANA (96 cases) (Table 3), and AQP4-IgG (70 cases of double-positive MOG-IgG and AQP4-IgG) (Table 2) respectively.

We found 9 cases of overlapping MOGAD and positive Glycine-Receptor (Gly-R)-antibody (Table 3), and one case of each of the following: double positive MOG-IgG and anti-NMDAR-EN with positive anti-contactin-associated protein-like 2 (CASPR2) antibody, glial fibrillary acidic protein (GFAP) astrocytopathy with double positive AQP4-IgG and MOG-IgG, MG with double positive AQP4-IgG and MOG-IgG, MOGAD with positive CASPR2-IgG

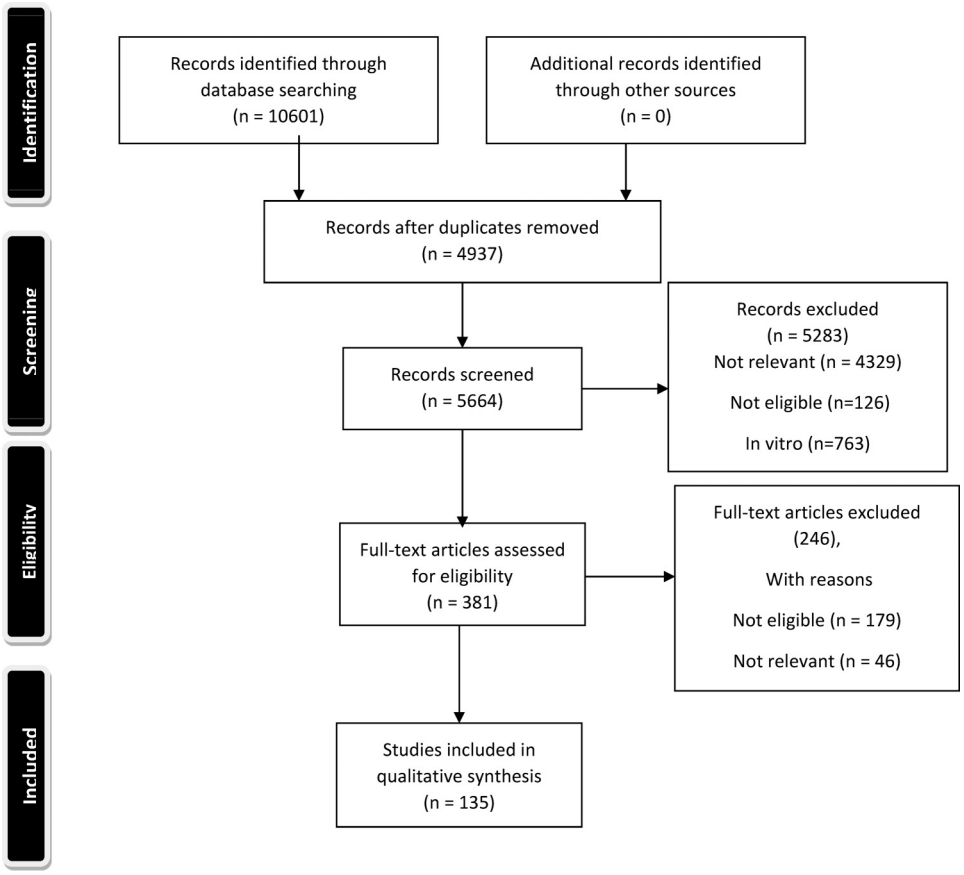

Figure 1. PRISMA flow chart of the study.

and leucine-rich glioma-inactivated 1 (LGI1) IgG, MOGAD with atopic dermatitis and asthma bronchiole, and MOG-ON with Sjogren's syndrome with positive Sjogren's syndrome antigen A antibody (SSA), ANA, and rheumatoid factor (RF).

A review of neoplasms and paraneoplastic syndromes associated with MOGAD showed five sporadic cases of cancers (T-cell lymphoma, lung, and colon) and three cases of MOG-IgG-associated paraneoplastic encephalomyelitis in the setting of malignancies of the ovary, lungs, and breast. Results of overlap between MOGAD and cancers/paraneoplastic syndromes are summarized in Table 4.

Discussion

To our knowledge, this is the first literature review of coexisting autoimmune diseases and cancers with MOGAD. Unlike AQP4-IgG + NMOSD, MOGAD lacks clustering of autoimmune diseases and autoantibodies associated with systemic and organ-specific autoimmunity.[3] Kunchok et al. showed that the frequency of any autoimmune diseases was 33.5% in AQP4-IgG + NMOSD adult-onset patients, and 12.2% in MOGAD adult-onset patients. These frequencies were 14.3% and 3.6% in pediatric-onset AQP4-IgG + NMOSD, and MOGAD patients, respectively.[3] This finding implies a difference in the immunopathogenic mechanisms involved in the development of each entity, such that AQP4-IgG + NMOSD is more attributable to a defect in immune tolerance, while MOGAD is more likely to be an aberrant autoimmune response to certain triggers, such as infection, vaccination, or malignancy.[3,146]

The major finding of our study was that the most frequent autoimmune disorder that has been reported to co-exist with MOGAD is anti-NMDAR-EN, which is a similar finding to previous studies.[53] Some studies have referred to this co-occurrence as "overlapping syndrome of MOGAD and anti-NMDAR-EN, (MNOS)."[19,51] In our literature review, we captured a total of 200 patients who either met the diagnostic criteria for autoimmune encephalitis and were simultaneously seropositive for both anti-NMDAR-IgG, and MOG-IgG or who met the diagnostic criteria

Table 1. Overlapping MOGAD/positive MOG-IgG and anti-NMDAR-encephalitis/positive anti-NMDAR-IgG.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
Titulaer et al. (2014)[10]	12	F/4 F/6 F/18 F/45 F/43 F/48 M/17 F/27 M/34 M/10 M/29 M/38	• MOGAD/positive MOG-IgG • CBA and IHC, serum and/or CSF	• Anti-NMDAR-EN • CBA and IHC, serum and/or CSF	Presentation of encephalopathy symptoms with clinical and/or MRI features of demyelination (ON, myelitis, BS dysfunction), and/or T2-W/FLAIR multifocal, infratentorial, or extensive abnormalities, suggesting involvement of the WM associated with MOG-IgG, either simultaneously or separate in time.
Hacohen et al. (2014)[11]	3	F/6 M/9 M/8	• Positive MOG-IgG • CBA, serum	• Anti-NMDAR-EN/positive anti-NMDAR-IgG • CBA, serum	Presentation of BON and/or ADEM in patients with positive MOG-IgG and anti-NMDAR-IgG (with or without anti-NMDAR-EN). Brain MRIs were normal in patients with ON, and showed bilateral deep GM and WM, and further BS lesions in second relapse in the patient with ADEM.
Kaneko et al. (2014)[12]	1	F/27	• Positive MOG-IgG • CBA, serum and CSF	• Anti-NMDAR-EN • CBA, serum, and CSF	Presentation of anti-NMDAR-IgG positive limbic encephalitis followed by demyelinating lesions.
Yokoyama et al. (2016)[13]	1	F/9	• Positive MOG-IgG • NA, serum, and CSF	• Anti-NMDAR-EN • CBA, serum, and CSF	Presented with fever, somnolence, hemiplegia, dysphagia, and memory impairment with high signal intensity in the left

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					temporal and parietal lobes on FLAIR MRI, started on steroids (IV followed by an oral taper), which was followed by recurrent ON with right optic nerve enhancing lesion on T1-W MRI, about 17 days after oral steroid cessation.
Kaneko et al. (2017)[14]	3	NA/NA	• Positive-MOG-Ab • CBA and IHC, serum	• Anti-NMDAR-EN • CBA and IHC, serum	
Zhou et al. (2017)[15]	1	M/31	• MOGAD (BS-EN, ON, ADEM) • Fixed CB-IIFT, serum	• Anti-NMDAR-EN • Fixed CB-IIFT, CSF	Cortical encephalitis initially presented with fever, headache, and seizure, with FLAIR MRI showing high-intensity lesions of right temporal, parietal, and occipital cortex, followed by the development of MOG-IgG-EN, BS-EN, ON, and ADEM-like illness successively, indicating demyelination. MRI revealed ADEM-like multiple lesions in the right midbrain, pons, left brachium points and juxtacortical WM, and a focal serpentine lesion in the left parietal cortex.
Nagata, et al. (2018)[16]	1	F/20	• MOG-ON • NA, CSF	• Anti-NMDAR-EN • NA, CSF	Presentation of seizures, disorientation, leg weakness with MRI lesions in the bilateral cingulate

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					gyrus and the superior frontal gyrus with enhancement in the upper part of the corpus callosum, followed by newly developed ring-enhancing lesion on the left side of the cingulate gyrus, hyperintense lesion spreading in the subcallosal area and the brainstem, and BON on STIR images.
Zhou et al. (2018)[17]	1	M/54	• MOG-EN • CB-IIFT, serum	• Anti-NMDAR-EN • CB-IIFT, CSF	Progressive unsteadiness and narcoleptic attacks followed by behavioral change and psychosis, without visual disturbances or seizures, with FLAIR findings of multiple high-intensity lesions involving the cerebellum, brainstem, thalamus, and third ventricular peri-ependymal region, consistent with demyelination.
Dubey et al. (2018)[18]	1	M/2	• MOG-ADEM • FACS, serum	• Anti-NMDAR-EN • CBA, CSF	Recurrent encephalitis, seizures, ON, and multifocal MRI abnormalities in the context of double positive MOG-IgG and anti-NMDAR-IgG.

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
Fan et al. (2018)[19]	5	F/3 M/23 M/6 M/25 M/9	• MOGAD • CB-IIFT, serum, and/or CSF	• Anti-NMDAR-EN • CB-IIFT, serum and/or CSF	Overlapping anti-NMDAR-EN occurred: (a) prior to episode of MOGAD (18 months) (b) simultaneously with MOGAD (c) after the episode of MOGAD (44 and 240 months) All had supratentorial lesions and three had infratentorial lesions on MRI.
Zhou et al. (2019)[20]	2	NA/NA	• MOGAD • CBA, serum	• Anti-NMDAR-EN/positive anti-NMDAR-IgG • NA, CSF	Initial presentation of CNS demyelinating syndrome, followed by acute mania and FLAIR MRI findings of extensive dorsal brainstem lesion. The second patient did not show encephalitic symptoms.
Taraschenko et al. (2019)[21]	1	F/10	• MOG-EN • CBA, serum	• Anti-NMDAR-EN • CBA, CSF	BON with enhancing lesions and perineural sheath swelling in bilateral optic nerves, followed by recurrent encephalitis with focal seizures.
Wang et al. (2019)[22]	6	NA/NA	• MOG-EN • CBA, serum	• Anti-NMDAR-EN/positive anti-NMDAR-IgG • CBA, CSF	Presentation of typical encephalitis symptoms including headache, decreased level of

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Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					consciousness, lethargy, irritability, memory, or cognitive decline with the most frequent MRI findings of cortical lesions on T2-W and FLAIR images.
Kaneko et al. (2019)[23]	1	F/28	• MOG-ADEM • CBA, serum and CSF	• Anti-NMDAR-EN • CBA, serum and CSF	Development of demyelinating MRI lesions, gait instability, and saccadic eye movements during exacerbation of previously diagnosed limbic encephalitis with positive anti-NMDAR-IgG.
Ren et al. (2019)[24]	4	NA/31.5 (median age)	• Positive MOG-IgG/ MOGAD • CBA, serum and/or CSF	• Anti-NMDAR-EN /positive anti-NMDAR-IgG • CBA, serum and/or CSF	Presentation of demyelinating symptoms associated with MOG-IgG in patients with previous diagnosis of anti-NMDAR-EN, or presentation of MOGAD with double positivity for MOG-IgG and anti-NMDAR-IgG, or development of psychiatric symptoms and limb weakness in double positive patient.
Kunchok et al. (2019)[25]	3	NA/NA	• MOGAD • CBA, CSF	• Positive-anti-NMDAR-IgG • CBA, CSF	
Aoe et al. (2019)[26]	1	F/36	• MOGAD • CBA, serum	• Anti-NMDAR-EN • CBA, CSF	Recurrent typical anti-NMDAR-EN coexisting with unusual symptoms such as balance

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					disability, and MRI findings of bilateral medial frontal cortical lesions, and Broca's aphasia caused by involvement of the Broca's area and lower part of the precentral gyrus, further found to be MOG-IgG-positive.
Rojc et al. (2019)[27]	1	M/47	• MOG-EM • Serum and CSF	• Anti-NMDAR-EN • Serum and CSF	Development of ON associated with MOG-IgG in a patient with history of recurrent ON who was diagnosed with anti-NMDAR-EN. (He was found to be MOG-IgG-positive in serum sample of previous ON attack when positive for anti-NMDAR-IgG, while having hyper-intense lesions in right frontoparietal WM on of T2-W and FLAIR MRI).
Kaneko et al. (2019)[28]	3	NA/NA	• Positive MOG-IgG • NA/NA	• Anti-NMDAR-EN • CBA and IHC, Serum and/or CSF	Anti-NMDAR-EN with overlapping MOG-IgG presenting with multifocal demyelination ($N = 1$) and meningoencephalitis ($N = 2$).
Sarigecili et al. (2019)[29]	1	M/6	• MOG-EN • IF, serum	• Anti-NMDAR-EN • IF, CSF	Clinical presentation of autoimmune encephalitis with simultaneous detection of antibodies against MOG and NMDAR. Cranial and

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					spinal MRI showed no signs of encephalomyelitis.
Gao et al. (2020)[30]	2	F/32 M/50	• MOG-EN • Serum and/or CSF	• Anti-NMDAR-EN • CBA, serum and/or CSF	Presentation of cortical encephalitis and subsequent demyelination in patients positive for both NMDAR-IgG and MOG-IgG. The clinical manifestations were different from pure MOGAD or anti-NMDAR-EN.
Amano et al. (2020)[31]	1	F/22	• Positive MOG-IgG • CBA, serum and CSF	• Anti-NMDAR-EN • CBA, CSF	Initial presentation of headache, aphasia, and right hemiparesis associated with left frontal cortical lesions on MRI. Recurrent anti-NMDAR-EN episodes with dominant MRI finding of leptomeningeal enhancement, in which MOG-IgG was retrospectively identified in the CSF and in the archived serum samples drawn in previous EN attacks.
Sakamoto et al. (2020)[32]	1	M/12	• MOG-EN • NA, CSF	• Anti-NMDAR-EN • NA, CSF	Presentation of psycho-behavioral symptoms in the first episode, followed by seizures, speech and writing difficulties, with abnormal MRI findings in the left frontal cortex, in the second episode.

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
Martinez-Hernandez et al. (2020)[33]	17	F ($N = 6$) M ($N = 11$)/ median age of symptoms onset: 38 (5–57)	• Positive MOG-IgG • IHC and CBA, serum and/or CSF	• Anti-NMDAR-EN • IHC and CBA, serum and/or CSF	Sixteen patients presented with anti-NMDAR-EN symptoms (two of them with additional BS-cerebellar symptoms, one limb numbness and weakness, and one pyramidal signs); and one patient with BON. FLAIR MRI abnormalities included cortical lesions in six, subcortical WM lesions in three, and meningeal enhancement in one patient.
Ma et al. (2020)[34]	1	M/12	• MOGAD • CBA, serum	• Anti-NMDAR-EN • CBA, serum and CSF	Development of emotional lability, irritability, insomnia, dysphagia, and MRI abnormalities with positive anti-MOG and anti-NMDAR antibodies, about 40 days of an initial presentation of fever, headache, loss of consciousness, and seizure with abnormal T2-W and FLAIR MRI findings (hyperintense signals in the left frontotemporal parietal occipital lobes and right temporal-parietal lobes) which was diagnosed with viral encephalitis.
Du et al. (2020)[35]	4	M/25 F/48	• MOGAD • IIF, serum and/or CSF	• Anti-NMDAR-EN • CBA, serum and/or CSF	Overlapping occurred sequentially in three and

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
		F/37 M/21			simultaneously in one patient.
Gong et al. (2020)[36]	11	F ($N=7$) M ($N=4$)/age of onset: 10.4 ± 2.3	• MOGAD • NA, NA	• Anti-NMDAR-EN • NA, NA	From 29 episodes of 11 patients with overlapping MOGAD and anti-NMDAR-EN, the common symptoms were convulsions, psychosis, lethargy, and the most frequent MRI findings were cortical focus, subcortical WM lesions, and BS lesions.
Zheng et al. (2020)[37]	1	NA/NA	• MOGAD • NA, NA	• Anti-NMDAR-EN • NA, CSF	One pediatric patient with anti-NMDAR-EN and optic nerve damage combined with positive MOG-IgG.
Wegener-Panzer et al. (2020)[38]	2	F/14 M/10	• MOG-EN • Live CBA, serum	• Positive anti-NMDAR-IgG/ Anti-NMDAR-EN • Commercially available biochips	
Zhu et al. (2020)[39]	2	NA/NA	• MOGAD • NA, NA	• Anti-NMDAR-EN • NA, NA	
Hou et al. (2020)[40]	7	M/4 F/7 F/6 F/5 F/6 F/8 M/6	• MOGAD/positive MOG-IgG • CBA, serum	• Anti-NMDAR-EN • CBA, CSF	Overlapping anti-NMDAR-EN occurred: (a) one year after recovery from MOG-ADEM (b) simultaneous with MOG-ADEM (c) Simultaneous with positive MOG-IgG The most frequent MRI findings were subcortical WM lesions

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					in bilateral frontal and parietal lobes, and patchy abnormal signal in bilateral basal ganglia region.
Perez et al. (2020)[41]	1	M/29	• MOGAD • CBA, serum	• Anti-NMDAR-EN • NA/CSF	Development of BON with positive MOG-IgG, and non-enhancing T2-W hyperintense lesions in the left thalamus and left medulla, mild enhancement of the optic chiasm, left intra-orbital optic nerve, and the optic tracts bilaterally, in a patient with previous episode of anti-NMDAR-EN presented with headaches, blurry vision, urinary retention, gait instability, slurred speech, fever, hypertension, and confusion.
Cherian et al. (2020)[42]	1	M/30	• MOG-EN • CBA, serum	• Anti-NMDAR-EN and positive Anti-CASPR2-Ab • CBA, serum and CSF	Presented with neuropsychiatric, cognitive symptoms, and long tract signs with a strong positivity of all three antibodies, and MRI findings of bilateral cingulate and hippocampal lesions.
Zhang et al. (2020)[43]	1	NA	• MOGAD • CBA, serum	• Positive anti-NMDAR-IgG • NA, serum and CSF	Clinical and MRI findings not specified, but reported to be more in line with ADEM than with anti-NMDAR-EN.

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
VJ Lopez et al. (2021)[44]	1	M/26	• MOGAD • NA, serum	• Anti-NMDAR-EN • NA, CSF	Development of behavioral changes (impaired executive function and memory, paranoia, and grandiose ideations), BON, nystagmus, pronator drift, and ataxia in the left arm, with MRI findings of multifocal, contrast-enhancing FLAIR hyperintensities involving the right pons, midbrain, thalami, and optic nerves.
Guzman et al. (2021)[45]	2	NA/NA	• MOG-EN • CBA, serum	• Positive anti-NMDAR-IgG • NA, CSF	
Weiss et al. (2021)[46]	1	F/20	• MOGAD • NA, serum	• Anti-NMDAR-EN • NA, CSF	Development of anti-NMDAR-EN and an overlapping demyelinating disorder with optic nerve involvement, gait disturbance, and MRI showing enhancing lesion in the dorsal column of the cervical spinal cord associated with anti- MOG-IgG, in a patient with a borderline personality disorder.
Cao et al. (2021)[47]	1	M/37	• MOGAD • CBA, serum and CSF	• Positive NMDAR-IgG • CBA, serum and CSF	Presentation of recurrent headaches with recent worsening, and seizures in a patient with a history of ON, and FLAIR MRI showing abnormal hyperintense signal in the bilateral frontal lobes,

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					cingulate cortex, and enhancement in meninges the parts above the lesions.
Ren et al. (2021)[48]	1	M/38	• MOGAD • CB-IIF, serum and CSF	• Anti-NMDAR-EN • CB-IIF, serum and CSF	Epileptic seizures following recurrent episodes of cross-sensory disturbance and dizziness with a demyelinating lesion in the right brainstem on MRI, followed by further relapses, progression, and new serpentine lesions with extensive involvement of cerebral cortex.
Shen et al. (2021)[49]	18	NA/NA	• Positive MOG-IgG • NA, NA	• Positive anti-NMDAR-IgG • NA, NA	Double-positive patients had more seizures, abnormal mental behavior, and visual impairment compared to MOG-IgG-positive group, and had more mental and behavioral abnormalities, and ataxia compared to anti-NMDAR-IgG positive group.
Fujimori et al. (2021)[50]	1	M/3	• MOG-EN • CBA, CSF	• Anti-NMDAR-EN • CBA, CSF	Relapsing anti-NMDAR-EN with initial MRI findings of bilateral medial frontal cerebral cortical lesions, and positive MOG-IgG at the same time.
Chen et al. (2021)[51]	5	F/63 M/41 F/18 M/19 M/31	• MOGAD • CBA, serum	• Anti-NMDAR-EN • CBA, CSF	Most frequent clinical presentations were headache, epileptic seizure, cognitive impairment, disturbance of consciousness, psychiatric

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					disorders, motor deficit, and involuntary movement. The dominant MRI findings were subcortex and cortical lesions, followed by lesions in basal ganglia and brainstem.
Teng et al. (2021)[52]	6	NA/NA	• MOG-EN • NA, NA	• Anti-NMDAR-EN • NA, NA	The most common clinical attack of the first and all MOGAD in children was ADEM, and the most common clinical syndrome was NMOSD. The most frequently involved areas in MRI were subcortical WM, cortex, and periventricular WM.
Nan et al. (2021)[53]	1	M/19	• MOG-EM • CBA, serum	• Anti-NMDAR-EN • CBA, CSF	First episode of anti-NMDAR-EN with positive CSF and negative serum anti-NMDAR-IgG and normal brain MRI (MOG-Ab not tested at the time), followed by a demyelinating episode (lethargy, facial pruritus, and right hemiplegia) with T2 and FLAIR hyperintense lesions in the left frontal lobe, basal ganglia, thalamus, and pons, and positive CSF and negative serum anti-NMDAR-IgG. MOG-IgG

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
					was positive in serum and negative in CSF at this time.
Caparo-Zamalloa et al. (2021)[54]	1	M/27	• MOGAD • IHC and CBA, CSF	• Anti-NMDAR-EN • IHC and CBA, CSF	Presentation of speech disorder, irritability, aggressive behavior, and disorientation followed by psychomotor agitation, seizure, generalized rigidity associated with ataxic gait and right hemiparesis in one month. T2-W/FLAIR MRI showed faint lesions in both middle cerebellar peduncles, midbrain and some supratentorial lesions, with no enhancement.
Guang et al. (2021)[55]	27	F ($N = 16$), M (N = 11)/ Median age at disease onset: 8.25 (5.44–9.86)	• Positive MOG-IgG • NA, CSF	• Anti-NMDAR-EN • NA, CSF	Overlapping cases showed milder conditions compared to patients with typical anti-NMDAR-EN and were more inclined to relapse.
Chen et al. (2021)[56]	3	NA/NA	• MOGAD • NA, cytometric beads array	• Positive anti-NMDAR-IgG • NA, cytometric beads array	
Li et al. (2021)[57]	3	NA	• MOGAD • CBA, serum and CSF	• Positive anti-NMDAR-IgG • NA, NA	Two patients presented with ON, and one with myelitis. None met the diagnostic criteria for anti-NMDAR-EN.
Jia et al. (2021)[58]	1	NA/NA	• Positive MOG-IgG • CBA, serum and/or CSF	• Anti-NMDAR-EN • CBA, serum and/or CSF	Presented with epilepsy and encephalopathy, where epileptic seizures were the prominent feature.
Wang et al. (2021)[59]	3	NA	• MOGAD/positive MOG-IgG	• Anti-NMDAR-EN • NA, serum and CSF	One patient presented with memory decline, seizures,

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
			• NA, serum		and multifocal neurological symptoms; the other two presented with acute encephalitic symptoms without focal neurological signs.
Han et al. (2022)[60]	1	M/3	• MOG-EN • NA, NA	• Positive Anti-NMDAR-IgG • NA, NA	Presented with drowsiness, generalized and focal seizures, abnormal behavior, aphasia, left arm weakness. Normal initial MRI, and development of diffuse brain atrophy in follow up MRI. MOG-IgG turned negative at month 37 of follow-up.
Wang et al. (2022)[61]	3	NA	• MOGAD • CBA, Serum	• Anti-NMDAR-EN/ Positive Anti-NMDAR-IgG • NA, Serum and CSF	
Lei et al. (2022)[62]	1	M/24	• MOGAD • CBA, Serum	• Positive Anti-NMDAR-IgG • NA, serum	Presented with recurrent encephalitis; also had ON during the first attack.
Guo et al. (2022)[63]	3	M/25	• MOGAD • NA, Serum	• Anti-NMDAR-EN • NA, Serum and CSF	A case of overlapping Anti-NMDAR-IgG and MOG-IgG associated refractory encephalitis mimicking the radiological characteristics of CLIPPERS.
Weihau et al. (2022)[64]	12	F ($N=9$), M ($N=3$), median age at disease onset: 8.6 (6.7–11.4)	• Positive MOG-IgG • CBA, serum	• Anti-NMDAR-EN • CBA, CSF	Patients with overlapping Anti-NMDAR-EN and MOG-IgG were more prone to relapse through the disease course.

(continued)

Table 1. Continued.

Reference	Number of cases	Sex/Age	• MOG-IgG-associated clinical phenotype or laboratory status • MOG-IgG testing assay, source (serum/CSF)	• Anti-NMDAR-IgG-associated clinical phenotype or laboratory status • Anti-NMDAR-IgG testing assay, source (serum/CSF)	Notable clinical features, or MRI findings
Yin et al. (2022)[65]	1	M/29	• MOGAD • NA, serum	• Anti-NMDAR-EN • NA, serum and CSF	Presentation of abnormal behavior and loss of consciousness, with MRI showing punctate abnormal signals in the left parietal lobe, followed by a demyelinating episode in one year, as of hoarseness and double vision, and MRI lesions in the medulla oblongata, left pons arm, left cerebellum and right midbrain, and thalamus.
Wang et al. (2022)[66]	1	M/36	• MOGAD • CBA, serum and CSF	• Anti-NMDAR-EN • IIFT, serum and CSF	Initial presentation of left facial numbness and slurred speech with FLAIR MRI showing lesions in left pontine and cerebral peduncle (MOG-IgG positive, NMDAR-IgG negative) followed by an episode of behavioral changes and psychosis with MRI showing deep white matter demyelinating lesions in the left lateral ventricle frontal angle and right lateral ventricle occipital angle (positive for both antibodies).

Abbreviations: MOGAD: myelin oligodendrocyte antibody associated disease; NMDAR: N-methyl-D-aspartate; MRI: magnetic resonance imaging; EN: encephalitis; CBA: cell-based-assay; IHC: immunohistochemistry; CSF: cerebrospinal fluid; ON: optic neuritis; BS: brainstem; T2-W: T2 weighted image; FLAIR: fluid-attenuated inversion recovery; WM: white matter; NA: not available, DS: demyelinating syndrome; BON: bilateral optic neuritis; ADEM: acute disseminated encephalomyelitis; GM: gray matter; T1-W: T1 weighted image; IIFT: indirect immunofluorescence test; STIR: short tau inversion recovery; FACS: fluorescence-activated cell sorting; EM: encephalomyelitis; IF: immunofluorescence; CASPR2: contactin-associated protein-like 2

Table 2. Overlapping MOGAD/positive MOG-IgG and AQP4-IgG + NMOSD.

References	Number of cases	Sex/age	Clinical phenotype or laboratory status	Antibody testing assay, source (serum/CSF), notable clinical features, or MRI findings
Mader et al. (2011)[67]	1	F/39	NMOSD	AQP4-IgG: live CBA, NA MOG-IgG: live CBA, NA NMOSD with 4 relapses of ON, TM, LETM, and no MRI cerebral lesions.
Kezuka et al. (2012)[68]	6	F/48 F/35 F/41 F/50 F/21 F/48	Unilateral or bilateral retrobulbar neuritis	AQP4-IgG: live CBA, serum MOG-IgG: ELISA, serum
Xu et al. (2012)[69]	1	F/60	NMOSD	AQP4-IgG: NA, serum MOG-IgG: NA, serum Multiple episodes of thoracic TM, ON with T2-hyperintense spinal cord lesion in T6-T9 and no typical MS lesions in the brain, initially misdiagnosed as MS and treated with IFNβ-1a, further found to be double positive for AQP-IgG and MOG-IgG.
Woodhall et al. (2013)[70]	1	F/34	NMOSD	AQP4-IgG: live CBA, serum MOG-IgG: live CBA, serum MOG-IgG and AQP4-IgG double-positive patients had significant disabilities. She had eight attacks over the disease duration of 12 years, EDSS = 9, progression index = 0.8, and no history of simultaneous ON + TM.
Cavus et al. (2013)[71]	1	NA/NA	NMOSD	AQP4-IgG: CBA, serum MOG-IgG: CBA, serum GlyR-IgG: CBA, serum Patients with simultaneous ON and TM were more prone to have antibodies against the Glycine receptor.
Martinez-Hernandez et al. (2014)[72]	2	NA/NA	NMOSD	AQP4-IgG: CBA, serum MOG-IgG: CBA, serum
Waters et al. (2015)[73]	11	NA/NA	Non-MS CNS demyelinating disease	AQP4-IgG: CBA, serum MOG-IgG: CBA, serum, using either full-length (FL-MOG) or the short-length (SL-MOG) human MOG-Ab. The SL-MOG assay detected Abs in 1 patient (low

(continued)

Table 2. Continued.

References	Number of cases	Sex/age	Clinical phenotype or laboratory status	Antibody testing assay, source (serum/CSF), notable clinical features, or MRI findings
				positive SL-MOG), who was strongly positive for AQP4-IgG.
Sepúlveda et al. (2015)[74]	2	NA/NA	NMOSD	AQP4-IgG: CBA, serum MOG-IgG: CBA, serum
Matsuda et al. (2015)[75]	2	F/41 F/48	NMOSD	AQP4-IgG: CBA IIF, serum MOG-IgG: CBA, serum From MOG-IgG-positive patients, 2 were AQP4-IgG-positive BON (NMO), of which one had 4 relapses with vision improvement after treatment with residual visual field deficit. The other one had eight relapses, no vision improvement after treatment, and residual visual field deficit.
Hoftberger et al. (2015)[76]	2	F/58 F/50	NMOSD	AQP4-IgG: CBA IIF, serum MOG-IgG: CBA, serum Patients with both antibodies presented with a classic NMO clinical picture of simultaneous BON and LETM.
Di Pauli et al. (2015)[77]	1	M/71	MOG-EMwith double positive AQP4-IgG and MOG-IgG	AQP4-IgG: Live CBA IIF, serum (AQP4 antibodies were absent at disease onset but seroconverted to low-titer positive at week nine with a titer of 1:40) MOG-IgG: Live CBA IIF, serum Presentation of MOG-EM with predominant optic and spinal involvement (acute visual and gait disturbance that dramatically worsened to bilateral amaurosis, tetraplegia, and respiratory insufficiency within a few days). MRI showed multiple supra- and infratentorial lesions with marked diffusion restriction, only slight hyperintensity on T2-W images, and intramedullary lesions (lesions marginally enhanced contrast).
Yan et al. (2016)[78]	10	F ($N = 10$)/median age at first attack: 32 (age range 15–60)	NMOSD	AQP4-IgG: FACS-based cell-binding assay, serum MOG-IgG: FACS-based cell-binding assay, serum The double-positive patients had a multiphase disease course with a high annual relapse rate, and severe residual disability compared to patients with only MOG-IgG. Of the double-positive patients, 70% had MS-like brain lesions, more severe edematous, multifocal regions on spinal MRI, pronounced decreases of retinal nerve fiber layer thickness and

(continued)

Table 2. Continued.

References	Number of cases	Sex/age	Clinical phenotype or laboratory status	Antibody testing assay, source (serum/CSF), notable clinical features, or MRI findings
				atrophy of optic nerves. 80% of double-positive patients had clinical evidence of spinal cord involvement at the first attack of NMOSD. The conus was significantly more likely to be involved in the double-positive group and MOG-IgG-positive patients.
Idiman et al. (2017)[79]	5	NA/NA	NMOSD with atypic aggressive demyelinating disease	AQP4-IgG: CBA, serum MOG-IgG: CBA, serum
Vural et al. (2017)[80]	2	NA/NA	NMOSD with positive MOG-IgG and MOG-IgM	AQP-IgG: NA, NA MOG-IgG: CBA and flow cytometry, serum
Yang et al. (2018[81]	1	F/41	GFAP astrocytopathy with double positive AQP4-IgG and MOG-IgG	AQP4-IgG: IIF, CSF MOG-IgG: IIF, serum Presented with SIADH and fever, vertigo, quadriplegia, dementia, and psychosis. Brain MRI showed lesions in bilateral thalamus, right hypothalamus and midbrain, left hippocampus, right parietal, frontal lobe, and T1-W "radial enhancing" lesions in the cerebellum. Spinal cord MRI was normal.
Kunchok et al. (2019)[25]	6	NA/NA	CNS inflammatory demyelinating disorders	AQP4-IgG: live CBA, serum MOG-IgG: live CBA, serum All double-positive cases had high titer AQP4-IgG and low titer MOG-IgG.
Kunchok et al. (2020)[82]	10	F ($N=9$) M ($N=1$)/median age: 47 (age range: 30–61 years)	CNS inflammatory demyelinating disorders	AQP4-IgG: flow cytometric assay, serum MOG-IgG: flow cytometric assay, serum All 10 patients with dual positivity had high-titer AQP4-IgG (median, 1:10 000; range, 1:100 to 1:100 000) and low-titer MOG-IgG (median, 1:40; range, 1:20 to 1:100).
Ishikawa et al. (2019)[83]	1	F/24	Unilateral or bilateral optic neuritis	AQP4-IgG: IIF, serum MOG-IgG: CBA, serum Presented with right ON at age 13, followed by left ON at age 18 (positive AQP4-IgG), and left ON at age 22 (positive MOG-IgG).
Bates et al. (2020)[84]	1	F/54	Myasthenia gravis (MG) with double-positive AQP4-IgG and MOG-IgG	AQP4-IgG: CBA, NA MOG-IgG: CBA, NA Presentation of acute right-sided weakness in a patient

(continued)

Table 2. Continued.

References	Number of cases	Sex/age	Clinical phenotype or laboratory status	Antibody testing assay, source (serum/CSF), notable clinical features, or MRI findings
				with long-standing MG.MRI showed longitudinally extensive edema beginning at the level of T1-T2 and extending up into the obex and area postrema with associated contrast enhancement of levels C1-C4.
Mori et al. (2020)[85]	1	F/NA	NMOSD	AQP4-IgG: NA MOG-IgG: NA Presentation of two episodes of right ON and two episodes of left ON resulting in severe visual function disability.
He et al. (2020)[86]	1	NA	NMOSD	AQP4-IgG: ELISA or IIFT, NA MOG-IgG: IIFT, NA
Zhang et al. (2021)[87]	1	F/79 months	NMOSD	AQP4-IgG: commercial kit, serum (assay not specified) MOG-IgG: commercial kit, serum (assay not specified)
Mason et al. (2021)[88]	1	F/66	Isolated sequential bilateral optic neuritis	AQP4-IgG: NA, serum MOG-IgG: NA, serum MRI of the brain and orbits revealed enhancement of the left optic nerve with no cerebral WM plaques.

Abbreviations: NMOSD: neuromyelitis optica spectrum disorder; CBA: cell-based assay; ON: optic neuritis, TM: transverse myelitis; LETM: longitudinally extensive transverse myelitis; MRI: magnetic resonance imaging; ELISA: enzyme-linked immunosorbent assay; MS: multiple sclerosis; EDSS: expanded disability status scale; GlyR-Ab: glycine receptor antibody; FL-MOG: full-length human MOG-Ab; SL-MOG: short-length human MOG-Ab; BON: bilateral optic neuritis; EM: encephalomyelitis; FACS: fluorescence-activated cell sorting; IIF: indirect immunofluorescence; MG: myasthenia gravis.

Table 3. Overlapping MOGAD/positive MOG-IgG and other autoimmune diseases and/or autoantibodies.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies	Notable clinical features, or MRI findings
Mader et al. (2011)[67]	2	NA/NA	• Positive MOG-IgG • Live cell staining IIF assay	SLE	
Mader et al. (2018)[89]	3	NA/NA	• Positive MOG-IgG • Live CBA, serum	Demyelinating or non-demyelinating NPSLE	All patients with demyelinating NPSLE met the ACR 1999 criteria for demyelination, and all had either TM occurring at 2 different time points or TM with WM demyelination or ON occurring at two different time points.
Bilodeau et al. (2019)[90]	1	F/32	• MOG-LETM • FACS, NA	SLE	Presented with severe TM (paraplegia) shortly after SLE onset in the post-partum period. FLAIR MRI showed lesions in medulla, pons, hypothalamus, subcortical WM, and T2-hyperintense lesions adjacent to the third and fourth ventricle. Spinal MRI showed LETM in the entire cervical and thoracic cord.
Pröbstel et al. (2019)[91]	14	M/34 F/33 F/61 F/35 F/63 F/49 F/51 F/34 M/54 F/26 F/35 F/31 F/25 M/40	• Positive MOG-IgG • CBA, serum	NPSLE ($N=6$) and non-NPSLE ($N=8$) ANA Anti-ds-DNA Anti-Phospholipid Anti-Sm Anti SSA/SSB	Presentation of visual disturbance, bladder dysfunction, seizure, and psychosis.
Seth et al. (2020)[92]	11	NA/NA	• Positive MOG-IgG • ELISA, serum	NPSLE ($N=8$) and non-NPSLE ($N=3$)	MOG-IgG was positive in a significantly higher proportion of patients having mood disorder as compared to patients without it.

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies		Notable clinical features, or MRI findings
Chawla et al. (2021)[93]	1	F/33	• MOG-LETM • NA, NA	Development of MOG-LETM in a patient with SLE, during active COVID-19		Presents with numbness and weakness in bilateral lower extremities with associated diplopia, urinary retention, 10 days after diagnosis of COVID-19 infection.T2-FLAIR brain MRI showed hyperintense lesions in the superior and middle cerebellar peduncles. MRI of spine showed LETM involving C4-C7 and T11-T12.
Kunchok et al. (2019)[25]	51	NA/NA	• MOGAD • CBA, serum	ANA Ds-DNA ENA Anti-CASPR2-IgG and LGI1-IgG antibodies GABA-A-R-IgG ATD RA Vitiligo Sjogren Pernicious anemia APS	$N=17$ $N=3$ $N=7$ $N=1$ $N=1$ $N=14$ $N=1$ $N=1$ $N=1$ $N=1$ $N=1$	
Kunchok et al. (2021)[3]	38 (from total of 170 MOGAD patients)	NA/NA	MOGAD Either flow cytometric assay or CBA	ANA ds-DNA Anti-Sm RNP RA SLE ATD Sjogren	$N=13$ $N=5$ $N=1$ $N=3$ $N=2$ $N=1$ $N=12$ $N=1$	
Malli et al. (2021)[94]	17	NA/NA	• MOGAD • IIF	ANA ATD/thyroid Autoantibody Atopic dermatitis	$N=9$ $N=5$ $N=4$	All autoantibodies: IIF, Presenting with NMOSD, recurrent TM, recurrent ON, and ADEM.
Jarius et al. (2016)[95]	19	NA/NA	• MOGAD • CBA, serum and/or CSF	ANA ANCA	$N=14$ $N=1$	

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies		Notable clinical features, or MRI findings
				ACA or B2GP	$N=2$	
				RF	$N=1$	
				anti-TPO	$N=2$	
				anti-TG	$N=1$	
				Anti-TSHR (perinuclear)	$N=1$	
				tTg-IgA	$N=1$	
				RA	$N=2$	
				ATD	$N=2$	
				Atopic dermatitis	$N=1$	
				Asthma bronchiole	$N=1$	
Ramanathan et al. (2018)[96]	4	NA/NA	• MOGAD • CBA, serum	Type 1 DM and Hashimoto's thyroiditis	$N=1$	
				Elevated thyroid Abs	$N=1$	
				Henoch-Schönlein purpura	$N=1$	
				Anti-LGI1 Abs	$N=1$	
Sato et al. (2014)[7]	5	M/29 F/15 M/49 M/70 M/38	• MOGAD • CBA, serum	ANA	$N=1$	
				Anti-SSA	$N=1$	
				Anti-TG	$N=1$	
				Anti-TPO	$N=1$	
Zhou et al. (2019)[20]	6	NA/NA	• MOGAD • CBA, serum	ANA	$N=5$	
				Anti-TPO	$N=1$	
Veselaj et al. (2021) [97]	7	F/25 F/21 M/66 F/40 F/58 F/17 M/55	• MOG-EM • CBA, serum	ANA	$N=3$	
				Autoimmune hepatitis	$N=1$	
				Undifferentiated connective tissue disease with positive ANA	$N=1$	
				Chronic juvenile arthritis	$N=1$	
				ANA and P-ANCA	$N=1$	

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies		Notable clinical features, or MRI findings
Chen et al. (2021)[56]	8	NA/NA	• MOGAD • Cytometric beads array	Anti-TPO/TG ANA Anti-SSA Anti-SSB Anti-RO-52	$N=4$ $N=1$ $N=1$ $N=1$ $N=1$	
Song et al. (2019)[98]	23	NA/NA	• MOG-ON • CBA, serum	ANA Anti-centromere Abs Anti-SSA/SSB Anti-CL/β 2GPI Anti-perinuclear factor Abs Anti-TG/TPO	$N=6$ $N=2$ $N=3$ $N=6$ $N=2$ $N=4$	
James et al. (2020)[99]	1	F/39	• MOGAD • CBA, serum	ANA + anti-SSB Abs		
Stathopoulos et al. (2019)[100]	9	NA/NA	• Positive MOG-IgG • CBA, serum	RA Type 1 DM SLE	$N=5$ $N=2$ $N=2$	
Zhao et al. (2018)[101]	4	NA/NA	• MOG-ON • CBA, serum	ANA Anti-SSA/SSB	$N=3$ $N=1$	
Ciotti et al. (2020)[102]	6	NA/NA	• MOGAD • CBA, serum	ANA ENA	$N=5$ $N=1$	
Papathanasiou et al. (2020)[103]	4 M/40 M/23	NA/NA	• MOGAD • CBA, Serum	ANCA P-ANCA with PR3-ANCA GAD-Ab	$N=3$ $N=1$ $N=1$	
Soelberg et al. (2018)[104]	1	NA/NA	• MOG-ON • CBA, NA	Anti-Sm antibody (weakly positive)		
Cross et al. (2021)[105]	3 M/39 F/33 M/40		• MOGAD • CBA, serum	ATD, psoriasis		
Liu et al. (2021)[106]	2	NA/NA	• MOGAD • CBA, serum	ATD Psoriasis	$N=1$ $N=1$	
Cobo-Calvo et al. (2016)[107]	1	NA/NA	• MOG-LETM • CBA, serum	MG		
Chen et al. (2017)[108]	1	F/10	• MOG-ADEM • NA, NA	Hashimoto thyroiditis encephalitis		Preceding autoimmune thyroid disease with elevated circulating anti-thyroid Abs, followed by subacute onset of multifocal CNS dysfunction.

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Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies	Notable clinical features, or MRI findings
					Brain MRI showed asymmetric, multifocal regions of T2-W and FLAIR signal hyperintensity in the subcortical white matter, left globus pallidus, thalami, midbrain, left pons, and cerebellum.
Nagahata et al. (2022)[109]	1	F/63	• MOG-ON • CBA, NA	• Granulomatosis with polyangiitis • MPO-ANCA: NA, NA	Presentation of recurrent poor vision and hearing loss with MRI showing low contrast-enhanced right optic nerve sheath and contrast-enhanced cavernous sinusitis.
Vural et al. (2017)[80]	2	NA/NA	• Positive MOG-IgG • CBA and flowcytometry, NA	Sjogren's syndrome	
Jobling et al. (2019)[110]	1	M/13	• Positive MOG-IgG • NA, NA	Sjogren's syndrome with positive anti-Ro and anti-La	Presentation of TM following a severe upper respiratory system infection. MRI showed a subtle TM of conus medullaris.
Ling et al. (2020)[111]	1	F/53	• MOG-ON • NA, serum (medium positivity)	Sjogren's syndrome and positive SSA, ANA, RF	Acute ON with MRI finding of mild right optic nerve enhancing lesion and normal brain MRI, 8 years after diagnosis of Sjogren which presented with BON.
Mittal et al. (2021)[112]	1	F/32	• MOG-ON • N/A, serum	Sjogren disease with positive ANA, anti-Ro, anti-RNP	Left optic neuritis with intraretinal and subretinal hemorrhages. MRI showed T2 hyperintensity and expansion of the left optic nerve and nerve sheath, along with flattening of the globe at the nerve insertion, and contrast enhancement of left optic nerve and sheath on T1.
Stamenova et al. (2021)[113]	1	F/31	• MOG-EN • N/A, serum	Crohn's disease treated with TNF-alpha inhibitors (adalimumab) and azathioprine	Development of headache, fever, and left-sided focal motor seizures, which progressed to bilateral tonic-clonic seizures

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies	Notable clinical features, or MRI findings
					during pregnancy. MRI showed bilateral cortical FLAIR-hyperintense lesions.
Philippart et al. (2019)[114]	1	M/33	• MOGAD • CBA, serum	Crohn's disease treated with TNF-alpha inhibitors (adalimumab) and azathioprine	Recurrent myelitis and brainstem syndrome with non-enhancing T2 hyperintense lesion from T4 to T6, and a second T2 hyperintensity at the T2 level. Brain MRI unremarkable.
Luo et al. (2021)[115]	1	F/26	• MOGAD • CBA, serum	Ankylosing spondylitis treated with TNF-alpha inhibitors (adalimumab)	Left ON and TM in postpartum setting, in a patient with AS treated with TNF-alpha inhibitors (adalimumab). T2-W MRI showed high signal intensity within the left optic nerve and the spinal cord from C5 to T2. No lesions were found in the brain.
Lommers et al. (2018)[116]	1	M/40	• MOGAD • CBA, serum	Pustular psoriasis treated with TNF-alpha inhibitors	MRI showed LETM at the bulbo-medullar junction with gadolinium enhancement in first attack, and T2 hyperintense signal in the intra-orbital part of the left optic nerve with a discrete enhancement in second attack.
Takei et al. (2017)[117]	1	F/18	• MOGAD • CBA, serum	Atopic dermatitis	Development of left-side dominant diffuse muscle weakness and numbness with bilateral ankle pseudo clonus along with deterioration of atopic dermatitis, followed by left ON one month later. MRI showed no abnormal lesions except for slight enlargement of the central canal at T7.
Adhikari et al. (2021)[118]	1	F/26	• MOGAD • CBA, serum	Opsoclonus myoclonus syndrome	Progressive bilateral hand tremors, intermittent myoclonus, ataxia, vertigo, and opsoclonus for two weeks in postpartum setting, found to be MOG-IgG positive in

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies	Notable clinical features, or MRI findings
					the second relapse, four months later. MRI showed non-enhancing right periventricular and left globus pallidus T2 FLAIR hyperintensities in initial presentation, and two new bilateral subcortical hyperintensities, one with postcontrast enhancement in second relapse.
Baptista et al. (2017)[119]	20	NA/NA	• High levels of MOG-IgG • ELISA, NA	RA	Patients had significantly higher levels of anti-MOG-IgG (5.68 ± 1.34 vs 0.51 ± 0.49 ng/mL), than controls.
Martinez-Hernandez et al. (2015)[72]	3 F/27 M/11	M/27	• MOGAD • MOG-Ab: CBA, serum and/or CSF	• GlyRs-Ab • CBA, serum, and/or CSF	
Hacohen et al. (2016)[120]	1	NA/NA	• MOG-EN • IF CBA, serum	• GlyRs-Ab • IF CBA, serum	
Armangue et al. (2017)[121]	4	NA/NA	• Positive MOG-IgG • NA, serum and/or CSF	• GlyRs-Ab • NA, NA	Children with acquired demyelinating syndrome with positive MOG-IgG had less frequent periventricular and tectum lesions, but more diffuse cerebellum involvement.
Ding et al. (2020)[122]	1	M/20	• MOGAD • CBA, serum	• GFAP astrocytopathy • CBA, CSF	
Fang et al. (2021)[123]	4	NA/NA	• Positive MOG-IgG • NA, NA	• GFAP astrocytopathy • CBA, serum, and CSF	
Ji et al. (2021)[124]	1	23/F	• MOGAD • CBA, CSF	• GFAP astrocytopathy • CBA, serum	Presentation of transient convulsions, a loss of consciousness, persistent fever, and vomiting, misdiagnosed as infectious meningoencephalitis. MRI showed asymmetric lesions of cerebellum, corona radiata, and enhancing white matter lesions.

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies	Notable clinical features, or MRI findings
Zhao et al. (2020)[125]	1	F/30	• MOGAD • CBA, CSF	• Anti-GFAP Abs • CBA, CSF	Normal brain and orbital imaging
Martin et al. (2022)[126]	1	M/53	• MOGAD • CBA, CSF	• Anti-GFAP Abs • CBA, CSF	Presentation of isolated meningitis and papillitis.
Shimizu et al. (2019)[127]	10	NA/NA	• MOGAD • CBA, NA	• GRP78 antibody • Western blots	The rate of GRP78 antibody positivity observed in acute MOG groups (10/15, 66%) was significantly higher than that in the disease control groups (3/27, 11%) or the healthy control groups (0/9, 0%).
Liu et al. (2020)[128]	1	F/48	• MOGAD • CBA, CSF	• CASPR2-IgG associated autoimmune encephalitis • NA, serum and CSF	Decreased vision in the right eye and subsequent episodes of neuropsychiatric disturbance including hypersomnia, agitation, apatheia, and memory impairment, with T2 and FLAIR hyperintense multiple lesions scattered in brain, brainstem, and cervical and thoracic spinal cord, with heterogenous patchy or ring-like enhancement in the majority of lesions.
Rauer et al. (2006)[129]	15	NA/NA	• Clinically isolated syndrome (CIS) • Western blot, serum	• Anti-MBP- Abs • Western blot, serum	There was no increased risk for developing definite MS in CIS patients with positive anti-MOG/MBP antibodies.
Tomassini et al. (2007)[130]	13	F ($N=11$), M ($N=2$) Median age: 27 years (range 25–33)	• MOGAD • Western blot, serum	• Anti-MBP- Abs • Western blot, serum	Presentation of MOG-IgG-associated ON ($N=4$), BS-cerebellar syndrome ($N=3$), myelitis ($N=3$), and multifocal neurologic deficit ($N=3$). Patients with double seropositivity had a higher risk of second relapse compared to seronegative or single seropositive patients.

(continued)

Table 3. Continued.

References	Number of cases	Sex/age	• MOG-IgG-associated clinical phenotype or laboratory status • Antibody testing assay, source (serum/CSF),	Overlapping autoimmunity and/or autoantibodies	Notable clinical features, or MRI findings
Rinaldi et al. (2021)[131]	4	F/30 F/29 M/58 F/31	• MOGAD • Live CBA, serum	• CASPR2 Ab (Live CBA, serum), • NF155 antibody (live CBA, serum), • GM1-Ab (ELISA, serum)	Coexistence of central and peripheral nervous system involvement in MOGAD patients.
Li et al. (2022)[132]	1	M/33	• MOG-EN • CBA, serum	• Anti-IgLON5-Ab • CBA, serum and CSF	Presentation of FLAIR hyperintense lesions in MOG-IgG-associated encephalitis and seizures (FLAMES).

Abbreviations: SLE: systemic lupus erythematosus; NPSLE: neuro-psychiatric SLE; ANA: anti nuclear antibody; ds-DNA: double-strand DNA; Anti-Sm: Anti-smith; ENA: extractable nuclear antigen antibodies; GABA-A-R: gamma aminobutyric acid receptor; CASPR2: contactin-associated protein-like 2; LGI1: leucine-rich glioma-inactivated 1; ATD: autoimmune thyroid disease; RA: rheumatoid arthritis, APS: anti-phospholipid syndrome; RNP: ribonucleoprotein; ANCA: antineutrophil cytoplasmic antibodies; ACA: anticardiolipin; B2GP: beta(2) glycoprotein; RF: rheumatoid factor; TPO: thyroid peroxidase antibody; TG: thyroglobulin; TSHR: thyroid stimulating hormone receptor; tTg: tissue transglutaminase; Anti-CL: anti-cardiolipin; PR3: proteinase3; GAD: Glutamic acid decarboxylase-antibody; MG: Myasthenia gravis; MPO: Myeloperoxidase; AS: Ankylosing spondylitis; GlyRs-Ab: Glycine receptor antibody; GRP78: Glucose regulated protein 78; MBP: myelin basic protein; NF155: neurofascin155; GM1: ganglioside epitope.

Table 4. Overlapping MOGAD/positive **MOG-IgG** and cancers or paraneoplastic syndromes

Reference	Number of cases	Sex/age	MOG-IgG-associated clinical phenotype	Overlapping cancer/paraneoplastic syndrome
Kwon et al. (2020)[133]	1	F/64	MOG-EM	T-cell lymphoma
Li et al. (2020)[134]	1	F/49	MOG-EM	Lung adenocarcinoma
Cohen et al. (2020)[135]	1	F/52	MOGAD	Colon adenocarcinoma
Kwon et al. (2020)[136]	NA	NA	MOG-EM	From 63 patients with MOG-EM, 11.3% had a history of cancer, and 6.5% had concurrent cancer. Types of cancers not specified.
Ajam et al. (2020)[137]	1	M/25	MOG-ON	Meningioma
Delgado et al. (2021)[138]	1	F/37	MOG-ON	Pituitary macroadenoma
Cirkel et al. (2021)[139]	1	F/44	MOG-EM	Paraneoplastic encephalomyeloradiculitis with multiple autoantibodies against ITPR-1, GFAP, and MOG Underlying malignancy: borderline tumor of the ovary
Cherian et al. (2021)[140]	1	F/37	MOG-LETM	MOG-LETM as paraneoplastic myelopathy Underlying malignancy: breast carcinoma
Rodenbeck et al. (2021)[141]	1	F/64	MOG-MY	MOG-MY as paraneoplastic myelopathy Underlying malignancy: lung adenocarcinoma

Abbreviations:EM: encephalomyelitis; ON: optic neuritis; LETM: longitudinally extensive transverse myelitis; MY: myelitis

for each of MOG-associated encephalitis (MOGAD-EN) or anti-NMDAR-EN and was seropositive for the antibody associated with the other one, at the same time.[10] Given the rarity of both MOGAD and anti-NMDAR-EN, the co-occurrence of these two entities may happen in the context of an association or could be just a coincidence. This overlap could be categorized as one of the following chronological phenotypes: (1) cases that start with a presentation of anti-NMDAR-EN who develop demyelinating events consistent with MOGAD later; (2) cases with a known diagnosis of MOGAD who later present with anti-NMDAR-EN; and (3) cases of autoimmune encephalitis with concurrent positive MOG-IgG and anti-NMDAR-IgG, demonstrating that antibodies against NMDAR and MOG can be simultaneously detected in one patient.[10,54] One possible explanation could be an immunological activation (e.g. an infection) driving the production of different subclasses of neuronal autoantibodies (anti-MOG-IgG, anti-NMDAR-IgG), resulting in the development of two different diseases.[13,53] Another hypothesis is that since both MOG and NMDAR are expressed on oligodendrocytes, an immune attack against one may provoke targeting the other one by antigen spreading.[22,53]

Considering the different disease courses and the outcomes of the two diseases, the management of the co-existing MOGAD and anti-NMDAR-EN could be challenging. MOGAD was perceived primarily as a monophasic CNS inflammatory disease, but further observations showed that a relapsing course occur in 28–60% of patients, and in up to 83% of cases with long-term observation, especially those with persistent positive MOG-IgG status.[99,143] Some evidence of subclinical disease activity and progression has been shown to be present in some of the patients with MOGAD.[2] Patients with anti-NMDAR-EN tend to have more severe neurologic presentations, and poor long-term functional outcomes, and the mortality rate has been reported between 5% and 7%.[144–146] In their study of 23 patients with overlapping demyelinating syndrome and anti-NMDAR-EN, Titaluer et al. demonstrate that the demyelinating episodes are more difficult to treat than anti-NMDAR-EN and often required more aggressive therapies, emphasizing the importance of its prompt diagnosis and treatment.[10]

We found 70 patients reported to be double-positive for MOG-IgG and AQP4-IgG, suggesting the co-existence of these two antibodies in the same patient is not as rare as previously thought.[25] Patients with double positive AQP4-IgG and MOG-IgG have been reported to be more refractory to therapies, relapse frequently, and

progress rapidly.[68,75] Matsuda et al. reported one double seropositive case which had recurrent ON attacks with residual visual field deficit, resulting in no improvement in visual acuity despite courses of treatments. They concluded that regardless of the method of measurement, double-positive cases were found to have a significantly poorer visual outcome, suggesting that anti-AQP4 and anti-MOG antibodies may indicate the prognosis of visual function in ON.[75] Kezuka et al. noted that 50% of double-positive patients did not respond to corticosteroid pulse therapy and plasmapheresis, and show no post-treatment improvement in visual function, whereas the visual acuity improved significantly in the double negative group following the same treatment.[68–75] Overall, these findings suggest that MOG-AQP4-IgG double seropositivity might be a marker of a poor outcome in NMO spectrum disorders, possibly because of the involvement of both astrocytes and oligodendrocytes.[68,75] Given the overall rarity of double positive cases based on the results of the larger studies, and considering the superior specificity of the AQP4-IgG test compared to the MOG-IgG test, and that a reliable cell-based-assay (CBA) for detecting MOG-IgG has been available just recently (2017), there is a high possibility that most of the double positive cases may likely be AQP4-IgG + NMOSD with false positive MOG-IgG test results, especially in reports made before this time with using enzyme-linked immunosorbent assay (ELISA).[68,75–147] False positive AQP4-IgG test results may occur rarely as well.

We found 35 cases of coexisting MOGAD and SLE, or SLE patients harboring MOG-IgG, some of which had a presentation of neuropsychiatric SLE (NPSLE). It is worthy of mentioning that, since the denominator to get to 35 cases is unknown, therefore the causal connection between MOGAD and SLE is either unclear or not present.

The potential role of anti-tumor necrosis factor (TNF)-alpha inhibitors in the development of CNS demyelination has been postulated to explain the overlapping MOGAD and some cases of Crohn's disease, ankylosing spondylitis, and psoriasis.[114–116] Other autoimmune disorders and autoantibodies that have been shown to overlap with MOGAD come from case reports and observational studies, which seem to be a coincidence and comparable with their frequency in the general population. Reports of MOGAD in patients with malignancies may represent the development of MOGAD as a paraneoplastic syndrome or could be an incidental co-occurrence, or false positive results of the MOG-IgG test.

Other than anti-NMDAR-EN and perhaps AQP4-IgG + NMOSD, the evidence thus far does not support the need for routine screening of overlapping autoimmunity and neoplasms in a patient with MOGAD. On the other hand, the antibody screening test for MOG-IgG may be helpful in cases of development of any atypical symptoms for encephalitis, or MRI findings attributable to demyelination, especially in patients with a diagnosis of anti-NMDAR-EN.

Most of our data come from case reports, case series, and observational studies, and only a few large studies looking at the association between MOGAD and autoimmune diseases are available, so we do not have a total denominator to report a fixed ratio or percentage of overlapping MOGAD and autoimmune disorders or cancers, and without the denominator, studies that found little or no association can make it appear like there is an association. The publication bias can inflate the potential connection between MOGAD and other autoimmune diseases or autoantibodies such as SLE or double AQP4-IgG and MOG-IgG positive cases, and this makes it hard to judge if there is a true connection between these two entities. As a result, we cannot draw a precise conclusion by only assessing and contrasting the total number of reported cases, which does not actually indicate the prevalence of concomitant autoimmune entities in MOGAD. In summary, our study suggests that the connection between autoimmune diseases and MOGAD is low or at least less than AQP4-IgG + NMOSD, and further studies are needed to establish the predisposing factors in the development of concurrent autoimmune diseases or autoantibodies in patients with MOGAD.

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Supplemental material

Supplemental material for this article is available online.

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