

Frequency of New or Enlarging Lesions on MRI Outside of Clinical Attacks in Patients With MOG-Antibody–Associated Disease

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Abstract

Background and Objective

To determine the frequency of new or enlarging T2-hyperintense or enhancing lesions outside of clinical attacks in myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD) vs multiple sclerosis (MS) and aquaporin-4 antibody positive neuromyelitis optica spectrum disorder (AQP4+NMOSD).

Methods

We retrospectively included Mayo Clinic patients with MOGAD with: (1) MOG-Immunoglobulin-G positivity by live cell–based assay, (2) fulfilling proposed MOGAD diagnostic criteria, and (3) baseline and follow-up paired MRIs without interval attacks. A neurologist and neuroradiologist reviewed MRIs (T2-fluid attenuated inversion recovery brain, T2 spine, and T1-postgadolinium brain and spine) to identify new or enlarging lesions. A MOGAD subset was then compared to patients with MS and AQP4+NMOSD, based on broadly similar interscan intervals.

Results

We included 105 patients with MOGAD (median age, 31 years [range, 2–80]; 60% female) with 373 paired MRIs. In total, 10/105 (9.5%) patients and 13/373 (3%) scans had one or more new T2 lesions (brain, 12/213 [6%]; spine, 1/160 [0.6%]); and 8/367 (2%) had enhancing lesions. New brain lesions were less in MOGAD (1/25 [4%]) than MS (14/26 [54%], $p < 0.0001$) but did not differ from AQP4+NMOSD (1/13 [8%], $p = 1.0$) in subgroup analysis. New spinal lesions were rare across groups (0%–4%).

Discussion

New or enlarging MRI lesions rarely develop outside of clinical attacks in MOGAD differing from MS. Surveillance MRIs in MOGAD have limited utility with implications for clinical practice and trial design.

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Glossary

AQP4+NMOSD = aquaporin-4 antibody-positive neuromyelitis optica spectrum disorder; **MOGAD** = myelin oligodendrocyte glycoprotein antibody-associated disease; **MS** = multiple sclerosis.

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a demyelinating disease distinct from multiple sclerosis (MS) and aquaporin-4 IgG-seropositive neuromyelitis optica spectrum disorder (AQP4+NMOSD).¹ In MS, MRI surveillance is standard of care,² and new asymptomatic lesions often developed before high-efficacy therapy availability, but in AQP4+NMOSD, such lesions are rare.³ Details on new lesion frequency outside of clinical attacks in MOGAD is limited.^{4,5} Herein, we determined new lesion frequency in MOGAD and compared it with MS and AQP4+NMOSD.

Methods

Patients were retrospectively identified from the Mayo Clinic MOGAD database (January 1, 2000–October 30, 2020), and inclusion criteria were (1) serum MOG-Immunoglobulin G (IgG) positive by live cell-based assay any time during disease course, (2) fulfilling current proposed MOGAD diagnostic criteria,⁶ and (3) a baseline and follow-up MRI without interval attacks. Paired MRIs (baseline and follow-up) were categorized as attack-to-remission or remission-to-remission scans. A neurologist and neuroradiologist compared follow-up T2-fluid attenuated inversion recovery (brain), T2 (spine), and T1-postgadolinium (brain and spine) images with a reference MRI to identify new or enlarging T2-hyperintense lesions or enhancing lesions with consensus reached in discordant cases. We compared the frequency of such lesions in a MOGAD subset (selected based on broadly similar interscan intervals to the other subgroups) to an MS and AQP4+NMOSD subgroup of patients negative for MOG-IgG from a previous study.⁷ Continuous variables were evaluated using the paired *t* test or Mann–Whitney *U* test, categorical variables with the Fisher exact test, and Kaplan–Meier curve for time to next relapse. All tests were two-sided, and $p \leq 0.05$ was considered statistically significant (SAS, Inc., Cary NC, version 9.4).

Standard Protocol Approvals, Registrations, and Patient Consents

Mayo Clinic's institutional review board approved the study. All participants consented to use of their medical records for research.

Data Availability

Anonymized data from this study are available on request.

Results

We included 105 patients with 373 paired MRIs (brain, 213; spine, 160). New or enlarging MRI T2-lesions outside of clinical attacks occurred in 10/105 patients with MOGAD (9.5%; new, 9; enlarged, 1) representing 13/373 (3.5%) scans

(brain, 12/213 [5.6%]; spine, 1/160 [0.6%]). Table 1 compares patients with and without new or enlarging lesions and Figure 1 shows MRI examples. New or enlarging T2-lesions occurred in attack-remission scans (8/171 [4.7%]) and remission-remission scans (5/202 [2.4%]), but future relapse risk did not differ based on this parameter (data not shown). New lesions were single (6/13 [46%]) or multiple concurrent (7/13 [54%]). New or enlarged gadolinium enhancing lesions occurred in 8/367 scans (2%; new, 7; enlarged, 1). New or enlarging T2-lesions did not predict future relapse (eFigure 1, links.lww.com/WNL/C367). In the MOGAD subset with broadly similar interscan interval to the comparison groups, new or enlarging brain T2-lesions were less frequent than in MS but similar to AQP4+NMOSD (Table 2). Spinal lesions were similarly rare in the MOGAD subset (0/21 [0%]) as MS (1/23 [4.4%]; $p = 1.0$) and AQP4+NMOSD (0/13 [0%]; $p = 1.0$) with similar interscan intervals in months (MOGAD, median 17 [range, 12–43]; MS, median 20 [range, 6–201]; AQP4+NMOSD, median 30.5 [range, 6–138]).

Discussion

We found new or enlarging lesions outside of attacks rarely developed on surveillance brain MRI in MOGAD differing from MS. The frequency in patients with MOGAD (12/213 [5.6%]) was similar to a Canadian pediatric study (16/483 [3.3%]) and UK report that included all ages (5/137 [3.4%]).^{4,5} As noted previously, new or enlarging lesions were more frequent at first follow-up MRI after an attack (attack-to-remission scan) than with remission-to-remission scans.^{4,5} This may reflect lesions accumulated during the prior clinical attack, but after the attack MRI was undertaken. New or enlarging MOGAD spinal lesions were rare (<1%) consistent with a prior study.⁴ New or enlarging lesions did not predict subsequent relapse although prior data are conflicting, and further studies are needed.^{4,5} Our inclusion of USA data, an adult MS comparison group, and pediatric spine MRI details are novel and add to knowledge on this topic.

The rarity of new or enlarging lesions suggests that MRI surveillance outside of attacks should not be recommended routinely in MOGAD differing from current MS practice with potential cost savings.² Moreover, surveillance MRI as a surrogate biomarker of disease activity in clinical trials will have much lower utility in MOGAD than MS.

The lower frequency of new or enlarging lesions in MOGAD than MS emphasizes its separate pathogenesis and may reflect less subclinical disease, greater T2-lesion resolution, monophasic course in 50%, greater potential for disease restricted to the optic

Table 1 Comparison of Patients With MOGAD Without or With New Lesions

	MOGAD total (n = 105)	MOGAD without asymptomatic lesions (n = 95)	MOGAD with asymptomatic lesions (n = 10)	Asymptomatic lesions vs No asymptomatic lesions (p value)
Median age at attack onset, years (range)	31 (2–80)	31 (2–80)	34 (9–66)	0.4677
Children, younger than 18 y (%)	36 (34.3)	33 (34.7)	3 (30.0)	1.00
Sex, female (%)	63 (60.0)	58 (61.1)	5 (50.0)	0.5162
Ethnicity, Caucasian (%)	83 (79.1)	75 (79.0)	8 (80.0)	1.00
Monophasic disease (%)	52 (49.5)	48 (50.5)	4 (40.0)	0.7415
High titer MOG-IgG (%)	58 (59.2) ^a	52 (59.1) ^a	6 (60.0) ^a	1.00
Persistent MOG-IgG (%)	52 (74.3) ^b	46 (73.0) ^b	6 (85.7) ^b	0.67
Treatment with DMT (%)	44 (41.9) ^c	41 (43.2) ^d	3 (30.0) ^e	0.5148
Treatment with acute therapy during previous clinical attack (%)	95 (91.3) ^f	86 (91.5) ^g	9 (90.0) ^h	1.00
Median no. of paired MRIs (range)	3 (1–16)	3 (1–13)	5.5 (1–16)	0.0032
Median interval from disease onset to reference MRI in months (range)	16 (1.5–314) ⁱ	16 (1.5–314) ⁱ	17 (1.5–111) ⁱ	0.97
Median interval between scans in months (range)	7 (0.5–133) ⁱ	7 (0.5–133) ⁱ	4 (1–31) ⁱ	0.31
Median length of follow-up in months (range)	27 (1–168)	27 (1–168)	32 (3–97)	0.8914

Abbreviations: DMT = disease-modifying treatment; IVIG = intravenous immune globulin; MOG-IgG = myelin oligodendrocyte glycoprotein antibody; MOGAD = myelin oligodendrocyte glycoprotein antibody-associated disease; PLEX = plasma exchange.

^a High titer $\geq 1:100$; Titers available for 98 patients with MOGAD, 88 patients with MOGAD with no asymptomatic lesions, all 10 patients with MOGAD with asymptomatic lesions.

^b Persistent seropositivity was defined as positive MOG IgG titers separated by at least 6 mos. Repeat titers were available for 70 patients with MOGAD, 63 with no asymptomatic lesions, and 7 with asymptomatic lesions.

^c Including one or more of azathioprine, 14; mycophenolate mofetil, 10; prednisone, 10; rituximab, 9; IVIG, 7; glatiramer acetate, 3; interferon beta, 2.

^d Including one or more of azathioprine, 12; mycophenolate mofetil, 10; prednisone, 9; rituximab, 9; IVIG, 7; glatiramer acetate, 3; interferon beta, 2.

^e Including one or more of azathioprine, 2; prednisone, 1.

^f Acute therapy information was available for 104/105 patients, treatment included one or more of steroids, 90; PLEX, 11; IVIG, 7.

^g Acute therapy information was available for 94/95 patients, treatment included one or more of steroids, 81; PLEX, 10; IVIG, 7.

^h Including one or more of steroids, 9; PLEX, 1.

ⁱ These data were calculated by scan pair rather than by patient, patients with MOGAD n = 373, patients with MOGAD with no asymptomatic lesions n = 360, patients with MOGAD with asymptomatic lesions n = 13.

nerve, and most patients with MS receiving lower-efficacy medications in this cohort.^{1,6,7} Our limitations include the retrospective nature, potential bias of patients with asymptomatic lesions receiving more scans, lack of standardized imaging protocols and intervals, and inability to control for treatment effects for which larger studies are needed. However, this closely mirrors clinical practice, and the results remain generalizable.

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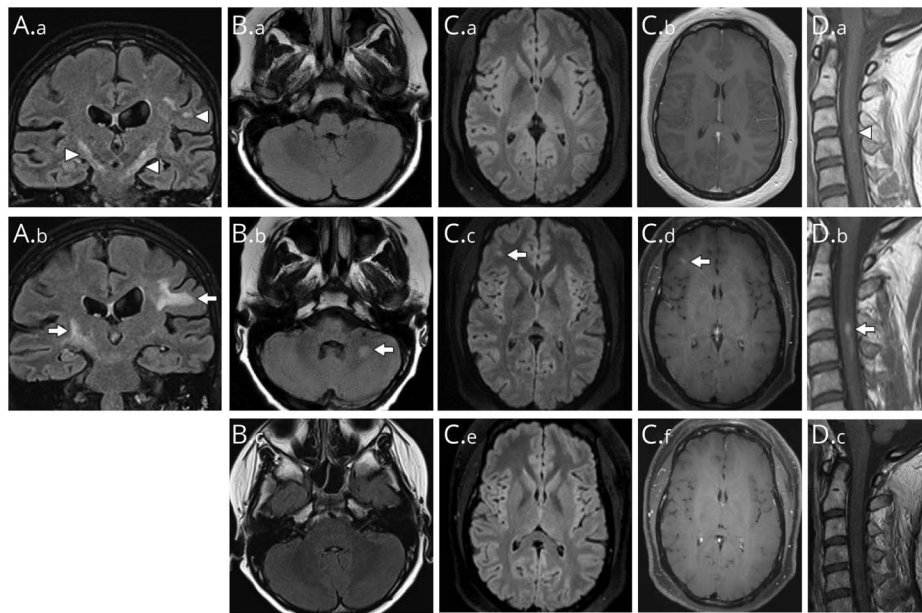
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Disclosure

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Figure 1 Examples of New or Enlarging Lesions Occurring Between Attacks in Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease



(A) The reference coronal MRI (T2-FLAIR) image reveals bilateral internal capsule and a left hemispheric T2-hyperintense lesion (A1, arrowheads) that on follow-up showed enlargement of the right internal capsule and left subcortical white matter T2-hyperintense lesions (A.b, arrows) in the absence of a new attack. (B) The reference axial MRI T2-FLAIR image (B.a) reveals normal brainstem and cerebellum signal while the follow-up image shows a new T2-hyperintensity in the left middle cerebellar peduncle (B.b, arrow) in the absence of a new clinical attack. The T2-lesion resolved completely and was no longer visible on a subsequent MRI FLAIR image (B.c) highly consistent with the expected evolution of a MOGAD lesion. (C) The reference axial MRI T2-FLAIR image (C.a) and axial T1 postgadolinium image (C.b) of the supratentorial region reveals no abnormalities but on follow-up show a new T2-hyperintensity had developed in the right frontal region (C.c, arrow) that enhanced after gadolinium (C.d, arrow) in the absence of a new attack. The lesion had resolved completely and was no longer visible on FLAIR (C.e) or T1 postgadolinium (C.f) images on a subsequent MRI highly consistent with the expected evolution of a MOGAD lesion. (D) The reference sagittal MRI cervical spine T1-weighted images postgadolinium revealed some subtle gadolinium enhancement (D.a, arrowhead) that increased in size in the follow-up (D.b, arrow) in the absence of a new attack despite no change in the T2-hyperintense cord lesion (not shown). The enhancement had resolved completely and was no longer visible on subsequent T1 postgadolinium image (D.c) highly consistent with the expected evolution of a MOGAD lesion. Abbreviations: FLAIR = fluid attenuated inversion recovery; MOGAD = myelin oligodendrocyte glycoprotein antibody-associated disease.

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(Application#12-678350, Neuromyelitis Optica Autoantibodies as a Marker for Neoplasia)—issued; a patent, Patent# 9,891,219B2 (Application#12-573942, Methods for Treating Neuromyelitis Optica [NMO] by Administration of Eculizumab to an individual that is Aquaporin-4 (AQP4)-IgG Autoantibody positive)—issued; E.P. Flanagan has served on advisory

Table 2 Comparison of New Brain T2-Lesions in Subset of MOGAD to MS and AQP4+NMOSD With Broadly Similar Interscan Intervals

	MOGAD (n = 25)	MS (n = 26)	MOGAD vs MS, p value	AQP4+NMOSD (n = 13)	MOGAD vs AQP4+NMOSD p value
New asymptomatic brain T2-lesions (%)	1 (4)	14 (53.9)	<0.0001	1 (7.7)	1.0000
Median age at attack onset, years (range)	31 (11-64)	41.5 (19-64)	0.1199	55 (22-73)	0.0048
Sex, female (%)	16 (64)	19 (73.1)	0.5551	11 (84.6)	0.2679
Treatment with DMT (%)	13 (52) ^a	22 (84.6) ^b	0.0167	13 (100) ^c	0.0026
Median interval between scans in months (range)	21 (16-133)	22.5 (7-138)	0.8801	44 (6-137)	0.4322

Abbreviations: AQP4+NMOSD = aquaporin-4 IgG-positive neuromyelitis optica; DMT = disease-modifying treatment; IVIG = intravenous immune globulin; MOGAD = myelin oligodendrocyte glycoprotein antibody-associated disease; MS = multiple sclerosis.

^a Including one or more of azathioprine, 4; mycophenolate mofetil, 3; prednisone, 3; rituximab, 3; interferon beta, 2; glatiramer acetate, 1; IVIG, 1.

^b Including one or more of interferon-beta, 14; fingolimod, 5; dimethyl fumarate, 4; glatiramer acetate, 2; natalizumab, 2; teriflunomide, 2; ocrelizumab, 1.

^c Including one or more of prednisone, 9; azathioprine, 6; rituximab, 6; mycophenolate mofetil, 5; cyclophosphamide, 1; methotrexate, 1.

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