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MOG IgG3-Subclass Antibodies in MOG-Associated Disease

Insights From a Pediatric Case With IgG1 Deficiency and Literature Review

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Abstract

Objectives

This report details a pediatric case of myelin oligodendrocyte glycoprotein antibody disease (MOGAD) characterized by isolated MOG-IgG3 positivity and IgG1 deficiency, highlighting a unique serologic profile and exploring its immunologic significance. In addition, a review of MOGAD cases with isolated MOG-IgG3 antibodies is included.

Methods

A 12-year-old boy presenting with a progressive neurologic syndrome underwent clinical, radiologic, and laboratory evaluation.

Results

MRI showed extensive brain and spinal lesions. MOG-IgG3 antibodies were identified in serum and CSF by live cell-based assay, while other MOG-IgG subclasses were not detected. Serum IgG1 was low at baseline and throughout follow-up. First-line immunotherapy treatment led to significant improvement, with no relapses at 2-year follow-up.

Discussion

This case highlights the potential role of subclinical IgG1 deficiency in shaping the predominance or selective detection of MOG-IgG3 in MOGAD. While most MOG antibodies are predominantly of the IgG1 isotype, our review identified a few cases with isolated MOG-IgG3 antibodies. A possible contribution to pathogenesis cannot be excluded, and the patient's inability to produce a balanced IgG subclass profile could have favored a sustained IgG3 response. Larger studies are needed to clarify the clinical significance of MOG-IgG3 in MOGAD.

Introduction

Detection of myelin oligodendrocyte glycoprotein (MOG) antibodies (Abs) has a central role in the recently proposed diagnostic criteria for MOG-Ab disease (MOGAD).[1] Cell-based assays (CBA) are the preferred method, yet substantial interlaboratory variability persists in transfection and fixation protocols and secondary antibodies selection. Notably, some groups measure total MOG-IgG using high-titer cut-offs, while others focus on MOG-IgG1, the predominant subclass.[2-4]

Limited evidence suggests the potential role of other IgG subclasses, particularly IgG3, in diagnosing MOGAD, though their significance remains unclear.[4-6]

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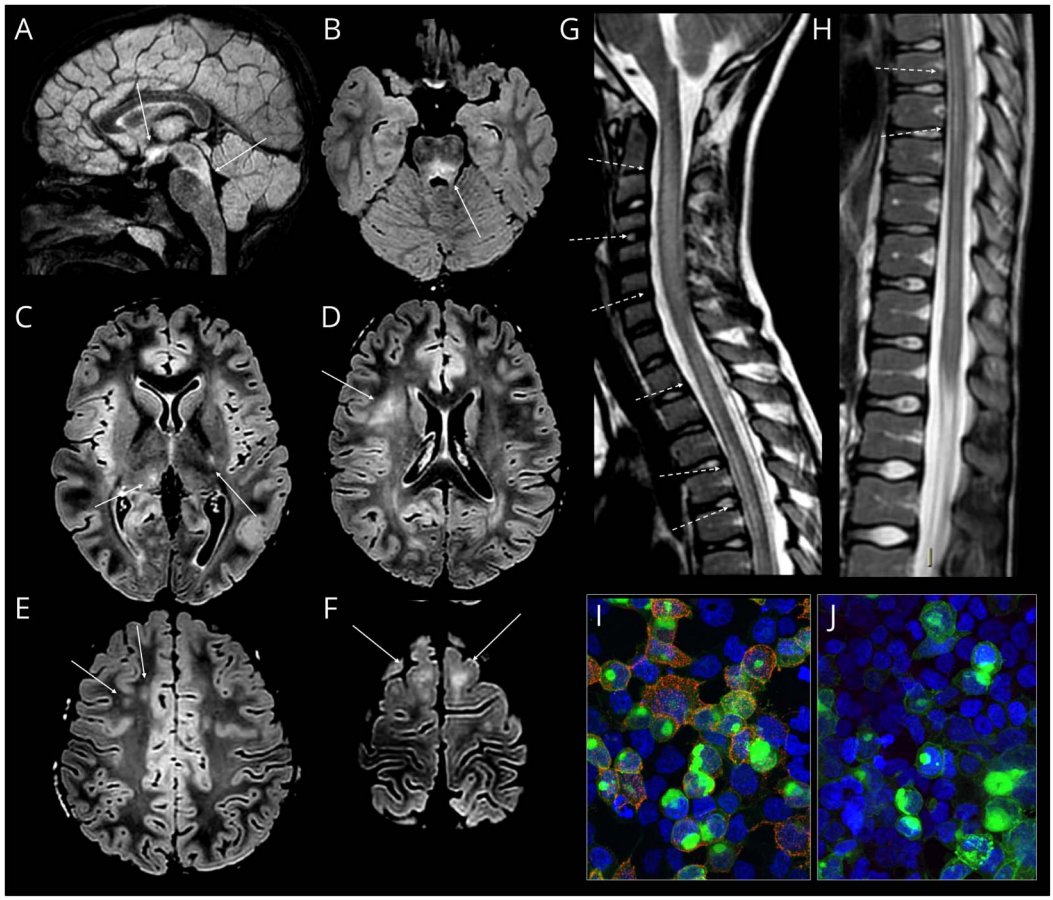
Figure 1 Brain MRI at Onset and Follow-Up and CBA Analysis



Sagittal 3D fluid-attenuated inversion recovery (FLAIR) T2-weighted images (A) reformatted on axial plane (B–F). T2 FLAIR hyperintense lesions are depicted at supratentorial and infratentorial brain locations (white arrows), in the hypothalamic region, in the mesencephalic tegmentum, in the right thalamus and in the posterior limb of the contralateral internal capsule, as well as in the deep and subcortical hemispheric cerebral white matter, bilaterally. Sagittal T2-weighted images (G and H) display spinal cord abnormality extending over multiple vertebral levels (white dotted arrows). (I and J) Live cell-based assay (CBA) showing serum human IgG3 antibody binding. HEK cells were transfected with full-length myelin oligodendrocyte glycoprotein (MOG) fused to an emerald green fluorescent protein (EGFP) tag. Right: Specific binding of serum IgG3 antibodies (Invitrogen, MH1031; 1:500) detected using a red-fluorescent goat anti-mouse secondary antibody (1:1000). Left: Control condition without antibody binding. Nuclei are counterstained with 4′,6-diamidino-2-phenylindole (DAPI; blue).

Methods

We report a pediatric case of MOGAD characterized by isolated MOG-IgG3 positivity and persistent IgG1 deficiency, together with a systematic literature review of previously reported cases. The study was approved by the local ethic committee, and informed consent for publication was obtained according to the Declaration of Helsinki. The datasets generated and/or analyzed during the current study are available from the corresponding authors on reasonable request.

Results

Case Presentation

A previously healthy 12-year-old boy presented subacute neurologic symptoms, including cervical pain, limb weakness, paresthesia, and intermittent fever (up to 38.7°C), with no recent infections or relevant exposures. MRI revealed multiple T2-fluid-attenuated inversion recovery (FLAIR) hyperintense supratentorial, infratentorial, and longitudinally extensive spinal cord lesions (Figure 1, A–H).

Neurologic examination showed severe tetraparesis (BMRC scale lower limbs 2/5, upper limbs 3/5), pyramidal signs, sensory loss in the lower limbs, and neurogenic bladder (EDSS 8). Consciousness and behavior were not impaired. EEG was normal. CSF analysis showed pleocytosis (304 white cells/mmc, 96% mononuclear), elevated protein (59 mg/dL), IgG (4.5 mg/dL), IL6 (310 pg/mL), and albumin quotient (8.4×10^{-3}), normal QIgG (5.6×10^{-3}), and negative oligoclonal bands. Infectious and rheumatological etiologies were excluded. Visual acuity was preserved (10/10), while optical coherence tomography showed left borderline thinning of the retinal nerve fiber layer and ganglion cell complex, reflecting possible prior damage.

First-line immunotherapy with 30 mg/Kg IV methylprednisolone and 2 g/Kg IV immunoglobulin led to significant improvement (EDSS 2.5 at discharge). Oral prednisone was

Table 1 Total IgG and Subclasses, Complement and Lymphocyte Immunophenotyping With Reference Values

Parameter	Unit	Onset (day 3)	Onset (day 18)	3 mo FU	8 mo FU	12 mo FU	20 mo FU	Reference for age: Median (* = 95% CI or ° = 10th −90th percentile)[7,8]
Complement C3	mg/dL	94	—	133		—	—	141.5 (97.36–195.9)*
Complement C4	mg/dL	17	—	41		—	—	19.72 (11.43–36.66)*
IgG	mg/dL	—	611	514	567	607	576	1,045 (581.4–1652)*
IgA	mg/dL	—	160	117	94	95	105	124.3 (41.59–344.9)*
IgM	mg/dL	—	72	107	68	65	77	98.6 (47.41–251.8)*
IgG1	mg/dL (% of total IgG)	—	293.2 (52.2%)	238.9 (48.3%)	275.6 (53.5%)	322.5 (57.6%)	311,5 (54.3%)	640 (315.6–1076)*
IgG2	mg/dL (% of total IgG)	—	144.1 (25.7%)	177.8 (36%)	141.1 (27.4%)	124.7 (22.3%)	136.5 (23.7%)	268.6 (85.71–509.4)*
IgG3	mg/dL (% of total IgG)	—	89.1 (15.9%)	55.6 (11.2%)	59.8 (11.6%)	65.1 (11.6%)	72.6 (12.6%)	77.25 (14.39–201.2)*
IgG4	mg/dL (% of total IgG)	—	35.3 (6.2%)	22 (4.5%)	38.9 (7.5%)	48 (8.6%)	54.1 (9.4%)	47.05 (0.69–102.7)*
WBC	$x10^3/\mu L$	12.96	14.93	8.25	7.18			
Lymphocytes (absolute)	/mmc	2,825	4,520	3,968	3,683			2,400 (1,710–3,060)°
Lymphocytes (relative)	% of WBC	21.8	30.3	48.1	51.3			38 (29–47)°
$CD3^+$ (T cells)	% of lymphocytes	67	70	80	78			70 (60–79)°
$CD3^+CD4^+$ ($CD4^+$ T cells)	% of lymphocytes	33	39	37	37			42 (31–53)°
$CD3^+CD8^+$ ($CD8^+$ T cells)	% of lymphocytes	25	25	33	27			23 (18–33)°
$CD56^+CD16^+CD3^-$ (natural killer)	% of lymphocytes	2	3	11	6			13 (8–22)°
$CD19^+$ (B cells)	% of lymphocytes	30.5	25.43	8.74	14.66			14 (8–21)°
$CD4^+CD45RA^+CCR7^+$ (naïve T $CD4^+$)	% $CD4^+$		79		70			51 (25–63)°
$CD4^+CD45RA^+CCR7^-$ (terminal effector T $CD4^+$)	% $CD4^+$		<1		0			7 (4–24)°
$CD4^+CD45RA^-CCR7^+$ (central memory T $CD4^+$)	% $CD4^+$		16		19			18 (11–25)°
$CD4^+CD45RA–CCR7^-$ (effector memory T $CD4^+$)	% $CD4^+$		5		11			22 (12–30)°
$CD8^+CD45RA^+CCR7–$ (late effector T $CD8^+$)	% $CD8^+$		7		15			12 (7–26)°
$CD8^+CD45RA^-CCR7^+$ (central memory T $CD8^+$)	% $CD8^+$		6		9			4 (2–15)°
$CD8^+CD45RA^-CCR7^-$ (effector memory T $CD8^+$)	% $CD8^+$		17		31			39 (24–58)°
$CD8^+CD45RA^+CCR7^+$ (naïve T $CD8^+$)	% $CD8^+$		69		45			41 (22–58)°
$CD19^+CD21lowCD38^-$ (activated B)	% $CD19^+$		1		0.8			3 (2–7)°
$CD19^+IgM^{-/+}CD38^{++}$ (plasmablasts)	% $CD19^+$		<1		0.2			0 (0–0)°

Continued

Table 1 Total IgG and Subclasses, Complement and Lymphocyte Immunophenotyping With Reference Values *(continued)*

Parameter	Unit	Onset (day 3)	Onset (day 18)	3 mo FU	8 mo FU	12 mo FU	20 mo FU	Reference for age: Median (* = 95% CI or ° = 10th −90th percentile)[7,8]
$CD19^+IgD^+CD27^+$ (unswitched memory B)	% $CD19^+$		12.8		9			7 (4–14)°
$CD19^+IgD–CD27^+$ (switched memory B)	% $CD19^+$		5.9		5			9 (6–16)°
$CD19^+IgD^+CD27^-$ (naïve B)	% $CD19^+$		78		82			76 (64–84)°
$CD19^+IgM^{++}CD38^{++}$ (transitional B)	% $CD19^+$		<1		6			13 (8–21)°

Abbreviations: mo = months; FU = follow-up; WBC = white blood cells.
At disease onset, total IgG and IgG1 were below age-specific reference values, and this deficiency remained stable over time. IgG1 had 7.5% decreasing and IgG3 had 29.6% decreasing between the first and second time points (immunosuppressive treatment). IgG2 and IgG4 fluctuated over time, reflecting dynamic changes in subclass distribution. By day 18, immunophenotyping revealed expanded B cells, and T cells within normal ranges, both with normal subset distribution. Eight months after onset, blood counts and T/B lymphocyte subsets normalized.

tapered over 6 weeks. At 2-year follow-up, the patient remained relapse-free with a normal neurologic examination (EDSS 0).

Live Cell-Based Assay

Serum and CSF samples were tested for MOG-Abs (full-length recombinant human MOG α1 isoform, EGFP tagged) and AQP4-Abs using a live-CBA, as previously described (eMethods).[4] AQP4-IgG was negative. MOG-IgG were positive in serum (titer 1:2560) and CSF (titer 1:32), using anti-human IgG-Fcγ-specific secondary antibody (Jackson Scientific). The MOG-IgG-specific antibody index was 2.2, indicating no intrathecal synthesis. MOG-IgG1 was undetectable (serum 1:10, CSF undiluted). Subclass analysis revealed isolated MOG-IgG3 positivity in serum (dilution 1:20) and CSF (dilution 1:2), while MOG-IgG2 and IgG4 were undetectable (Figure 1, I–J). Six months after onset, serum MOG-IgG titers had decreased to 1:1280, with persistent MOG-IgG3 positivity; at the 1-year follow-up both MOG-IgG and MOG-IgG3 were undetectable (1:20); MOG-IgG1, IgG2, and IgG4 remained undetectable throughout.

Immune Profile and Antibody Responses

Lympocyte immunophenotyping, total serum Ig, and complement levels at onset and during follow-up are reported in Table 1 and the eFigure. Despite complete vaccination according to the national schedule, the patient exhibited protective responses to measles and rubella, borderline response to mumps, and nonprotective antibody titers to hepatitis B, diphtheria, tetanus, and pertussis at disease onset and at 8-month follow-up. No increased susceptibility to infections was observed.

Literature Review

A systematic review following PRISMA 2020 guidelines was conducted using PubMed, Scopus, and Web of Science with the terms: ("Myelin oligodendrocyte glycoprotein" OR "MOG antibody disease" OR "MOGAD") AND ("IgG3" OR "immunoglobulin G3"). Only studies reporting serologically confirmed MOGAD with isolated IgG3 subclass were included. After screening 30 records (including one manual addition) and excluding duplicates and noneligible articles, 4 studies met inclusion criteria (Table 2).[2,4-6]

Discussion

This case describes a MOGAD presentation with a typical clinical and neuroradiologic pediatric phenotype,[10] but an unusual serologic profile with high-titer MOG-IgG, exclusively of the IgG3 isotype.

In most reports, MOG-Abs are predominantly IgG1,[1,2] and our review identified only a few cases, mainly adults, with isolated MOG-IgG3.[2,4,5] Recently, Jarius et al. examined serum samples that had previously shown negative or discrepant results in standard H&L-specific or Fc-specific assays for MOG-IgG and found IgG3 exclusivity in 2 patients and dominance in 6 with weak IgG1 signal. They discussed whether this finding might reflect dynamic subclass evolution over the disease course, treatment effect, different disease triggers, and/or distinct pathogenic subtypes.[6] Importantly, all samples in that study were from patients previously clearly positive for total MOG-IgG, with documented seroreversion and established MOGAD diagnosis. Similarly, our patient tested positive for total MOG-IgG by standard assay and met both the 2018 and 2023 proposed diagnostic criteria for MOGAD.[1,9]

Although all previously described phenotypes in IgG3-dominant MOGAD exhibited transverse myelitis and/or optic neuritis, our case, besides the longitudinally extensive

Table 2 Literature Review of the Patients With MOG-IgG With Negative IgG1 Subclass and Predominance of the IgG3 Subclass

Ref.	Sex	Age	Clinical phenotype	Outcome and follow-up	Total MOG IgG on serum		MOG-IgG subclass assay on serum (technique; titer or titer or semiquantitative; cut-off)	Subclass distribution among whole study population of reported articles
					Fixed CBA	Live CBA (titer; cut-off)		
Waters et al. 2015	M	51	TM (patchy lesions) +ON	Full recovery, no relapses	n/a	H + L: pos (semiquantitative cut-off: score >1 at 1: 20 patient sera dilution)	IgG3 pos (live CBA with subclass-specific secondary Abs; endpoint titer 1:3200; semiquantitative cut-off: score >1 at 1:20 patient sera dilution)	n/a
Gastaldi et al. 2020	F	5	TM	22 mo FU; no relapse; final diagnosis: possible MOGAD	Fcγ: pos	H + L: pos (titer 1:160; cut-off ≥1:160) Fcγ: neg (titer n/a; cut-off ≥ 1:160)	IgG3 pos (live CBA with subclass-specific secondary Abs; cut-off ≥1:20)	Among 18 IgG1-neg: 5 IgG2 (3 high titer, 2 low-titer) 3 IgG3 (low-titer)
Gastaldi et al. 2020	F	55	ON	23 mo FU; no relapse; final diagnosis: possible MOGAD	Fcγ: pos	H + L: pos (titer 1:160; cut-off ≥1:160) Fcγ: neg (titer n/a; cut-off ≥ 1:160)	IgG3 pos (live CBA with subclass-specific secondary Abs; cut-off ≥1:20)	
Mariotto et al. 2023	F	55	TM	n/a	n/a	H + L: neg (titer, 0; cut-off ≥1:160)	IgG3 pos 1:20 (live CBA with subclass-specific secondary Abs; cut-off ≥1:20)	H + L-positive samples: 22/22 IgG1 5/22 IgG2 9/22 IgG3 3/22 IgG4 H + L-negative samples: 8/132 IgG1 1/132 IgG2 3/132 IgG3 0/132 IgG4
Mariotto et al. 2023	F	31	TM	n/a	n/a	H + L: neg (titer 1:80; cut-off ≥1:160)	IgG3 pos 1:40 (live CBA with subclass-specific secondary Abs; cut-off ≥1:20)	
Mariotto et al. 2023	F	28	TM	Final diagnosis: clinically isolated syndrome	n/a	H + L: neg (titer 1:40; cut-off ≥1:160)	IgG3 1:20 (live CBA with subclass-specific secondary Abs; cut-off ≥1:20)	
Jarius et al. 2024	F	n/a	ON+ LETM	Final diagnosis: MOGAD[9]	Fcγ: pos	H + L: neg (titer 1:40; cut-off 1:160)	IgG3 ++++ (>1:320); IgG2 +; IgG4 + weak (fixed CBA with subclass-specific secondary Abs; cut-off ≥1:10)	IgG1: 25/27 IgG2: 8/27 IgG3: 14/27 (predominant or exclusive in 8/27)[a] IgG4: 2/27
Jarius et al. 2024	F	n/a	LETM	Final diagnosis: MOGAD[9]	Fcγ: neg	H + L: pos (titer 1:160; cut-off 1:160) Fcγ: neg (titer 1:80; cut-off 1:160)	IgG3 ++; IgG2 +/−; IgG1 +/1 in one sample; in another (fixed CBA with subclass-specific secondary Abs; cut-off ≥1:10)	
Our patient	M	12	LETM + subcortical and deep brain lesions	48 mo FU; full recovery, no relapses; final diagnosis: MOGAD[1,9]	Fcγ: pos	Fcγ: pos (titer 1:2560; cut-off ≥1:160)	IgG3 pos 1:32 (live-CBA with subclass-specific secondary Abs; cut-off ≥1:20)	

Abbreviations: CBA = cell-based assay; F = female; Fcγ = antibody detection against Fc gamma receptor; FU = follow-up; H + L = antibody detection against heavy + light chains; LETM = longitudinally extensive TM; M = male; n/a = not available; neg = negative; ON = optic neuritis; pos = positive; TM = transverse myelitis.
[a] No significant IgG3 reactivity in 60 control samples.

transverse myelitis, had diffuse typical subcortical and deep brain lesions, arguing against the possibility that MOG-IgG3 denotes a distinct clinical phenotype.

Alongside established T-cell–mediated mechanisms, the pathogenic role of MOG-Abs is increasingly recognized, while the interplay between humoral and cellular immunity remains incompletely understood.[11,12] MOG-IgG primarily exerts pathogenic effects through antibody-dependent cell-mediated cytotoxicity, complement-dependent cytotoxicity (CDC), and, potentially, antibody-dependent cellular phagocytosis.[11] IgG3 subclass, with strong C1q binding and a longer hinge region promoting hexamer formation at low antigen densities, is particularly effective at initiating CDC in vitro, as demonstrated using both monoclonal Abs and pooled human serum in target cell assays.[13] Consistent with this, in vitro studies have shown that both IgG1 and IgG3 MOG-Abs can induce CDC, either alone or in combination.[14] However, these results may not fully reflect in vivo effector mechanisms and the potential pathogenic role of MOG-IgG3 remain to be clarified.

In our case, reduced C3 and C4 within the lower limit of normal supported complement activation, while B-cell expansion and neutrophilic leukocytosis suggested humoral and innate immune activation; T-cell counts remained normal, contrasting with previous reports of reduced T-helper cells.[15]

Persistently low total IgG1 suggested a constitutional or transient deficiency. The absence of infection susceptibility and normal immunophenotype make a CVID-like primary immunodeficiency unlikely, pointing instead to selective IgG1 deficiency, often asymptomatic but associated with reduced responses to protein-based vaccines, as in our patient. Although total IgG1 partially recovered post-treatment, it remained below the lower bound of the 95% CI for age at follow-up, potentially explaining undetectable MOG-IgG1. Because MOG-IgG1 and IgG3 co-occur in up to half of patients with MOGAD,[5,6] our case suggests that isolated MOG-IgG3 positivity may, in some instances, reflect an epiphenomenon of subclinical selective IgG1 deficiency. Alongside this, our patient's failure of the normally unidirectional IgG3-to-IgG1 switch[6] may reflect a "two-hit" mechanism where pre-existing IgG1 deficiency created a permissive environment for a sustained IgG3-skewed response. In turn, inflammation may have reinforced the imbalance further inhibiting the subclass switching. This hypothesis is supported by the one-third reduction in total IgG3 after immunosuppression (greater than declines in total IgG or IgG1), the progressive, although subnormal, increase in total IgG1, and the restoration of subclass proportions at follow-up. Unfortunately, prior reports of isolated MOG-IgG3 lack IgG subclass profiling during follow-up[2,5,6], leaving the consistency of this pattern in other cases uncertain.

Conclusions

Our findings suggest that subclinical IgG1 deficiency may underlie MOG-IgG3 predominance or selective detection, highlighting how variability total IgG subclass composition could affect the interpretation of subclass-specific antibody assays in MOGAD. They also point to a possible pathogenic contribution for MOG-IgG3, particularly in the context of altered subclass expression or switching and/or in the early phases of the disease.

The single-case nature limits generalizability of these findings and larger cohorts are needed to clarify the role of IgG3 in MOGAD pathogenesis, phenotype and diagnosis, and its potential as a therapeutic target.

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Author Contributions

A. Fetta: drafting/revision of the manuscript for content, including medical writing for content; study concept or design. F. Conti: study concept or design. A. Lopalco: drafting/revision of the manuscript for content, including medical writing for content. C. Corsini: drafting/revision of the manuscript for content, including medical writing for content. M. Moratti: analysis or interpretation of data. L. Morelli: analysis or interpretation of data. F. Ricciardiello: analysis or interpretation of data. F. Toni: analysis or interpretation of data. M. Lanari: drafting/revision of the manuscript for content, including medical writing for content. R. Liguori: drafting/revision of the manuscript for content, including medical writing for content. D. Maria Cordelli: drafting/revision of the manuscript for content, including medical writing for content. M.P. Giannoccaro: drafting/revision of the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data.

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Disclosure

The authors report no relevant disclosures. Go to Neurology.org/NN for full disclosures.

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