

# Myelin Oligodendrocyte Glycoprotein Antibody Persistency in a Steroid-Dependent ADEM Case

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Myelin oligodendrocyte glycoprotein (MOG) is a candidate target antigen in demyelinating central nervous system diseases, including acute disseminated encephalomyelitis (ADEM), neuromyelitis optica, and multiple sclerosis. It may give prognostic information regarding monophasic or recurrent course of the disease. MOG antibodies have been shown to be positive in high titers during the first episode of ADEM with rapidly decreasing to undetectable limits after recovery. However, persistent MOG antibodies are considered as a predicting factor for multiple sclerosis, optic neuritis relapses, and incomplete recovery of ADEM. Here we report a unique case with persistent MOG antibodies presented with multiphasic ADEM-like attacks. A 6-year-old girl was consulted with encephalopathy, gait disturbance, and oculomotor nerve palsy. Periventricular white matter lesions were seen on cranial magnetic resonance imaging studies. ADEM was diagnosed and treated with steroid. During follow-up, she experienced repeated episodes after steroid therapy termination. We were able to search MOG antibody at the ninth attack. The positivity of this antibody remained. It was thought to be associated with steroid-dependent course, and azathioprine and intravenous human immunoglobulin treatment were added. Patients with persistent MOG antibodies may benefit from addition of immunosuppressant agents, which may decrease the number of attacks.

## CASE

A 6-year-old girl was consulted with multiple encephalopathy episodes and neurologic findings. Until admission she had 4 similar attacks. Family history was unremarkable. First attack occurred at the age of 18 months with drowsiness, fever, and gait disturbance (Table 1). Radiologic studies showed multiple supratentorial and basal ganglia lesions without contrast enhancement (Fig 1A). She was treated with pulse methylprednisolone. She had 3 similar attacks accompanied by encephalopathy during the first year of disease, which were treated with intravenous immunoglobulin (IVIG)

or methylprednisolone at another hospital. Except cerebral spinal fluid (CSF) oligoclonal band positivity, other laboratory findings, including complete blood cell count and biochemical, metabolic, infectious, immunologic, and serologic tests, were normal. At the fifth attack, she had drowsiness, strabismus, gait disturbance, and ataxia. Her neurologic examination showed hyperactive deep tendon reflexes, positive Babinski sign, lethargy, and abnormal cerebellar tests. Cranial magnetic resonance imaging (MRI) revealed mild cerebral atrophy with supratentorial lesions (Fig 1B). Diffusion MR, MR angiography,

## abstract

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Dr Polat conceptualized and designed the report, coordinated and collected the clinical data, reviewed the literature, and wrote and drafted the initial manuscript; Dr Yiş conceptualized and designed the report, made the initial analysis and interpretation, coordinated and supervised the data collection, and critically reviewed and revised the manuscript; Drs Karaoğlu, Ayanoğlu, and Öztürk made the initial analysis, collected the data, and reviewed the literature; Dr Gülerüz made the initial analysis, coordinated and collected the data, and critically reviewed and revised the manuscript; Dr Kurul made the initial analysis and interpretation, coordinated and supervised the data collection, and critically reviewed and revised the manuscript; and all authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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MR spectroscopy, orbital MR, and spinal MR revealed no abnormality. Hemogram, biochemical studies, thyroid autoantibodies, immunoglobulins, metabolic investigations including lactic acid, pyruvic acid, ammonia, amino acid chromatography (blood, urine, CSF), tandem mass spectrometry, and urine organic acids were normal. Infectious markers including toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus; hepatitis; HIV; mycoplasma pneumonia; Chlamydia pneumonia; Brucella; lyme; adenovirus; varicella zoster virus; and enterovirus were negative. Antinuclear antibody, double-stranded DNA antibody, and extractable nuclear antigens panel were negative. Autoimmune encephalitis panel, including N-methyl-D-aspartate receptor, antiglutamate receptor 1, antiglutamate receptor 2, contactin-associated protein-like 2, leucine-rich glioma-inactivated protein 1, and  $\gamma$ -aminobutyric acid receptor, was normal. Routine, infectious studies and lactic acid of CSF was normal. CSF oligoclonal band, which was detected by isoelectric focusing, was negative. CSF immunoglobulin (Ig) G index was 2.61 (normal: 0.3–0.7). She was successfully treated with methylprednisolone with tapering doses. Six weeks after the cessation of steroid she developed a sixth attack, with drowsiness, oculomotor palsy, and gait disturbance. MR revealed new lesions on the left thalamus and brainstem (Fig 1C). She was treated with 5 days of pulse methylprednisolone with tapering doses. Examinations between the attacks were normal, but after this episode there was mild gait disturbance. After an 8-week steroid-free period, she had a seventh attack with the same symptoms with new brainstem lesions (Fig 1D). After the pulse methylprednisolone treatment, mycophenolate mofetil (MMF) was started at a dose of 300 mg/m<sup>2</sup> and increased to the dose of

600 mg/m<sup>2</sup>. She developed a new encephalopathic attack 2 weeks after the cessation of steroid with new periventricular lesions on MR (Fig 1E). As a new demyelinating-like attack occurred after the initiation of MMF, progressive multifocal encephalopathy was suspected. CSF studies for John Cunningham and BK polyoma viruses were negative. MMF was stopped. She was considered to have steroid-dependent multiphasic disseminated encephalomyelitis (ADEM) attacks. Azathioprine (AZA) was started at a dosage of 0.5 mg/kg per day and increased to 2 mg/kg per day and steroid continued for 9 months. She could not take AZA regularly because of vomiting due to a urinary tract infection and developed a new attack with bilateral basal ganglia, periventricular lesions (Fig 1F). At this attack, we searched for aquaporin 4 antibody (AQP4) and myelin oligodendrocyte glycoprotein (MOG) antibodies. AQP4 antibody was negative but anti-MOG IgG was positive (1:100). Anti-MOG antibody was detected by immunofluorescence testing. After pulse methylprednisolone treatment, she again recovered well. Prednisolone treatment was continued with a dosage of 0.125 mg/kg per day with AZA 2 mg/kg per day. With this treatment, she was attack free for 7 months. Repeated anti-MOG IgG remained positive (1:100 titers) during this remission phase. During the course of a new infection, she experienced left hemiplegia which was followed by facial palsy and encephalopathy. Repeat MRI showed periventricular white matter and basal ganglia lesions (Fig 1G). IVIG and pulse methylprednisolone were given. Repeated anti-MOG IgG was still positive (1:100). Prednisolone 0.25 mg/kg per day with AZA 2 mg/kg per day was continued with monthly IVIG administration. Latest neurologic examination showed mild weakness on the left side with mild difficulty of speech and walking.

## DISCUSSION

ADEM, neuromyelitis optica (NMO), and multiple sclerosis (MS) are the most common inflammatory and demyelinating central nervous system (CNS) diseases.<sup>1</sup> Both the cellular and humoral immune system mechanisms may play role in the pathogenesis.<sup>2</sup> Reactive T cells may damage the blood-brain barrier leading to access of pathogenic antibodies to the brain.<sup>1</sup> The first demyelinating event with encephalopathy is termed as ADEM, which is classically monophasic disease and often recovers completely with corticosteroid, IVIG, or plasmapheresis therapy.<sup>3</sup> Our patient was diagnosed as multiphasic pediatric ADEM with criteria including a new event of ADEM, which is a polyfocal, clinical CNS event with presumed inflammatory demyelination, encephalopathy, and MRI findings including diffuse large T2/Flair hyperintensities in white matter and basal ganglia that occurred  $\geq 3$  months after the initial event and associated with new or reemergence of previous clinical and MRI findings.<sup>4</sup> Although the multiphasic ADEM criteria reported in 2007 included the condition of the occurrence of new attack 1 month after the interruption of steroid therapy, the revised 2012 multiphasic ADEM criteria did not mention timing of steroid therapy cessation as pertinent.<sup>4</sup> Our patient had 10 relapses with involvement of new areas on MRI and the attacks occurred within 4 weeks of tapering steroid therapy, so was considered as steroid-dependent multiphasic ADEM.<sup>4,5</sup> Revised ADEM diagnosis report indicates that relapsing diseases after ADEM will be no longer consistent with multiphasic ADEM but rather indicate a chronic disorder most often leading to the diagnosis of MS or NMO. Although our patient developed multiple attacks after the first ADEM attack, none of these clinical and radiologic

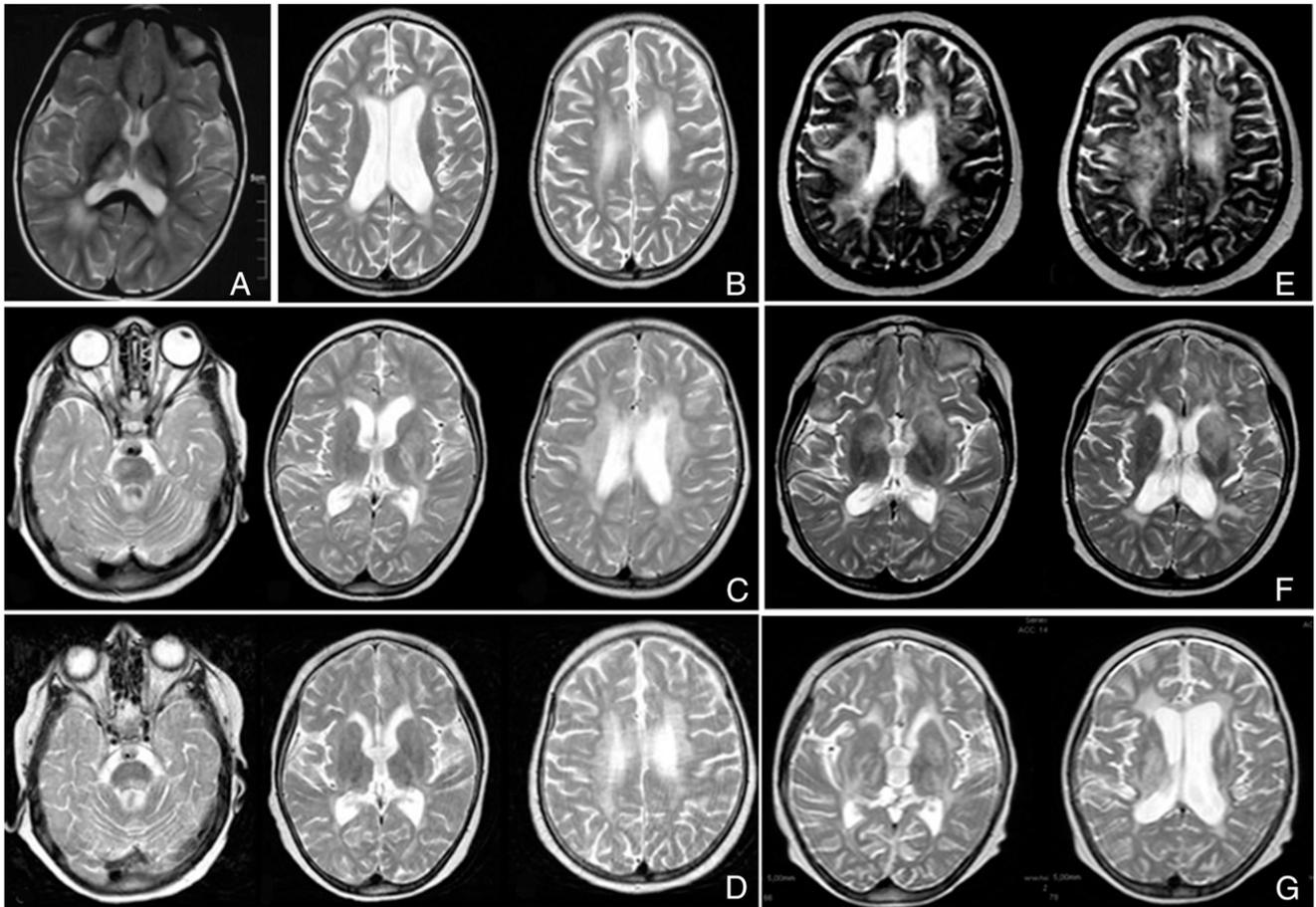
**TABLE 1** Timeline Table of Attacks with Clinical Features, MR Findings, Treatment Options, and Durations of Our Case

Episode	1	2	3	4	5	6	7	8	9	10
Months from onset	0.	10.	25.	34.	38.	41.	44.	48.	58.	64.
Pulse methylprednisolone (days)	↑↑↑	NT	↑↑↑	↑↑↑	↑↑↑	↑↑↑↑↑	↑↑↑↑↑	↑↑↑	↑↑↑↑↑	↑↑↑↑↑
IVIg (days)	NT	↑↑	NT	NT	NT	NT	NT	NT	NT	↑↑
Nonsteroid period before attack, wk	NS	NS	NS	NS	6	8	2	2	—	—
MMF	NT	NT	NT	NT	NT	NT	+	NT	NT	NT
AZA	NT	NT	NT	NT	NT	NT	NT	+	+	+
Unconsciousness	+	+	+	+	+	+	+	+	+	+
Fever	+	+	-	-	-	-	-	+	+	-
GTC seizure		+								
Gait disturbance	+	-	+	+	+	+	+	+	+	+
Speech difficulty	-	-	-	-	-	-	-	+	+	+
Cranial nerve palsy	-	-	+	+	+	+	+	+	+	+
Ataxia	-	-	-	+	+	+	+	+	+	+
MRI	ST, BG	ST, BS	ST	ST, BG	ST	BG, BS	BS	ST	ST, BG	ST, BG
Other MRI	ND	Orbital MR N, Spinal MR N, MRA N	Spinal MR N	Diffusion MR N	Spinal MR N, Orbital MR N, MRA N,	ND	ND	Spinal MR N, Orbital MR N	Diffusion MR N, MRA N	ND
CSF OCB	ND	ND	+	-	-	-	ND	-	ND	ND
CSF IgG index	ND	ND	N	Elev	Elev	N	N	N	ND	ND
Other	ND	EEG N	ND	ND	AQP4 (-) <sup>a</sup> VEP N, BAEP N, SEP defect +	ND	ND	JCV (-) <sup>b</sup>	AQP4 (-) <sup>a</sup>	MOG (+) <sup>a</sup>
		EMG N						BKV (-) <sup>b</sup>	MOG (+) <sup>a</sup>	

Summary of attacks with clinical features, MR findings, treatment options, and durations of our case. BAEP, brainstem auditory evoked potentials; BG, basal ganglia/thalamus; BKV, polyoma BK virus; BS, brainstem; EEG, electroencephalography; Elev, elevated; EMG, electromyography; GTC, generalized tonic clonic; JCV, polyoma John Cunningham virus; MRA, magnetic resonance angiography; MRS, magnetic resonance spectroscopy; N, normal; ND, not done; NS, not specified; NT, not treated; OCB, oligoclonal band; SEP, somatosensory evoked potentials; ST, supratentorial white matter; VEP, visual evoked potentials; +, positive; -, negative; -, treatment course and number of doses.

<sup>a</sup> Antibodies detected in serum.

<sup>b</sup> Investigated in CSF.



**FIGURE 1**

A, At the first attack, transverse T2-weighted fast spin-echo MRI demonstrates bilateral hyperintense lesions located in the periventricular and subcortical white matter. Involvement of bilateral thalami is also seen. B, Transverse T2-weighted fast spin-echo MRIs show new periventricular hyperintense lesions at the fifth attack. Thalamic lesions are regressed. C, Transverse T2-weighted fast spin-echo MRIs show new lesions located at brainstem, left thalami, and left basal ganglia at the sixth attack. D, At the seventh attack, transverse T2-weighted fast spin-echo MRIs reveal new lesion located at dorsal pons. Left basal ganglia and thalamic lesions seen on previous attack are resolved. E, Follow-up MRIs obtained at the eighth attack. Transverse T2-weighted fast spin-echo MRIs show new asymmetric large hyperintense lesions located in the deep periventricular white matter in addition to subcortical white matter involvement. F, At the ninth attack, transverse T2-weighted fast spin-echo MRIs reveal new lesions located at bilateral lentiform nucleus and left caudate nucleus. G, At the last attack, transverse T2-weighted fast spin-echo MRIs reveal new lesion located at bilateral thalamus and basal ganglia with periventricular white matter involvement.

features of attacks were consistent with childhood MS or NMO. First, all of the attacks were accompanied with encephalopathy that did not match the nonencephalopathic event criteria of MS. None of the clinical and radiologic features of attacks revealed MS, optic neuritis (ON), or transverse myelitis. Contrast enhancement was reported in 14% to 30% of ADEM cases. Periventricular lesions were found less commonly in patients with ADEM compared with MS cases. It was reported that lesion locations and number might vary and absence of hypointense lesions

and large lesions are hallmarks for differential diagnosis. Involvement of thalamus and brain stem support ADEM diagnosis.<sup>5</sup> AQP4 antibody was negative, which made us exclude the diagnosis of NMO.<sup>4</sup> Oligoclonal bands are known to be present and transient in 29% of patients with ADEM.<sup>5</sup> CSF oligoclonal band was positive only at the third attack. Although IgG index remained elevated, CSF oligoclonal bands were found negative in follow-up tests. Oligoclonal bands and elevated IgG index are well established in patients with MS, but oligoclonal bands are

rarely observed in demyelinating and chronic inflammatory diseases.<sup>2,5</sup> Extensive search for other infectious, metabolic, and autoimmune diseases were negative. The normalization of MRI lesions between the attacks and improvement of clinical findings after the initial episodes, made us exclude leukodystrophies. Recent studies proposed that myelin basic protein, myelin proteolipid protein, myelin-associated glycoprotein, and MOG are the targets of the immune system in demyelinating diseases.<sup>2,6</sup> MOG is a candidate target antigen in demyelinating CNS diseases. It is

expressed on the outer surface of myelin, which leads to be accessible to autoantibodies from damaged blood-brain barrier.<sup>6</sup> Diseases with MOG IgG antibodies include childhood MS, ADEM, AQP4-negative NMO, recurrent ON, and rarely adult MS. Detection of MOG antibodies may help to differentiate autoimmune and infectious CNS disease and may give prognostic information regarding monophasic or recurrent course.<sup>1</sup> Brilot et al<sup>6</sup> reported that 40% of children with ADEM in their study had high anti-MOG IgG titers. The antibody response against MOG was primarily of the IgG isotype and anti-MOG IgM was rarely found. The exact mechanism of MOG antibodies is not understood but exposure to viral antigens can lead to autoantibody formation via molecular mimicry.<sup>6</sup> It was shown that some children with ADEM had high titers of MOG antibodies ( $\geq 1:160$ ) that rapidly decreased to the undetectable levels after the first attack.<sup>7,8</sup> Strikingly, persistence with fluctuations of anti-MOG IgG was noted in children with MS.<sup>7</sup> MOG antibodies rapidly declined in patients who completely recovered from ADEM. Decreasing MOG antibody titers show favorable clinical outcome in patients with ADEM. In contrast, persistence and fluctuations of MOG antibodies are associated with incomplete recovery of ADEM. Di Pauli et al<sup>3</sup> reported that the patients with full recovery of ADEM were accompanied by a decrease of MOG antibody levels. Follow-up studies showed that positivity of MOG antibodies continued between 4 and 69 months. Patients with ADEM with incomplete recovery had a tendency to stay at positive titer with small fluctuations.<sup>3</sup> Baumann et al<sup>9</sup> showed that 44% of patients with ADEM revealed positive MOG titers (median titer 1:80) during the follow-up period of 9 to 72 months.

High and persisting anti-MOG IgG titers are likely to represent a distinct subgroup of demyelinating diseases with important clinical and therapeutic implications. Rostasy et al<sup>8</sup> showed that the presence of high-titer anti-MOG IgG in a subgroup of patients with recurrent ON as compared with children with monophasic ON. In their study, the patients with recurrent attacks needed to be treated with immunomodulators, including AZA, rituximab, and IVIG. It was mentioned that the children with recurrent ON episodes have persistent high-titer MOG antibodies and the children with anti-MOG IgG-positive NMO had more benign disease course with response to milder immunomodulation therapies.<sup>8</sup> Sato et al<sup>10</sup> reported that NMO spectrum disorders with positive MOG and negative AQP4 antibodies more often have single attack and better functional recovery. According to recent reports, it cannot be said with certainty that antibody positivity predicts the subtype of demyelinating disease. Our patient was admitted to our clinic 38 months after the first attack. We were able to investigate MOG antibody level at the ninth attack, which occurred almost 5 years after the first attack. Transfected MOG cells with a formalin fixation are used for the anti-MOG IgG immunofluorescence assay and the standard value is  $< 1:10$ , and takes place in PBS Tween.<sup>11</sup> The positive titers (1:100) still persisted while she was in remission. She responded less well to the combination of low-dose steroid and AZA treatment. AZA treatment led us to use steroid at low doses and significantly decreased the number of attacks. MOG antibody was found positive but it did not decrease to undetectable levels. Due to the usage of immunosuppressant agents for a long period, we might not find very

high-titer MOG antibodies. Although bystander activation cannot be completely excluded, MOG antibody low-titer persistency suggests that recurrent attacks and steroid-dependent course of the disease may be associated with it.

In conclusion, MOG antibodies should be searched in children with multiphasic ADEM and steroid-dependent cases. These patients also may benefit from addition of AZA, mycophenolate mofetil, or IVIG treatment, which may decrease the number of attacks. Thus, persistent MOG antibodies promise early appropriate treatment choice for a patient with ADEM who has the risk of recurrence or incomplete recovery of ADEM.

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#### ABBREVIATIONS

ADEM: acute disseminated encephalomyelitis  
AQP4: aquaporin 4 antibody  
AZA: azathioprine  
CNS: central nervous system  
CSF: cerebrospinal fluid  
Ig: immunoglobulin  
IVIG: intravenous immunoglobulin  
MMF: mycophenolate mofetil  
MOG: myelin oligodendrocyte glycoprotein  
MR: magnetic resonance imaging  
MS: multiple sclerosis  
NMO: neuromyelitis optica  
ON: optic neuritis

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