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Original article

# MRI findings in pediatric neuromyelitis optica spectrum disorder with MOG antibody: Four cases and review of the literature

Azusa Ikeda [a,b,*], Yoshihiro Watanabe [a], Hikari Kaba [a,b], Kimihiko Kaneko [c,d], Toshiyuki Takahashi [c,e], Saoko Takeshita [a]

[a] Department of Pediatrics, Yokohama City University Medical Center, Japan
[b] Department of Neurology, Kanagawa Children's Medical Center, Japan
[c] Department of Neurology, Tohoku University Graduate School of Medicine, Japan
[d] Department of Neurology, Miyagi National Hospital, Japan
[e] Department of Neurology, Yonezawa National Hospital, Japan

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**Abstract**

*Background:* Myelin oligodendrocyte glycoprotein antibodies (MOG Abs) are frequently detected in pediatric acquired demyelinating syndrome (ADS), and MOG-Ab-positive ADS differs from multiple sclerosis (MS) and aquaporin-4 (AQP4)-Ab-positive neuromyelitis optica spectrum disorder (NMOSD) in terms of age distribution, therapeutic response, and prognosis.

*Methods:* Based on medical records, we retrospectively evaluated patients with MOG-Ab-positive NMOSD treated in the acute phase who were followed up in the chronic phase at our hospital from January 2011 to December 2017.

*Results:* The patients comprised two boys and two girls aged 3–12 (median, 8) years. Peak MOG-Ab titers were 1:2048 to 1:32768 (median, 1:10240), and the relapse rate ranged from 0 to 1.25 times/year (median, 0.59 times/year); no sequelae were observed in any cases. Lesions other than those of optic neuritis were distributed at the cortex in one patient, subcortical white matter in four, deep white matter in three, and brainstem in one, all of which were disseminated lesions. No lesions were found in the corpus callosum, periventricular white matter, diencephalon, and regions adjacent to the third and fourth ventricles. The lesions tended to be asymptomatic, and two patients aged >5 years had well-demarcated lesions.

*Conclusion:* All the patients showed disseminated lesions in the subcortical region to deep white matter, which were different from those found in MS and AQP4-Ab-positive NMOSD and were consistent with the characteristics of brain lesions in MOG-Ab-positive ADS, including other disease types.


*Keywords:* Myelin oligodendrocyte glycoprotein antibody; Neuromyelitis optica spectrum disorder; Children; Magnetic resonance imaging; Brain

## 1. Introduction

Myelin oligodendrocyte glycoprotein antibodies (MOG-Abs) are frequently detected in pediatric acquired demyelinating syndromes (ADSs), such as acute disseminated encephalomyelitis (ADEM), multiphasic disseminated encephalomyelitis (MDEM), optic neuritis (ON), and myelitis [1–3]. In adult neuromyelitis optica spectrum disorder (NMOSD), MOG Abs are detected in 7%–20% of cases [4]; however, it is presumed to be more frequent in children.

Recent studies on ADS with MOG Abs have revealed a good response to steroids in the acute phase [5], poor

* Corresponding author at: Department of Neurology, Kanagawa Children's Medical Center, 2-138-4, Mutsukawa, Minami-ku, Yokohama, Kanagawa 232-8555, Japan.
*E-mail address:* azusaikeda39@gmail.com (A. Ikeda).

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response to disease-modifying drugs used in multiple sclerosis (MS) [6], and better prognosis than that of MS or aquaporin-4 antibody (AQP4-Ab)-positive NMOSD; however, there are recurrences [7]. Thus, MOG-Ab-positive ADS differs from MS and AQP4-Ab-positive NMOSD in terms of age distribution, therapeutic reactivity, and prognosis [8]; a distinction between MOG-Ab-associated diseases and other ADSs has been proposed [7].

There have been recent reports on the neuroradiological findings of MOG-Ab-associated disease; lesions are frequently observed in the cortex, subcortical and deep white matter, basal ganglia, thalamus, and infratentorial region in pediatric MOG-Ab-positive MDEM [9]. Infants tend to exhibit large and poorly demarcated lesions in MOG-Ab-positive demyelinating diseases [10], although there are only a few reports supporting this implication. The present study aimed to clarify the features of the brain neuroradiological findings in MOG-Ab-positive NMOSD.

## 2. Subjects and methods

Based on medical records, we retrospectively evaluated patients with MOG-Ab-positive NMOSD treated in the acute phase and followed up in the chronic phase at our hospital from January 2011 to December 2017. Patients aged <15 years at disease onset were included. The diagnosis of NMOSD was made according to the revised NMOSD diagnostic criteria (Wingerchuck et al. Neurology 2015 [11]). The MOG-Ab was detected using a cell-based assay [12]. MOG-Ab titers of ≥1:160 were classified as seropositive, as previously described [13].

We investigated the age at onset, sex, lesion distribution, clinical symptoms, peak MOG Ab titer, cerebrospinal fluid findings at onset, treatment in the acute and chronic phases, relapse rate, and prognosis [presence or absence of visual impairment and Expanded Disability Status Scale (EDSS) findings]. Furthermore, MRI brain lesion parameters, such as the detailed site, size (whether it exceeded 2 cm axially), demarcation, and gadolinium enhancement, were examined.

This research was approved by the ethics committee of Yokohama City University Center for Novel and Exploratory Clinical Trials (approval number: B180300062).

## 3. Results

The subjects comprised two boys and two girls aged 3–12 (median, 8) years. Table 1 shows the lesion distribution, clinical symptoms, peak MOG Ab titer, treatment, relapse rate, and prognosis for each patient. All the patients showed transverse myelitis extending over three contiguous segments, and three patients showed optic neuritis. All the patients had a high MOG Ab titer, which peaked at the disease onset. AQP4 Ab findings were negative in all cases. In the acute phase, all the patients were treated with intravenous methylprednisolone and three with plasma exchange. In the chronic phase, all the patients were

Table 1
Clinical information, treatment, and prognosis of patients.

| | | Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|---|---|
| Age at onset, sex | | 3y10m, F | 6y4m, M | 10y2m, F | 12y7m, M |
| MRI lesions | Optic neuritis | + | + | − | + |
| | Myelitis | + (C2–7, Th2-6) | + (C3–Th12) | + (C2–5) | + (C2–4, C7–Th4) |
| | Cerebral lesions (Supratentorial) | + | + | + | + |
| | Cerebral lesions (Infratentorial) | − | − | + | − |
| Initial symptoms | | Optic neuritis | Lower extremity weakness | Upper extremity paresthesia<br>Lower extremity weakness | Optic neuritis<br>Psychological symptom |
| Symptoms of encephalopathy | | − | − | − | − |
| Peak MOG-Ab titer | | 1:32768 | 1:16384 | 1:2048 | 1:4096 |
| Acute phase treatment | | IVMP<br>PE | IVMP<br>PE | IVMP | IVMP<br>PE |
| Chronic phase treatment | | PSL, TAC, and AZA | PSL | PSL | PSL and AZA |
| Relapse rate (per year) | | 1.25 | 0.52 | 0.67 | 0 |
| Follow-up period (months) | | 87 | 46 | 18 | 19 |
| EDSS | | 0.0 | 0.0 | 0.0 | 0.0 |
| Visual disturbance | | none | none | none | none |

M = male; F = female; AZA = azathioprine; EDSS = Expanded Disability Status Scale; IVMP = intravenous methylprednisolone; MOG Ab = myelin oligodendrocyte glycoprotein antibody; PE = plasma exchange; PSL = prednisolone; TAC = tacrolimus.

treated with oral prednisolone, two with azathioprine, and one with tacrolimus. The relapse rate ranged from 0 to 1.25 times/year (median, 0.59 times/year), and no sequelae were observed in any case. The cerebrospinal fluid findings at onset showed pleocytosis in three patients and positive myelin basic protein (MBP) in three. The oligoclonal band (OCB) results were negative in all the cases (Table 2).

Brain MRI findings for each case are shown in Fig. 1. Case 1 had bilateral ON at onset, with asymptomatic disseminated lesions in the bilateral temporal subcortical region to deep white matter. Three years after the recurrence of right ON and transverse myelitis, asymptomatic disseminated lesions were newly detected in the bilateral parietal subcortical white matter. Case 2 had C3-Th12 transverse myelitis at onset, which recurred with C2-7 transverse myelitis. During the second episode, asymptomatic disseminated lesions in the bilateral frontal and temporal subcortical white matter were detected. Case 3 had C2-5 transverse myelitis at onset, with asymptomatic lesions in the left temporal white matter. The lesions disappeared once; however, after 4 months, disseminated lesions appeared in the bilateral parietal lobes, accompanied with headache. Case 4 had bilateral ON and psychiatric symptoms at onset, with bilateral cerebral cortex and subcortical white matter lesions. Although he had one remission, after 6 months, asymptomatic disseminated lesions in the bilateral white matter near the trigone of the lateral ventricle were detected.

Table 3 summarizes brain lesions other than those in ON in each case. Lesions were distributed in the cortex in one patient, subcortical white matter in four, deep white matter in three, and brainstem in one, all of which were disseminated lesions. No lesions were found in the corpus callosum, periventricular white matter, diencephalon, and regions adjacent to the third and fourth ventricles. Well-demarcated lesions were detected in Cases 2 and 4 and poorly demarcated lesions in Cases 1 and 3. A large lesion of >2 cm was detected only in Case 1 (age, 3 years). No symptoms of encephalopathy were observed in any of the patients. In Cases 1 and 2, the lesions were asymptomatic; in total, five of seven occurrences of brain lesions were asymptomatic, and all showed a trend toward disappearance with disease amelioration.

Table 2
CSF findings at onset.

| | Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|---|
| Cell count (/μL) | 30 | 152 | 8 | 36 |
| Protein (mg/dL) | 23 | 32 | 35 | 40 |
| MBP (pg/mL) | 290.0 | 399.5 | 514.0 | Negative |
| OCB | Negative | Negative | Negative | Negative |

CSF = cerebrospinal fluid; MBP = myelin basic protein; OCB = oligoclonal band.

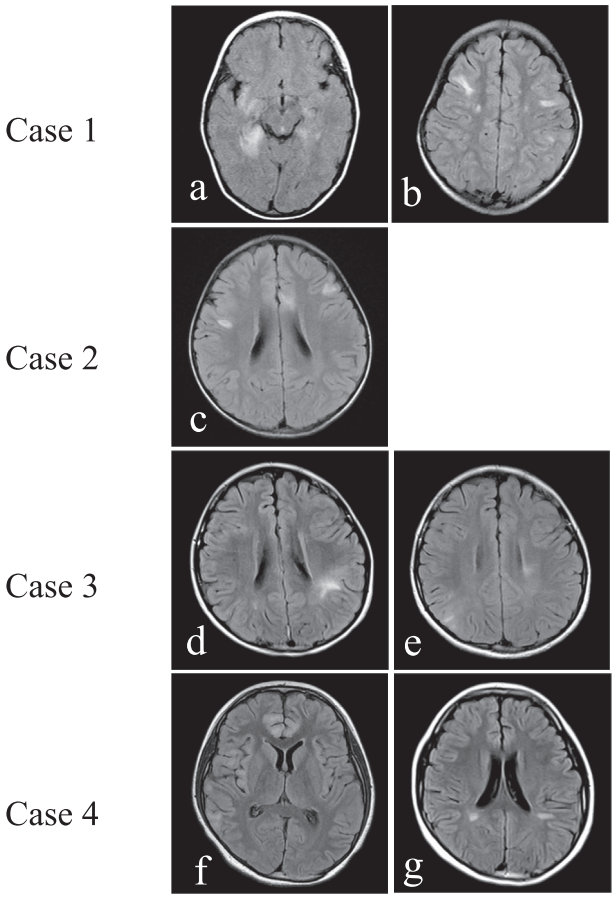


Fig. 1. Brain MRI findings. All are T2 FLAIR images. a) First episode of Case 1 with optic neuritis. A large (>2 cm) poorly demarcated lesions in the right temporal white matter and disseminated lesions in the bilateral subcortical region to deep white matter were detected. (b) New brain lesions without clinical symptoms in Case 1. Disseminated poorly demarcated lesions were detected in the parietal subcortical white matter. (c) Second episode of Case 2 with transverse myelitis. Well-demarcated lesions were detected in the bilateral frontal to temporal subcortical white matter. (d) First episode of Case 3 with transverse myelitis. Poorly demarcated lesions were detected in the left temporal white matter. (e) Second episode of Case 3 with headache. Faint disseminated lesions were newly detected in the parietal cortex to subcortical white matter. (f) First episode of Case 4 with optic neuritis and psychiatric symptoms. Poorly demarcated lesions were detected in the bilateral cortex to subcortical white matter. (g) New brain lesions without clinical symptoms in Case 4. Well-demarcated lesions were detected in the bilateral white matter.

## 4. Discussion

Brain lesions in NMOSD associated with AQP4 abs are localized to areas of high AQP4 expression. In this study, it was suggested that brain neuroradiological findings in MOG-Ab-positive NMOSD were different from those in AQP4-Ab-positive NMOSD and MS in four pediatric patients. In all the patients, brain lesions other than those of optic neuritis, were mainly distributed in the subcortical region to deep white matter, and all were supra- or infratentorial disseminated

Table 3
Distribution and features of brain MRI lesions.

| | Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|---|
| Cortical | − | − | − | + |
| Subcortical white matter | + | + | + | + |
| Deep white matter | + | − | + | + |
| Lesions surrounding lateral ventricle | − | − | − | − |
| Corpus callosum | − | − | − | − |
| Thalamus or hypothalamus | − | − | − | − |
| Brainstem | − | − | + | − |
| Cerebellum | − | − | − | − |
| Lesions surrounding cerebral aqueduct or third or fourth ventricle | − | − | − | − |
| Large lesion (>2-cm axial) | + | − | − | − |
| Well-demarcated lesion | − | + | − | + |
| Gadolinium enhancement | − | ND | ND | − |

ND = not done.

lesions. No lesions were detected in the corpus callosum or periventricular white matter, which are frequently observed in MS [14] or in the diencephalon or regions adjacent to the third and fourth ventricles, which are commonly observed in AQP4-Ab-positive NMOSD. Furthermore, the brain lesions were frequently asymptomatic, with five of seven occurrences being asymptomatic in total. Notably, well-demarcated lesions were detected in two children aged >5 years.

It has been reported that the frequency of occurrence of brain lesions in NMOSD is 55%, and children have complicated brain lesions more frequently than those in adults [15]. Brain lesions in NMOSD are localized to areas of high AQP4 expression, such as periventricular lesions of the third (diencephalon) and fourth (brainstem) ventricles, supra- and infratentorial white matter, midbrain, and cerebellum [4]. Particularly, lesions in the diencephalon, hypothalamus, and dorsal medulla are specific to AQP4-Ab-positive cases [16]. Our four MOG-Ab-positive cases did not show these distributions despite exhibiting the same NMOSD, which suggested that the pathological mechanism in these cases was different from that in the AQP4-Ab-positive cases. The precise function of MOG is not clear, but the expression of MOG in mature oligodendrocytes suggests a role in maturation, myelin integrity, and cell surface interaction [17]. Histopathological findings of demyelination caused by an antibody reaction against MOG expressed on the cell surface have been shown [18], and MOG Ab is recognized as a cause of demyelinating disease.

A summary of the past reports on brain MRI findings in pediatric ADS with MOG-Ab is shown in Table 4. The disease type included ADEM, MDEM, NMOSD, ON, transverse myelitis, clinically isolated syndrome,

Table 4
Brain MRI features of pediatric acquired demyelinating syndromes with MOG-Ab.

| Reference | Total (n) | Children (n) | Age at initial presentation (years) | Disease type (n) | Brain MRI features |
|---|---|---|---|---|---|
| Baumann, 2015 [23] | 19 | 19 | 5 (1–17) (median, range) | ADEM (19) | Supratentorial white matter, thalamus/basal ganglia, spinal, bilateral, hazy, >2 cm |
| Baumann, 2016 [9] | 8 | 8 | 3 (1–7) (median, range) | MDEM (8) | Juxtacortical, deep white matter, brainstem, blurred, >2 cm |
| Fernandez-Carbonell, 2016 [19] | 13 | 13 | 7.7 (median) | MS (4), ADEM (3), CIS (2), NMO (2), non-specific demyelinating diseases (2) | subcortical, absence of corpus callosum |
| Lechner, 2016 [21] | 25 | 25 | 7 (2–15) (median, range) | NMO (7), LETM (6), ON (12) | Less periependymal, nonspecific, ADEM-like (blurred, hazy, and large) |
| Cobo-Calvo, 2017 [22] | 27 | 13 | 6.8 (1.7–13.8) (median, range) | ADEM (6), ON (3), NMOSD (2), TM (1), MS (1) | Bilateral thalamic lesions in ADEM |
| Hacohen, 2017 [7] | 26 | 26 | No data | NMOSD (15), MDEM (9), ON (2) | Leukodystrophy pattern, cortical gray matter, thalamus, juxtacortical, deep white matter, cerebellar peduncle |
| Juryńczyk, 2017 [24] | 21 | 6 | 9.1 (mean) | No data | Brainstem, thalamus, basal ganglia, periventricular, subcortical, bilateral, large, fluffy |
| Baumann M, 2018 [10] | 69 | 69 | 2 (1–7) (median, range) | ADEM(36), ON(16), TM (8), NMOSD(5), CIS(3), MS(1) | Poorly demarcated lesion in ADEM and NMOSD, poorly defined and widespread lesions in younger children |

ADEM = acute disseminated encephalomyelitis; CIS = clinically isolated syndrome; LETM = longitudinally extensive transverse myelitis; MDEM = multiphasic disseminated encephalomyelitis; MS = multiple sclerosis; NMO = neuromyelitis optica; NMOSD = neuromyelitis optica spectrum disorders; ON = optic neuritis; TM = transverse myelitis.

and MS. Several reports indicated that lesions are distributed in the cortex, subcortical region, deep white matter, brainstem, thalamus, and basal ganglia and are characterized by poor demarcation and large size. These features have been found particularly in ADEM and MDEM, but similar ADEM-like lesion distribution has also been observed in other disease types. Hacohen et al reported that pediatric AQP4-Ab-positive ADS cases were more likely to have lesions restricted to the brainstem and hypothalamus, and that lesions in the dorsal medulla were more frequently observed in AQP4-Ab-positive cases, whereas cerebellar peduncle and leukodystrophy-like lesions were observed only in MOG-Ab-positive cases [7]. Fernandez-Carbonell et al reported the absence of corpus callosum lesions in pediatric MOG-Ab-positive ADS [19]. The disease type in our four cases was NMOSD, but the brain lesion features were similar to those in MOG-Ab-positive ADS, including all disease types.

Baumann et al reported that the lesion features in pediatric MOG-Ab-positive ADS were strongly influenced by age [10]. In particular, children aged <5 years tended to have poorly demarcated and widespread lesions, whereas those age >5 years tended to have well-demarcated lesions. In this study, well-demarcated lesions were observed in two patients who were aged >5 years, which is consistent with the previous report. Myelin development and changes in the MOG expression site are thought to influence age-dependent lesion distribution [20,21]; a similar mechanism is presumed for the association between age and disease type because younger children mostly account for ADEM cases and older children for mostly ON cases [19].

Although most brain lesions in this study showed a distribution similar to that in ADEM, all the cases were not accompanied by encephalopathic symptoms, such as impaired consciousness or seizure. There are reports on asymptomatic white matter lesions in MOG-Ab-positive NMOSD [21] and the frequency of encephalopathic symptoms in younger children with MOG-Ab-positive ADS [22]. The lesions in our patients were thought to be asymptomatic due to the fact that lesions in the white matter are more likely to be asymptomatic and that the progression of myelination with age affects the occurrence of lesions.

Our study was limited by the small number of patients and its retrospective, single-center design. However, in this study, the features of brain lesions in the unified pathology of MOG-Ab-positive NMOSD were suggested.

In conclusion, all patients with MOG-Ab-positive NMOSD had disseminated lesions distributed in the subcortical region to deep white matter, which were different from those found in patients with MS and AQP4-Ab-positive NMOSD but consistent with those in MOG-Ab-positive ADS, including other disease types. These features suggested a new spectrum of MOG-Ab-related diseases.

## Conflicts of interest

The authors have no financial or personal relations that could pose a conflict of interest.

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