


Clinical Features of Patients with Myelin Oligodendrocyte Glycoprotein Antibody–Associated Disease and Isolated Seizure Symptoms

Yun Yang^{1,2}, Chao Zhang¹, Chen Cao³, Wenhua Su¹, Na Zhao¹, Wei Yue^{1,4} 

¹Department of Neurology, Clinical College of Neurology, Neurosurgery, and Neurorehabilitation, Tianjin Medical University, Tianjin Huanhu Hospital, Tianjin, People's Republic of China; ²Department of Neurology, The Second Hospital of Tianjin Medical University, Tianjin, People's Republic of China; ³Department of Medical Imageology, Tianjin Huanhu Hospital, Tianjin, People's Republic of China; ⁴Department of Biomedical Engineering, Tianjin University, Tianjin, People's Republic of China

Correspondence: Wei Yue, Department of Neurology, Tianjin Huanhu Hospital, 6 Jizhao Road, Tianjin, 300350, People's Republic of China, Fax +862259065662, Email hhyuewei2021@163.com

Background: Myelin oligodendrocyte glycoprotein (MOG) antibody–associated encephalitis is a new clinical phenotype of inflammatory demyelinating diseases. Some MOG antibody–positive patients with central nervous system demyelinating events present with isolated seizures. However, there are gaps in the epidemiological knowledge regarding seizures with MOG antibody–associated encephalitis in adults. This study characterized the clinical features and treatment of MOG antibody–positive patients with isolated seizures.

Methods: We reviewed all the patients admitted to Tianjin Huanhu Hospital between Jan. 1st 2017 and Jan. 1st 2022, to screen the MOG antibody–positive patients with isolated seizures, and collected the concerned patients' information regarding epidemiology, clinical presentations, laboratory and radiological characteristics, electroencephalogram (EEG), treatments, and prognoses.

Results: We collected six MOG antibody–positive adult patients who had isolated symptomatic seizures. The mean age of the patients was 33 years (range, 29–40 years), and five (83.3%) were men. All patients presented with motor seizures, five (83.3%) had cognitive dysfunction, and only one (16.7%) had status epilepticus. Five (83.3%) patients had a good response to immunotherapy and antiseizure medications; only one had a sequela. The cerebrospinal fluid or serum anti-MOG antibody test turned negative over time.

Discussion: The most common seizure type in patients with MOG antibody–associated encephalitis with isolated seizures was focal to bilateral tonic-clonic seizures, and most patients had a good prognosis. Adding antiseizure medications were beneficial for MOG antibody–positive patients with seizures. Relapses and sequelae were associated with low-dose, short-time, or delayed therapy, and wide-range demyelinating brain damage.

Plain Language Summary: We reported six adult patients with seizures (as the isolated symptom without any ophthalmoneuromyelitis symptom) associated with MOG antibodies. Focal to bilateral tonic-clonic seizures are the common seizure type in patients with myelin oligodendrocyte glycoprotein (MOG) antibodies. Antiseizure medications and immunotherapy are beneficial for treating MOG antibody–associated encephalitis with seizures. Many patients had good or complete recovery; low-dose, short-time, or delayed therapy is associated with relapses and sequelae. Emphasized the MOG antibodies testing should be relevant in patients with isolated seizures, particularly occurred in clusters, to guarantee the acute management timely.

Keywords: myelin oligodendrocyte glycoprotein, antibody-associated disease, seizure, clinical features, antiseizure medications

Introduction

Myelin oligodendrocyte glycoprotein (MOG) is a membrane protein uniquely expressed in the outermost lamellae of the myelin sheath in oligodendrocytes of the central nervous system (CNS).^{1–3} MOG antibody–associated disease is a new clinical phenotype of inflammatory demyelinating disease.^{4–6} In particular, MOG antibody–associated encephalitis is mostly characterized by acute-disseminated encephalomyelitis, multiphasic disseminated encephalomyelitis, neuromyelitis optica spectrum disorder, optic

neuritis, myelitis, encephalitis, brainstem encephalitis, and a series of less typical syndromes such as encephalitis-like seizures, involuntary movement, nausea, vomiting, hiccups, and cranial neuritis.^{7–10} Compared with multiple sclerosis and aquaporin-4 antibody-associated disease, MOG antibody-associated disease is an age-dependent demyelinating disease; adults have a lower prevalence than children (the positive frequency in adults is nearly half of that in children [22–23% vs 39–40%]), and many patients present with the ophthalmoneuromyelitis phenotype.^{11–13} Recent evidence increasingly indicates that patients with CNS demyelinating events who are MOG antibody-positive present with isolated seizures.^{14,15} However, there are gaps in the epidemiological knowledge regarding seizures in adults with this disease. In this study, we reported and analyzed the occurrence and evolution of isolated symptomatic seizures in MOG antibody-positive patients to characterize the clinical symptoms, treatment effects, and prognoses.

Method

Study Design, Population and Statistical Analysis

The retrospective study was conducted to characterize the clinical features. We reviewed all the patients admitted to Tianjin Huanhu Hospital between Jan. 1st 2017 and Jan. 1st 2022. A total of 57 patients with seropositive and/or cerebrospinal fluid (CSF)-positive were screened out. Six patients with epileptic seizures as an isolated symptom were included for further analysis. All patients underwent MOG antibody examinations during the acute and follow-up phases. We collected information regarding epidemiology (age, sex and head trauma history), clinical presentations (the neurological and seizure symptoms), cerebrospinal fluid (CSF) examinations, MOG-immunoglobulin G (IgG) serum and CSF titers, laboratory and radiological characteristics, electroencephalogram (EEG), treatments, and prognoses. Included patients were follow-up by telephone at April 2022. All concerned information about the patients in this case series was collected from hospital notes and self reporting. These data were summarized into a table for further analysis.

Definition of Variables

Form of onset was grouped by the presented epileptic seizure time from the first clinical symptoms; acute indicated less than 15 days, sub-acute indicated between 15 days to 3 months. The seizure symptoms were analyzed in three aspects, whether it is a status seizure, motor symptom, and impaired awareness or not. Status seizure is defined as a seizure more than 5 minutes or the patient does not regain consciousness in between the continuous seizure activity. Clinical relapse was defined as sudden seizure onset and/or the presence of new lesions on magnetic resonance imaging (MRI) after treatment during the acute phase. Seizure free was defined as without any epileptic seizure during follow-up.

Results

We described six adult patients who experienced isolated symptomatic seizures and MOG antibody-associated encephalitis and were found to have MOG antibodies in the CSF and/or serum via cell-based assays. The mean age was 33 years (29–40 years), and five (83.3%) patients were men. All six patients presented with motor seizures, and five (83.3%) presented with impaired awareness. One patient had a status epilepticus (case 1). Five (83.3%) patients had focal to bilateral tonic-clonic seizures, and the other one had a focal seizure. All seizures occurred in a cluster over a short interval (2–3 days), and the patients developed anti-MOG antibody-positivity afterwards; one patient was positive for anti-MOG antibody in the third lumbar CSF puncture 4 months after the first seizure (case 6). The frequency of general clinical symptoms among the patients was as follows: five (83.3%) patients reported headaches, three (50%) reported fever, and five (83.3%) reported preceding infection history—three patients had an upper respiratory infection and three patients had a gastrointestinal infection. The disease duration ranged from 0 to 40 days (Table 1, Supplementary Table 1).

Examination of the CSF revealed increased intracranial pressure (range, 231–333 mmH₂O) in five (83.3%) patients, increased leukocyte levels (range, 10–224*10⁶ /L) in all patients, and increased protein levels (range, 0.58–1.25 g/L) in three (33.6%) patients. Three (50.0%) patients had CSF positive for anti-MOG antibodies, and five (83.3%) had seropositivity for the antibodies. Other autoimmune antibodies, including the anti-AQP4 antibody, anti-N-methyl-D-aspartate receptor antibody, anti-leucine-rich glioma inactivated 1 (LGI1) antibody, the anti γ -aminobutyric acid (GABA) type A receptor antibody, the anti-GABA_B receptor antibody, antinuclear antibody, and oligoclonal band (OB) were negative.

Table I Clinical, Treatment and Follow-Up Data in Patients with MOG-IgG-Associated Seizures

| Characteristic | Value |
|-------------------------------------------------------------------|-------------|
| Age, years, mean (range) | 33 (29–40) |
| Male, n/total (%) | 5/6 (83.3%) |
| Disease duration at presentation, days, mean (range) | 17 (0–40) |
| Headache, n/total (%) | 5/6 (83.3%) |
| Fever, n/total (%) | 3/6 (50.0%) |
| Temperature higher 38.5 degrees, n/total (%) | 2/3 (66.7%) |
| Preceding infection, n/total (%) | 5/6 (83.3%) |
| Form of onset | |
| Acute, n/total (%) | 2/6 (33.3%) |
| Sub-acute, n/total (%) | 4/6 (66.7%) |
| Seizure Symptom | |
| Status seizure, n/total (%) | 1/6 (16.7%) |
| Motor symptom, n/total (%) | 6/6 (100%) |
| Impaired awareness, n/total (%) | 5/6 (83.3%) |
| Seizure Type | |
| Focal to bilateral tonic-clonic seizure, n/total (%) | 5/6 (83.3%) |
| Focal seizure, n/total (%) | 1/6 (16.7%) |
| Anti-MOG antibody positive (CSF), n/total (%) | 3/6 (50.0%) |
| Anti-MOG antibody positive (Serum), n/total (%) | 5/6 (83.3%) |
| CSF examination | |
| High pressure (normal: 80–180 mmH ₂ O), n/total (%) | 5/6 (83.3%) |
| Leukocytes increased (normal: <8*10 ⁶ /L), n/total (%) | 6/6 (100%) |
| Protein increased (normal: 0.15–0.45 g/L), n/total (%) | 3/6 (50.0%) |
| Brain MRI | |
| FLAIR hyperintensity, n/total (%) | 6/6 (100%) |
| Treatment | |
| Intravenous methylprednisolone, n/total (%) | 6/6 (100%) |
| Immunoglobulin, n/total (%) | 1/6 (16.7%) |
| Follow-up, months, mean (range) | 35 (6–62) |
| Disease course | |
| Monophasic, n/total (%) | 4/6 (66.7%) |
| Relapsing, n/total (%) | 2/6 (33.3%) |

Abbreviations: CSF, cerebrospinal fluid; FLAIR, fluid attenuated inversion recovery.

Reverse transcription–polymerase chain reaction (RT-PCR) analysis of the CSF, as well as next-generation sequencing (NGS), for the presence of herpes simplex virus, cytomegalovirus, and Epstein–Barr virus yielded negative results.

All seizure patients had a focal demyelinating event in the cortical and subcortical areas, and five (83.3%) had single or multiple demyelinations of the white matter; three (50.0%) events were located in the brainstem, one (16.7%) in the basal ganglia, and one (16.7%) in the cerebellum. EEG at the time of admission showed symmetric diffuse slowing waves in five (83.3%) patients. All patients were treated with intravenous methylprednisolone (three patients, 1000 mg; three patients, 500 mg; and one patient, 120 mg), and then with the slowly tapered drug oral methylprednisolone tablets. All patients received antiseizure medications (ASMs), and two (33.3%) patients were treated with two or more ASMs. Levetiracetam was the most commonly administered ASMs, received by all patients.

During follow-up (mean, 35 months; range, 6–62 months), two (33.3%) patients experienced relapse after the acute episode (case 1 relapsed at the second month and case 6 at the fourth month). All patients remained seizure-free with ASMs withdrawal, and only one patient had sequelae with neurological symptoms, performed with behavioral and personality changes. Repeat brain MRI showed the disappearance of T2 hyperintensity (3/6, 50.0%) and reduction of brain cortex swelling (1/6, 16.7%), and the patients turned negative for anti-MOG antibody over time (3/6, 50.0%). The patients' disease durations are shown in [Supplementary Figure 1](#), and we described the details of case 3 in [Supplementary Case Description](#) and [Supplementary Figure 2](#).

Discussion

The present study reported six adult cases of epileptic seizures and MOG antibody–associated encephalitis and investigated the seizure type, epidemiology, comorbidities, imaging data, CSF examination results, treatments, prognoses, and sequelae. Patients with MOG antibody–associated encephalitis are more likely to manifest seizures than those with AQP4-IgG-associated encephalitis.^{16,17} Several studies with large cohorts have determined that seizures occur in different phenotypes of MOG antibody–associated diseases.^{4,18} Clarifying the coexistence of epileptic seizures and MOG antibodies is challenging, especially because of the association of epileptic seizures with multiple symptoms and prognoses. Our study indicated that the most common seizure type was generalized epileptic seizure, which is consistent with previous results.^{14,16} Additionally, seizures occur as single episodes or clusters, and few patients have status epilepticus (only one patient did in our study); most patients present with headaches and fever.^{19–21} Many patients have diffuse signal changes in the cerebral cortex, brainstem, and deep grey matter, and interictal epileptiform discharges (slow waves) are seen in most patients.^{22,23} Based on the current findings, MOG antibody testing should be a part of neuroimmunology laboratory testing in patients with isolated seizures, especially those with MRI abnormalities.

Patients with isolated seizures, which is a clinical symptom of MOG antibody–associated diseases, showed a marked and prompt clinical and radiological improvement after immunotherapy and ASMs therapy.^{14,24} Some cases are seizure free by immunotherapy, even without ASMs administration.^{4,20} Due to the lack of randomized controlled trials, it is difficult to evaluate the efficacy and necessity of ASMs in MOG antibody–associated diseases, and the use of immunotherapy and ASMs is based on case series and clinical experience.^{25,26} However, patients may benefit from ASMs with or without immunotherapy.^{17,27,28} All patients in our study were treated with ASMs and immunotherapy and were advised to discontinue ASMs if they were seizure-free and had a normal EEG during follow-up. We propose adding ASMs to the treatment regimen for MOG antibody–associated encephalitis with seizures, when necessary. This result has also been confirmed in autoimmune encephalitis; while emphasizing the use of immunotherapy to control acute seizures, the use and avoidance of early withdrawal of ASMs are recommended, especially in patients at risk of developing epilepsy.^{29–31}

According to multi-center studies, most patients (ranging from 50% to 90% in different studies) with MOG antibody–associated diseases had a good or complete recovery if they were administered intravenous methylprednisolone at large doses over short periods.^{14,23,32} We also recorded three patients with fully recovered, who had a negative MOG antibody test, and their MRI lesions subsequently disappeared. Compared with AQP4 antibody–positive patients, MOG antibody–positive patients are at a lower risk of further clinical relapse.⁴ Relapses frequently occur during steroid weaning, especially in patients with low-dose steroids (prednisone < 20 mg/day adults), short-term therapy (<3 months), and high MOG antibody–positive titers.^{11,12,16,33–35} After acute attack treatment, treating patients with an additional immunosuppressor (eg, rituximab or

mycophenolate mofetil), can significantly reduce the risk of recurrence and the occurrence of seizures.^{17,36–38} In our study, case 6 showed cerebral cortical demyelination that was progressive (the relapsed phase lesion range was larger than the first phase); the patient's MOG antibody result was positive in the third CSF test (which was negative in sero/CSF during the first and second lumbar punctures), and the seizures were temporally diverse throughout the disease duration. This progressive and relapsed course may have been associated with delayed methylprednisolone therapy. For patients with a high progressive risk and clinical features that are highly compatible with MOG antibody-associated encephalitis, early and full-dose immunotherapy is recommended, even without a positive MOG antibody test.

Some pediatric patients present with various sequelae, including cognitive impairment, severe brain atrophy, and refractory epilepsy; moreover, several cases of catastrophic brain injury and death caused by intracranial hypertension have been reported.^{3,32} In our study, only one patient had sequelae with personality changes, and the symptoms were similar to those reported in a previous case.³⁹ We speculate that the relapse course and irreversible and wide-range demyelinating brain damage affected the outcome. Additional disease-modifying treatments, regular re-examination, and second-line treatments should be used to achieve further substantial improvement.^{14,32,40}

However, this study has limitations that could affect the application of the results. This study was limited by its single-center retrospective analysis design, the small sample size, and a short follow-up period, which may have affected the estimates and was a source of potential bias. Besides, our center is neurological specialized hospital, it is possible that we have encountered only the severe symptoms of MOG antibody-associated encephalitis. This study was retrospective, and the details of seizure were self-provided. Inaccurate recording or reporting about may have affected the result, although we relied on hospital notes whenever possible, which is still a source of potential bias. Future studies should investigate the relevance of MOG antibodies and neurological disorders to improve the recognition of these patients with rare syndromes and determine the pathophysiology, optimal treatment regimens, and long-term prognoses.

In conclusion, we recorded six adult patients presented isolated seizures with MOG antibody-associated encephalitis. All patients had motor seizures, and the most common seizure type was focal to bilateral tonic-clonic seizures. CSF examination showed significantly increased intracranial pressure, increased leukocyte levels (the increases varied from several times to dozens of times), and slightly elevated protein levels. Slow waves were observed in the central, frontal or parietal region. Almost all patients had a good or complete recovery. Low-dose, short-term, or delayed therapy is associated with relapses and sequelae. ASMs are beneficial for treating MOG antibody-associated encephalitis with seizures. Regular re-examinations are recommended to assess illness representations. Immunosuppressors can be used to halt disease progression and prevent relapse, if necessary. We suggest that patients with isolated seizures and MRI abnormalities may be tested for MOG antibodies to ensure timely acute management.

Data Sharing Statement

There are no additional data availability statement in our study.

Ethical Approval and Consent to Participate

Although this research involving human participants, it is an observational study without any intervention. Therefore, this study was granted exemption by the ethics committee of Tianjin Huanhu Hospital. All patients provided written informed consent. The written informed consent has been provided by the patients to publish the case details and accompanying images. We certify that the study was performed in accordance with the 1964 declaration of Helsinki and later amendments.

Acknowledgments

We all appreciated the participated patients in our study.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article

has been submitted; and agree to be accountable for all aspects of the work. Yun Yang and Chao Zhang contributed to the work equally and should be regarded as co-first authors.

Funding

Tianjin Key Medical Discipline (Specialty) Construction Project (No. TJYXZDXK-052B).

Disclosure

All authors declare that they have no conflicts of interest. We confirm that we have read the appropriate checklists and affirm that this report is consistent with the guidelines.

References

- Johns TG, Bernard CC. The structure and function of myelin oligodendrocyte glycoprotein. *J Neurochem*. 1999;72(1):1–9. doi:10.1046/j.1471-4159.1999.0720001.x
- Reindl M, Di Pauli F, Rostásy K, Berger T. The spectrum of MOG autoantibody-associated demyelinating diseases. *Nat Rev Neurol*. 2013;9(8):455–461. doi:10.1038/nrneuro.2013.118
- Ramanathan S, Dale RC, Brilot F. Anti-MOG antibody: the history, clinical phenotype, and pathogenicity of a serum biomarker for demyelination. *Autoimmunity Rev*. 2016;15(4):307–324. doi:10.1016/j.autrev.2015.12.004
- Cobo-Calvo A, Ruiz A, Maillart E, et al. Clinical spectrum and prognostic value of CNS MOG autoimmunity in adults: the MOGADOR study. *Neurology*. 2018;90(21):e1858–e1869. doi:10.1212/WNL.0000000000005560
- Krishnamoorthy G, Lassmann H, Wekerle H, Holz A. Spontaneous opticospinal encephalomyelitis in a double-transgenic mouse model of autoimmune T cell/B cell cooperation. *J Clin Invest*. 2006;116(9):2385–2392. doi:10.1172/JCI28330
- Patterson K, Iglesias E, Nasrallah M, et al. Anti-MOG encephalitis mimicking small vessel CNS vasculitis. *Neurol Neuroimmunol Neuroinflamm*. 2019;6(2):e538.
- Cobo-Calvo A, Vukusic S, Marignier R. Clinical spectrum of central nervous system myelin oligodendrocyte glycoprotein autoimmunity in adults. *Curr Opin Neurol*. 2019;32(3):459–466. doi:10.1097/WCO.0000000000000681
- Hennes EM, Baumann M, Schanda K, et al. Prognostic relevance of MOG antibodies in children with an acquired demyelinating syndrome. *Neurology*. 2017;89(9):900–908. doi:10.1212/WNL.0000000000004312
- Jarius S, Ruprecht K, Kleiter I, et al. MOG-IgG in NMO and related disorders: a multicenter study of 50 patients. Part 2: epidemiology, clinical presentation, radiological and laboratory features, treatment responses, and long-term outcome. *J Neuroinflammation*. 2016;13(1):280. doi:10.1186/s12974-016-0718-0
- Huang C, Tian X, Jiang S, et al. Comparisons between infectious and autoimmune encephalitis: clinical signs, biochemistry, blood counts, and imaging findings. *Neuropsychiatr Dis Treat*. 2020;16:2649–2660. doi:10.2147/NDT.S274487
- Reindl M, Waters P. Myelin oligodendrocyte glycoprotein antibodies in neurological disease. *Nat Rev Neurol*. 2019;15(2):89–102.
- Hegen H, Reindl M. Recent developments in MOG-IgG associated neurological disorders. *Therap Adv Neurol Disord*. 2020;13:1756286420945135. doi:10.1177/1756286420945135
- Thompson AJ, Baranzini SE, Geurts J, Hemmer B, Ciccarelli O. Multiple sclerosis. *Lancet*. 2018;391(10130):1622–1636.
- Ogawa R, Nakashima I, Takahashi T, et al. MOG antibody-positive, benign, unilateral, cerebral cortical encephalitis with epilepsy. *Neurol Neuroimmunol Neuroinflamm*. 2017;4(2):e322.
- Ikeda T, Yamada K, Ogawa R, et al. The pathological features of MOG antibody-positive cerebral cortical encephalitis as a new spectrum associated with MOG antibodies: a case report. *J Neurol Sci*. 2018;392:113–115. doi:10.1016/j.jns.2018.06.028
- Hamid SHM, Whittam D, Saviour M, et al. Seizures and encephalitis in myelin oligodendrocyte glycoprotein IgG disease vs aquaporin 4 IgG disease. *JAMA Neurol*. 2018;75(1):65–71. doi:10.1001/jamaneurol.2017.3196
- Yao Y, Xu Y, Ren H, et al. Acute epileptic seizures in myelin oligodendrocyte glycoprotein encephalomyelitis and neuromyelitis optica spectrum disorder: a comparative cohort study. *Mult Scler Relat Disord*. 2019;27:281–288. doi:10.1016/j.msard.2018.11.007
- de Mol CL, Wong Y, van Pelt ED, et al. The clinical spectrum and incidence of anti-MOG-associated acquired demyelinating syndromes in children and adults. *Mult Scler*. 2020;26(7):806–814.
- Budhram A, Mirian A, Le C, Hosseini-Moghaddam SM, Sharma M, Nicolle MW. Unilateral cortical FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures (FLAMES): characterization of a distinct clinico-radiographic syndrome. *J Neurol*. 2019;266(10):2481–2487. doi:10.1007/s00415-019-09440-8
- Wang YF, Liu XW, Lin JM, Liang JY, Zhao XH, Wang SJ. The clinical features of FLAIR-hyperintense lesions in anti-MOG antibody associated cerebral cortical encephalitis with seizures: case reports and literature review. *Front Immunol*. 2021;12:582768.
- Cobo-Calvo A, Ruiz A, D'Indy H, et al. MOG antibody-related disorders: common features and uncommon presentations. *J Neurol*. 2017;264(9):1945–1955. doi:10.1007/s00415-017-8583-z
- Tokumoto K, Nishida T, Kawaguchi N, Kaneko K, Takahashi T, Takahashi Y. Electroclinical features of seizures in myelin oligodendrocyte glycoprotein antibody-associated cerebral cortical encephalitis: a case report and literature review. *Seizure*. 2022;98:13–18. doi:10.1016/j.seizure.2022.04.001
- Foiadelli T, Gastaldi M, Scaranzin S, Franciotta D, Savasta S. Seizures and myelin oligodendrocyte glycoprotein (MOG) antibodies: two paradigmatic cases and a review of the literature. *Mult Scler Relat Disord*. 2020;41:102011. doi:10.1016/j.msard.2020.102011
- Mariotto S, Monaco S, Peschl P, et al. MOG antibody seropositivity in a patient with encephalitis: beyond the classical syndrome. *BMC Neurol*. 2017;17(1):190. doi:10.1186/s12883-017-0971-6

25. Titulaer MJ, McCracken L, Gabilondo I, et al. Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study. *Lancet Neurol.* 2013;12(2):157–165. doi:10.1016/S1474-4422(12)70310-1
26. Thompson J, Bi M, Murchison AG, et al. The importance of early immunotherapy in patients with faciobrachial dystonic seizures. *Brain.* 2018;141(2):348–356. doi:10.1093/brain/awx323
27. Cabezedo-García P, Mena-Vázquez N, Villagrán-García M, Serrano-Castro PJ. Efficacy of antiepileptic drugs in autoimmune epilepsy: a systematic review. *Seizure.* 2018;59:72–76. doi:10.1016/j.seizure.2018.05.004
28. Ramanathan S, O'Grady GL, Malone S, et al. Isolated seizures during the first episode of relapsing myelin oligodendrocyte glycoprotein antibody-associated demyelination in children. *Dev Med Child Neurol.* 2019;61(5):610–614. doi:10.1111/dmcn.14032
29. Zhong R, Zhang X, Chen Q, Li M, Guo X, Lin W. Acute symptomatic seizures and risk of epilepsy in autoimmune encephalitis: a retrospective cohort study. *Front Immunol.* 2022;13:813174. doi:10.3389/fimmu.2022.813174
30. Husari KS, Dubey D. Autoimmune Epilepsy. *Neurotherapeutics.* 2019;16(3):685–702.
31. Dubey D, Singh J, Britton JW, et al. Predictive models in the diagnosis and treatment of autoimmune epilepsy. *Epilepsia.* 2017;58(7):1181–1189. doi:10.1111/epi.13797
32. Armangue T, Olivé-Cirera G, Martínez-Hernandez E, et al. Associations of paediatric demyelinating and encephalitic syndromes with myelin oligodendrocyte glycoprotein antibodies: a multicentre observational study. *Lancet Neurol.* 2020;19(3):234–246. doi:10.1016/S1474-4422(19)30488-0
33. Jurynczyk M, Messina S, Woodhall MR, et al. Clinical presentation and prognosis in MOG-antibody disease: a UK study. *Brain.* 2017;140(12):3128–3138. doi:10.1093/brain/awx276
34. Ramanathan S, Mohammad S, Tantsis E, et al. Clinical course, therapeutic responses and outcomes in relapsing MOG antibody-associated demyelination. *J Neurol Neurosurg.* 2018;89(2):127–137.
35. Konuskan B, Yildirim M, Gocmen R, et al. Retrospective analysis of children with myelin oligodendrocyte glycoprotein antibody-related disorders. *Mult Scler Relat Disord.* 2018;26:1–7. doi:10.1016/j.msard.2018.07.022
36. Fujimori J, Takai Y, Nakashima I, et al. Bilateral frontal cortex encephalitis and paraparesis in a patient with anti-MOG antibodies. *J Neurol Neurosurg.* 2017;88(6):534–536. doi:10.1136/jnnp-2016-315094
37. Hacoen Y, Banwell B. Treatment approaches for MOG-Ab-ASSOCIATED DEMYELINATION IN CHILDREN. *Curr Treat Options Neurol.* 2019;21(1):2. doi:10.1007/s11940-019-0541-x
38. Durozard P, Rico A, Boutiere C, et al. Comparison of the response to rituximab between myelin oligodendrocyte glycoprotein and Aquaporin-4 antibody diseases. *Ann Neurol.* 2020;87(2):256–266. doi:10.1002/ana.25648
39. Yılmaz Ü, Edizer S, Songür ÇY, Güzin Y, Durak FS. Atypical presentation of MOG-related disease: slowly progressive behavioral and personality changes following a seizure. *Mult Scler Relat Disord.* 2019;36:101394. doi:10.1016/j.msard.2019.101394
40. Wen X, Wang B, Wang C, Han C, Guo S. A retrospective study of patients with GABAR encephalitis: therapy, disease activity and prognostic factors. *Neuropsychiatr Dis Treat.* 2021;17:99–110. doi:10.2147/NDT.S289942

Neuropsychiatric Disease and Treatment

Dovepress

Publish your work in this journal

Neuropsychiatric Disease and Treatment is an international, peer-reviewed journal of clinical therapeutics and pharmacology focusing on concise rapid reporting of clinical or pre-clinical studies on a range of neuropsychiatric and neurological disorders. This journal is indexed on PubMed Central, the 'PsycINFO' database and CAS, and is the official journal of The International Neuropsychiatric Association (INA). The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <https://www.dovepress.com/neuropsychiatric-disease-and-treatment-journal>