Brain Neurorehabil. 2022 Mar;15(1):e9
https://doi.org/10.12786/bn.2022.15.e9
pISSN 1976-8753·eISSN 2383-9910

Brain & NeuroRehabilitation

Case Report

Multidisciplinary Rehabilitation for Relapsing Myelin Oligodendrocyte Glycoprotein Antibody-associated Disease: A Case Report

Jong Mi Park, Yongwook Kim, Soojin Choi

Received: Aug 13, 2021
Revised: Oct 21, 2021
Accepted: Oct 22, 2021
Published online: Nov 22, 2021

Correspondence to
Soojin Choi
Department of Rehabilitation Medicine and Research Institute of Rehabilitation Medicine, Yonsei University College of Medicine, 50-1 Yonsei-ro, Seodaemun-gu, Seoul 03722, Korea.
E-mail: sjc8070@yuhs.ac

HIGHLIGHTS

- Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a neuroinflammatory disorder of the central nervous system.
- MOGAD is a relapsing disease, requiring continuous monitoring and rehabilitation.
- Tailored rehabilitation treatment for each symptom is required for MOGAD patients.

Brain Neurorehabil. 2022 Mar;15(1):e9
https://doi.org/10.12786/bn.2022.15.e9
pISSN 1976-8753·eISSN 2383-9910

bnr
Brain & NeuroRehabilitation

Case Report

Check for updates

Multidisciplinary Rehabilitation for Relapsing Myelin Oligodendrocyte Glycoprotein Antibody-associated Disease: A Case Report

Jong Mi Park, Yongwook Kim, Soojin Choi

Department of Rehabilitation Medicine and Research Institute of Rehabilitation Medicine, Yonsei University College of Medicine, Seoul, Korea

OPEN ACCESS

Received: Aug 13, 2021
Revised: Oct 21, 2021
Accepted: Oct 22, 2021
Published online: Nov 22, 2021

Correspondence to
Soojin Choi
Department of Rehabilitation Medicine and Research Institute of Rehabilitation Medicine, Yonsei University College of Medicine, 50-1 Yonsei-ro, Seodaemun-gu, Seoul 03722, Korea.
E-mail: sjc8070@yuhs.ac

ORCID iDs
Jong Mi Park
https://orcid.org/0000-0003-4272-7489
Yongwook Kim
https://orcid.org/0000-0002-5234-2454
Soojin Choi
https://orcid.org/0000-0001-6117-8353

Funding
None.

Conflict of Interest
The authors have no potential conflicts of interest to disclose.

ABSTRACT

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an inflammatory central nervous system disease that is driven by antibodies of the immunoglobulin G1 class. MOGAD has recently been recognized as an autoimmune disease; therefore, little is known about its rehabilitation. Here, we present a case of MOGAD that showed significant recovery after rehabilitation. A 58-year-old woman developed weakness in all extremities, dysarthria, and dysphagia. She visited the neurology department, and early brain and spine magnetic resonance imaging showed multifocal high intensity in the subcortical and periventricular white matter and the cervical cord. The patient's serum tested positive for anti-MOG antibodies. She was diagnosed with MOGAD and received intravenous steroid pulse therapy. After pharmacologic therapy, the patient was transferred to the rehabilitation department. Initially, her Functional Independence Measure (FIM) motor score was 26, allowing her to stand independently for only a few seconds. After 5 weeks of rehabilitation involving physical therapy, occupational therapy, and balance training, her FIM motor score improved to 60. However, 4 months after discharge, the disease relapsed with symptoms of motor weakness in all extremities, and steroid treatment was initiated. On the second admission, her FIM motor score was 42, but after continuous multidisciplinary rehabilitation, it improved to 76. Computerized cognitive therapy improved her cognitive function, from a Korean version of the Mini-Mental State Examination score of 23 on the first admission to 30 on final discharge. Since MOGAD is a relapsing disease, a favorable outcome can be achieved with continuous monitoring and multidisciplinary, symptom-specific rehabilitation.

Keywords: Myelin-Oligodendrocyte Glycoprotein; Demyelinating Diseases; Rehabilitation; Recurrence; Functional Status

INTRODUCTION

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is distinct from other neuroinflammatory disorders such as multiple sclerosis (MS) or AQP4-associated optic neuromyelitis spectrum disorder and has a relatively mild disease course. Myelin oligodendrocyte glycoprotein (MOG) is a central nervous system (CNS)-specific integral membrane protein that is expressed only in the myelin sheath and plasma membrane of oligodendrocytes [1]. Serological antibodies against MOG mediate T cell activation

and signal natural killer cells to kill MOG-expressing cells [2]. Therefore, MOGAD is an inflammatory, demyelinating condition of the CNS.

MOGAD shows a monophasic or recurrent course of neurological dysfunction and presents various symptoms due to CNS demyelination, each of which is seen at different frequencies. Optic neuritis is the predominant phenotype (41%–63%), followed by longitudinally extensive transverse myelitis (29%–31%), neuromyelitis optica (6%–24%), and acute disseminated encephalomyelitis (ADEM) (2%–6%) [3]. The clinical phenotypes of MOGAD are age-dependent; ADEM occurs more frequently in children, whereas optic neuritis and myelitis are more common in adults [4].

The clinical phenotype can vary from benign to fulminant, and due to the small patient population, proper rehabilitation for MOGAD has not been fully established thus far. Here, we present a case of a patient with repeated relapse and remission after MOGAD diagnosis, whose condition was significantly improved through multidisciplinary rehabilitation.

CASE REPORT

A 58-year-old woman was admitted to the hospital emergency room for recurrent falls due to weakness in all extremities, dysarthria, and dysphagia that began 2 months prior. The first symptoms started 8 years ago, but they worsened and improved about once or twice a year. Despite being hospitalized at every occurrence of the symptoms, the patient could not obtain an accurate diagnosis. She was then admitted to our hospital for the first evaluation.

When the patient was first admitted to the Department of Neurology, the neurologist assessed her initial strength using the Medical Research Council (MRC) scale, scoring 1 for her right deltoid, biceps, gluteus and quadriceps femoris muscles, 3 for the left deltoid and biceps muscles, and 2 for her left gluteus and quadriceps femoris muscles. The bilateral biceps and patella showed hyperactive tendon reflexes. Hoffman signs, Babinski signs, and ankle clonus were observed on both sides. There were no specific findings on the cerebrospinal fluid (CSF) test, and the anti-AQP4 antibody specific for optic neuritis and the CSF oligoclonal band test specific for MS were negative. The absence of optic neuritis was confirmed by ophthalmic examination through ophthalmology consultation. The patient's serum was also seropositive for anti-MOG antibody in a live cell fluorescence-activated cell sorting assay. Initial brain magnetic resonance imaging (MRI) showed multifocal T2 hyperintense lesions in the subcortical and periventricular white matter and brainstem (**Fig. 1A**). Initial spine MRI revealed multifocal T2 high signal intensity in the spinal cord at C1–2, C2–3, C5–6, and C7–T1 levels, suggesting demyelination disease (**Fig. 2A**). MOGAD was diagnosed after excluding other CNS inflammatory diseases according to the brain and spine MRI findings, CSF study, serum anti-body study, and clinical features. Based on the diagnosis of MOGAD, oral prednisolone and azathioprine were administered as maintenance therapy after intravenous steroid pulse therapy. After the initial steroid therapy, follow-up brain MRI showed significantly decreased T2 hyperintense lesions in the brain (**Fig. 1B**). On spine MRI, the T2 high-signal intensity lesion of the cervical cord had disappeared (**Fig. 2B**).

A month after admission to the neurology department, the patient was transferred to our department for rehabilitation. The patient initially showed motor weakness and functional impairment; she could stand independently for only a few seconds. **Table 1** shows the initial

https://doi.org/10.12786/bn.2022.15.e9

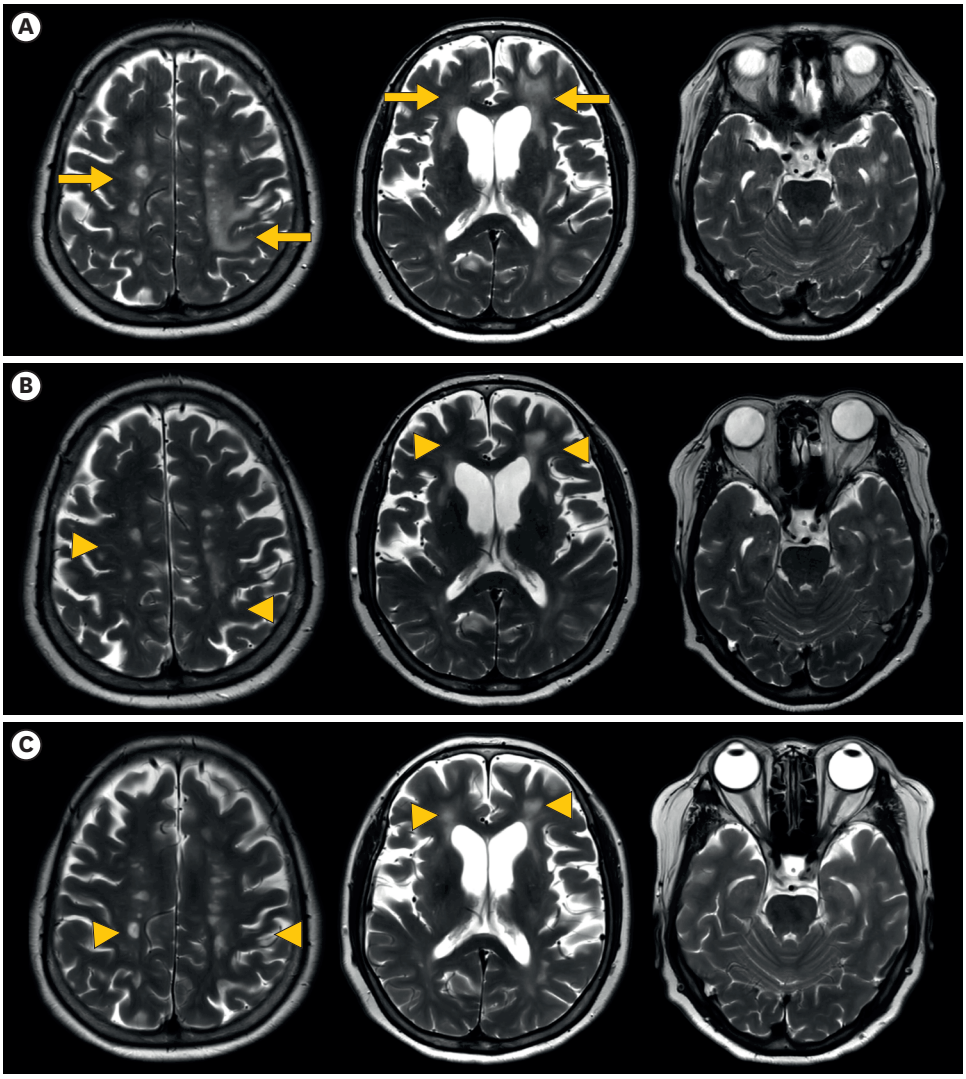

Fig. 1. (A) Brain MRI at first admission showing multifocal hyperintense lesions in the cortical, subcortical, periventricular white matter (arrows) and brainstem on a T2-weighted image. (B) Two-week follow-up brain MRI showing significantly decreased hyperintense lesions in the cortical, subcortical, periventricular white matter (arrowheads) and brainstem on a T2-weighted image after steroid therapy. (C) Brain MRI at second hospitalization (7 months after the first brain MRI) revealing newly developed tiny active lesions in bilateral white matter (arrowheads) on a T2-weighted image.
MRI, magnetic resonance imaging.

evaluation of muscle power measured on the MRC scale, Functional Independence Measure (FIM) score, and the Korean version of the Mini-Mental State Examination (K-MMSE) at the time of transfer. The patient's FIM score was 55, and the motor FIM score was 26 at the time of the initial evaluation. The patient's initial K-MMSE score was 23, with impaired time orientation, recent memory, calculation, and visual construction. The patient received physical therapy, occupational therapy, balance training, and strengthening with equipment twice a day for 30 minutes each, 5 times a week. Computerized cognitive therapy was also provided. At the time of first discharge from our department, the patient showed improvement in motor and functional levels and could walk on her own using an anterior walker. The patient's total and motor FIM scores improved, and her cognitive function also improved to a K-MMSE score of 30 points (**Table 1**). Dysarthria and dysphagia also improved,

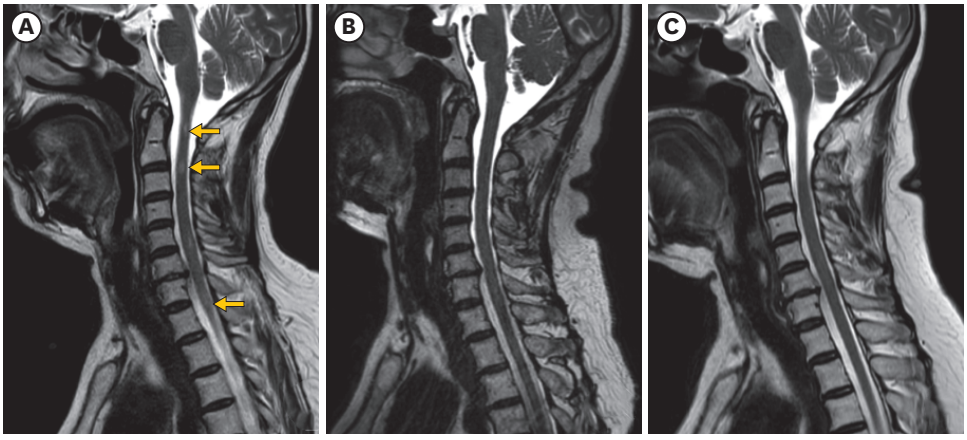

Fig. 2. (A) Cervical spine MRI at first admission revealing multifocal high signal intensity in the spinal cord at C1–2, C2–3, C5–6, and C7–T1 levels (arrows) on a T2-weighted image. (B) Seven-week follow-up cervical spine MRI showing that the signal in the cervical cord after steroid therapy disappeared. (C) Cervical spine MRI at second hospitalization (7 months after the first spine MRI) showing no significant signal change or enhancement.
MRI, magnetic resonance imaging.

Table 1. Muscle power measured using the Medical Research Council scale, FIM, and K-MMSE scores of the patient at first and second admissions

Characteristics	First admission	First discharge	Second admission	Second discharge
Muscle power				
Upper extremities (right/left) (0–5)	3/4	3/4	3/3	4/4
Lower extremities (right/left) (0–5)	3/3	4/4	3/4	4/4
FIM score				
Total (range: 18–126)	55	93	67	108
Motor (range: 13–91)	26	60	42	76
K-MMSE (0–30)	23	30	27	30

FIM, Functional Independence Measure; K-MMSE, Korean version of the Mini-Mental State Examination.

along with motor symptoms. The patient was discharged and followed-up on an outpatient basis. One month after discharge, she was able to walk independently even on the ground and perform household chores.

However, 4 months after discharge, symptoms of dragging in the right foot, weakness in the right hand, and dysarthria recurred. Decreased muscle strength and impaired functional levels were detected in the outpatient rehabilitation clinic. She was re-admitted to the neurology department of our hospital for re-evaluation. Brain MRI revealed newly developed tiny active lesions of anti-MOG encephalitis in the bilateral white matter (**Fig. 1C**). Spine MRI showed no significant T2 high-intensity lesions in the spinal cord (**Fig. 2C**). Anti-MOG antibody tests also showed persistent seropositivity. Based on the diagnosis of MOGAD recurrence, intravenous steroid pulse therapy was initiated.

Considering the fast recovery rate of MOGAD, the patient was transferred to the rehabilitation department within a week of admission and began early rehabilitation treatment. As her gait function deteriorated, she could only walk with the support of a caregiver holding both hands. The right foot clearance was decreased, and a weakened right ankle dorsiflexor and genu recurvatum were noted. Her FIM score was 67 and her motor FIM score was 42, which was lower than that at the time of the previous discharge. Her K-MMSE score was 27, with mildly impaired calculation (**Table 1**). Gait training and strengthening continued considering the previous speed of recovery. At the second discharge after 4 weeks of intensive rehabilitation, the patient's muscle strength and functional level improved

https://doi.org//10.12786/bn.2022.15.e9

significantly. She was able to walk independently using a cane intermittently, with a FIM score of 108 and a motor FIM score of 76. Her cognitive impairment also improved, with a K-MMSE score of 30 (**Table 1**).

DISCUSSION

We present the case of a patient with MOGAD who was diagnosed after repeated symptom recurrence and remission for several years, whose condition improved significantly through customized rehabilitation treatment for each symptom.

MOGAD presents with a combination of optic neuritis and transverse myelitis with brain lesions consistent with ADEM, commonly involving the brainstem [5]. Spinal cord involvement and ADEM were also observed in our case, but without optic neuritis. The disease course of MOGAD can be monophasic, relapsing, and remitting. Recurrent MOGAD is associated with old age, female sex, persistence of MOG-immunoglobulin G seropositivity, severe cognitive deficits, persistent seizures, and certain MRI findings [5,6]. On brain MRI, poorly demarcated, sparse lesions (< 3) located infratentorially in the brainstem and cerebellar peduncles are typically seen in MOGAD [7]. MOGAD can involve white matter, gray matter, or both [8]. Our patient had encephalitis with involvement of the white matter and brainstem.

As MOGAD is the newest member of the antigen-specific autoimmune diseases of the CNS and presents with a wide variety of clinical manifestations, limited information is available regarding its mode of rehabilitation. In studies on patients with MS, another neuroinflammatory disease, multidisciplinary rehabilitation has been performed for multiple deficits (motor, sensory, cognitive, behavioral, and communication problems) [9-11]. Rehabilitation was performed in patients with MOGAD with reference to existing MS rehabilitation studies, based on the fact that they both relapse and remit neuroinflammatory diseases [9,10,12]. However, there are differences in the clinical features between MOGAD and MS, which are summarized in **Table 2** [13-20]. Unlike MS, MOGAD has less occurrence of sensory impairment and a higher relapse rate with shorter intervals between attacks, as well

Table 2. Characteristics of patients with MOGAD and MS

Characteristics	MOGAD	MS
Demographics		
Median age at onset (yr)	31–37	20–30
Female sex	63%–74%	70%–75%
Clinical presentation at onset		
Optic neuritis	60%–74%	15%–20%
Bilateral optic neuritis	35%–41%	0%–1%
Myelitis	18%–23%	< 1%
Brain stem encephalitis	8%–14%	< 1%
Sensory impairment	< 1%	28%
Prognosis		
Annualized relapse rate, median (range)	0.83 (0.05–6.92)	0.25 (0.10–1.18)
Recovery time (mon)	< 1	2–12
Interval between first and second attacks (mon), median (range)	3.6 (0.5–182.0)	17.0 (1.9–152.1)
Treatment		
Acute treatment	IV steroid, plasma exchange, IVIG	IV steroid, IFNB, GA
Maintenance treatment	Azathioprine, low-dose prednisone	Fingolimod, teriflunomide

MOGAD, myelin oligodendrocyte glycoprotein antibody-associated disease; MS, multiple sclerosis; IV, intravenous; IVIG, intravenous immunoglobulin; IFNB, interferon beta; GA, glatiramer acetate.

https://doi.org//10.12786/bn.2022.15.e9

as a shorter recovery time. While recovery from MS takes 2–3 months to a maximum of 12 months, it generally takes only a few weeks in MOGAD [13,19,21].

First, considering the similarities between MOGAD and MS, exercise intensity and frequency were determined according to previous studies [9,10,12]. In patients with MS, 30 minutes of moderate-intensity aerobic exercise and strength training for major muscle groups has been shown to reduce fatigue, improve mobility, and improve health-related quality of life [9,10,12]. Therefore, our patient received moderate-intensity physical therapy for 30 minutes, twice a day, 5 times a week for 4 weeks at each admission. Aerobic exercises such as walking and cycling were also performed daily with an intensity of 2–3 ratings of perceived exertion on the Borg scale, which is a light to moderate-intensity aerobic activity [9]. Strengthening exercises were performed 2–3 times per week, at an intensity of 10 to 12 repetitions maximum (RM) (up to 70%–80% of 1 RM) [9]. Cognitive training was also performed. Previous studies have shown that computer-assisted cognitive rehabilitation improves cognitive abilities and induces changes in brain function connections in patients with relapsing–remitting MS [11]. Therefore, we conducted computer-assisted cognitive rehabilitation for the patient 30 minutes each, 5 times a week for a total of 4 weeks. The K-MMSE score improved at final discharge through combined occupational therapy and computer-assisted cognitive rehabilitation.

Second, considering the different clinical aspects of MOGAD and MS, and the faster recovery rate of MOGAD, the rehabilitation therapy of our patient focused on early strength recovery and early functional improvement through gait training. Walking aids such as an anterior walker, quad cane, and mono cane were serially used for early mobilization of the patient. In addition, since MOGAD rarely shows sensory impairment, occupational therapy focused more on performance of daily activities and cognitive therapy rather than sensory stimulation. After 4 weeks of customized multidisciplinary rehabilitation, improved mobility, strength, functional outcomes, and reduced fatigue were observed, which ultimately improved her quality of life.

While the patient showed significant overall improvement, it was difficult to fully determine whether the improvement was due to the effect of steroid therapy or rehabilitation treatment. To date, there have been no randomized controlled trials comparing steroid monotherapy with rehabilitation monotherapy in patients with MOGAD or MS. However, there was a randomized controlled trial comparing steroid monotherapy with a combination of steroid and rehabilitation therapy in patients with MS, and there was a more significant improvement in the activity profile and functional level in the combination therapy group [22]. Therefore, it can be assumed that rehabilitation treatment will induce additional beneficial effects in patients with MOGAD. However, it cannot be considered as the effect of rehabilitation treatment alone, and this remains a limitation of this study.

Although MOGAD has a high recurrence rate in the short term, there is still a lack of consensus on the proper treatment regimen to prevent recurrence. Previous studies have suggested that continuous administration of oral steroids and immunosuppressants are helpful. Therefore, in the case of relapsed patients, it is necessary to combine continuous drug treatment and customized rehabilitation treatment for functional recovery. In the UK cohort, post-onset motor recovery was complete or good in 78%, but 28% maintained permanent bladder dysfunction, 21% maintained erectile dysfunction, 20% maintained bowel dysfunction, and 16% maintained decreased visual acuity [23]. Therefore, patients and caregivers should be educated about the course and symptoms of the disease known from

previous studies, and if symptoms worsen after discharge, they should be instructed to visit the hospital immediately to check for recurrence.

In addition, in order to diagnose a patient with a disability through regular follow-up, such as a patient with relapsing MS, it is thought that it should be performed 6 months after the onset of the disease because the symptoms do not fully recover. In addition, since MOGAD patients have a higher recurrence rate and faster recovery than MS patients, physicians will need to consider disability diagnosis based on more medical knowledge and clinical experience in the future.

In conclusion, MOGAD has a diverse clinical phenotype and disease course of relapse and remission. After comprehensive rehabilitation therapy, the prognosis of our patient was favorable with respect to muscle strength, functional ability, and cognitive function. Therefore, continuous monitoring for recurrence and a multidisciplinary, symptom-specific approach are required. In addition, the physiatrist should continuously follow up the patient after acute inpatient rehabilitation to monitor compliance to previous treatment regimen and outpatient exercise program to reduce the risk of recurrence, check muscle weakness and functional deterioration, and provide education to patients and caregivers.

REFERENCES

1. Kroepfl JF, Viise LR, Charron AJ, Linington C, Gardinier MV. Investigation of myelin/oligodendrocyte glycoprotein membrane topology. J Neurochem 1996;67:2219-2222.
PUBMED | CROSSREF
2. Brilot F, Dale RC, Selter RC, Grummel V, Kalluri SR, Aslam M, Busch V, Zhou D, Cepok S, Hemmer B. Antibodies to native myelin oligodendrocyte glycoprotein in children with inflammatory demyelinating central nervous system disease. Ann Neurol 2009;66:833-842.
PUBMED | CROSSREF
3. Chen JJ, Flanagan EP, Jitprapaikulsan J, López-Chiriboga AS, Fryer JP, Leavitt JA, Weinshenker BG, McKeon A, Tillema JM, Lennon VA, Tobin WO, Keegan BM, Lucchinetti CF, Kantarci OH, McClelland CM, Lee MS, Bennett JL, Pelak VS, Chen Y, VanStavern G, Adesina OO, Eggenberger ER, Acierno MD, Wingerchuk DM, Brazis PW, Sagen J, Pittock SJ. Myelin oligodendrocyte glycoprotein antibody-positive optic neuritis: clinical characteristics, radiologic clues, and outcome. Am J Ophthalmol 2018;195:8-15.
PUBMED | CROSSREF
4. Reindl M, Waters P. Myelin oligodendrocyte glycoprotein antibodies in neurological disease. Nat Rev Neurol 2019;15:89-102.
PUBMED | CROSSREF
5. Narayan R, Simpson A, Fritsche K, Salama S, Pardo S, Mealy M, Paul F, Levy M. MOG antibody disease: a review of MOG antibody seropositive neuromyelitis optica spectrum disorder. Mult Scler Relat Disord 2018;25:66-72.
PUBMED | CROSSREF
6. López-Chiriboga AS, Majed M, Fryer J, Dubey D, McKeon A, Flanagan EP, Jitprapaikulsan J, Kothapalli N, Tillema JM, Chen J, Weinshenker B, Wingerchuk D, Sagen J, Gadoth A, Lennon VA, Keegan BM, Lucchinetti C, Pittock SJ. Association of MOG-IgG serostatus with relapse after acute disseminated encephalomyelitis and proposed diagnostic criteria for MOG-IgG-associated disorders. JAMA Neurol 2018;75:1355-1363.
PUBMED | CROSSREF
7. Jurynczyk M, Geraldes R, Probert F, Woodhall MR, Waters P, Tackley G, DeLuca G, Chandratre S, Leite MI, Vincent A, Palace J. Distinct brain imaging characteristics of autoantibody-mediated CNS conditions and multiple sclerosis. Brain 2017;140:617-627.
PUBMED | CROSSREF
8. Netravathi M, Holla VV, Nalini A, Yadav R, Vengalil S, Oommen AT, Reshma SS, Kamble N, Thomas PT, Maya B, Pal PK, Mahadevan A. Myelin oligodendrocyte glycoprotein-antibody-associated disorder: a new inflammatory CNS demyelinating disorder. J Neurol 2021;268:1419-1433.
PUBMED | CROSSREF

9. Latimer-Cheung AE, Pilutti LA, Hicks AL, Martin Ginis KA, Fenuta AM, MacKibbon KA, Motl RW. Effects of exercise training on fitness, mobility, fatigue, and health-related quality of life among adults with multiple sclerosis: a systematic review to inform guideline development. Arch Phys Med Rehabil 2013;94:1800-1828.e3.
PUBMED | CROSSREF
10. Khan F, Turner-Stokes L, Ng L, Kilpatrick T, Amatya B. Multidisciplinary rehabilitation for adults with multiple sclerosis. Cochrane Database Syst Rev 2007:CD006036.
PUBMED | CROSSREF
11. Bonavita S, Sacco R, Della Corte M, Esposito S, Sparaco M, d'Ambrosio A, Docimo R, Bisecco A, Lavorgna L, Corbo D, Cirillo S, Gallo A, Esposito F, Tedeschi G. Computer-aided cognitive rehabilitation improves cognitive performances and induces brain functional connectivity changes in relapsing remitting multiple sclerosis patients: an exploratory study. J Neurol 2015;262:91-100.
PUBMED | CROSSREF
12. Latimer-Cheung AE, Martin Ginis KA, Hicks AL, Motl RW, Pilutti LA, Duggan M, Wheeler G, Persad R, Smith KM. Development of evidence-informed physical activity guidelines for adults with multiple sclerosis. Arch Phys Med Rehabil 2013;94:1829-1836.e7.
PUBMED | CROSSREF
13. Ciotti JR, Eby NS, Wu GF, Naismith RT, Chahin S, Cross AH. Clinical and laboratory features distinguishing MOG antibody disease from multiple sclerosis and AQP4 antibody-positive neuromyelitis optica. Mult Scler Relat Disord 2020;45:102399.
PUBMED | CROSSREF
14. Tajfirouz DA, Bhatti MT, Chen JJ. Clinical characteristics and treatment of MOG-IgG–associated optic neuritis. Curr Neurol Neurosci Rep 2019;19:100.
PUBMED | CROSSREF
15. Weber MS, Derfuss T, Metz I, Brück W. Defining distinct features of anti-MOG antibody associated central nervous system demyelination. Ther Adv Neurol Disorder 2018;11:1756286418762083.
PUBMED | CROSSREF
16. Healy S, Elhadd KT, Gibbons E, Whittam D, Griffiths M, Jacob A, Huda S. Treatment of myelin oligodendrocyte glycoprotein immunoglobulin G–associated disease. Clin Exp Neuroimmunol 2021;12:22-41.
CROSSREF
17. Goldenberg MM. Multiple sclerosis review. P&T 2012;37:175-184.
PUBMED
18. Langer-Gould A, Popat RA, Huang SM, Cobb K, Fontoura P, Gould MK, Nelson LM. Clinical and demographic predictors of long-term disability in patients with relapsing-remitting multiple sclerosis: a systematic review. Arch Neurol 2006;63:1686-1691.
PUBMED | CROSSREF
19. Flanagan EP, Tillema JM. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): clinical features and diagnosis. Available at https://www.uptodate.com/contents/myelin-oligodendrocyte-glycoprotein-antibody-associated-disease-mogad-clinical-features-and-diagnosis [accessed on 1 October 2021].
20. Dubey D, Pittock SJ, Krecke KN, Morris PP, Sechi E, Zalewski NL, Weinshenker BG, Shosha E, Lucchinetti CF, Fryer JP, Lopez-Chiriboga AS, Chen JC, Jitprapaikulsan J, McKeon A, Gadoth A, Keegan BM, Tillema JM, Naddaf E, Patterson MC, Messacar K, Tyler KL, Flanagan EP. Clinical, radiologic, and prognostic features of myelitis associated with myelin oligodendrocyte glycoprotein autoantibody. JAMA Neurol 2019;76:301-309.
PUBMED | CROSSREF
21. Hirst CL, Ingram G, Pickersgill TP, Robertson NP. Temporal evolution of remission following multiple sclerosis relapse and predictors of outcome. Mult Scler 2012;18:1152-1158.
PUBMED | CROSSREF
22. Craig J, Young CA, Ennis M, Baker G, Boggild M. A randomised controlled trial comparing rehabilitation against standard therapy in multiple sclerosis patients receiving intravenous steroid treatment. J Neurol Neurosurg Psychiatry 2003;74:1225-1230.
PUBMED | CROSSREF
23. Jurynczyk M, Messina S, Woodhall MR, Raza N, Everett R, Roca-Fernandez A, Tackley G, Hamid S, Sheard A, Reynolds G, Chandratre S, Hemingway C, Jacob A, Vincent A, Leite MI, Waters P, Palace J. Clinical presentation and prognosis in MOG-antibody disease: a UK study. Brain 2017;140:3128-3138.
PUBMED | CROSSREF