Journal of Neuroimmunology 337 (2019) 577078

Contents lists available at ScienceDirect

Journal of Neuroimmunology

journal homepage: www.elsevier.com/locate/jneuroim

Review Article

The clinical presentation and treatment of MOG antibody disease at a single academic center: A case series

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ARTICLE INFO

Keywords:
Myelin oligodendrocyte glycoprotein
Optic neuritis
Transverse myelitis
Neuromyelitis optica spectrum disorder
Demyelination
CNS inflammation

ABSTRACT

Objectives: To describe the clinical presentation of MOG antibody disease (MOG-AD) in a series of patients at a single academic center.
Methods: We performed a retrospective review of patients with MOG antibodies.
Results: We review the clinical presentation of 11 patients with MOG antibodies. In patients seen at Duke University Health System with MOG antibodies, the most common presentation was optic neuritis. Rituximab was the most used treatment for long-term management.
Conclusions: Our case series highlights the common presentation of MOG antibody disease (MOG-AD) at a single academic medical center.

1. Introduction

Myelin oligodendrocyte glycoprotein (MOG) was first identified as a target for demyelination in 1989 in guinea pigs (Reindl and Waters, 2019). This raised interest into MOG antibodies and their potential role in neurological diseases. While initially thought to be associated with multiple sclerosis (MS), further investigations distinguished MOG antibody disease (MOG-AD) as separate from other demyelinating diseases.

Myelin oligodendrocyte glycoprotein (MOG) is uniquely expressed in mammalian oligodendrocytes with high conservation across species. Although its exact role remains unclear, studies in experimental models have shown that it can elicit a demyelinating immune response (Reindl and Waters, 2019). Clinically, the presence of MOG antibodies – better detected by cell-based assays in the serum (de Seze, 2019) – has been associated with a number of neurological manifestations that are categorized as MOG-antibody disease. The most common clinical manifestation is optic neuritis, followed by transverse myelitis (with longitudinally extensive TM being more frequent) (Jarius et al., 2016a). In the pediatric population, acute disseminated encephalomyelitis (ADEM) is most common presentation (Jurynczyk et al., 2019). MOG-AD has been connected to several atypical presentations including pseudotumor cerebri (Narayan et al., 2019; Lotan et al., 2018), encephalitis with seizures (Jurynczyk et al., 2019; Salama et al., 2019), small vessel CNS vasculitis (Patterson et al., 2019), relapsing lumbosacral myeloradiculitis (Sundaram et al., 2019), mimicking multiple sclerosis (Breza et al., 2019), and aseptic meningitis (Nagabushana et al., 2019).

Cell-based assays for MOG antibodies were first developed in 2001, but they did not become commercially available until 2017. This allowed a broader understanding of the variability of the initial presentation of MOG-AD. Due to the variability in presentation and limited availability of long-term data on MOG-AD, there are different approaches to management of the disease. Herein, we aim to present the specific characteristics of patients with MOG-AD within our institution, focusing on their initial presentation, age of onset, gender predominance, and management both acutely and over the long term (Tables 1 and 2).

2. Methods

A retrospective chart review of patients testing positive for MOG antibody at our institution was performed. Patient characteristics including age at presentation, gender, MOG antibody titers, clinical presentation, treatment course, and disease outcome were recorded. The MOG antibody titer testing was performed at the same laboratory. This study was approved by the Duke University IRB. Protocol number: Pro00101469.

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https://doi.org/10.1016/j.jneuroim.2019.577078
Received 21 August 2019; Received in revised form 24 September 2019; Accepted 30 September 2019

Table 1
Demographics of patients presenting with MOG-AD.

Patient characteristics	
Sex, N (%)	
Male	6 (54.5)
Female	5 (45.5)
Age at initial presentation, year	
Mean (SD)	41.5 (13.6)
Range	25–69
MOG antibody titer	
Mean (SD)	1:240 (1:377)
Range	1:20–1:1000
Initial presentation, N (%)	
Optic neuritis	9 (81.8)
Bilateral	3
Unilateral	6
Longitudinally Extensive Transverse myelitis (LETM)	2 (18.2)
Disease course, N (%)	
Monophasic	5 (45.5)
Recurrent	6 (64.5)

3. Patient presentations

3.1. Patient 1

A 53-year-old woman with no significant past medical history presented to the emergency department with acute vision loss in her left eye superimposed upon 10 days of progressive vision loss in her right eye. Ophthalmologic evaluation demonstrated decreased visual acuity bilaterally, left relative afferent pupillary defect (RAPD), and bilateral optic disc edema. Magnetic resonance imaging (MRI) of the brain revealed bilateral optic nerve enhancement and lumbar puncture (LP) demonstrated 5 nucleated cells/μL, 53 red blood cells/μL, protein of 31 mg/dL, and glucose of 58 mg/dL. She was treated with 1000 mg of intravenous methylprednisolone for five days followed by oral prednisone 80 mg daily to be taken for seven days. She had improvement in her vision after receiving the intravenous steroid infusion. MOG testing resulted positive with a titer of 1:20. The patient has remained both clinically and radiographically stable for two years following her initial presentation without chronic disease modifying therapy.

3.2. Patient 2

A 69-year-old man with type 2 diabetes mellitus and hypertension presented to the emergency department for evaluation with one week of right periorbital pain followed two days later by complete vision loss in his right eye over a period of 24 h. Brain MRI revealed right optic nerve enhancement and lumbar puncture demonstrated 1 nucleated cell/μL, 205 red blood cells/μL, protein of 76 mg/dL, and glucose of 61 mg/dL. He was treated with 500 mg of intravenous methylprednisolone twice a day for five days and discharged on oral prednisone 60 mg daily for seven days. Six months later, he experienced a decline in his visual acuity in his right eye. Repeat MRI of the brain demonstrated right optic nerve enhancement. MOG testing returned positive with a titer of 1:100. He was treated with a three-month taper of oral prednisone. He was then started on maintenance therapy with rituximab and as had no new neurologic or vision changes.

3.3. Patient 3

A 49-year-old-man with hypercholesterolemia presented for outpatient evaluation of numbness and cramping that started 2–3 weeks after initiation of rosuvastatin. He also reported vision loss in the right eye, paresthesias, and imbalance that had started in the same time period. Neurologic exam revealed hemisensory loss on the torso, upper and lower extremities with mild weakness in his left upper extremity, diffuse hyperreflexia, and an upgoing toe on the left. Brain and cervical

Table 2
Acute and disease modifying treatments for MOG-AD patient with disease course.

Patient	Acute treatment	Oral steroid taper	Disease modifying treatment	Recurrences and outcomes
1	IVMP 1000 mg for 5 days	Prednisone 80 mg with slow wean over three months	None	Clinically and radiographically stable
2	IVMP 500 mg twice a day for 5 days	Prednisone 60 mg with a very prolonged taper given multiple recurrences	Rituximab	Multiple relapses prior to DMT initiation. Clinically and radiographically stable since.
3	None, presented for evaluation after acute setting.	None	Rituximab	Relapses prior to DMT initiation
4	IVMP 1000 mg for 5 days	Prednisone 60 mg over 12 days	Rituximab	N/A[a]
5	IVMP 1000 mg for 2 days, then oral dexamethasone 80 mg for 1 day	Prednisone 60 mg for 18 days	Mycophenolate mofetil	N/A[a]
6	IVMP 1000 mg for 3 days	Deferred due to severe hyperglycemia	Rituximab	N/A[a]
7	IVMP 1000 mg for 3 days	Multiple prednisone tapers for prior relapses	Rituximab	Multiple relapses prior to DMT initiation. Now, clinically and radiographically stable
8	IVMP 1000 mg for 3 days	None	Deferred- watchful waiting	N/A[a]
9	IVMP 1000 mg for 3 days	Prednisone 60 mg for 6 days	Rituximab	Relapse prior to DMT initiation. Now, clinically and radiographically stable
10	IVMP 1000 mg for 3 days, followed by IVIg 2 g	Prednisone 40 mg over 4 weeks	Rituximab	Relapse prior to DMT initiation.
11	IVMP 1000 mg for 3 days	Prolonged prednisone taper over weeks	Rituximab	N/A[a], Patient with coexistent AQP-4 antibodies.

[a] These patient were identified as having MOG-AD in the acute setting and information regarding outcome is not available at the time of writing.

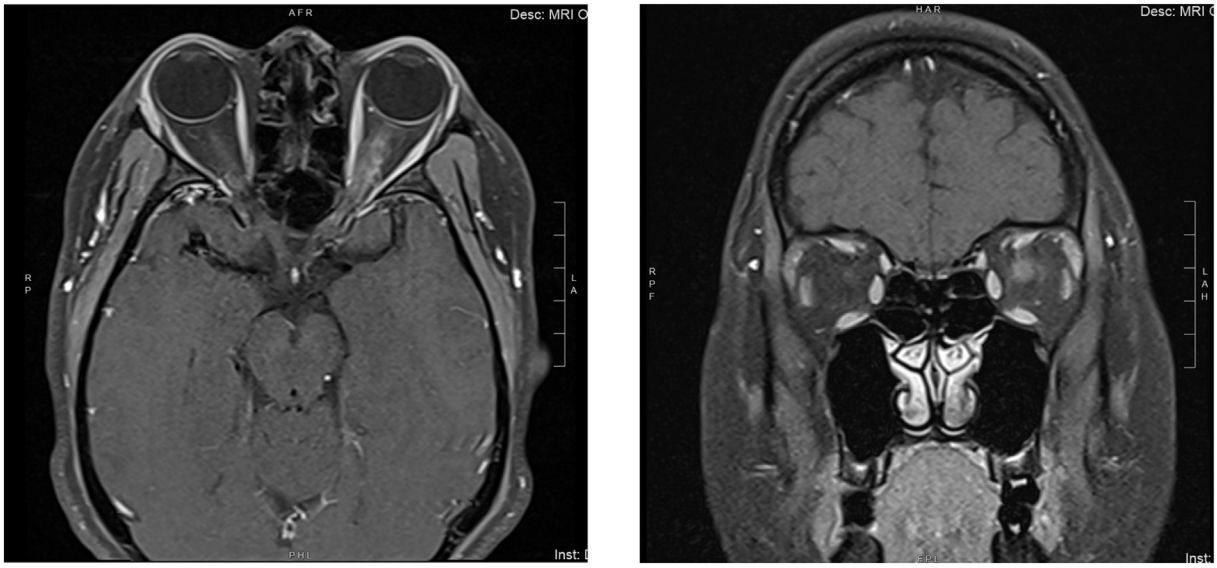

Fig. 1. MOG-AD Optic Neuritis in Patient 8. Enhancement of the anterior portion of the left optic nerve as can be seen with MOG-AD optic neuritis.

spine MRI showed enhancement in the medulla and upper cervical spine (Fig. 2). Visual evoked potentials were abnormal with moderate prolongation of P100 distal latencies bilaterally. Lumbar puncture demonstrated 28 nucleated cells/μL (91% lymphocytic predominance), 17 red blood cells/μL, protein of 50 mg/dL, glucose of 49 mg/dL, and no oligoclonal bands. MOG antibody titer returned 1:40. The patient later noted he had experienced decreased visual acuity and weakness about twenty years prior. He was recommended to start treatment with rituximab, which is yet to be initiated.

3.4. Patient 4

A 46-year-old woman with history of a spindle cell tumor involving the brainstem resected three years prior without residual deficits presented to the emergency department with one month of progressive, painful vision loss in her left eye, described as a "windshield covered by snow" followed by loss of color vision and eventually only being able to see shadows. Ophthalmologic exam revealed optic disc edema in the left eye. MRI of her orbits demonstrated enhancement of her left optic nerve. She was started on intravenous methylprednisolone 1000 mg daily for five days followed by oral prednisone 60 mg with a 12-day taper with improvement in her visual acuity. MOG titer was 1:40. The patient was recommended to start treatment with rituximab, which is yet to be initiated.

3.5. Patient 5

A 37-year-old man presented to an ophthalmologist with two weeks of vision loss. One day prior to symptom onset he experienced sinus pressure and headaches. Initial evaluation diagnosed dry eyes versus giant papillary conjunctivitis and he was treated with steroid drops. Within a week, his symptoms progressed and he developed pain with eye movements. Repeat evaluation found decreased visual acuity bilaterally, a right RAPD, and bilateral optic disc edema. MRI of the brain demonstrated bilateral optic nerve enhancement, worse on the right. He was treated with intravenous methylprednisolone 1000 mg daily for two days with dexamethasone 80 mg on the third day due to patient request for expedited discharge. He was discharged with an oral prednisone taper over 18 days starting at 60 mg. One month later he had regained almost normal visual acuity. MOG antibody titer was 1:100. Chronic therapy with mycophenolate mofetil was recommend.

3.6. Patient 6

A 54-year-old woman presented to her ophthalmologist due to pain with left eye movement. Approximately six days later, the pain persisted and she developed blurry vision, prompting a presentation to the emergency department. MRI revealed left optic nerve enhancement consistent with optic neuritis. She received intravenous methylprednisolone 1000 mg daily for three days without a prednisone taper (due to severe hyperglycemia). MOG antibody titer was 1:1000. She reported improvement in her vision during her follow-up appointment 8 weeks later. Rituximab was recommended but has yet to be initiated.

3.7. Patient 7

A 43-year-old woman presented to the neurology clinic for episodes of diplopia, numbness and weakness. At 36, she had experienced diplopia which resolved without medical evaluation. Four years later, she had a recurrence of the diplopia for which she received steroids and no further diagnostic evaluation. A few months prior to her current presentation, she developed progressive numbness in both hands followed by numbness in her right leg. MRI brain and cervical spine revealed a T2 signal abnormality within the posterior limb of the left internal capsule and extensive FLAIR abnormality within the ventral medulla and cervical cord. Spinal fluid evaluation did not demonstrate any unique oligoclonal bands. She was treated with 1000 mg of intravenous methylprednisolone daily for three days with improvement in her symptoms. Two months later, she developed a third episode of diplopia and was treated with oral steroids. MOG testing was positive with a titer of 1:100, and she was started on rituximab. Surveillance MRI obtained a year after starting treatment showed new thoracic spine lesions compared to imaging obtained prior to initiating treatment, but she has remained clinically stable.

3.8. Patient 8

A 25-year-old woman with childhood asthma presented to neuro-ophthalmology clinic after an urgent referral from her local ophthalmologist for vision loss and papilledema in the left eye. Her symptoms started three days prior with acute onset of blurry vision in the left eye. In addition, patient reported some intermittent headaches, left shoulder numbness, and neck tightness. Her ophthalmologic exam was interpreted as atypical left optic neuritis given the pattern of peripheral vision loss and presence of peripapillary hemorrhages. MRI brain and

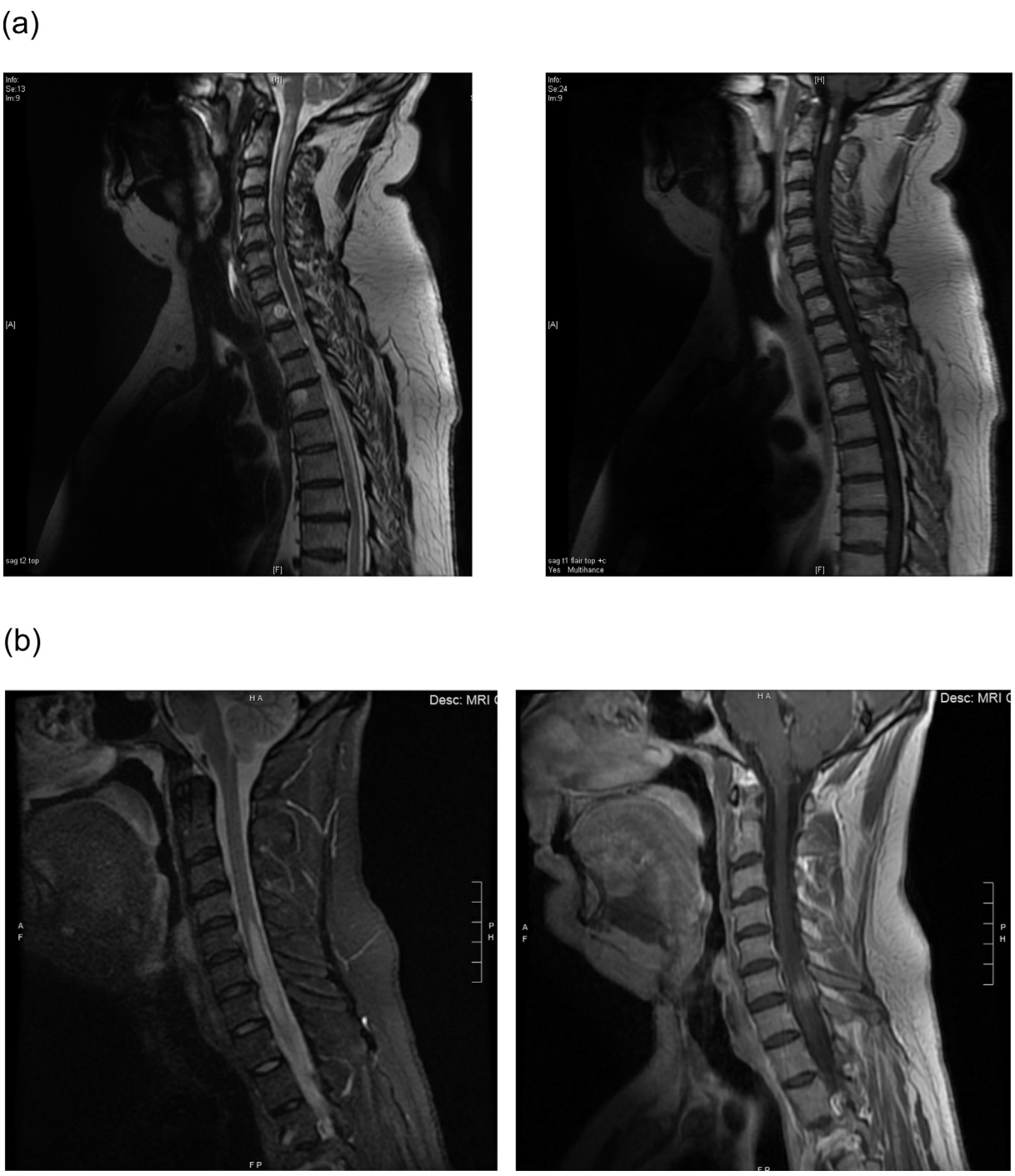

Fig. 2. a. MOG-AD Longitudinally Extensive Transverse Myelitis (LETM) in Patient 3 – T2 hyperintense signal extending from the medulla to the lower cervical spine with evidence of enhancement in the upper cervical spine. Fig. 2b MOG-AD Longitudinally Extensive Transverse Myelitis (LETM) in Patient 10 – T2 hyperintense signal extending C4 to T2 with evidence of enhancement around C6-C7. Longitudinally extensive lesions are very common in MOG-AD.

orbits demonstrated left optic neuritis (Fig. 1). She was started on 1000 mg of intravenous methylprednisolone daily for three days with significant improvement in her vision and was discharged home without an oral steroid taper. MOG antibodies titer was 1:100. The patient elected to defer chronic treatment.

3.9. Patient 9

A 34-year-old man with a previous episode of monocular vision loss two years prior presented to the emergency department with a three-day history of blurred vision and pain in the left eye that worsened with eye movement. On examination, he was found to have a relative afferent pupillary defect and red desaturation in the left eye in addition to hyperreflexia in the bilateral upper extremities. MRI of the brain and orbits demonstrated changes consistent with left optic neuritis with perineural inflammation. Symmetric T2 FLAIR signal along the bilateral corticospinal tract was also noted. Spinal fluid analysis revealed 4 nucleated cells/μL, 2 red blood cells/μL, protein of 38 mg/dL, and glucose of 53 mg/dL. MRI imaging of his cervical and thoracic spine was performed with no evidence of cord lesions. He was started on 1000 mg of intravenous methylprednisolone for three days with rapid improvement of his visual symptoms. He was then treated with a six-day taper of oral

prednisone starting at 60 mg. MOG antibodies returned with 1:1000 titer. Patient had near-complete vision recovery. He was started on rituximab and has continued on this for over a year with clinical and radiographic stability.

3.10. Patient 10

A 31-year-old man with no significant past medical history presented to the emergency department for evaluation for acute weakness in his bilateral upper extremities, bilateral lower extremities, as well as stool and urinary retention. MRI of his cervical and thoracic spine showed a patchy lesion from C7 to T8 and some lesions in the conus medullaris (Fig. 2b). MRI of his brain did not demonstrate any evidence of inflammation. Patient had an extensive work up for autoimmune and inflammatory causes, which was unrevealing. At the time of this presentation, commercial MOG antibody testing was not available. He received 1000 mg of intravenous methylprednisolone for three days, followed by 2 g of IVIg with clinical improvement. However, after discharge he was started on a slow steroid taper starting at 40 mg over four weeks due to subjective worsening of his symptoms. Six years later, he developed acute worsening of his left lower extremity weakness and recurrence of urinary retention. MRI of his thoracic spine showed a focal area of enhancement at T5-T6. He was subsequently admitted and started on 1000 mg of IV methylprednisolone for five days followed by a prolonged steroid taper starting at 60 mg. MOG antibody titer was 1:100. Recommendation was made for patient to start rituximab.

3.11. Patient 11

A 41-year-old man with myasthenia gravis, stable after thymectomy, presented with vision loss and pain in his left eye consistent with optic neuritis based on clinical findings and MRI of the brain showing enhancement of the left optic nerve. He had clinical improvement after treatment with 1000 mg of intravenous methylprednisolone daily for three days followed by oral prednisone taper over 10 days. For two years, the patient remained clinically stable. He was then lost to follow-up but presented to the emergency department at age 51 with vision loss in the left eye and pain with eye movements. In the interim, the patient had been diagnosed with lymphoplasmacytic malignant lymphoma and there was concern for a paraneoplastic process. MRI showed left optic nerve enhancement and spinal fluid analysis demonstrated 3 nucleated cells/μL, protein of 38 mg/dL, glucose of 74 mg/dL, and negative flow cytometry and cytology. The patient was treated for optic neuritis and received intravenous methylprednisolone 1000 mg daily for four days and discharged with an oral prednisone taper starting at 60 mg. Anti-AQP4 antibodies returned positive at > 1:10000 and MOG antibodies were positive at 1:40. After consulting with the patient's oncologist, the decision was made to start patient on rituximab.

3.12. Patient 12

A 64-year-old woman with history of type 2 diabetes mellitus, hypertension, hyperlipidemia, coronary artery disease, obstructive sleep apnea, and hepatitis C and cirrhosis presented to neurology clinic for evaluation of progressive dysphagia over a year and a half. This started as trouble swallowing pills, and progressed to solid food then liquids, with patient requiring a modified diet as she aspirated both thin and nectar thick liquids. She also reported that her voice had changed and was now raspy and hoarse. She was evaluated by gastroenterology and upper endoscopy showed no structural lesions. Otolaryngological evaluation showed normal fiber optic nasal laryngoscopy. Initial neurologic work up evaluated for myasthenia gravis, but her electrodiagnostic studies and antibodies were not consistent with this diagnosis. MRI brain demonstrated encephalomalacia of the right paracentral gyrus. Inflammatory workup was then pursued with lumbar puncture, and serologies including antibody testing for AQP-4 and MOG. Anti-MOG antibodies returned positive at a titer of 1:20. She was started on oral prednisone 30 mg daily with limited improvement in her dysphagia and she elected to have a gastrostomy tube placed. She continued to have worsening of her dysphagia, and MRIs of her brain, cervical and thoracic spine were repeated to evaluated for possible lesion progression. However, there was no evidence of any demyelinating lesions besides the initially reported encephalomalacia. As such, given the atypical presentation for MOG-AD, as well as the absence of demyelination, MOG antibody was retested to confirm initial diagnosis and returned negative. Patient receives symptomatic management of her dysphagia from her primary care provider.

4. Discussion

Similar to what has been previously observed, 81.8% of our patients (nine of the eleven) with MOG-AD had a clear initial presentation of optic neuritis. While bilateral optic neuritis has been described in MOG-AD, only three of our eleven patients demonstrated this at initial presentation, with a remaining six presenting with monocular optic neuritis. MOG-AD has a predilection for the anterior optic nerve with resulting disc edema as opposed to AQP4 –associated neuromyelitis optic spectrum disorder (NMOSD), which tends to involve the posterior optic nerve and chiasm (Jurynczyk et al., 2019). This tendency for anterior involvement was observed in three out of nine of our patients with optic neuritis. Two of our patients presented with longitudinally extensive transverse myelitis (LETM). MOG-AD has been described as having a predilection for the lower thoracic and conus rather than the cervical region (Jurynczyk et al., 2019) and one patient exhibited this pattern while the other had an upper cervical lesion with extension into the medulla. Studies of different patient cohorts show that disease course can be either monophasic or relapsing, with optic neuritis being the most common type of relapse regardless of the initial presentation (Jurynczyk et al., 2019; Nagabushana et al., 2019). Five of the patients (45.5%) had a monophasic course. Of the six patients who experienced recurrences, three patients had only optic neuritis, one had both optic neuritis and LETM, and two had only LETM. Three patients experienced recurrences shortly after tapering oral steroids. This steroid dependency has been previously reported in MOG-AD (Cobo-Calvo et al., 2017). MOG-AD demonstrates no gender preference to a slight female predominance in MOG-AD (Reindl and Waters, 2019). Our patient group shows a similar gender distribution with 5 females and 6 males.

Though MOG antibodies have been associated with acute disseminated encephalomyelitis, none of our patients had this presentation. This could be related to two factors. First, the ADEM phenotype has been shown to be associated with higher MOG antibody titers than the remaining phenotypes with median titers reported as 1:10,000 (Cobo-Calvo et al., 2017). Our patients had titers ranging from 1:20 to 1:1000. Second, all of our patients were adults with age of first attack ranging from 25 to 69 with a mean of 43 years. ADEM predominantly affects a pediatric population, and if present in adults, occurs in younger individuals (Cobo-Calvo et al., 2017).

The significance of MOG antibodies in the setting of coexisting autoimmunity is yet to be determined. When patient 11's optic neuritis recurred, antibodies to both AQP4 and MOG returned positive, with a significantly higher titer for AQP4. It is more likely that his presentation represents a classical AQP4 seropositive case of NMOSD and should be managed as such, with the MOG antibodies representing either a false positive or potential cross reactivity. Coexistence of these titers is quite uncommon, with one study demonstrating no coexisting antibodies in the serum testing of 459 patients (Jarius et al., 2016b). Interestingly, this patient had previously been diagnosed with myasthenia gravis, suggesting an underlying predisposition to immune mediated disease.

During our review, we encountered one patient whose testing resulted in a false positive MOG antibody titer. This patient's clinical presentation was atypical for MOG-AD as she presented with

progressive dysphagia and hoarseness. Imaging of her neuraxis only demonstrated encephalomalacia of her right paracentral gyrus, but there was no evidence of lesions consistent with MOG-AD (Jurynczyk et al., 2017). Initial MOG titer was 1:20 and it returned negative on retesting. While the cell-based assays for MOG antibodies in serum are highly specific (≥99%), the true positive results can easily be outnumbered by false positive results given the low prevalence of MOG-AD in the population if indiscriminate screening is performed (Reindl and Waters, 2019).

MOG antibody testing should be based on clinical and radiographic findings that have been associated with a high pre-test probability. Recommendation has been made against testing if patient has a progressive disease course with imaging findings without evidence of demyelination. The laboratory uses a flow cytometry cell-based assay that was developed using the MOG construct provided by Dr. Waters from the Oxford University group that first established the methods for cell-based assays for measurement of MOG antibodies (Waters et al., 2015).

Moreover, if testing returns positive with a low titer, repeat testing is recommended (up to three times) with consideration of a different cell-based assay (Jarius et al., 2018).

There is no clear consensus on the management of patients with MOG-AD. Acute treatment has been largely adopted from the management of AQP4-associated NMOSD. This consists of intravenous methylprednisolone (IVMP) at a high dose of 500–2000 mg for three to seven days (Wynford-Thomas et al., 2019). This is typically followed by an oral steroid taper; however, no specific recommendations exist on starting dose and duration of taper. In our case series, 7 of the 10 patients who presented acutely received IVMP followed by an oral prednisone taper of varied lengths (7 days to several months). In addition, one patient also received 2 g of IVIg. One patient who presented for outpatient evaluation after his initial presentation never received steroids. It is unclear if the degree of improvement in the patients' symptoms after intravenous methylprednisolone therapy should guide the duration of the oral taper. While all patients in this series had improvement in their presenting symptoms with IVMP, it has been suggested that plasma exchange should be used as a second line treatment, followed by IVIg (Tsantes et al., 2019).

Disease modifying therapies typically used for multiple sclerosis tend to be ineffective (Nagabushana et al., 2019). However, no prospective studies have been completed to assess long-term disease modification in MOG-AD. Recommendations range from long-term oral steroids, monthly IVIg, rituximab, mycophenolate mofetil, methotrexate, and azathioprine (Jurynczyk et al., 2019). The ideal duration of continuing immunosuppressive treatment is unclear. Some experts suggest treatment course based on antibody titer levels while others recommend relying on clinical and radiographic evidence of no relapses. The majority of patients in this report were counseled to start treatment with rituximab. One patient was offered mycophenolate mofetil, while two other patients have been followed clinically without starting disease modifying treatment.

At our center, if patients present acutely with either optic neuritis or LETM, we recommend testing for MOG antibodies in additionn to AQP4 antibodies and starting IVMP dosed at 1000 mg daily for at leaset 3 days.At our center, if patients present in acutely with either optic neuritis or LETM, raising suspicion for MOG-AD, we recommend testing for MOG antibodies and starting IVMP at 1000 mg for at least 3 days. We expect that serum is drawn before the first dose of IVMP and sent to the same laboratory for testing. Depending on the degree of improvement in the patient's presenting symptoms, IVMP may be extended to a total of 5 days or IVIg could be considered. Most patients are typically discharged home on an oral prednisone taper, scheduled over the course of 10–14 days. Patients are usually scheduled for follow up in clinic within 4–6 weeks after their discharge. At that time, if their acute presentation represented a first instance of MOG-AD, we offer initiation on disease modifying treatment or clinical monitoring as MOG-AD can have a monophasic disease course. However, if the patient's history reveals a recurring pattern of localizing neurologic symptoms, we typically recommend initiation of immunomodulatory treatment. Most of our patients who have elected to start treatment have chosen to start on rituximab. At the time of writing, three of our patients have been on rituximab for more than a year with no evidence of recurrence.

5. Conclusion

In the last several years, several clinical descriptions of MOG-AD have been published. Our case series presents the clinical phenotype of MOG antibody positive patients at our academic center and is consistent with prior experience with MOG-AD. Prospective studies are needed to help guide clinical decision making regarding the appropriate immunologic targets, duration and intensity of treatment. Additionally, further studies are needed to gather outcomes information on patients treated with immunomodulatory therapies so as to better guide management.

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