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Ocular Myasthenia Gravis; a Case Report


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Ocular Myasthenia Gravis; a Case Report

Abstract

Abstract

Background: Myasthenia gravis (MG) is an autoimmune disease disorder that affects the skeletal muscles causing increased fatigability and weakness. When it affects the ocular muscles only, it is known as ocular myasthenia gravis (OMG). Patients with OMG will often have symptoms of diplopia and ptosis, which will be worse towards the end of the day. This case report will discuss the common symptoms of OMG as well as the appropriate treatment and management. It will also review the diagnostic tests used to aid in the diagnosis of OMG and discuss the prognosis of OMG. **Case Report:** A 67-year-old male presented with a chief complaint of diplopia that was variable throughout the day. Additionally, he complained that his left eyelid was drooping. Clinical examination revealed a mild ptosis of the left eye and right adduction deficit. An MRI/MRA of the head and orbits was ordered, along with a myasthenia panel. His MRI and MRA were unremarkable, and he tested negative for acetylcholine receptor antibodies. A neurologic exam was completed, and the patient was diagnosed with late-onset, triple antibody negative, ocular myasthenia gravis. His symptoms improved once he was placed on prednisone.

Conclusion: When a patient presents with pupil-sparing, painless, diplopia, ptosis or ophthalmoplegia, OMG should always be considered. While the diagnosis relies heavily on history and physical examination, serologic, radiographic, and electrophysiologic testing are used to support the diagnosis. First line treatment for patients with OMG is pyridostigmine; however, patients will often require corticosteroids. Some may continue to have diplopia or ptosis even with maximal medical therapy. When this occurs, prism or surgery is an option. The prognosis for OMG is good with most patients reaching remission.

Keywords

ptosis, diplopia, single fiber electromyography, acetylcholine receptor antibodies

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Background

Myasthenia gravis (MG) is an autoimmune disease that affects the post-synaptic neuromuscular junction causing fatigable weakness. When it affects the extraocular muscles only, it is known as ocular myasthenia gravis (OMG). Patients with OMG will often have symptoms of diplopia and ptosis, which will be worse towards the end of the day. This case report will provide a review of the existing literature with a case of OMG to illustrate it.

Case Report

History

A 67-year-old male presented to the eye clinic at the Minneapolis Veterans Affairs Medical Center (VAMC) with complaints of double vision. Diplopia was noted to be both horizontal and vertical in nature, variable throughout the day, and alleviated by closing one eye. It had been ongoing for the prior six months. Additionally, the patient noted that his left eyelid was drooping. His last exam was two months prior, outside the VA by a local ophthalmologist who obtained an MRI of his brain and brainstem which did not show any evidence of an acute abnormality, no evidence of a cause for the patient's symptoms, and no intracranial metastatic disease. The diagnosis at that time was thought to be decompensating phoria as a result of chemotherapy for his lymphoma. His ocular history was positive for an alternating exotropia but was otherwise unremarkable as was his family ocular history. His medical history was positive for B cell lymphoma, which was in remission for five years.

Exam at presentation

Upon presentation to the VA eye clinic, best corrected visual acuities were recorded to be 20/20 in the right eye and 20/25 in the left eye. Pupils were round, regular, and reactive with no afferent pupillary defect. Muscle balance indicated a 10 prism diopter (pd) alternating exotropia with a 5pd left hypertropia. Extraocular muscle motility showed a right adduction deficit. Confrontation visual fields were full to finger counting. Goldmann applanation tonometry findings were 16 mm Hg right eye and 17 mm Hg left eye. An undilated fundus

examination was unremarkable with a cup to disc ratio of 0.35 in the right eye and 0.30 in the left eye, good perfusion, and clear distinct margins.

External evaluation indicated a left ptosis, marginal reflex distance 1(MRD1) in the right eye 4 mm and MRD1 in the left eye was 0.5 mm. His corneas were clear, and he had a deep and quiet anterior chamber with early nuclear sclerotic cataracts in both eyes.

The patient's diplopia was neutralized 90% of the time with 20 pd BI and 5 pd BD OS. This was placed in a trial frame, and the patient experienced an improvement with his symptoms. An MRI with and without contrast of the brain, brainstem and orbits along with an MRA of the head was ordered by the optometrist who first saw the patient to rule-out an aneurysm or compressive lesion involving multiple cranial nerves. Additionally, a myasthenia panel was ordered. The results of his MRI and MRA came back negative with no evidence of intracranial mass, hemorrhage, or aneurysm. Additionally, he tested negative for acetylcholine receptor antibodies

The patient was scheduled to follow-up in one week for completion of his examination including a dilated funduscopy examination. The patient was referred to neuro-ophthalmology within 2 weeks for further evaluation and work up.

One-week follow-up

At his one-week follow-up, the patient reported that his double vision was not improved with his glasses with prism that were prescribed at his last visit; the prism did not provide any relief. The patient added that his symptoms were stable, but worse toward the end of the day. By evening, the ptosis became so bad that his left eye would close. Visual acuities were stable in each eye, a cover test at distance suggested 8pd exotropia with 5 pd left hypertropia, with some variability noted on testing. Marginal reflex distance 1 was 4 mm in the right eye and 0.5 mm in the left eye. Intraocular pressures were 19 mm Hg OD and 20 mm Hg OS with Goldmann applanation tonometry. A dilated fundus exam revealed cup to disc ratios of 0.35 round in the right eye and 0.3 round in the left eye, posterior and peripheral fundus exam was normal in both eyes. Due to the variability in his diplopia and minimal improvement with prism in his glasses, we recommended he wear a patch and alternate the patched eye each day to provide some functional relief. He was scheduled for his neuro-ophthalmology

consultation in 2 days and recommended to follow-up with the VA eye clinic in 2 months.

Neuro-ophthalmology consultation

A thorough neurologic exam was completed by the neuro-ophthalmologist. Myasthenia Gravis (MG) was highly suspected thus muscle-specific tyrosine kinase (MuSK) antibodies and low-density lipoprotein receptor-related protein 4 (LRP4) antibodies were tested. Both antibodies came back negative. Due to the association of a thymoma with MG, a CT of the chest was ordered and revealed no evidence of thymic tissue or thymoma. At this point, although all testing for MG was negative, OMG was still highest on the differentials. A single fiber electromyography was eventually completed which resulted in abnormal jitter in the orbicularis oculi suggesting the diagnosis of OMG.

Although the patient did not have any symptoms of autoimmune thyroid disease, due to the strong correlation between MG and autoimmune thyroid disease, thyroid testing was completed. Results were negative for autoimmune thyroid disease.

The patient was diagnosed with late-onset, triple antibody negative, non-thymomatous, ocular myasthenia gravis, which was considered a diagnosis of exclusion since all other neurological testing was found to be normal. Treatment was initiated with the traditional anticholinesterase therapy, pyridostigmine 60 mg three times a day (TID) for one month without any improvement of his symptoms. At this time the decision was made to switch to immunosuppressive treatment. Prednisone was started at 20 mg for a month with no resolution in symptoms. The patient finally noticed a remarkable improvement in his symptoms once the prednisone was increased to 60 mg.

2-month VA Follow-up

The patient presented back to the VA for a follow-up and reported that his ocular symptoms improved since taking prednisone 60 mg daily. Visual acuities remained stable in each eye, and there was no ptosis noted at this exam even after sustained up gaze, extraocular motilities were full, cover test suggested resolution of exotropia at distance with only 5 pd left hypertropia remaining. At this visit, intraocular pressures were 30 mm Hg in the right eye and 29 mm Hg in the left eye, and a diagnosis of steroid induced ocular hypertension was made. The patient

was started on 2% dorzolamide hydrochloride two times a day (BID) in each eye, 0.5% timolol maleate twice a day in each eye, and was scheduled for a follow-up in 2 months for the management of his ocular hypertension. He was referred to a neuromuscular physician for consideration of long-term steroid sparing immunosuppressive therapy.

Two months after starting ocular hypotensive treatment, the patient was doing well on 2% dorzolamide hydrochloride 0.5% timolol maleate.. Intraocular pressures were reduced by over 20% and were 21 mm Hg in the right and left eye. He had begun a steroid taper and was down to 15 mg of prednisone daily with the hopes of stopping prednisone soon. He maintained a 5 pd left hypertropia even with treatment and therefore it was determined to be unrelated to the OMG. The patient was prescribed glasses with 5 pd ground in prism (split between the two eyes); he reported that he was doing well with the glasses and was no longer wearing his patch over his left eye.

Over the course of two years, he continued his care with his neuromuscular physician and eventually was able to reduce his prednisone to 5 mg daily. His symptoms relapsed approximately one year into treatment and the dose was adjusted up to 60mg again with a slow taper back down eventually to 5 mg daily with good symptom control. During this time intraocular pressures remained well controlled on 2% dorzolamide hydrochloride 0.5% timolol maleate BID OU. Eventually as the steroid was tapered, and out of extreme caution for timolol to exacerbate myasthenia symptoms, this was switched to latanoprost 0.005% in the evening (QHS) OU with continued good control.

Neurology follow-up visit

The patient was last seen in late 2021 by neurology. He had decreased his prednisone to 2.5mg daily and was doing well. He continued to deny diplopia, difficulty with speech, swallowing, weakness or any other focal neurologic symptoms. The goal is to eventually try to taper the prednisone completely over time.

Discussion

Myasthenia gravis is an uncommon autoimmune disorder affecting the neuromuscular junction resulting in fatigable weakness of the affected muscle.

MG is reported to have an annual incidence of approximately 2.2 per 100 000 and 1.1 per 100 000 incidence of OMG.¹ Studies are highly variable, but suggest 40-85% of patients initially present with ocular symptoms, isolated to the extraocular muscles and the orbicularis oculi, resulting in variable diplopia and ptosis.¹⁻⁶

Approximately 50% of these patients will develop generalized myasthenia gravis (GMG) within 2 years, exhibiting systemic symptoms including dysphagia, dysarthria, dyspnea, dysphonia, and neck or extremity weakness.¹⁻⁶

Approximately 15% of all MG patients will be diagnosed with purely ocular MG.⁵ MG can affect both men and women at any age. Women show a bimodal distribution peaking at the age of 30 and again at the age of 60, whereas men typically show a peak incidence at 70.⁴ Although OMG is considered non-life threatening, it is critical to continue to monitor for conversion to GMG as this can have potentially life-threatening complications including respiratory failure. If the disease remains purely ocular after 2 years, there is a roughly 90% chance that generalization will never occur.⁴⁻⁶

Testing and diagnosis of OMG can be challenging due to the highly variable nature of the disease and the complex pathophysiology.² Most often autoantibodies against the AChR have been implicated in the disease pathway. Recent research has demonstrated that with advanced knowledge of the neuromuscular junction, other proteins, including muscle-specific kinase (MuSK), and low density lipoprotein receptor-related protein 4 (LRP4) have been identified in the motor end plate.² These proteins may be implicated in the disease process and may be a potential target for disease modification or treatment.² Ultimately, a reduced transmission of acetylcholine leads to fatigable muscle weakness, a hallmark of the disease.³⁻⁶ Many patients with OMG produce no detectable AChR antibodies, which is considered seronegative myasthenia gravis or antibody negative myasthenia gravis.^{2,3,7}

The clinical presentation is weakness or fatigue of the extraocular eye muscles and/or orbicularis oculi, resulting in variable double vision and or ptosis in the setting of normal pupillary function, sensory function and visual acuity.³⁻⁴ Weakness is variable, increases with repeated or sustained muscle use throughout the day, and improves with rest, sleep and with cold temperatures.^{3,8,9} It is crucial to assess pupils in every patient suspected of having OMG as it is important to rule-out other alternative potentially life-threatening conditions, such as chronic progressive external ophthalmoplegia, mitochondrial myopathy, and single or multiple cranial nerve palsies.⁴

It is important that clinicians recognize iatrogenic sources of MG. Immune checkpoint inhibitors have become powerful in the management of malignancy and with their increasing use, have become recognized as having the potential to induce MG.^{2,10} It is important to screen patients on immune checkpoint inhibitor for signs and symptoms of MG and to review systemic medication carefully in anyone presenting with signs of variable ptosis and or double vision for possible drug induced MG.

Diagnosis

While the clinical diagnosis heavily relies on the history and physical examination, non-pharmacological, pharmacological, laboratory and electrophysiological testing may all be used to aid with the diagnosis.³ Commonly known non-pharmacological tests include the fatigue test, sleep test, and ice pack tests. The fatigue test is done by having the patient sustain upgaze for a few minutes and noting an increase in ptosis or double vision. The rest test looks for improvement with the ptosis after the patient closes their eyes for two-five minutes.¹¹ With the ice pack, ice is applied to the closed eyelid. After a few minutes, improved ptosis is considered a positive test.¹¹ While this test is not diagnostic, it does have an 80% sensitivity in those with OMG and can significantly aid with the diagnosis of OMG.¹¹ The ice pack test works on the principle that neuromuscular transmission is improved at lower muscle temperatures.¹¹ Cogan's lid twitch is another useful clinical sign that can point toward the diagnosis of MG.¹¹ Patients are asked to look downward for 15 seconds and then return to primary gaze, if the affected lid shows an overshoot or an upward twitch and then drops back down, this is a positive result and suggestive of MG.

Pharmacological testing involves administering a short-acting reversible acetylcholinesterase inhibitor known as the edrophonium (Tensilon) intravenously.³ Intravenous edrophonium will transiently increase the availability of acetylcholine at the receptor sites.^{3,12} The edrophonium test has the potential to have severe cardiovascular side effects and because of this, this test is no longer available in the United States and many other countries.³

More reliable tests include serologic tests for autoantibodies as well as electrophysiologic studies.⁷ Serological testing tends to be more helpful in the diagnosis of MG; however, it is not as helpful with OMG.^{3,7} This is because the sensitivity of acetylcholine receptor antibody (AChR-Ab) testing in OMG is only

45 to 60% where it can be as high as 90% in those patients with GMG.^{3,12} There are additional antibodies which can be tested which include anti-muscle specific kinase (MUSK) and low-density lipoprotein receptor-related protein 4 (LRP4).⁷ However, many patients with purely OMG will be seronegative for all three antibodies.^{3,7,13}

Electromyography (EMG) is another diagnostic test used to support a clinical diagnosis of OMG, especially in seronegative OMG.³ Repetitive nerve stimulation (RNS) is the most frequently used electrodiagnostic test for MG because of its wide availability.¹¹ While the sensitivity of RNS is relatively high for MG, the sensitivity is much lower in patients with OMG.¹¹

A more sensitive test than the traditional EMG or RNS is single fiber EMG (SF-EMG).¹⁴ SF-EMG is an advanced method of determining neuromuscular defects.¹⁴ It is often abnormal for patients with both MG and OMG even when RNS and serologic testing show no abnormalities.¹⁵ It records the action potentials of two muscle fibers innervated by the same motor axon and measures the jitter, which is the variability in time of the second action potential relative to the first.¹¹ Abnormal jitter is found in up to 99% of MG patients and up to 80% for OMG patients.^{3,4} Muscles commonly tested include the orbicularis oculi, the extensor digitorum communis or the frontalis muscle.⁴ While SF-EMG plays an important role in the diagnosis of MG and OMG, it is not universally available and it is time consuming.¹⁴ Further, it requires more patient cooperation and takes a skilled clinician to administer this test.¹⁴

It is especially important to obtain neuroimaging studies to rule out an intracranial mass in atypical cases.¹⁴ These atypical cases include those that are unilateral or seronegative.^{3,14} Additionally, a CT or MRI of the chest should be obtained for every patient with suspected myasthenia gravis to assess the thymic status, looking for a thymoma.⁴ The majority of MG patients found to have a thymoma will have positive AChR-Ab. It is important to keep in mind that seronegativity does not exclude the possibility of finding a thymoma.⁴

Approximately 15% of patients with MG will develop other autoimmune diseases.^{4,7,16} The most common condition being autoimmune thyroid disease.^{4,7,16} Because of this, thyroid testing should be included as part of the initial work-up for all patients suspected of having MG.^{7,16}

Treatment

The goal of therapy is to minimize the patient's symptoms in both OMG and GMG.¹⁷ Another goal of treatment is to prevent the progression of OMG to GMG.¹⁷ Patients with OMG are often initially started on acetylcholinesterase inhibitors.^{2-4,8,16,18} The most commonly prescribed drug within this class of medication is pyridostigmine, brand name Mestinon.⁴ Dosing generally starts low but can be increased if tolerated and needed. A patient's ptosis will generally improve, but often will continue to have motility deficits with this therapy.⁴ As a result, many patients with OMG will require immunosuppressive treatment. The first line immunosuppression medication is most often prednisone.^{4,16,18} Exact dosing of prednisone can vary amongst practitioners, but commonly is dosed low initially and with gradual increase until therapeutic achievement is met.^{2,16,18,19} To avoid long term steroid use, steroid sparing agents may be introduced throughout the disease course.^{4,18} The most common steroid sparing agent used is azathioprine.^{4,20} Unfortunately, therapeutic effects can be delayed many months with azathioprine.⁴

Intravenous immunoglobulin and plasmapheresis are both mainstays in treatment for GMG but are limited by administrative challenges. Neither of these treatments are typically used for OMG.¹⁸ A thymectomy is also not typically recommended in patients with OMG without thymoma.⁴ Rarely, OMG patients positive for acetylcholine receptor antibodies and unresponsive to medications could be considered for thymectomy. This would be an individualized decision and is somewhat controversial.^{4,18,21} In December of 2021, the FDA approved intravenous efgartigimod (Vyvgart) for the treatment of GMG in adults who are anti-AchR antibody positive. Efgartigimod is an Fc receptor antagonist.²² Fc receptors function to prolong the life-span of immunoglobulin G (IgG) which binds to the postsynaptic AchRs resulting in impaired neuromuscular function. By targeting the Fc receptor less IgG is available to bind to the AchRs and less inhibition of acetylcholine-dependent signaling. Efgartigimod is administered as weekly hour-long infusions over the course of four weeks.^{22,23} Additional treatment cycles are then administered based on the clinical response. Efgartigimod offers benefits with its selective targeting of IgG autoantibodies resulting in early onset of action and has shown a favorable tolerance profile.^{22,23} Side effects to efgartigimod use have been found to be mild to moderate with the most common side effects being respiratory tract infections, headache and urinary tract infections.^{22,23} Corticosteroids and non-steroidal immunosuppressive therapies, on the other hand, will broadly suppress the immune system often

resulting in more burdensome side-effects.²³ While there continues to be an ongoing open-label extension study, the results thus far show that efgartigimod is an effective and well tolerated treatment for patients with GMG.²³

Prognosis

With the use of short-term corticosteroids, the prognosis of OMG is generally good.³ Resolution of symptoms has been reported in many patients.³ Some patients may continue to have diplopia despite medical therapy.^{3,24,25} In these cases, prism or occlusion therapy is an option.²⁵ Strabismus surgery may be a possibility when the ocular deviation has been stable for 6 months; however, the prognosis is variable.^{3,25} Although persistent ptosis can be surgically repaired, outcomes are variable and considerations should be made for ptosis crutches and or lid taping.³

Conclusion

When a patient presents with variable pupil-sparing, painless, diplopia, ptosis or ophthalmoplegia, OMG should always be considered. For atypical cases, neuroimaging needs to be obtained. Thyroid function tests and a chest CT, to rule out thyroid disease and thymoma, should also be obtained. While the diagnosis relies heavily on history and physical examination, serologic, radiographic, and electrophysiologic testing are used to support the diagnosis. First line treatment for patients with OMG is typically pyridostigmine; however, for patients who do not respond to treatment, oral immunosuppressive therapy can be helpful. The prognosis for OMG is relatively good with most patients reaching remission at some point during their disease. It is critical to continue to monitor OMG patients for conversion to GMG for years after diagnosis and review systemic medications like checkpoint inhibitors that may exacerbate or induce disease.

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