# The peculiar case of myasthenia gravis disguised as Miller Fisher syndrome

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## Introduction

Miller Fisher syndrome (MFS) is a rare variant of Guillain-Barré syndrome (GBS). Miller Fisher syndrome is an autoimmune phenomenon that occurs after infection with C. jejuni, CMV, or HIV.1,2 During infection, the viral or bacterial proteins closely resemble host proteins of the body, which leads to antibodies being created against host cells or proteins. This type of antibody formation is known as molecular mimicry. In the case of MFS, the anti-GQ1b antibody is created against gangliosides within the nervous system, which leads to demyelination. The classic symptoms secondary to the demyelination are ophthalmoplegia, ataxia, and areflexia. Several of these clinical signs are also common in Myasthenia Gravis.

Myasthenia Gravis (MG) is another autoimmune disease that occurs secondary to antibodies that are formed against the acetylcholine receptor (anti-AchR) located at the postsynaptic membrane of skeletal muscles. The anti-AchR antibodies compete against acetylcholine for binding at the neuromuscular junction, which leads to an increasing weakness of skeletal muscles over time of use.4 The classic presentation of MG is ophthalmoplegia, dysarthria, skeletal muscle weakness, and ptosis that typically get worse as the day progresses. Given the similarities between these two autoimmune diseases, diagnosis can be difficult. Here, we present a case of Myasthenia Gravis that was initially disguised as Miller Fisher syndrome.

## Case Description

71-year-old male complaining of week-long worsening dysphagia accompanied by fatigue, generalized weakness, weight loss and the inability to swallow solids and eventually liquids. He denied any recent travel, outdoors activities, chest pain, throat pain, voice or visual changes. Initial physical exam was positive for tachycardia and hypertensive urgency with systolic blood pressure of greater than 200 which improved with labetalol. Hours later the physical exam developed to include diplopia, muffled voice, ptosis, ataxia, hyporeflexia, bilateral facial paralysis (CN VII) hyporeflexia and bilateral ophthalmoplegia affecting cranial nerves II, III, and VI. Electrocardiogram showed sinus tachycardia while chest x-ray and computed tomography (CT) imaging studies of the head, neck, chest and abdomen conducted within the emergency department were unremarkable for acute findings, except for diverticulitis within the bowel.

## Case Description

MRI of brain was unremarkable for acute findings, but did expressed mild cerebral atrophy and signs of chronic small vessel ischemia. Labs revealed anti-Acetylcholine receptor (anti-AchR) binding antibodies, leukocytosis, hyponatremia, and hypokalemia. Patient's labs were negative for rickettsia antibodies, Lyme antigens, anti - muscle-specific kinase (anti-Musk) antibodies and oligoclonal bands and cryptococcal antigen on CSF analysis. GQ1b antibody screening was not tested following results.

Empiric symptomatic treatment with pyridostigmine and IVIG occurred before antibody panels resulted. After antibody panel resulted we continued treatment with IVIG for a total of five days. He exhibited acute improvements of symptoms within hours of empiric treatment.

## MRI of brain

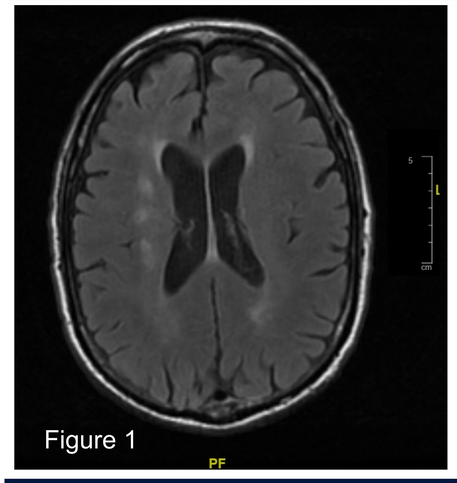
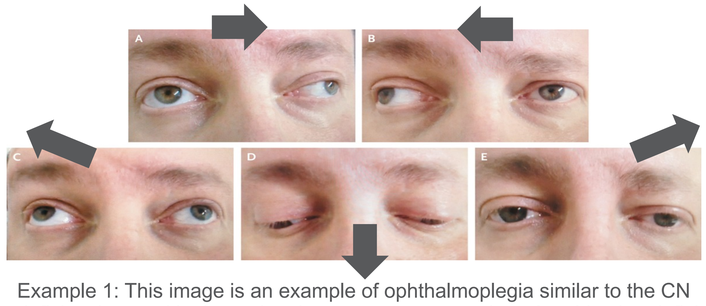



Figure 1:
Axial View of MRI of brain showing mild cerebral atrophy, right non-specific changes with chronic small vessel ischemic disease.

## Example of Ophthalmoplegia




Example 1: This image is an example of ophthalmoplegia similar to the CN deficits observed in the patient in our case presentation. A. adduction deficit of right eye, B. adduction deficit of left eye. C. Trochanteric deficit of left and right (E) eye. D. looking down. Source: Hassen et al.

## Discussion

Acute neurological deficits carry a wide range of diagnoses that must take into account the patient's age, environmental exposures, history and physical. The primary differential diagnoses for this patient included cerebrovascular accident, myasthenia gravis (MG), multiple sclerosis, GBS and Miller Fisher syndrome (MFS). Diverticulitis in addition to ophthalmoplegia of CN II, III, and VI, ataxia and hyporeflexia were factors which favored MFS disorder. The lack of Anti-Musk antibodies were an interesting finding in the setting of elevated Anti-AchR antibodies. Anti-Musk receptor antibodies are seen in 30-40% of Anti-AchR seronegative patients with MG and if present commonly changes the therapy for treating MG.

By keeping our differential diagnosis broad and empirically treating this patient for MG we were able to prevent delay in appropriate treatment and management. A delay in treatment by awaiting antibody results could have resulted in a fatal worsening of myasthenic crisis. MG and MFS have been found in co-existence and presents with ophthalmologic similarities. MFS cannot be ruled out due to the lack of antibody testing in this case. Although GQ1b antibodies were not tested, the likelihood of an individual having both MG and MFS synonymously is very low. MFS often self resolves over several weeks but symptoms have been shown to improve with IVIG.

## Conclusion

Although this patient's presentation resembled MFS, it is important to keep common differentials such as myasthenia gravis or GBS in mind. Considering common life threatening diagnoses and providing swift empirical management is important to improve mortality related to these conditions.

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