An Atypical Presentation of Miller Fisher Syndrome: A Case of Diagnostic Dilemma and Successful Treatment

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Introduction

Miller-Fisher Syndrome (MFS) is an atypical variant of Guillain-Barre Syndrome (GBS), an acute immune-mediated polyradiculopathy. The typical variant of GBS usually presents with ascending symmetrical paralysis and areflexia. However, MFS usually presents with a **triad of ophthalmoplegia (initial symptom in 80% cases), followed by ataxia with variable onset of areflexia.**

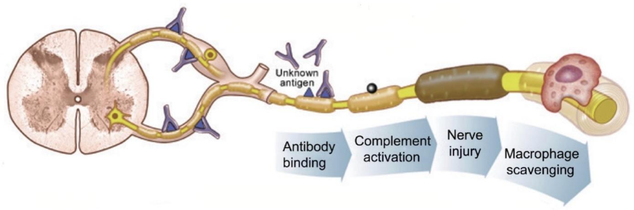

Fig 1. Miller Fisher Syndrome

Classically, **GQ1b autoantibodies** are involved in the pathogenesis. *We present an atypical case of MFS in a middle-aged male with a complex medical history who initially presented with progressive generalized weakness, ataxia and dysarthria – **but notably without ophthalmoplegia or areflexia.***

Patient Description

Presentation	Elevated home blood glucose, weakness, and shakiness
Characteristics	African-American male in his 50s
Past Medical History	Bipolar I, Schizophrenia, Hypertension Type 2 DM, Chronic Kidney Disease, Hyperlipidemia, Substance use disorder
Medications	Paliperidone, Metformin, Glipizide Carvedilol, Amlodipine, Losartan Prescribed but mostly nonadherent

Table 1: Patient Presentation upon admission.

Clinical Course

Admission
- Hyperosmolar Hyperglycemic State, Hypertensive Crisis and Acute Kidney Injury
- Generalized weakness and lightheadedness upon ambulation

Day 4 -6
- Significant bilateral extremity (UE > LE) and truncal ataxia
- Dysdiadochokinesia and dysmetria; Dysarthria and dysphagia
- Imaging

Day 8
- Reduced reflexes with unchanged dsyarthria and ataxia; full ocular motility
- Imaging/Labs
- Interventions: **5 day course of Empiric Steroids**

Day 14
- Unchanged ataxia, dysarthria and dysphagia with areflexia
- New dysconjugate upward gaze, bilateral distal UE weakness and UE/LE dysesthesias
- Interventions: **5 day course of Empiric IVIG**

5 Months Post-Discharge
- Patient reported complete resolution of symptoms
- Normal full neurological physical examination

Fig. 3: Clinical Timeline and Management of a Patient with MFS; dysdiadochokinesia = difficulty performing rapid alternating movements; dysmetria = difficulty with movement accuracy.

Labs/Imaging

Evaluation	Results
MRI Brain w and wo contrast	**No acute abnormalities;** mild cerebral and cerebellar atrophy
CSF WBC count	**48** (lymphocytic predominance)
CSF Protein	**33 mg/dL**
Culture CSF with gram stain	**No growth**
GQ1b antibodies	**1:1600 titer**

Table 3: Relevant Diagnostic Findings.

References

Investigations

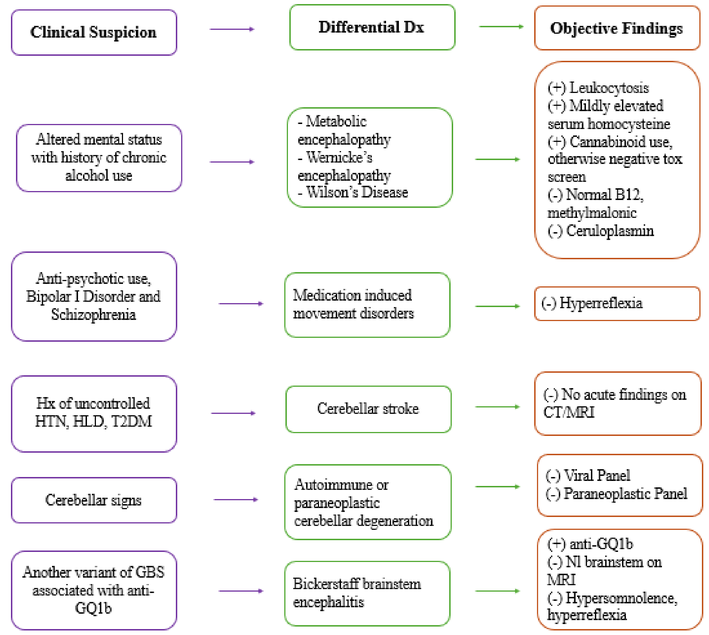

Fig. 4: Differential Diagnosis of MFS with Supporting Labs, Imaging and Objective Findings. Pertinent positives (+) and negatives (-).

Discussion/Conclusion

Typical MFS	Patient Presentation
Early onset ophthalmoplegia	**Delayed** ophthalmoplegia
Lower extremities ataxia	Pronounced **upper** extremities ataxia
Albuminocytologic dissociation on CSF	**Absent**
GQ1b antibodies	**Present**
IVIG treatment	**Improved**

Table 3: Typical MFS presentation vs Patient Presentation.

The patient was ultimately diagnosed with MFS. The GQ1b antibodies are highly specific and sensitive for MFS. Additionally, CSF albuminocytologic dissociation is not required for diagnosis, although it provides high diagnostic certainty. Therefore, this case demonstrates the importance of combining clinical reasoning and diagnostic testing in the setting of atypical clinical manifestations.