Lower Extremity Manifestations in Miller Fisher Syndrome, an Atypical Variant of Guillain-Barré Syndome: Case Report

Tayyaba Hasan DPM[1], Nelson Maniscalco DPM[1], Angela Costa DPM[1], James Sullivan DPM[2]
1 Resident Physician, HMH Jersey Shore University Medical Center, Neptune, NJ
2 Attending, Podiatric Medicine and Surgery, HMH Jersey Shore University Medical Center, Neptune, NJ

Hackensack Meridian *Health*

LEARNING OBJECTIVE

This presents a case of Miller Fisher syndrome, an atypical variant of Guillain-Barré syndrome (GBS) with lower extremity manifestations.

LITERATURE REVIEW

Guillain-Barré syndrome (GBS) is a heterogeneous syndrome encompassing acute inflammatory polyneuropathies of the peripheral nervous system. Seventy percent of cases follow acute infection and typically present with ascending, progressive, symmetric muscle weakness with absent or depressed deep tendon reflexes (DTR). GBS is theorized to be immune mediated from a preceding infection that cross-reacts with peripheral nerve components (1). Miller Fisher syndrome, originally described by James Collier in 1932, is a rare variant of GBS classically presenting with ophthalmoplegia, ataxia, and areflexia. One quarter of these patients develop extremity weakness (2, 3). Miller Fisher syndrome accounts for one to five percent of all GBS cases in Western countries and has a higher incidence in men. It is often preceded by an upper respiratory tract infection with *Campylobacter jejuni* and *Haemophilus influenzae* as the most common pathogens (1,4). Antibodies against GQ1B, a ganglioside component of nerves, are present in 85-90% of patients with Miller Fisher syndrome, serving as a diagnostic tool (5).

CASE STUDY

22-year-old male presented with complaints of difficulty walking and leg tingling for two weeks duration. He reported worsening of symptoms over the last several days, prompting his hospital visit. He also reported left ear pain that began just prior to his lower extremity symptoms. The laboratory testing revealed normal values for complete blood count and comprehensive metabolic panel. On physical examination, neurologic abnormalities were noted including finger-to-nose dysmetria, positive horizontal nystagmus, as well as ataxic gait, difficulty standing, and bilateral ocular abduction deficits. DTRs were +1 of the biceps, brachioradialis, patella but ankle DTR was 0. A brain computed tomography (CT) showed no acute pathology and serum TSH, B12, and folate levels were within the normal range. A panel of infectious markers (including HIV, West Nile, Lyme, HSV) was negative. A lumbar puncture was performed and the cerebrospinal fluid (CSF) analysis demonstrated normal levels of protein with a mild increase in white blood cells (9/ul) and a negative culture. Given a high index of suspicion for Miller Fisher syndrome, a GQ1B autoantibody test was obtained and returned with a strong positive result, leading to the diagnosis of Miller Fisher syndrome preceded by otitis media. The Miller Fisher syndrome and otitis media were treated with IVIG and a ten-day course of Augmentin (respectively). He was discharged from the hospital after five days of IVIG treatment, with significantly improved gait stability, DTRs, ocular movements, and resolved nystagmus.

ANALYSIS/DISCUSSION

Miller Fisher syndrome is an atypical variant of GBS, with the clinical triad of ophthalmoplegia, ataxia, and areflexia. Patients with these symptoms can often be mistakenly diagnosed with a brainstem stroke or Wernicke encephalopathy. These are differentiated from Miller Fisher syndrome by the acute onset of symptoms or altered mental status associated respectively (6). GBS and Miller Fisher syndrome are frequently preceded by an upper respiratory tract infection, however can also be present following other infectious processes, immunization, surgery, trauma, or bone marrow transplantation (4). Additionally, in 60% of GBS cases, a lumbar puncture will reveal elevated CSF protein and normal CSF white blood cell count, a finding known as albuminocytologic dissociation (7). As in other forms of GBS, the neurologic process can affect the facial and respiratory muscles, thus necessitating a swift and accurate diagnosis. This case is a classic presentation of a rare pathology, noting the importance of meticulous history taking and fastidious examination to diagnose.

Figure 1a and 1b –Laboratory Findings

B12	675 pg/ml
Folate	17.3 ng/ml
HIV 1&2Ag/AB	Negative
HSV	Negative
Lyme titer	0.75
Stool culture	Negative
GQ1b Antibody	245 IV

CSF Character	Clear, colorless
CSF WBC	9
CSF Protein mg/dL)	15
CSF Glucose (mmol/L)	62
CSF Lymphocytes (%)	96
CSF Culture	No growth

Laboratory findings and cerebrospinal fluid analysis.

REFERENCES

1. Snyder LA, Rismondo V, Miller NR. The Fisher variant of Guillain-Barré syndrome (Fisher syndrome). J Neuroophthalmol. 2009, 29:312-324.
2. Fisher M. An unusual variant of acute idiopathic polyneuritis (syndrome of ophthalmoplegia, ataxia, and areflexia). N Engl J Med 1956; 255:57.
3. Fross RD, Daube JR. Neuropathy in the Miller Fisher syndrome: clinical and electrophysiologic findings. Neurology 1987; 37:1493.
4. Mori M, Kuwabara S, Fukutake T, et al. Clinical features and prognosis of Miller Fisher syndrome. Neurology. 2001, 56:1104-1106.
5. Lee SH, Lim GH, Kim JS, et al. Acute ophthalmoplegia (without ataxia) associated with anti-GQ1b antibody. Neurology 2008; 71:426.
6. Bukhari S, Taboada J. A Case of Miller Fisher Syndrome and Literature Review. Cureus 9(2): e1048.
7. Yuki N, Hartung HP. Guillain- Barré syndrome. N Engl J Med 2012; 366:2294.