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REVIEW ARTICLE

Miller Fisher syndrome: A comprehensive review and clinical insights

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Abstract

Miller Fisher syndrome (MFS) is a variant of Guillain-Barré syndrome (GBS), characterized by the clinical triad of ophthalmoplegia, ataxia and areflexia. Anti-ganglioside GQ1b antibodies, detected in 80–90% of cases, play a central role in molecular mimicry-driven pathogenesis. While albuminocytological dissociation in cerebrospinal fluid (CSF) and electrophysiological findings (e.g., sensory axonal neuropathy, absent H-reflex) aid diagnosis, overlap syndromes with GBS or Bickerstaff brainstem encephalitis (BBE) necessitate early recognition to guide management. This review summarize current evidence on MFS pathogenesis, diagnostic challenges, and treatment paradigms, while proposing novel hypotheses on genetic predispositions and calcium-dependent antibody mechanisms.

Keywords: Miller Fisher syndrome, anti-GQ1b antibody, molecular mimicry, overlap syndrome, immunotherapy

INTRODUCTION

Guillain-Barré syndrome (GBS) encompasses acute immune-mediated neuropathies, characterized by progressive weakness of upper and lower limbs and cranial nerves.Its primary variants include acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN) and Miller-Fisher Syndrome (MFS).[1] MFS distinguishes from other GBS variants by the clinical triad of ophthalmoplegia, ataxia and areflexia. Bickerstaff's brainstem encephalitis (BBE) represents another variant of GBS, defined by acute ophthalmoplegia, ataxia, altered consciousness or brisk reflexes. Overlap syndromes refer to conditions where MFS coexists with features of GBS (e.g., limb weakness) or BBE (e.g., altered consciousness), complicating diagnosis and prognosis. This review integrates recent advances in serological, genetic, and neurophysiological research to refine diagnostic criteria and therapeutic strategies for MFS.[2]

EPIDEMIOLOGY

In contrast to GBS, MFS is relatively uncommon; however, epidemiological data regarding its prevalence and incidence remain scarce. A male predominance (1.5–2:1) and median onset age of 43.6 years are consistently reported.[3] MFS accounts for 15- 25% of GBS in Asian populations, compared to 1-5% in Western cohorts.[4,5] Emerging data suggest regional variations in anti-GQ1b antibody prevalence, potentially linked to pathogen exposure patterns.

PATHOGENESIS

Approximately two-thirds of individuals diagnosed with MFS report antecedent symptoms consistent with upper respiratory or gastrointestinal infections. It is hypothesized that MFS results from an aberrant acute autoimmune response triggered by preceding infections or neoplastic disorders. *Haemophilus influenzae* has been implicated in approximately 8% of cases, while *Campylobacter jejuni* accounts for roughly 21%. In addition to these pathogens, dengue virus has emerged as a significant antecedent infection in

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MFS, particularly in endemic regions of Asia and Latin America. Epidemiological studies indicate that up to 20% of cases in these areas may be attributable to dengue infection, underscoring the influence of regional pathogen exposure on the disease spectrum.[6,7] However, the causative pathogen remains unidentified in the majority of instances.[3,8-10] Molecular mimicry between microbial epitopes (e.g., *Campylobacter jejuni* lipo-oligosaccharides) and human gangliosides drives autoantibody production. The antigenic structures of pathogens and human cells exhibit a significant degree of similarity, which can trigger an autoreactive responses in T or B lymphocytes following infection, thereby contributing to disease pathogenesis.[2,11,12] Nevertheless, the precise mechanisms of pathogenesis are not yet fully elucidated.

Lipo-oligosaccharide (LOS) constitutes a major cell-surface structure expressed by *C. jejuni* or *H. influenzae.*[13] The Campylobacter sialyltransferase II enzyme (Cst-II) palys a crucial role in synthesizing ganglioside-like LOS. Cst-II Thr51 enzyme produces GM1-like and GD1a-like Lipo-oligosaccharides, whereas Cst-II Asn51 generates GQ1b-like and GT1a-like Lipo-oligosaccharides.[3,14] Abundant levels anti-GQ1b epitopes exist within the oculomotor, trochlear and abducens nerves, dorsal root ganglia, muscle spindle afferents, and paranodal regions of peripheral nerves, as well as with in brainstem reticular formation.[5,15-17]

Anti-GQ1b IgG binds to neuromuscular junctions in ocular nerves, impairing acetylcholine release (Figure 1). In addition, calcium-dependent anti-GQ1b antibodies have been reported in approximately 12% of seronegative cases, which may contribute to paraneoplastic associations, particularly in males. Electrophysiological studies indicate that some patients develop significant limb weakness due to axonopathy. This is associated with Cst-II Thr51–induced production of anti-GM1 (15%) and anti-GD1a (28%) antibodies.[18] Anti-GQ1b antibodies frequently cross-react with other gangliosides, including GT1a IgG (75%), GD1a (30%), GM1 (20%), and GD1b (20%), potentially underlying the heterogeneous clinical spectrum observed in MFS and related overlap syndromes.[19,20] Based on these antibody profiles, various clinical phenotypes may manifest, including pure MFS, BBE, and overlaps of MFS with BBE or GBS. Epidemiological studies suggest that overlaps between MFS and GBS or BBE occur in approximately 15% and 12% of cases, respectively, supporting the conceptualization of BBE, MFS, and GBS as a continuous spectrum.

CLINICAL FEATURES

Classic MFS typically presents with a triad of ophthalmoplegia, ataxia, and areflexia, observed in approximately 80% of patients .Apart from the classical triad, up to 30% of patients with

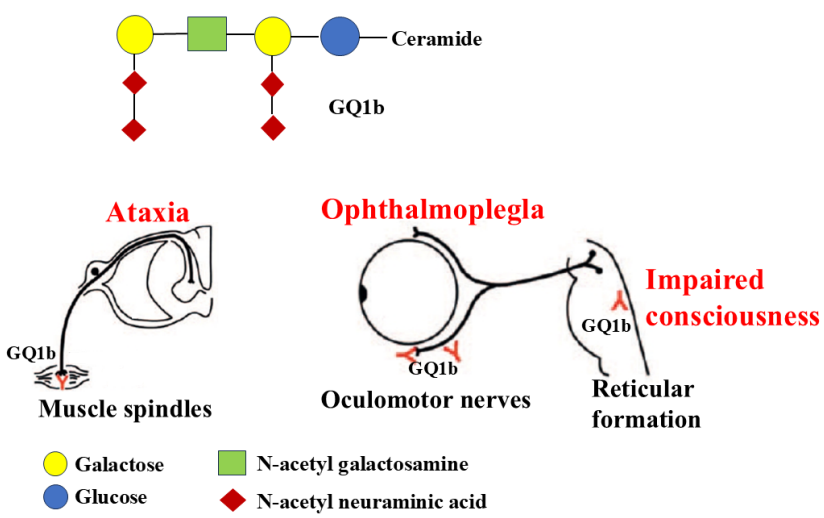

Figure 1. Mechanism of molecular mimicry in MFS
Pathogens (e.g., *C. jejuni*) express GQ1b-like epitopes. Anti-GQ1b antibodies cross-react with homologous structures in ocular nerves, dorsal root ganglia, and brainstem reticular formations, causing ophthalmoplegia, ataxia, and BBE overlap.

Miller Fisher Syndrome (MFS) may exhibit atypical or isolated presentations, including optic neuritis, taste disturbances, headache (12–16% of cases), acute vestibular syndrome, convergence insufficiency, and internal ophthalmoplegia secondary to parasympathetic fiber involvement. Early recognition of these atypical manifestations is essential, as they may closely resemble other neurological disorders.[21] The most common initial symptoms include ophthalmoplegia and ataxia, with some patients experiencing both on the same day. Ataxia may lead to an inability to walk unassisted despite normal strength. Areflexia is less prevalent than ophthalmoplegia and ataxia, being absent in 18% of cases.[22] This symptomatological triad is frequently accompanied by additional signs such as ptosis (60%), facial nerve palsy (30–50%), sensory deficits (20–50%), and hyposthenia (20–25%). Other associated symptoms may include blepharoptosis, weakness affecting the facial, oculomotor, or bulbar muscles, mild limb weakness, and disturbance in micturition.

Autonomic dysfunctions such as dysphagia or extensor plantar responses requiring ventilatory support are rare occurrences. Typical neurological symptoms manifest within 8-10 days following onset, with the median time to reach the most severe clinical manifestations estimated at approximately 1-2 weeks (ranging from 2 to 21 days).[3,4,9,22-24] A significant proportion of MFS patients face risks for disease progression. Red flags for overlap syndromes include: GBS overlap: Progressive limb weakness within 7 days of onset (3–15% of cases). BBE overlap: Altered consciousness or hyperreflexia (12% of cases). Early recognition requires serial anti-GQ1b antibody testing and MRI to detect brainstem involvement. These overlaps commonly arise within 7 days following MFS onset; however, predicting overlap syndromes is challenging due to similarities among clinical presentations as well as electrophysiological and serological features. Therefore, recognizing overlapping signs within the first week is crucial for reducing the need for mechanical ventilation and improving prognosis.[25,26]

The antiganglioside anti-GQ1b IgG antibody serves as a specific maker for MFS but has also been detected in BBE patients. Elevated serum levels of anti-GQ1b antibodies exhibit high specificity during the initial week following MFS onset.[2,27] Seropositivity for anti-GQ1b antibodies can be found in approximately 80%-90% of MFS cases, particularly those exhibiting ocular involvement.[3-5,28] Forms of GBS characterized by ophthalmoparesis or acute isolated ophthalmoplegia alongside BBE also present positive anti-GQ1b antibodies, thus constituting what is referred to as "Anti-GQ1b syndrome".[29] Approximately 12% of individuals diagnosed with MFS are seronegative, which may reflect the requirement for calcium-dependent ligands necessary for antibody binding, a phenomenon predominantly observed in male patients and often associated with paraneoplastic conditions.[30] Notably, in addition to anti-GQ1b antibodies, anti–glutamic acid decarboxylase (anti-GAD) antibodies have been detected in seronegative MFS cases, suggesting that autoimmune mechanisms beyond ganglioside reactivity may contribute to MFS pathogenesis in a subset of patients.[26,31] Collectively, these findings support the concept that a broader immunologic basis, rather than solely autoantibody-mediated responses against ganglioside complexes, may underlie MFS. Consequently, a negative anti-GQ1b result cannot definitively exclude the diagnosis of MFS.

Lumbar puncture remains essential for clinicians aiming to rule out alternative disorders. During peak of disease stages, cerebrospinal fluid (CSF) albuminocytological dissociation occurs in approximately 80% of cases. The dissociation does not appear early on but tends to be more frequent among overlap syndromes. Normal protein levels do not necessarily exclude the diagnosis either. CSF mild pleocytic changes have been noted occurring among approximately 5% patients. No specific CSF markers exist currently available for diagnosing MFS.[3,28,30,32]

Electrophysiological assessments provide valuable diagnostic insights along with prognostic information. Absent H-reflex and reduced sensory nerve action potentials remain hallmarks. Beyond the characteristic findings of absent H-reflexes and reduced sensory nerve action potentials (SNAPs), additional peripheral neurophysiological abnormalities have been reported. These include reversible conduction failure in upper-limb SNAPs and H-reflexes, as well as neuromuscular junction dysfunction in anti-GQ1b syndrome, attributed to terminal motor axonal impairment and reflected by abnormal responses on repetitive nerve stimulation.In addition, central motor pathway involvement has been demonstrated by transcranial magnetic stimulation (TMS) studies, which revealed impaired inhibitory interneuronal circuits and motor neuron hyperexcitability in patients with preserved or brisk reflexes.[14,33] F-wave variability may indicate early GBS

overlap. Early electrophysiological manifestations reveal sensory axonal neuropathy coupled with absent H-reflex without demylination indicators especially evident among those presenting ataxia and areflexia.[33-35] Absence of soleus H-reflex combined with body sway analysis suggests that the ataxia and areflexia in MFS stem from dysfunction within proprioceptive afferent systems rather than cerebellar pathways. Ia muscle spindle afferents displaying elevated concentrations anti-GQ1b antibodies may account for the absence of H-reflex.[3,36]

MRI enhancements involving third and sixth cranial nerves can be observed in among patients diagnosed with MFS.[10] Although classified under peripheral neuropathies, some individuals diagnosed with MFS have exhibited abnormal brain MRI and EEG recordings reported around 1% and 25%, respectively. Specific cases presenting spinal thickening alongside dorsal spinal nerve roots enhancement or cauda equina abnormalities.[37] Microscopic examinations revealed segmental demyelination characterized by minimal perivascular infiltration, while both spinal cords and brain stem stem structures remained unaffected.[2] These observations imply that the pathology primarily affects peripheral components although central elements may occasionally be involved in MFS.[3,14,24]

TREATMENT / MANAGEMENT

For the majority of patients with MFS, plasma exchange and intravenous immunoglobulin (IVIG) facilitate recovery from ophthalmoplegia and ataxia; however, they do not significantly impact overall outcomes. This phenomenon is likely attributable to the favorable spontaneous recovery observed in MFS patients.[19] Nevertheless, plasma exchange and IVIG show equivalent efficacy, but are reserved for overlap syndromes, despite a lack of supporting evidence from randomized double-blind placebo-controlled trials.[3,5,38]

Previous studies have indicated that oral nor intravenous glucocorticoids confer benefits on outcomes of MFS or GBS. The combination of IVIG and glucocorticoids or plasma exchange does not demonstrate superior efficacy compared to IVIG alone.[39] Glucocorticoids are recommended solely for cases of MFS accompanied by visual impairment or neuropathic pain.[16] Treatment responses appear comparable between antibody-positive and -negative MFS patients.[20]

PROGNOSIS

MFS is generally regarded as a self-limiting condition with an optimistic prognosis characterized by a case fatality rate of less than 5%. Mean recovery times typically range from 8 to 12 weeks. Reports of residual symptoms and serious complications are rare. Ataxia and ophthalmoplegia usually resolve within 1-3 months post-onset, with near-complete recovery anticipated within six months.[40] Recovery often follows a sequence involving ataxia resolution after approximately 35 days (range:10–121), followed by areflexia after about 64 days (range:10–650), and finally ophthalmoplegia resolving around 93 days (range:18–244).[22] Recurrence occurs in 11–14% of cases, often linked to HLA-DR2 or HLA-Cw3 alleles. The clinical and electrophysiological characteristics in these recurrent cases mirror those seen in non-recurrent forms. Younger patients (median age 22 vs. 37 years) exhibit milder recurrent episodes, suggesting immunogenetic modulation of disease severity. Recurrence is observed in

Table 1: The differences between GBS and MFS

	Typical GBS	Typical MFS
Clinical features	Progressive symmetrical weakness of upper and lower limbs; decreased/ absent deep tendon reflexes	Ataxia,ophthalmoplegia, areflexia
Electromyogram findings	Demyelinating,axonal polyneuropathy	majority normal; possibly appear reduced sensory nerve action potentials and absent H reflexes
Antibodies	GM1a,GM1b,GD1a,GT1a,GT1b	GQ1b
Treatment	IVIg and plasma change	negating the necessity for treatment unless overlap syndrome features
Prognosis	80% walk independently; 3% mortality rate	favorable spontaneous recovery

Abbreviations: GBS, Guillain-Barré syndrome; MFS, Miller Fisher syndrome

approximately 11–14% of MFS cases. Beyond genetic susceptibility, recurrent episodes have been linked to anti-GAD antibodies, HIV infection, and underlying malignancies. Although these episodes typically mirror the initial presentation, they often exhibit a milder clinical course, supporting the notion of heterogeneous immunopathogenic mechanisms. These recurrent cases typically manifest similar symptoms to initial occurrences but tend to be less severe.[41,42]

CONCLUSION

MFS represents a rare variant of the GBS characterized by the acute onset of ophthalmoplegia, ataxia and areflexia following antecedent infections. It may overlap with other forms such as GBS or BBE. Anti-ganglioside GQ1b antibodies can be detected in serum samples from approximately 80%-90% of patients aiding clinicians in ruling out alternative diagnoses while confirming this specific diagnosis. The presence of albuminocytological dissociation in CSF appears later and is more frequent among overlapping syndromes. Sensory axonal neuropathy alongside absent H-reflex constitutes key electrophysiological features. In most instances, MFS demonstrates favorable spontaneous recovery negating the necessity for treatment unless overlap syndrome features are present. Patients with positive human leukocyte antigens may exhibit frequent recurrences. Unresolved questions include: 1. Role of anti-GT1a/GD1a antibodies in atypical MFS. 2.Impact of gut microbiota on antibody production. 3. Utility of complement inhibitors in refractory cases. Future studies should prioritize biomarker discovery and genotype-phenotype correlations.

DISCLOSURE

Conflict of interest: None

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