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A Rare Case Of Miller Fisher Syndrome In An Elderly Female

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Abstract

Miller Fisher Syndrome (MFS) is a rare variant of Guillain-Barré Syndrome (GBS), characterized by the classical triad of ophthalmoplegia, ataxia, and areflexia. It is strongly associated with anti-GQ1b IgG antibodies and is typically seen in middle-aged individuals. Its occurrence in elderly patients is uncommon and often leads to diagnostic challenges due to overlapping clinical features with other neurological conditions. We report a case of a 75-year-old female presenting with classical features of MFS, confirmed serologically. Early recognition and prompt treatment with intravenous immunoglobulin (IVIG) resulted in significant clinical improvement. This case highlights the importance of considering MFS in elderly patients to ensure timely diagnosis and management.

Keywords: Miller Fisher Syndrome, Guillain-Barré Syndrome, Anti-GQ1b antibody, Elderly, IVIG

Introduction

Miller Fisher Syndrome (MFS) is an uncommon immune-mediated neuropathy first described by Fisher in 1956 as a variant of Guillain-Barré Syndrome (GBS) (1). It is classically defined by a triad of ophthalmoplegia, ataxia, and areflexia, which distinguishes it from other variants of GBS (2). The pathophysiology of MFS is closely linked to autoimmune mechanisms, particularly the production of anti-GQ1b IgG antibodies that target gangliosides located in cranial nerves, leading to the characteristic clinical manifestations (3). MFS accounts for approximately 1–5% of GBS cases in Western countries, with a higher prevalence reported in Asian populations (4). The syndrome commonly affects middle-aged adults, and its occurrence in elderly individuals is relatively rare (5). This atypical age distribution can contribute to delays in diagnosis, as symptoms may mimic other neurological disorders such as cerebrovascular accidents, cerebellar ataxia, or vestibular dysfunction (6).

Clinically, patients often present with acute onset diplopia, gait instability, and loss of deep tendon reflexes, frequently following a preceding infection (7). Early diagnosis is essential because, although MFS is generally self-limiting, timely treatment with immunotherapy such as IVIG can accelerate recovery and prevent complications.

This report presents a rare case of MFS in an elderly female, emphasizing the importance of early recognition, appropriate diagnostic evaluation, and prompt management.

Case Presentation

A 75-year-old female with a known history of hypertension presented with complaints of sudden onset giddiness, tingling sensations in both upper and lower limbs, and progressive unsteadiness of gait. She also reported diplopia associated with restricted eye movements. There was no history of fever, trauma, recent infection, or prior neurological illness.

On clinical examination, the patient was conscious and oriented. Neurological examination revealed normal motor strength (5/5) in all four limbs and intact sensory function. However, deep tendon reflexes were absent in all limbs, indicating areflexia. Cranial nerve examination showed ophthalmoplegia with restricted horizontal gaze and dysconjugate eye movements, without ptosis or facial weakness. The presence of ophthalmoplegia, ataxia, and areflexia suggested a clinical diagnosis of Miller Fisher Syndrome.

Investigations

Cerebrospinal fluid (CSF) analysis revealed mildly elevated protein levels (35 mg/dL) with no pleocytosis, consistent with albuminocytological dissociation. Nerve conduction studies showed no significant abnormalities in motor or sensory conduction; however, bilateral absence of H reflexes was noted, which is considered an early supportive finding in MFS. Serological testing demonstrated borderline positivity for GM3, positivity for GD1b, and strong positivity for anti-GQ1b IgG antibodies, which confirmed the diagnosis.

Treatment and Outcome

The patient was initiated on intravenous immunoglobulin (IVIG) therapy at a dose of 0.4 g/kg/day for five consecutive days. In addition, supportive care including physiotherapy, monitoring of respiratory function, and autonomic stability was provided. The patient showed gradual and significant clinical improvement, with resolution of ophthalmoplegia and improvement in gait over time. No complications were observed during the treatment course.

Discussion

Miller Fisher Syndrome is a rare variant of GBS characterized by immune-mediated damage to peripheral nerves, particularly cranial nerves. The presence of anti-GQ1b antibodies is highly specific and plays a crucial role in the pathogenesis by targeting gangliosides in the oculomotor nerves (8). Molecular mimicry following antecedent infections, particularly with *Campylobacter jejuni*, is believed to trigger this autoimmune response (9). The classical triad of ophthalmoplegia, ataxia, and areflexia is present in the majority of cases; however, variations in presentation can occur, especially in elderly patients (10). In this age group, symptoms are often misattributed to more common conditions such as stroke or cerebellar degeneration, leading to delayed diagnosis (11). The absence of motor weakness, as seen in this case, is a distinguishing feature that helps differentiate MFS from other forms of GBS.

CSF findings in MFS may initially be normal, with mild protein elevation developing later, as observed in this patient (12). Similarly, nerve conduction studies may not show significant abnormalities in early stages, making serological testing for anti-GQ1b antibodies a critical diagnostic tool (13). Treatment with IVIG or plasmapheresis has been shown to be effective in reducing recovery time and preventing complications (14). The prognosis of MFS is generally favorable, with most patients achieving complete recovery within weeks to months, even in elderly populations (15). Early recognition and intervention remain key factors in improving outcomes.

Conclusion

Miller Fisher Syndrome should be considered in elderly patients presenting with acute ophthalmoplegia, ataxia, and areflexia. Due to its rarity in this age group, a high index of suspicion is required to avoid misdiagnosis. Anti-GQ1b antibody testing plays a pivotal role in confirming the diagnosis, and early treatment with IVIG leads to excellent clinical outcomes.

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