

The stroke that wasn't: Miller Fisher Syndrome - a neurological masquerade uncovered in the emergency department

Gina Rami Ibrahim Abdelmesih¹

1. Department of Emergency Medicine, Zayed Military Hospital, Abu Dhabi, UAE

Correspondence to: Gina Rami Ibrahim Abdelmesih

*Department of Emergency Medicine, Zayed Military Hospital, Abu Dhabi, UAE.

Email: ginarami82@gmail.com

DOI: 10.24911/SJEMed.12-2616

Background: Miller Fisher Syndrome (MFS) is a rare, immune-mediated variant of Guillain-Barré Syndrome (GBS), classically presenting with the triad of ophthalmoplegia, ataxia, and areflexia. A viral or gastrointestinal illness usually precede MFS.

MFS incidence is 0.09 per 100,000 annually and accounts for roughly 1%-5% of GBS cases in Western populations. It predominantly affects males and typically presents in middle age. Although the initial presentation can mimic other serious neurological conditions such as brainstem stroke, MFS usually has a benign course with good recovery if treated early and appropriately.

Case Presentation: A 59-year-old previously healthy man presented to the Emergency Department (ED) with the sudden onset of tongue heaviness and dysarthria, blurry vision and dizziness, which began 1 hour prior to arrival. Based on the initial neurological assessment, a stroke was suspected.

Brain imaging, including a non-contrast computed tomography (CT) and magnetic resonance imaging (MRI), showed no evidence of acute ischemia or hemorrhage. After being cleared of stroke, the patient was admitted for further investigations. He was found to have complete ophthalmoplegia, bilateral ptosis, ataxia, dynamic dysarthria, and generalized areflexia, while motor strength fully preserved in all four limbs. Laboratory investigations and extensive work-up (lumbar puncture, nerve conduction studies, CT of the chest, abdomen, and neck, and MRI of the orbits) were unremarkable.

Given the clinical presentation and after ruling out structural and infectious causes, the patient was started empirically on intravenous immunoglobulin and corticosteroids.

Ganglioside antibody testing resulted later and revealed elevated levels of GD1a, GD1b, GT1a, and GQ1b antibodies, confirming the diagnosis of MFS. The patient continued to improve and had a favorable prognosis.

Conclusion: MFS, though rare, must be recognized by ED physicians as a potential cause of acute neurological deficit. Its presentation can mimic stroke, vasculitis, or intoxication/Wernicke's encephalopathy, leading to misdiagnosis or delayed care. Early identification in the emergency setting enables appropriate neurology referral, and initiation of management. Awareness of MFS in the ED is essential - not only to prevent unnecessary interventions (Intubation, investigations) but also to reassure patients of its typically favorable prognosis when accurately diagnosed.

Keywords: Miller Fisher Syndrome, stroke, diplopia, ataxia, Guillain-Barré Syndrome, brain imaging.