



# Guillain-Barré and Miller Fisher Overlap Syndrome Mimicking Alimentary Botulism

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Dear Editor,

We describe a patient presenting with atypical overlapping Guillain-Barré syndrome (GBS) and Miller Fisher syndrome (MFS), with positivity for anti-GM1, anti-GD1a, and anti-GD1b IgG antigangliosides, whose initial presentation evoked alimentary botulism (AB).

A 50-year-old male patient with no medical history presented at the emergency department with blurred vision, dry mouth, and asthenia that had evolved over 24 hours. He reported nausea, vomiting, and diarrhea for 4 days after eating an out-of-date sausage. A neurological examination revealed external ophthalmoplegia with bilateral third cranial nerve and sixth cranial nerve palsy, dysphagia, mild tetraparesis, and preserved deep tendon reflexes. Autonomic dysfunction with urinary retention, constipation, and hypotension were present. This presentation led to suspicion of AB. Stool and blood samples were obtained to screen for *Clostridium botulinum*. The patient received equine serum trivalent botulism antitoxin of types A, B, and E. He was transferred to our division of neurorehabilitation a few days later, and his clinical symptoms improved rapidly over 3 weeks.

Blood and stool tests were negative for *Clostridium botulinum*. Repetitive nerve stimulation showed no increment in the M-wave amplitude or signs of presynaptic junction disorder. Single-fiber electromyography did not reveal increased jitter. Considering the clinical symptoms, rapid improvement, and findings of ancillary tests, we diagnosed MFS. However, enzyme-linked immunosorbent assay screening (GanglioCombi<sup>TM</sup>, Bühlmann, Schönenbuch, Switzerland) of anti-GM1, anti-GM2, anti-GD1a, anti-GD1b, and anti-GQ1b IgG antigangliosides revealed high titers for the anti-GM1 (67%), anti-GD1a (78%), and anti-GD1b (91%) antigangliosides. Based on these findings, we concluded that the patient had atypical overlapping GBS-MFS. No relapse was observed during a 3-month follow-up, but the patient still complained about slight fatigue and persistent right sixth cranial nerve palsy. He was able to perform all of the basic activities of daily living without limitation, and resumed his employment as a security guard.

MFS is considered as a GBS variant characterized by ophthalmoplegia, ataxia, and areflexia, and is mostly associated with anti-GQ1b serum antibodies. However, Morgan et al.<sup>1</sup> identified a patient with ophthalmoparesis, ptosis, and ataxia presenting anti-GM1 and anti-GD1a antibodies. Fusco et al.<sup>2</sup> described a 6-year-old girl with isolated ophthalmoplegia and normal deep tendon reflexes with the same antibody profile as our patient.

The initial presentation of our patient mimicked AB, with multiple cranial nerve palsies, motor impairment, and autonomic dysfunction. However, the clinical course was too favorable for this diagnosis. In addition, bioassays of the toxin in the serum and stool were negative, and electroneuromyography did not reveal any facilitation of compound motor action potential amplitudes during tetanic stimulation or increased jitter. The antiganglioside profile in our patient is unusual for the observed clinical picture—anti-GD1a antibodies are usually associated with acute motor axonal neuropathy (AMAN), facial palsy, and the absence of sen-

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sory signs.<sup>3</sup> Nevertheless, a few patients with ophthalmoparesis or recurrent cranial nerve palsy have exhibited elevated anti-GD1a antibodies.<sup>1,4</sup> Anti-GM1 antibody is also associated with AMAN without sensory and cranial nerve involvement.<sup>3</sup> There have been only a few reports of the presence of this antibody in patients presenting with MFS<sup>1</sup> or overlapping GBS-MFS and Bickerstaff's syndrome.<sup>5</sup> The anti-GD1b antibody has been reported in conjunction with sensory impairment and ataxia.<sup>6</sup> A particularly interesting observation is that *C. botulinum* toxin seems to have a high affinity for GD1a oligosaccharide,<sup>7</sup> which could explain the typical phenotype of a neurological condition mimicking botulism in our case.

While the initial clinical presentation of this case was suggestive of AB, both the rapid improvement and antiganglioside profile pointed to overlapping GBS-MFS. This case highlights the importance of searching for an alternate diagnosis when encountering an atypical presentation and unusual clinical response.

#### Conflicts of Interest

The authors have no financial conflicts of interest.

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