Miller Fischer Syndrome as a Rare Mimic of Botulism

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BACKGROUND

- Botulism is a rare but life-threatening neuroparalytic illness caused by botulinum neurotoxin, often presenting with symmetric cranial neuropathies, descending muscle weakness, and autonomic dysfunction.
- Hallmark features include ptosis, ophthalmoplegia, facial weakness and preserved sensorium in the absence of fever or inflammatory markers.
- While classically associated with ingestion of improperly preserved food, botulism-like presentations can be mimicked by several neurologic conditions, including autoimmune neuropathies such as Guillain-Barré syndrome (GBS) and its variants.
- Miller Fisher Syndrome (MFS), a rare GBS variant, is characterized by the triad of ophthalmoplegia, ataxia, and areflexia, and is strongly associated with anti-GQ1b antibodies.
- Differentiating between these conditions is critical, as management and prognosis differ substantially.

CASE PRESENTATION

We present the case of a 19-year-old male construction worker with no significant medical history who developed binocular diplopia, bilateral mydriasis, extremity weakness, and gait ataxia after eating poorly refrigerated sandwiches and daily exposure to roofing materials. On examination, he exhibited external ophthalmoplegia, symmetric extremity weakness, and normal reflexes in an otherwise afebrile and appropriate teenager. Basic labs and inflammatory markers were unremarkable. He was treated with botulinum antitoxin without improvement. Respiratory panel was positive for rhinovirus. Head imaging was normal. Lumbar puncture revealed albumin cytologic dissociation concerning for a post-viral autoimmune condition. He was treated with IVIG with marked improvement and was discharged home without need for rehabilitation. Send-out CSF studies were later positive for anti-GQ1b antibodies (387), Asialo-GM1 IgG/IgM antibodies (51), and an elevated cerebrospinal fluid IgG synthesis index consistent with MFS, a rare variant of GBS.

DISCUSSION

- MFS often presents with ophthalmoplegia, ataxia and areflexia.
- However, early features such as diplopia, mydriasis, and symmetric weakness can mimic other neurologic conditions, including botulism.
- In this case, initial concern for foodborne botulism was based on the patient's dietary history and cranial nerve findings.
- Lack of response to antitoxin and CSF findings of albuminocytologic dissociation raised suspicion for an autoimmune process.
- The presence of anti-GQ1b antibodies confirmed MFS, and treatment with IVIG led to rapid clinical improvement, thus, shedding light on the diagnostic overlap between toxic, infectious, and autoimmune causes of acute neurologic deficits.

CONCLUSIONS

- This case highlights the importance of considering immunologic causes in addition to infectious and toxic exposures in healthy patients with new neurologic deficits.
- Intravenous immunoglobulins may be a suitable treatment in many of these diseases.