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A diagnostic dilemma: Overlapping features of Guillain-Barré syndrome and myasthenia gravis in an elderly patient

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Abstract

Background: Acute neuromuscular presentations often pose diagnostic challenges, especially when clinical features overlap between Guillain-Barré Syndrome (GBS) and Myasthenia Gravis (MG). Differentiating between these conditions is crucial for timely and appropriate management.

Case presentation: We present a 72-year-old male with acute onset dysphagia, limb weakness, and prior diarrheal illness. Initial findings were suggestive of the Acute Motor Axonal Neuropathy (AMAN) variant of GBS, confirmed by clinical examination and neurophysiology. However, serology revealed double antibody positivity for anti- Musk and anti-acetylcholine receptor antibodies. Despite initial treatment with intravenous immunoglobulin (IVIg), the patient experienced worsening bulbar weakness necessitating intubation and tracheostomy. The neostigmine test was negative, ruling out myasthenic crisis. The patient gradually improved with supportive care and a second IVIg course.

Conclusion: This case highlights the importance of considering overlapping neuromuscular syndromes in elderly patients presenting with acute neurological deficits. A systematic approach and vigilant monitoring are essential for accurate diagnosis and management.

Keywords: Guillain-Barré syndrome; Myasthenia gravis; Acute motor axonal neuropathy; Overlapping syndromes.

Introduction

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapid progressive limb weakness and areflexia. The global incidence of GBS is estimated at 1-2 cases per 100,000 population annually, with around 100,000 new cases reported worldwide each year [1]. GBS is commonly precipitated by infections, particularly Campylobacter jejuni, cytomegalovirus, and Epstein-Barr virus [2,3]. Myasthenia Gravis (MG), on the other hand, is a chronic autoimmune disorder of the neuromuscular junction, predominantly caused by Autoantibodies Against Acetylcholine Receptors (ACHR) or Muscle-Specific Kinase (MUSK) [4]. While GBS typically presents with ascending paralysis and hyporeflexia, MG is characterized by fluctuating muscle weakness, often involving ocular, bulbar, and limb-girdle muscles. However, overlapping clinical features can pose diagnostic challenges, especially in elderly patients [5,6]. Anti-ganglioside antibodies, such as anti-GQ1b, are frequently associated with variants of GBS, including Miller Fisher syndrome and AMAN, while AChR antibodies are specific for MG [7,8]. This case report discusses a rare presentation of an elderly male with overlapping serological features of GBS and MG, emphasizing the complexity of diagnosis and management.

Case presentation

A 72-year-old male presented to the emergency department with sudden onset dysphagia, primarily for liquids, difficulty rising from a seated position, and inability to lift his arms above shoulder level. He had a history of diarrheal illness 10 days prior. On neurological examination, he exhibited rapidly progressive pure motor lower motor neuron (LMN) quadriparesis with bilateral facial and bulbar involvement. Generalized hyporeflexia was noted. Bowel and bladder involvement were absent. The sensory examination was unremarkable. He was admitted to the intensive care unit (ICU) with a provisional diagnosis of Guillain- Barre syndrome for strict monitoring of vitals and plan for intubation in case of worsening of bulbar dysfunction. He was provisionally started on intravenous immunoglobulin (IVIg).

Electrodiagnostic studies confirmed the Acute Motor Axonal Neuropathy (AMAN) variant of GBS. Five doses of Intravenous immunoglobulin (IVIg) were given and after the fifth dose, he was shifted to the Neurology High Dependency Unit. Following this, he continued to remain hemodynamically stable and was discharged to go home on Ryle's tube feeding. The following day, he was again presented to the emergency department with worsening breathlessness and increased oral secretions. He was tachypneic and was using accessory muscles of respiration and was intubated in the Emergency Department itself in view of worsening bulbar muscle weakness and risk of aspiration. Neurological examination revealed drooping of eyelids and mild bilateral eye closure weakness. Incidentally, that very day, we received the results of the serological tests that were sent in the first admission which revealed positivity for Muscle Specific Kinase (MuSK) antibodies and anti-AChR antibodies, raising suspicion of a concurrent myasthenic component. He was again admitted to the intensive care unit (ICU). Electrodiagnostic studies were repeated which showed worsening of conduction parameters as compared to the previous study. A second course of IVIg was administered. Neostigmine testing was negative, ruling out a myasthenic crisis. Gradual improvement was noted over the following weeks. Currently, the patient remains tracheostomized and on percutaneous endoscopic gastrostomy (PEG) feeding.

Discussion

This case demonstrates the complex diagnostic landscape in acute neuromuscular presentations, particularly when clinical and serological findings overlap between Guillain-Barré Syndrome (GBS) and Myasthenia Gravis (MG). GBS is an acute, immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness, hyporeflexia, and often a preceding infection. Variants like Acute Motor Axonal Neuropathy (AMAN) predominantly involve motor axons with early axonal degeneration, leading to more severe presentations and slower recovery [9,10]. Myasthenia Gravis, in contrast, is a chronic autoimmune disease caused by antibodies against the neuromuscular junction, primarily anti-acetylcholine receptor (AChR) and anti-MuSK antibodies [11,12]. Clinically, MG is characterized by fatigable muscle weakness, often involving ocular, bulbar, and limb muscles. However, atypical or overlapping presentations can create significant diagnostic ambiguity. The co-existence of anti-GQ1b and anti-AChR antibodies, as seen in this patient, is rare. Anti-GQ1b antibodies are typically associated with Miller Fisher syndrome and other GBS variants, particularly those with cranial nerve involvement [13]. Serological positivity for AChR antibodies is considered a hallmark of MG but may occasionally be detected in patients with no overt clinical manifestations of MG, possibly due to subclinical disease or assay cross-reactivity [14,15].

In this case, the absence of hallmark MG features, such as fluctuating weakness, ocular involvement, and fatigability, alongside a negative neostigmine (edrophonium) test, effectively ruled out a concurrent myasthenic crisis [16,17]. Furthermore, the predominant pattern of pure motor lower motor neuron weakness, hyporeflexia, and antecedent diarrheal illness pointed towards an AMAN variant of GBS as the primary pathology. Managing such diagnostic dilemmas necessitates prioritizing the dominant clinical syndrome while remaining vigilant for signs of overlap. IVIg therapy is effective for both GBS and acute exacerbations of MG; however, treatment of MG often requires additional long-term immunosuppression and acetylcholinesterase inhibitors [18,19]. In this patient, clinical improvement following repeated IVIg courses without the need for MG-specific therapies further supports GBS as the primary diagnosis. Age-related immunosenescence may contribute to atypical presentations and increase the likelihood of serological cross-reactivity, complicating diagnosis in elderly patients [20]. Reports of serological overlap without clinical MG manifestations emphasize the importance of correlating laboratory findings with clinical presentation rather than relying on serology in isolation [22]. Respiratory failure remains a life-threatening complication of GBS, with up to 30% of patients requiring mechanical ventilation, especially those with bulbar involvement, rapid disease progression, or high disability scores on admission [22,23]. Early ICU admission and aggressive supportive management are crucial in such cases to prevent mortality and improve functional outcomes.

Conclusion

In conclusion, this case underscores the importance of a systematic and holistic approach when evaluating elderly patients with acute neuromuscular deficits. Recognizing the potential for overlapping syndromes, carefully interpreting serological findings, and timely initiation of supportive care are essential components in the management of such complex presentations.

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