Acute-onset myasthenia gravis mimicking Guillaine-Barrè Syndrome: a case report

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Introduction

Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction, characterized by fluctuating muscle weakness. Its pathogenesis is related to autoantibodies against post-synaptic antigens, such as the acetylcholine receptor (AChR) and muscle-specific kinase (MuSK). Guillain-Barrè Syndrome (GBS) is an acute inflammatory polyradiculoneuropathy often preceded by an infectious disease of the gastrointestinal or upper respiratory tract. The typical presentation is with muscle weakness spreading from lower to upper limbs and areflexia. We present a case of atypical presentation of MG mimicking GBS.

Case report

A 23-year-old primigravid woman developed acute endometritis two days after a cesarean section. Antibiotic therapy with i.v. vancomycin and meropenem was started and a laparoscopic peritoneal lavage was performed. After a few days, the patient developed an acute and rapidly progressive limb weakness. Neurological examination revealed generalized areflexia, severe generalized weakness (the patient was unable to stand up and walk unaided) also involving facial muscles. Electroneurography of the tibial nerve bilaterally showed normal values (right: CMAP 9.9 mV, DML 3.8 ms, F wave 51.9 ms; left: CMAP 9.6 mV, DML 4.2 ms, F wave 47.3 ms), except for a slight difference of the F wave mean latency between the two sides. GBS was suspected, and therapy with intravenous immunoglobulins (IVIg) was started.

High-dose intravenous Ig (IVIg) resulted in rapid and marked clinical improvement. Electroneurography was repeated and confirmed normal results. Repetitive nerve stimulation showed significant CMAP decrement, consistent with a post-synaptic disorder of the neuromuscular junction. Testing for AChR antibodies gave positive results.

Currently, MG symptoms are well controlled by pyridostigmine treatment. Retrospectively, the patient reported sporadic eyelid ptosis and fluctuating diplopia, that had not prompted medical advice.

Conclusions

In this patient, both clinical presentation and neurological examination were suggestive of acute polyradiculoneuritis. Indeed, normal neurophysiological studies at onset do not rule out such a diagnosis (1). On the other hand, areflexia and acute-onset weakness with an ascending pattern and sparing of extrinsic ocular muscles are exceedingly rare in MG (2)(3). In this patient, the abrupt onset and rapid progression of symptoms could be ascribed to MG deterioration induced by vancomycin.

Tibialis nerve			
	CMAP	**DML**	**F wave**
Right (R)	9,9 mV	3,8 ms	51,9 ms
Left (L)	9,6 mV	4,2 ms	47,3 ms

References

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