

Bilateral Vocal Chord Paralysis in new onset Myasthenia gravis

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INTRODUCTION

Myasthenia gravis is a common disorder of neuromuscular transmission. It is well studied to be an autoimmune disorder with a t cell mediated antibody attack on the post synaptic neuromuscular junction, typically Ach receptors. Most commonly these present with weakness of specific muscle groups. The extraocular muscles are the most common initial presentation at greater than 50%. Bulbar weakness and proximal limb weakness are less common.

CASE

Patient is a 69 year old male that originally presented with concern for airway obstruction to an ENT clinic. Patient had been having difficulty breathing. At the time the patient had no known history of muscle weakness but did have a positive family history for Myasthenia Gravis from his mother. Patient had a scope done by the ENT physician and found to have severe bilateral vocal cord paralysis. Patient was given a steroid shot from ENT and sent for urgent imaging of the head, neck, and chest. Within 24 hours the patient began decompensating. He was having stridor and slurred speech and presented to the emergency department. Imaging from the ENT the previous day was largely negative for cause of the paralysis. Tensilon test in the emergency department was profoundly positive. Patient was admitted and given IVIG therapy as well as started on prednisone and pyridostigmine and saw improvement of symptoms. Positive Ach receptor antibody at 50, this confirmed the diagnosis of Myasthenia Gravis in the

CLINICAL FINDINGS

Ptosis



Bilateral vocal cord paralysis



CONCLUSION

This case report points out an uncommon initial presentation of Myasthenia. Vocal cord paralysis causing stridor should be considered in any patient presenting with airway obstructive like symptoms. Quick response could very well prevent patients from going on the ventilator. Also of note is the potential that there is a hereditary component to Myasthenia as classically it is thought to have no hereditary component with few case reports showing any connection beyond an autoimmune link.

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Introduction

Myasthenia gravis is a common disorder of neuromuscular transmission. It is well studied to be an autoimmune disorder with a t cell mediated antibody attack on the post synaptic neuromuscular junction, typically Acetylcholine receptors. Most commonly these present with weakness of specific muscle groups. Extraocular muscle weakness is the most common initial presentation, representing more than 50% of the new cases. Bulbar weakness and proximal limb weakness are less common. Respiratory symptoms attributed to Myasthenia Gravis are more commonly a result of diaphragmatic or intercostal weakness.

Case

The patient is a 69 year old male with history of HTN and hyperlipidemia that originally presented with shortness of breath to an outpatient ENT clinic. He had been experiencing progressive difficulty breathing over the course of a few weeks. Direct nasopharyngoscopy performed by the ENT physician identified severe bilateral vocal cord paralysis. The patient was given IM Decadron and sent for urgent imaging of the head, neck, and chest. Within 24 hours he began decompensating—developing stridor and dysphonia. He presented to the ISMC Emergency Department in respiratory distress. Family history was noted to be positive for Myasthenia Gravis in his mother. The previous day's outpatient imaging did not reveal a cause for the symptoms. The patient was stabilized without intubation and evaluated by neurology while in the Emergency Department. A Tensilon test performed in the Emergency Department was profoundly positive. The patient was admitted for IVIG therapy with rapid clinical improvement. He was also started on Prednisone and Pyridostigmine. His Acetylcholine Receptor Binding Antibody test resulted at 50 nmol/L, confirming the diagnosis of Myasthenia Gravis prior to hospital discharge.

Clinical Findings



6 months prior to symptom onset



Lid Ptosis (not initial complaint)



Patient during Tensilon Test



Bilateral Vocal Cord Paralysis

Conclusion

The case report highlights an uncommon initial presentation of Myasthenia Gravis. Although dysphonia and shortness of breath can occur in crisis, bilateral vocal cord paralysis is an uncommon initial clinical manifestation. Vocal cord dysfunction resulting in stridor should be considered in any patient presenting with symptoms of upper airway obstruction. Targeting the correct pathophysiology resulted in avoiding emergent airway intervention. Also of note with this case is the unique family history. Myasthenia Gravis is classically thought to not have a hereditary component, with few case reports showing any connection beyond an autoimmune link.

References Available