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Case Report

Camouflaged Myasthenia Gravis: Case Report

Dr K P Jeswanth Kiran[1*], Dr Uma MA[2], Dr. Jagadish KL[3], Dr. Pillarsetty Pavan Kumar[4], Dr. N S Prasad[5]

[1*]Post Graduate, Department of General Medicine, PES Institute of Medical Sciences and Research Institute, Kuppam, Andhra Pradesh.
[2]Professor and HOD, Department of General Medicine, PES Institute of Medical Sciences and Research Institute, Kuppam, Andhra Pradesh.
[3]Assistant Professor, Department of General Medicine, PES Institute of Medical Sciences and Research Institute, Kuppam, Andhra Pradesh.
[4]Post Graduate, Department of General Medicine, PES Institute of Medical Sciences and Research Institute, Kuppam, Andhra Pradesh.
[5]Post Graduate, Department of General Medicine, PES Institute of Medical Sciences and Research Institute, Kuppam, Andhra Pradesh.
[*]**Corresponding Author**: Dr K P Jeswanth Kiran.
Email: kpjeswanthkiran@gmail.com
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Abstract
Myasthenia Gravis is an anti-acetylcholine receptor antibody-mediated neuromuscular junction disorder. Fluctuating fatiguability, diplopia, ptosis, dysphagia, and dysphonia are characteristic symptoms seen in patients with this disorder. Although uncommon, this illness can affect any skeletal muscle, from those in the neck to the proximal muscles of the limbs. Very few cases of myasthenia manifesting as neck weakness only have been documented.

INTRODUCTION

Myasthenia gravis is an autoimmune disease characterised by a disorder of the neuromuscular junction mediated by anti-acetylcholine receptor antibodies.[1] this disease is mediated by a type-II antibody reaction in which antibodies directed against postsynaptic nicotinic acetylcholine receptors attack and damage the myoneural junction postsynaptic membrane via complement fixation. The results is failure of action potential propagation across the neurons, eventually leading to neuromuscular weakness without stiffness.[2] Classically, the anticholinergic autoantibodies target the extraocular muscles, leading to fluctuating muscular fatiguability, diplopia, ptosis, which is typically worse at the end of the day, dysphagia, dysphonia are characteristic symptoms seen in patients with this disorder. Extraocular, bulbar, and proximal limb muscles are commonly involved, while the distal limb muscles are generally unaffected or minimally affected.[2]

Although it is uncommon, this illness can affect any skeletal muscle, including those in the neck or proximal limbs. Ocular and bulbar symptoms are common, while axial muscle involvement, such as neck flexor weakness, maybe under-recognised, particularly in elderly patients with comorbidities. Till now, very few cases of Myasthenia Gravis manifesting as a single neck weakness have been documented.

Case Presentation

A 61-year-old male with a history of well-controlled hypertension and type 2 diabetes mellitus presented with a 3-month history of gradually progressive bilateral visual blurring, more prominent in the evenings and improving after rest. Over the preceding 6 months, he also developed dull, aching neck pain localised to the nape of the neck, accompanied by fatigable head drop, which worsened with activity and improved with rest. There was no associated limb weakness, sensory disturbances, cerebellar signs, or autonomic dysfunction.

Clinical Examination

On admission, vital signs were stable. A general physical examination revealed bilateral arcus senilis, bilateral pterygium, muddy conjunctiva, unilateral senile cataract, and poor oral hygiene with tobacco staining. Head tremors were observed. Neurological examination showed right upper eyelid ptosis with external ophthalmoplegia and isolated weakness of the right lateral rectus muscle. There was no limb or bulbar muscle weakness. Other systemic examinations, including cardiovascular, respiratory, and abdominal systems, were unremarkable.

Investigations

Routine haematological and biochemical investigations were within normal limits. Serum

anti-acetylcholine receptor (AChR) antibody testing was positive. Nerve conduction studies done showed a decremental response in the right orbicularis oris muscle, which was consistent with a neuromuscular junction disorder. Contrast-enhanced computed tomography (CECT) of the chest revealed no evidence of thymoma.

Based on clinical features, serological positivity for AChR antibodies, and electrophysiological findings, a diagnosis of generalised Myasthenia Gravis was established. The patient was also noted to have hypertension, type 2 diabetes mellitus, and bilateral senile cataract.

Management

The patient was initiated on Tab. Pyridostigmine 60 mg orally three times daily, Prednisolone 20 mg once daily, and Mycophenolate mofetil 500 mg once daily. Supportive care and monitoring were provided. Symptomatic improvement in ocular and neck muscle weakness was noted during follow-up.

DISCUSSION

Myasthenia gravis (MG) is an autoimmune disease due to the presence of antibodies against the acetylcholine receptor (AChR), muscle-specific kinase (MuSK) or other AChR-related proteins in the postsynaptic muscle membrane, which leads to localised or general muscle weakness.[3] Therefore, the diagnosis is based on clinical findings, electrophysiology findings, and serology.[4,5]

In patients with MG, eyelid ptosis is often the first symptom to appear and may be accompanied by bulbar, midaxial, or proximal weakness.[6] Distal limb involvement usually occurs in the later stages of the disease. Early onset muscle weakness in the distal limbs as the primary manifestation of MG is infrequent

It commonly presents with ocular muscle weakness, progressing to limb involvement, which is observed in 85% of patients.[7,8] A clinical examination would reveal more than one extraocular muscle involvement, with pupillary sparing. 60% of patients with the disease would demonstrate bulbar muscle weakness, which can present with fatigable chewing and difficulty in deglutition.9 Respiratory muscle involvement is rarely seen during the first two years of onset of the illness.[10] It occurs in intercostal and diaphragmatic muscle involvement, and if present, leads to myasthenic crisis, which is a life-threatening consequence, often requiring mechanical ventilation. Several precipitating factors for the myasthenic crisis, such as medications and intercurrent infections, have been identified in the literature.[11]

Classically, it presents with fluctuating and fatigable skeletal muscle weakness, commonly affecting the extraocular muscles and the muscles of mastication to a lesser extent. There have been reports of myasthenia presenting as dysphagia in elderly males with rapid progression to respiratory failure.[2] Apart from prototypical muscular involvement, any set of muscles can be affected, including the proximal limb and neck musculature.

Significant delays in diagnoses or frequent misdiagnoses among elderly patients have been reported.[12] The estimated time for diagnosis in the elderly was 4.5 months in juxtaposition to 2.5 months in the younger age groups. [12]

Traditional treatment for stable myasthenia gravis involves the use of indirect-acting anticholinesterases, such as pyridostigmine or neostigmine.[13] Ample therapy with anticholinesterases and steroids directly leads to the desired survival rate in elderly populations.[14] Thymectomy is an appropriate option to control symptoms in younger patients, but it is not typically favoured in elderly patients, especially those above 60 years old. [15] Elderly patients have often shown a good response to therapies in general.[13]

This case depicts the diverse clinical spectrum of MG, especially in elderly, where axial muscle involvement may be the initial presenting feature. Early recognition and targeted immunosuppressive therapy are important for preventing progression and enhancing quality of life. in our patient, the presence of AChR antibodies and no thymoma on radiological imaging point towards a late-onset, non-thymomatous form of MG.

CONCLUSION

In elderly patients with unexplained ocular symptoms and fatigable neck weakness, myasthenia gravis should be considered. Very few cases of Myasthenia Gravis presenting solely as neck weakness have been reported to date, especially in males. It is essential to consider Myasthenia Gravis as a differential diagnosis in the elderly who present with the above symptoms in order to prevent misdiagnosis, initiate timely treatment, and alleviate the patient's distress. Prompt diagnosis and immunosuppressive therapy can lead to favourable outcomes even in the presence of multiple comorbidities.

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