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A typical Presentation of Myasthenia Gravis: A Clinical Sequelae of Thymoma

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1. Abstract

Myasthenia gravis (MG) is an autoimmune disorder in which antibodies block the acetylcholine receptors in the post synaptic membrane causing skeletal muscle weakness. The weakness can be localized or diffuse. MG predominantly affects the bulbar muscles and 15% of the cases are confined to the ocular muscles. Although MG is classically recognized as a disorder of muscle function, non-motor manifestations, particularly altered somatic sensation, are overlooked and underreported. This under recognition has contributed to a limited awareness among clinicians regarding the wider range of MG presentations. MG is a disease that can manifest with both motor and sensory symptoms. We present the case of a male patient subsequently diagnosed with thymoma, who initially exhibited abnormal sensory perceptions prior to the onset of muscular weakness. The diagnosis of MG was only established following the development of motor impairment. This case underscores the need for heightened clinical suspicion regarding atypical presentations of MG. The underappreciation of sensory symptoms may delay diagnosis and contribute to increased patient morbidity.

2. Introduction

Myasthenia gravis (MG) is an autoimmune disorder in which antibodies block the acetylcholine receptors in the post synaptic membrane causing skeletal muscle weakness. MG, along with its subtypes, is one of the primary conditions impacting the neuromuscular junction, with yearly incidence of 8 to 10 cases per million people and a prevalence ranging from 150 to 250 cases per million [1]. In 10% of the cases, MG is a result of paraneoplastic syndrome from a thymoma. The weakness can be localized or generalized and MG predominantly affects the bulbar muscles and 15% of the cases are confined to the ocular muscles [2]. Although rare, sensory symptoms are considered not to be features of MG in the minds of many physicians. In this case we report a case of a male with MG secondary to a thymoma, who presented with sensory symptoms along with the traditional motor symptoms and rapidly deteriorated.

3. Case Presentation

A male in hist 40s presented to the neurology clinic with complaints of severe generalized pruritis, paresthesia in the V3 distribution of the right trigeminal nerve and the left upper limb. He also complained of hyperesthesia and allodynia in the thorax, upper and lower limbs causing a daily struggle in wearing clothes. The patient reported no motor weakness or muscle fatigability which was also consistent with the physical exam. His appetite was poor and he had lost a significant amount of weight over the past few months. Preceding his symptoms, the patient had recent history of 6 months constitutional syndrome, constipation, nausea, self-limited jaundice, a diagnosis of stroke made in other facility due to visual disturbances but normal MRI. A recent admission due to acute kidney impairment of unknown cause (there is a pathology report an image from Mediclinic) treated successfully with 5 pulses of methylprednisolone. In view of the constellation of symptoms the patient was worked up extensively.

Workup revealed normal cell counts, liver fuction, renal fuction, electrolytes and C-reactive protein (CRP). A comprehensive Antinuclear antibody panel was negative. NCS/EMG was done which was normal. Body-CT scan revealed mediastinal mass suggestive of thymoma. Paraneoplastic syndrome secondary to a

thymoma was concluded. A paraneoplastic antibody panel was done and was unremarkable.He was treated symptomatically however was lost to follow up, until 40 days later when he presented in the clinic with a range of symptoms, including intermittent diplopia, difficulty with left gaze, and mild dysphagia that he related due to a recent esophagitis for candida admitted and treated in another facility. These symptoms prompted evaluation of Acetylcholine receptor (AChR) binding, blocking and modulating antibody. A Pet/CT revealed a 3cm thymic mass with standardized uptake value of 3.8. A diagnosis of myasthenia gravis (MG), secondary to thymoma was made. The patient was started on Pyridostigmine which improved his symptoms however he failed to follow up yet again. 30 days later, he presented with generalized weakness, exophthalmos and tachycardia. On further investigations showed altered thyroid function elevated T4 (3.81 ng/dL), decreased TSH (0.017 uIU/mL) and positive thyroid peroxidase antibody (360 IU/mL). The patient was admitted and started on Carbimazole, Propranolol, Prednisolone and IVIG. The patient continued to deteriorate with signs of respiratory failure requiring intubation and mechanical ventilation. After repeated failed extubation, the patient underwent thymectomy. Histopathology report confirmed thymoma, type B2. Thyroid parameter normalized and didn't require thyroid treatment after. MG was treated with steroids, Azathioprine unsuccessfully and further on with Rituximab with good outcome. The patient went on to make a full recovery.

Table 1:

Paraneoplastic Ab

Test	Current Result and Flag	Previous Result and Date	Units	Reference Interval
Interpretation[01]	Negative A negative paraneoplastic autoantibody result does not rule out all clinically relevant antibodies. If indicated, a phenotype-specific profile (For example, Encephalopathy, dementia, myelopathy, or axonal neuropathy) should be considered.			Negative
Anti-Hu Ab[A,01]	Negative			Negative
Anti-Ri Ab[A,01]	Negative			Negative
Antineuronal nuclear Ab Type 3 [A,01]	Negative			Negative
PCA Type-1 (Anti-Yo) Ab[A,01]	Negative			Negative
Purkinje Cell Cyto Ab Type 2[A,01]	Negative			Negative
Purkinje Cell Cyto Ab Type Tr [A,01]	Negative			Negative
Amphiphysin Antibody[A,01]	Negative			Negative
CRMP-5 IgG[A,01]	Negative			Negative
AGNA-1[A,01]	Negative			Negative
VGCC Antibody[01]	<1.0		pmol/L	0.0-30.0
CASPR2 Antibody,Cell-based IFA[A,01]	Negative			Negative
LGI1 Antibody, Cell-based IFA [A,01]	Negative			Negative

Table 2:

Test	Result	Reference ranges.
AChR binding antibody, serum	1.71 nmol/L	0.00-0.24
AChR blocking antibody, serum	35%	0-25%
AChR modulating antibody, serum	50%	0-45%

4. Discussion

Myasthenia gravis is a chronic autoimmune neuromuscular disorder characterized by fluctuating muscle weakness, most commonly affecting ocular, bulbar, limb and respiratory muscles [1,3,4]. Sensory symptoms are not considered a typical manifestation of MG and are rarely described [5]. Neuromuscular transmission relies on the release of acetylcholine from the presynaptic terminal, which then binds to receptors on the postsynaptic membrane. These acetylcholine receptors work in conjunction with other membrane-associated proteins such as Dok7 and rapsyn. Mutations in Dok7 and rapsyn contribute to certain forms of congenital myasthenia. In autoimmune myasthenia gravis, weakness is caused by antibodies targeting acetylcholine receptors, as well as muscle-specific kinase (MuSK) and lipoprotein receptor–related protein 4 (LRP4). Additionally, antibodies against intracellular proteins like titin and the ryanodine receptor serve as important biomarkers in specific subtypes of the disease. Acetylcholine is broken down

by acetylcholinesterase, and inhibiting this enzyme can provide symptomatic relief for patients with myasthenia gravis [1]. Thymomas are rare tumors that arise from thymic epithelial cells, but they are the most common type of anterior mediastinal mass in adults, making up about 50% of cases [6]. There is a well-known association between thymomas and autoimmune diseases (AD), particularly myasthenia gravis (MG) [7]. MG occurs in up to 44% of patients with thymoma [8]. Conversely, 75–80% of patients with MG have changes in the thymus, most commonly thymic hyperplasia (85%) or, less commonly, thymoma 15–20% [9]. pure red cell aplasia, autoimmune encephalitis, haemolyticanaemia, Good's syndrome, and autoimmune thyroiditis are some notable conditions associated with this tumour [10]. In our patient we first observed nonspecific signs of paraneoplastic syndrome, however later on he goes on to present with MG, the classic autoimmune disease associated with a thymoma and thyroiditis which is a much rarer manifestation.

Sensory manifestations are an important part of MG. There is a myriad of ways that sensory symptoms present in MG. Studies have found that 68% of patients with MG had higher tactile perceptual thresholds compared to healthy people, measured by grating orientation tasks [11]. Pain has been reported, moderate to severe pain scores were found in upto 50% of patients with MG [11], numbness and tingling are present in 10% of patients with MG [12]. Also reported are peripheral neuropathies, hearing loss, dizziness, abnormal thermoregulation and altered sensation of taste and smell [5]. In our patient, the initial presentation was predominantly sensory, which is atypical for MG. While MG was considered early in the differential diagnosis, the prominence of sensory features led us to prioritize alternate diagnoses. It was more upon the subsequent presentation with motor findings that MG became classically apparent.

5. Conclusion

MG is a disease that can manifest with both motor and sensory symptoms. The sensory symptoms are usually under reported and hence the lack of awareness of sensory symptoms. There should be mor focus placed on sensory manifestations, should be reported more and should be kept in mind so that treatment can be started early to prevent deterioration.

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