Case Report

Ocular myasthenia gravis in a setting of thyrotoxicosis

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ABSTRACT

Ocular myasthenia gravis in conjunction with thyroid disorders, although rare, has been reported in the past. However, the similarity in the presentation of both the entities and the tendency of myasthenia gravis to get overlooked easily, even by experienced clinicians, necessitates a thorough knowledge, a strong consideration, and a vigilant approach, to aid in its diagnosis. We discuss a case of a female in a thyrotoxic state, with symptoms of ocular myasthenia gravis, and a brief overview of this entity.

Key words: Graves' disease, hyperthyroidism, ocular myasthenia gravis, thyrotoxicosis

INTRODUCTION

Coexistence of Myasthenia Gravis (MG) and thyroid disorders is well known for the past few decades. It has been postulated that MG frequently occurs in patients with autoimmune thyroid disease, as compared to the general population and is mostly seen with hyperthyroidism (50 times more common with Graves' disease).^[1] Although autoimmune thyroid disorders are known to be present in 5-7.5% of the MG patients, MG is seen in only 0.2% of the patients with thyroid disease, commonly associated with thyrotoxicosis.^[2,3] We discuss herewith, a female patient presenting in a thyrotoxic state and thereafter developing ocular MG.

CASE REPORT

A 22-year-old female presented to the Neurology Clinic with a history of a mild, dull aching headache in the occipital area, which progressively increased over the past

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two years. She also complained of diplopia in all directions for the past six months. A positive history of aura, neck pain, restriction of movement in the neck, vomiting, photophobia, seizures, and weakness of limbs was present. There was also a positive history of amenorrhea since four months, and a 10 kg weight loss over the last four months was documented. Family history suggestive of any thyroid disorder or neuromuscular disease was negative. A general survey of the patient showed a thin built and poorly nourished female with pallor and pedal edema. Vitals recorded: Pulse – 96/minute, regular, with normal volume and character; Blood pressure - 130/90 mm of Hg. The patient was afebrile. Diffuse thyroid swelling was noticed during the examination. Systemic examination including neurological and all other systems documented no significant abnormality, except for decreased palpebral fissure of the left eye and lid lag noticed in the left eye [Figure 1]. Based on the history and examination findings and a battery of relevant investigations (Tensilon test), diagnosis of ocular MG was made, and



Figure 1: A 22-year-old female diagnosed with hyperthyroidism showing mild ptosis of the left eye

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physostigmine was initially prescribed to the patient. The patient was then referred to Internal Medicine for further evaluation for suspicion of hyperthyroidism. Thyroid function tests reported T3 levels of 3.4 (0.8-2.0) ng/ml, T4 levels of 20.2 (4.5-12.0) ug/dl, and TSH levels of <0.005 (0.3-5) uiu/ml. The hematological and other biochemical investigations were all within normal limits. Finally, in view of the thyrotoxic status of the patient, carbimazole was started at a dose of 10 mg, eight hourly. The patient was discharged with an advice to continue carbimazole and review after one month. Follow-up after one month showed both symptomatic and biochemical improvement, that is, left eye ptosis had decreased and T3, T4, and TSH levels were within normal range, respectively. Follow-up after three months demonstrated complete clinical and biochemical recovery. The further plan was to discuss the alternative of thymectomy with the patient, on the next follow-up after two months.

DISCUSSION

Myasthenia gravis is an autoimmune disorder characterized by fluctuating weakness of the skeletal muscles caused by impaired neuromuscular transmission due to circulating antiacetylcholine receptor antibodies. MG has a high incidence of association with autoimmune diseases, most common being Graves' disease and autoimmune thyroiditis. Graves' disease is commonly associated with the ocular form of MG, which has been supported by the increased presence of thyroid antibodies in patients with ocular MG (approximately 40%).^[4,5] Furthermore, the presence of thyroid antigens in ocular tissues has been proven.^[6]

This case report of ocular MG in conjunction with thyrotoxicosis supports the earlier reported association between the two entities.^[4,5] Treatment of thyroid disorder in patients with MG leading to myasthenia regression, in approximately two-thirds of the patients, also strengthens this correlation.^[7] However, the exact reason for this association remains elusive. Several hypotheses have been postulated. First, the possibility of immunological cross-reaction against epitopes or auto-antigens shared by the thyroid and the eye muscles. Second, higher frequency of ocular myasthenia in autoimmune thyroid disease could be attributed to genetic linkage.^[5] Human leukocyte antigen (HLA) specificity (B8, DR3, and BW46) between MG and thyroid disease has been reported.^[8] However, further studies to unearth the precise association between thyroid disease and MG is a must in the future, to understand the relation between both the entities.

In a majority of patients (~75%) with coexisting conditions,

hyperthyroid symptoms occur before or along with myasthenic symptoms.^[9] Our patient initially showed symptoms of thyrotoxicosis, that is, weight loss and amenorrhea, four months before developing left eye ptosis, lid lag, and diplopia. Accurate and early diagnosis in such circumstances is a challenge to the physician. Neuromuscular features are known to be associated with both the entities and the presentation of myasthenia. When it is present concomitantly with thyrotoxicosis, it is usually mild, and therefore, can get overlooked when considering differential diagnosis for a patient with hyperthyroid symptoms and muscular weakness. Ptosis is generally not seen in thyroid ophthalmopathy, in contrast to MG. A strong suspicion of MG should be considered while dealing with parallel scenarios. Appropriate diagnosis of MG can be made by a complete history and thorough examination, with the supplementation of laboratory investigations such as Edrophonium (Tensilon) test, radioimmunoassay, and electromyography.

A common treatment modality denotes the use of anticholinesterase medications supplemented by immunosuppression, plasmapheresis and/or thymectomy. It is also seen that any deviation from a euthyroid state will adversely affect MG and the treatment of thyroid dysfunction may alleviate the existing myasthenic symptoms when both disorders are present concomitantly.^[7] The presence of an enlarged thymus and thymic abnormalities have been seen in both thyrotoxic and MG patients. The beneficial effect of thymectomy in the management of MG, if done at an earlier stage in the disease course, has been documented.^[10] Thymectomy not only aids in improving the symptoms of myasthenia, but it also reduces the morbidity in severe cases and where large doses of anticholinesterases are required on a daily basis.

The main objective of this case is to highlight the importance of detecting a coexisting mild myasthenia gravis at an early stage in a patient with thyrotoxicosis with predominantly restricted ocular symptoms.

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