

Thyroid associated orbitopathy with ocular myasthenia in primary hypothyroidism: Keep those eyes open

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ABSTRACT

Thyroid associated orbitopathy, although seen most commonly with thyrotoxicosis, is also known to occur in primary hypothyroidism. Myasthenia gravis is an autoimmune condition with an established association with autoimmune thyroid disease. We report the case of a patient who presented with recent onset unilateral ptosis that was fatigable with a history of proptosis since a year. On examination, she had a goiter, bilateral proptosis, restriction of upward gaze and adduction both eyes and normal pupils. Investigations revealed primary hypothyroidism with anti-thyroid peroxidase positive and anti-acetylcholine receptor antibody positive. Computerized tomography orbit showed thickening of medial and inferior rectus characteristic of thyroid orbitopathy. A diagnosis of primary hypothyroidism with thyroid orbitopathy with ocular myasthenia gravis was made. Patient is on Levothyroxine and anticholinesterase medications and is on follow-up. We present this case to highlight that the presence of ptosis in a patient with thyroid orbitopathy should alert the clinician to the possible coexistence of myasthenia gravis.

Key words: Grave's ophthalmopathy, hypothyroidism, myasthenia

INTRODUCTION

Thyroid associated orbitopathy (TAO), although commonly seen in Graves' thyrotoxicosis, has also been known to occur in patients with primary hypothyroidism.^[1] Myasthenia gravis is an autoimmune disease involving the neuromuscular junction with an association with autoimmune thyroid disease.^[2] The clinical features of TAO and ocular myasthenia gravis have significant overlap and in the rare instance of their coexistence, recognition of the second entity may be missed, unless looked for specifically. We report here the case of a woman who presented with TAO with primary hypothyroidism with ocular myasthenia gravis to highlight the same.

CASE REPORT

A 62-year-old lady sought an ophthalmology consultation for complaints of drooping of right eyelid since 10 days. She was referred to our endocrinology clinic thereafter. The drooping of the right eyelid was sudden and was better at the start of the day and got worse as the day progressed. When she lifted the lid with her hands, she also noted double vision. On questioning, she had complaints of watering from both eyes and occasional redness with a gritty sensation since a year. Her daughter notes that her eyes have become more prominent since the last year. She had no past history or family history of thyroid illness and had no complaints suggestive of thyrotoxicosis although she had constipation and malaise. She had no difficulty in swallowing food or speaking. She had no difficulty in getting up from the squatting position, or climbing stairs or combing her hair. She had no history of cough, hemoptysis or shortness of breath.

On examination, she was a moderately built lady, conscious oriented and alert. General examination revealed a

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visible diffuse goiter, dry skin with delayed relaxation of ankle jerks. Examination of the eyes revealed bilateral proptosis (26 mm in the left eye and 24 mm in the right eye). The right eye revealed ptosis with transient improvement after rest [Figure 1]. There was no exotropia and esotropia. Ocular movements testing revealed restriction of upward gaze and adduction both eyes. Pupils were equal and reactive to light. Clinical activity score was 1/7 in both eyes (right – conjunctival congestion and left pain at rest). Vision was normal in both eyes. Examination of the central nervous system revealed no bulbar weakness or weakness in limbs. Sensory system examination was normal.

A provisional diagnosis of TAO, hypothyroidism and myasthenia gravis was made. Routine investigations were normal. Thyroid function tests revealed primary hypothyroidism (thyroid-stimulating hormone 38 mIU/L, Free T4 0.8 ng/dl, anti-thyroid peroxidase 189 IU/L). Computed tomography of the orbit [Figure 2] showed bilateral proptosis (left > right) with no mass lesions in the orbit. Figure 3 shows enlargement of extra ocular muscles both eyes, especially inferior recti and medial recti, findings characteristic of TAO.

A neurology consultation was obtained. Anti-acetylcholine receptor antibody (antiAChR) was positive 2.32 nmol/L ($N < 0.5$ nmol/L). A repetitive nerve stimulation test at the limbs was normal but the testing at the nasalis muscle was found to inconsistently positive. A diagnosis of ocular predominant myasthenia gravis was made. A computerized tomography thorax was normal. Magnetic resonance imaging of the brain was normal.

She was started on Levothyroxine 75 mcg/day and pyridostigmine 15 mg thrice daily and showed improvement in general well-being and ptosis.

DISCUSSION

It is recognized that about 5% of patients with thyroid associated ophthalmopathy have hypothyroidism as against the expected picture of thyrotoxicosis.^[1] The presence of ptosis in a patient of TAO should alert one to the coexistence of myasthenia gravis, a condition known to be associated to be associated with autoimmune thyroid disease although ptosis has been described as a rare feature of TAO in anecdotal case reports.^[3] Autoimmune thyroid disease occurs in about 3-8% of patients with myasthenia gravis and thyroid function tests are routinely recommended in all myasthenia patients.^[2]

Ocular myasthenia gravis is a subtype where the weakness remains restricted to the extra ocular muscles with no



Figure 1: Profile of patient showing bilateral proptosis (left > right) with the right eye ptosis

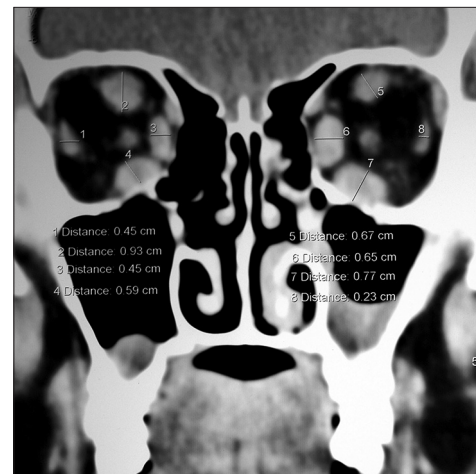


Figure 2: Computerized tomography orbits showing bilateral thickening of intraocular muscles, predominantly involving inferior and medial rectus followed by superior rectus and lateral rectus due to thyroid associated ophthalmopathy



Figure 3: Transverse section of computerized tomography orbits showing bilateral proptosis (left > right)

generalized weakness. Patients with myasthenia gravis with autoimmune thyroid disease are more likely to have milder clinical features with preferential ocular involvement as compared with myasthenia patients with none-autoimmune thyroid disease or no thyroid disease.^[4] The management of thyroid condition (hyperthyroidism or hypothyroidism) significantly improves response of myasthenia to treatment.

The presence of antiAChR antibody is virtually diagnostic of myasthenia gravis, but a negative test does not exclude the disease. Only about 50% of patients with disease restricted to ocular muscles have detectable antiAChR antibodies in serum.^[2] Our patient is on follow-up since a year and is doing well with no progression of myasthenia gravis.

CONCLUSION

We report this case to highlight the association of TAO, in this case with primary hypothyroidism with myasthenia

ocular variant. The presence of ptosis in a patient with TAO should alert the clinician to the presence of myasthenia gravis.

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