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Thyroid

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A Case Report Identifying Graves’ Ophthalmopathy in a Myasthenia Gravis Crisis and Response to Teprotumumab

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Background: Thyroid disease is relatively common in patients with Myasthenia Gravis (MG), affecting 5%–10% of patients, conversely, the prevalence of MG in patients with Graves’ disease is 0.14%. Of thyroid diseases seen in MG, Graves’ Disease (GD) is the most common. Graves ophthalmopathy (GO) is a known complication associated with GD, however, in patients with MG, its identification may be difficult, as both GD and MG may present with similar ocular signs and subtle differences. This case identifies GO in a MG crisis and its response to teprotumumab.

Clinical Case: A 53-year-old-woman with MG presented with one year of worsening bilateral eye and arm weakness, fatigue, eye pain, headaches and palpitations. Examination revealed bilateral ptosis, eyelid edema, and 4/5 bilateral upper extremity strength. Workup revealed suppressed TSH (<0.010, n 0.5-5.0 ml U/L) and elevated antibodies: Thyroid Stimulating Immunoglobulin (4.86, n <0.54 IU/L) and Thyrotropin Receptor Antibodies (TRAb) (8.08, n ≤1.75 IU/L). Thyroid ultrasound showed several bilateral cystic nodules and increased thyroid parenchyma vascularity without thyromegaly, suggestive of GD. Thyroid Uptake and Scan showed diffusely elevated 24-hour uptake (71%, n 10-30%) consistent with GD. Treatment was initiated with methimazole 5 mg daily. At follow up, she had symptoms of eyelid weakness, diplopia, headaches, and muscle weakness. Graves’ Ophthalmopathy Clinical Activity Score (CAS) was 5/10. Examination showed bilateral ptosis with eyelid edema, proptosis, conjunctival redness, and chemosis. Extraocular muscle movements intact without pain, normal pupils and visual fields. Ophthalmology review and orbital CT showed bilateral exophthalmos with extraocular muscle thickening consistent with thyroid eye disease (TED). The classic coke-bottle-sign of the anterior tendon of the inferior rectus muscles was noted. Given her presentation, clinical course and progression of TED, she commenced a teprotumumab treatment trial. Clinical improvement of GO was noted upon follow-up assessment after 5 doses of teprotumumab: CAS 2/10, decreasing proptosis on exam, and TRAb (3.86, n ≤1.75 IU/L).

Conclusion: This case highlights the potentially misleading clinical findings and difficult diagnoses of coexisting GO and MG. Although muscular weakness is common in GD, concomitant MG is rare and may cause profound additional morbidity. Ocular signs in both diseases may cause diagnostic confusion, although ptosis is more consistent with MG and lid retraction with GD alone. GO can mimic some of the neuromuscular and ophthalmologic signs of MG and can be easily missed or mistakenly attributed to either MG or GD. This may result in a delayed diagnosis/inappropriate treatment of GO, thus, clinicians...
should maintain suspicion for coexisting diagnoses.

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