Several years ago, one of my patients visited my office repeatedly for what appeared to be recurrent allergic dermatitis. Both of her eyes were experiencing episodes of periorbital edema that responded well to oral steroids. This patient was in her late 60s, which is well past the common time frame of Graves disease onset, and she had no relevant systemic diseases. At her third visit, however, not only were her eyelids swollen, but there was some proptosis in her left eye. After I ordered a CT scan and bloodwork, the answer was clear: She had Graves disease.

The American Thyroid Association describes Graves disease as “an autoimmune disease caused by antibodies directed against receptors present in the thyroid cells and also on the surface of the cells behind the eyes.” In simpler terms, it is an autoimmune attack on the thyroid, which is the butterfly-shaped gland located at the front of the neck. When the thyroid is overworked or is not working properly (known as hyperthyroidism), a patient can experience immense pain from muscle aches and cramps as well as abnormal weakness or fatigue. Graves disease can also cause a host of other problems, including loss of appetite, insomnia, changes in weight, memory loss, dizziness, and depression. And, as in the case of my patient, it can wreak havoc on the eyes.

Graves disease tends to affect more women than men (at a ratio of 5:1), and it most commonly presents during middle age. From an eye doctor’s perspective, one of the most problematic results of Graves disease is thyroid eye disease (TED). Up to one-half of patients who develop Graves disease eventually develop TED (see Seven Symptoms of Thyroid Eye Disease).

**Ocular Manifestations**

When bloodwork is performed on patients with hyperthyroidism, the thyroid hormones triiodothyronine (T3) and thyroxine (T4), thyroid-stimulating hormone (TSH), and TSH antibodies are measured to assess the thyroid’s functioning. Patients with Graves disease typically have increased levels of T3/T4, decreased levels of TSH, and the presence of TSH receptor antibodies. When those same TSH antibodies attack the extracocular muscles (EOMs), the result is enlarged EOMs, which can be seen on CT scan or MRI.

**Proptosis**

In physical evaluations, Graves disease can present in its early stages as...
Graves disease is an autoimmune disease caused by antibodies directed against receptors in the thyroid cells and on the surface of cells surrounding the eyes.

The most common ocular manifestation of Graves disease may be dry eye, which is typically more difficult to manage than when it results from other causes. It is important to educate patients on long-term management strategies. The ophthalmic dyes fluorescein and lissamine green can be useful for assessing dry eye in patients with Graves disease.

Dry eye induced by an autoimmune disease should be treated more aggressively than other forms of dry eye disease. Therapy with medications such as cyclosporine ophthalmic emulsion 0.05% (Restasis, Allergan) and lifitegrast ophthalmic solution 5% (Xiidra, Shire) should be considered sooner rather than later.

Lissamine green can also help to identify superior limbic keratoconjunctivitis (SLK), which can be associated with Graves disease. SLK can also result from excessive contact lens wear, but thyroid disease should always be considered when patients with SLK are encountered.

If SLK is a result of thyroid disease, it will not respond to topical steroids and may require treatment with silver nitrate.

A PLAN OF ACTION

The role of eye care providers in the management of patients with Graves disease is to preserve sight. The examination of patients with Graves disease should include: VA measurements, pupil testing, cover test, Hertel exophthalmometry, corneal staining, and visual field testing. If optic nerve compression is suspected, comanagement with an oculoplastic surgeon is the standard of care. Treatment of Graves disease can include orbital radiotherapy, steroid therapy, or a combination. In a recent study of 41 patients with moderate to severe Graves ophthalmopathy, patients responded well to radiation therapy plus steroids, and at 56 months only 21.9% had undergone orbital decompression for a relapse.1

Because detecting a drop in VA is the greatest concern for eye care providers managing patients with Graves disease, it is essential to advise patients to follow up with their eye doctor (ie, you) as soon as they notice any changes in their vision.

Looking for a positive afferent pupillary defect can help flag optic nerve damage. Identifying new or worsening vertical or horizontal tropias and measuring proptosis are key to determining if the disease is progressing. Finally, regular visual field testing can help optometrists monitor the health of the optic nerve to ensure that it is not being compressed.  


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