Puffy eyes

Cosmetic problem or thyroid eye disease?

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Early recognition of thyroid eye disease is the key to preventing disease progression. Advanced disease causes disfigurement, double vision and blindness. In this article, Dr Maloof presents his approach to diagnosis and management of this debilitating condition.

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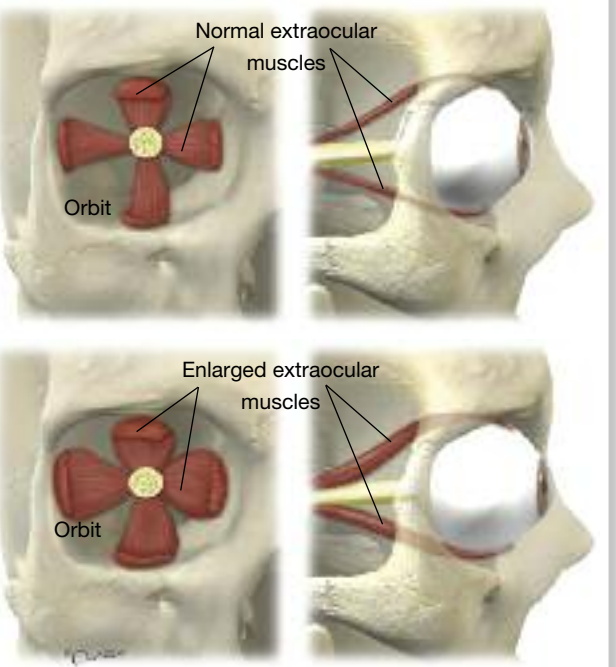
Thyroid eye disease (TED) is an inflammatory disease of the orbit (see box below) and is associated with thyroid disease such as Graves' disease. It presents in many different ways and is often misdiagnosed and undertreated.

The eye changes in TED usually progress slowly but sudden deterioration can occur. When severe, the disease causes disfigurement, double vision and blindness. The typical early warning symptoms of red eyes, recurrent or persistent eyelid swelling or puffiness around the eyes, or chronic watery eye (epiphora) should be heeded.

CAUSES

TED is an autoimmune disease and its exact cause and trigger remain unclear.[1] The disease is more common in females than males. It can be associated with diabetes or myasthenia gravis and is worse in cigarette smokers.

EXTRAOCULAR MUSCLES IN THYROID EYE DISEASE



Frontal (left) and lateral (right) orbital views showing the extraocular muscles in the normal orbit (top) and diseased orbit (bottom). In patients with thyroid eye disease, the belly of these muscles enlarges significantly, leading to bulging or protrusion of the eyeball. Additional inflammation causes puffiness of the eye.

CASE PRESENTATIONS

Case 1. A 42-year-old woman presents with puffiness of the eyes (Figures 1a and b). Her symptoms fluctuate but are getting worse over time. She complains of 'bags under the eyes'. She has been diagnosed with allergic eye disease related to her work environment, which led to workers compensation supported leave. She has tried over-the-counter allergy eyedrops, without success. The patient is of short stature and obese; she is being treated for mild hypertension. She has no history of thyroid disease or immune disease. A diagnosis of thyroid eye disease is made. The patient responds to systemic immunosuppression, with complete resolution of her presenting symptoms.

Figure 1a. The patient at presentation, with orbital inflammation and puffy eyes, stained with fluorescein, mimicking allergic eye disease.

Figure 1b. Swollen conjunctiva with dilated blood vessels.

Case 2. A 30-year-old woman presents with facial asymmetry of recent onset and a stare (Figure 2a). She is distraught – she has lost her position as bridesmaid at a friend's forthcoming wedding because of the changes in her appearance. She has no history of swelling around the face but she is being treated for hyperthyroidism. The patient undergoes thyroidectomy, followed by endoscopic orbital decompression and subsequent lowering of the upper eyelid. The surgical course took three months and restored her appearance (Figure 2b).

Figure 2a. The patient at presentation, with a bulging right eye (exophthalmos) and retracted upper eyelid. The changes were of rapid onset.

Figure 2b. Restored appearance after endoscopic orbital decompression and eyelid lowering.

Case 3. A woman with known hyperthyroidism presents with double vision (Figure 3a). She has been told she needs glasses. She is sent for ophthalmological review and is noted to have orbital inflammation. The patient declines orbital radiotherapy but undergoes thyroidectomy. The inflammation settles, but her double vision worsens because the orbital inflammatory disease has progressed (Figure 3b).

Figure 3a. The patient at presentation, with a stare and upper eyelid retraction.

Figure 3b. Worsening double vision due to progression of inflammatory orbital disease. The right eye is turned down due to tightening of the inferior rectus muscle.

PRESENTATIONS

TED is most common in women aged in their mid-40s. Patients typically complain of puffy eyelids, which they often interpret as a cosmetic problem rather than as evidence of a physical disease. They usually discuss their symptoms with friends but may be less inclined to mention these to their GP, feeling that such concerns would be interpreted as 'vanity'.

One difficulty with diagnosis is the variation in normal eye appearance between individuals. People of certain races are naturally more exophthalmic than others, and underdevelopment of the cheek can cause a pseudoexophthalmic picture. Unilateral TED is more easily detected than bilateral disease.

Some examples of TED are presented in case format in Figures 1 to 3 (see the box on this page).

CLINICAL FEATURES

The early orbital features of TED include the following:

- puffiness of the upper and/or lower eyelids (Figures 4a and b)
- redness or swelling of the conjunctiva (chemosis)
- watery eyes, typically associated with puffiness (Figure 5)
- eyelid retraction, typically involving the upper eyelid (Figures 6a and b)
- lid lag (Figure 7).

There may be systemic skin changes, including thickening of the pretibial skin (pretibial myxoedema) or enlargement of the terminal phalanges (thyroid acropachy). However, in my experience skin involvement is uncommon.

As the disease progresses, more established features may become present. These include exophthalmos (proptosis of the eyeball), diplopia, eyelid asymmetry and both upper and lower eyelid retraction. TED is more easily diagnosed clinically when these features are present. Loss of vision may occur at any time but is more typical with advanced disease.

DIAGNOSIS

TED can present before systemic thyroid disease has been diagnosed in the patient. Due to the array of soft and nonspecific symptoms and signs, the early diagnosis of TED can be difficult, and most reliably made by an ophthalmologist or oculoplastic surgeon. It relies on the clinical and radiological features.

All patients with an abnormal thyroid test should be questioned about orbital features and sent for thyroid antibody screening. Patients who have a positive test for thyroid antibodies should be questioned about orbital symptoms and examined for orbital features. All patients with orbital features should be referred to an oculoplastic surgeon or ophthalmologist.

The clinical diagnosis of TED is supported by studies of thyroid antibodies, which can be monitored as an indicator of disease activity.[1,2]

When the disease is severe, the characteristic eye bulge or stare makes the diagnosis obvious. However, by this stage the window has been missed for stopping disease progression and preventing disfigurement, double vision and blindness. The aim of early diagnosis is the prevention of full blown disease.

IMPORTANT CLINICAL SIGNS OF ORBITAL INFLAMMATION

Figures 4a and b. Orbital swelling in a younger patient (a, left) as unilateral disease and in an older patient (b, right) as bilateral disease. Age related skin changes can mask swelling.

Figure 5 (above). Increased volume of tears (epiphora), stained with fluorescein.

Figure 6a and b. Lid retraction (upper eyelids pulled up) in TED may be unilateral (a, top) or bilateral (b, above).

Figure 7 (left). Bilateral lid lag. The upper eyelid is higher than normal when the globe is in downgaze.

CT SCANNING

Ideally, an orbital CT scan should be performed in all patients with a clinical or suspected diagnosis of TED. The CT scan should be viewed as a supportive tool in the diagnosis of the disease. In reality, however, the scan may not be helpful because the presentation is so varied; the result may even be reported as normal in the presence of well developed TED. The scan is best ordered by the oculoplastic surgeon or ophthalmologist.

MANAGEMENT

Unless TED is severe with loss of vision, early systemic treatment aimed at thyroid control is an important first step to bring the disease under control. Management options include propylthiouracil or carbimazole and, if necessary, radioiodine therapy or thyroidectomy. Local treatment is supportive, with the use of tear replacement, ice packs and low dose topical steroids to address the inflammation. Cessation of smoking should be encouraged in all patients with TED who smoke.

If orbital disease does not respond to initial systemic therapy then the patient may need additional systemic treatment. Oral steroids, intravenous steroids, steroid sparing immunosuppression or orbital radiotherapy may be required. These treatments are initiated by an ophthalmologist or oculoplastic surgeon.

When radioiodine therapy is used to control systemic thyroid disease, transient exacerbation of the disease may occur in the immediate post-treatment period. Patients should be watched closely and, if necessary, provided with short-term steroid cover.

In my experience, low dose orbital radiotherapy provides a safe and effective means for controlling disease involving the orbit.

SURGERY

When the orbital inflammation has subsided and TED is in remission, surgical procedures are available to address any resulting disfigurement. These include eyelid surgery to reposition the eyelids, cosmetic procedures to restore eyelid appearance, and orbital decompression

THYROID EYE DISEASE: WHAT IS THE ROLE OF THE GP?

- The GP has a central role in the early diagnosis of TED, which is the key to managing the orbital component of the disease, and in looking for subclinical systemic disease.
- All patients with an abnormal thyroid test should be questioned about orbital features and sent for thyroid antibody screening.
- All patients with a positive test for thyroid antibodies should be questioned about orbital features.
- All patients with orbital features should be referred to an oculoplastic surgeon or ophthalmologist.
- GPs are involved in the investigation and management of systemic endocrine disease, where required.
- The GP can initiate supportive treatment to the eyes, including a trial of topical lubricants and a short course of low dose topical steroids.
- Cessation of smoking is a key factor in controlling TED. The GP has an important role in managing patients who smoke.
- For patients with elevated thyroid antibodies, the immune disease should be monitored with three-monthly thyroid antibody assays, along with thyroid function tests to monitor thyroid disease. For patients receiving immunosuppressive therapy, monitoring for side effects of immunosuppression, including osteopenia, mood changes, changes in blood glucose levels and cardiovascular disease, is critical.
- The GP may provide information about patient support groups for TED. Useful sources include The Australian Thyroid Foundation (www.thyroidfoundation.com.au) and the American Thyroid Association (www.thyroid.org).

to reposition the eyeball within the eye socket. Techniques for orbital decompression include external approach incisional surgery and endoscopic keyhole surgery, undertaken via the nasal cavity, which minimises tissue trauma and recovery time.[3] The postoperative pain of keyhole surgery is similar to that experienced following FESS (functional endoscopic sinus surgery).[3]

FINAL COMMENTS

TED is an inflammatory orbital disease that presents in many different ways and can be difficult to diagnose. Early diagnosis leads to prevention of debilitating disease, and involves recognition of orbital oedema, together with prompt assessment by an oculoplastic surgeon or ophthalmologist and collaboration with the GP. Keypoints for GPs in the diagnosis and management of TED are outlined in the box on this page. MT

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FURTHER READING

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COMPETING INTERESTS: None.