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CONSULTANT OPHTHALMIC, OCULOPLASTIC AND ORBITAL SURGEON

Patient information leaflet
Thyroid Eye Disease

What is Thyroid eye Disease?
Thyroid Eye Disease (TED) is also known as Graves' orbitopathy (GO), thyroid ophthalmopathy and thyroid-associated ophthalmopathy.

In TED, there is inflammation in and around the orbit (eye socket) caused by abnormal activity of the thyroid gland in the neck. The thyroid gland produces thyroid hormone, which helps to regulate our metabolism. Most patients with TED have over or underactive thyroid glands (hyper- or hypothyroidism) but some may have normal thyroid hormone levels (euthyroidism) at the time of presentation with TED. Thyroid gland dysfunction can develop before, after, or at the same time as TED.

How common is it?
Thyroid Eye Disease is the most common orbital disease in Europe. It is four times more common in women than men. This is because there is a higher preponderance of hyperthyroidism (overactive thyroid) in women. TED occurs in up to half of all patients with autoimmune thyroid disease. The incidence of TED is 16 per 100 000 females and 2.9/100 000 in males. Most patients have mild TED; only about 20% of patients will require more specific management over and above ocular lubricants.

What causes it?
This inflammation is a form of immune 'auto-reactivity' that is, an immune reaction by the body to thyroid stimulating hormone (TSH) receptors, involving the body's white blood cells (T-lymphocytes) which infiltrate the orbital tissue setting up an inflammatory cascade. This can cause swelling of muscles that move the eyeball (extra-ocular muscles), and an increased amount of fat in the orbit (eye socket). The exact mechanism linking thyroid dysfunction to thyroid eye disease however, still remains unclear and is the subject of on-going research.

During the course of the disease, there is usually progressive worsening over a few months in the initial phase followed by a peak before spontaneously

improving. It ends in a chronic 'burn-out' phase when further changes are unlikely.

Does anything increase the risk of TED?
Smoking is an important risk factor. It increases the risk of developing TED by seven- to eight-fold. The risk increases with the number of cigarettes smoked and reduces on quitting. Smoking also increases the risk of developing TED after radioiodine treatment for hyperthyroidism (overactive thyroid gland) but this can be reduced by corticosteroids. Smoking also reduces the impact (efficacy) of other methods of treatment such as steroids and radiotherapy.

Other risk factors include: being female. middle-age, uncontrolled thyroid dysfunction.

What are the symptoms?
Symptoms include: red, irritated dry eyes with tearing, grittiness and ache (worse in the mornings) behind the eye, as well as a staring appearance. Swelling of the eyelids, fatty tissue around the eyes and the eye muscles, can push the eye forward creating a bulging or protrusion of the eye and a staring appearance. The degree of eye protrusion can be variable and may involve one or both eyes. Swelling of the muscles that move the eyes may produce double vision. In severe cases, the clear window of the eye (the cornea) may ulcerate, or the optic nerve may be compressed resulting in loss of vision.

How is it diagnosed?
Diagnosis is usually straightforward in patients with the typical disease features mentioned above and a background abnormal thyroid function. Diagnosis may be confirmed with a blood test of thyroid function and imaging of the orbit (eye socket) for example, a CT or MRI scan of the orbits.

How is it treated?
Once an abnormal thyroid gland (over or underactive) is suspected, the thyroid function should be evaluated and appropriately treated by an endocrinologist (doctor that specialises in hormones). The eye disease should be managed by a ophthalmologist since the eye disease may continue to progress after the thyroid abnormality is treated and restored to normal.
Two phases of eye treatment should be considered. The first involves treating the active phase of the disease, which usually lasts about two years. This requires monitoring until stable. The second phase involves correcting any unacceptable permanent changes that persist following stabilisation of the active phase of the disease.

Treating the first (active) phase of TED

Mild active disease

In most patients with TED, the disease follows a mild course and artificial tears may be enough to control symptoms. The thyroid function should be optimised in all patients and smoking, a known risk factor for disease severity and response to treatment, should be stopped. Oral selenium supplements are now considered to be beneficial in mild forms of disease. They can be purchased from most health food shops. Prisms in spectacles may be given to control double vision.

Significant active disease

The inflammatory phase of the disease is self-limiting and usually lasts for one to two years. Treatment of the active phase focuses on preserving sight. Treatments specifically aimed at reducing active orbital inflammation include: oral or intravenous steroids (or other immunosuppressant medications such as azathioprine or rituximab), and low-dose orbital radiation (x-ray treatment of the eye socket). Orbital radiation may reduce the need for (and therefore the risks of) systemic immunosuppressive treatment and later, orbital decompression.

Severe TED

A very small number of patients suffer with marked inflammation and do not respond well enough to the treatments described above. In these cases, urgent surgery (within days or weeks) may be needed to protect vision. This surgery is called 'orbital decompression'. It relieves the inflammatory pressure around the eye itself and the optic nerve behind the eyeball.

Treating the second (inactive) phase of TED

In the second phase of treatment, surgery may be needed to correct any unwanted permanent changes caused by the eye disease for example, correction of eye protrusion (proptosis), double vision, or abnormal eyelid height. Since orbital decompression (used to treat protrusion of the eye) can affect double vision and eyelid height, this surgery if needed is done before eye muscle or eyelid surgery.

When is orbital decompression surgery required?

Orbital decompression may be indicated in the following circumstances:

1. Orbital decompression surgery may be needed if the optic nerve is being compressed by the inflamed orbital (eye socket) contents causing visual loss, which has not responded well enough to medical treatment (immunosuppression).

2. If the pressure in the orbit (eye socket) is causing high intraocular pressure (within the eye) known as secondary glaucoma.
3. Stable proptosis ('bulging' eyes) – if desired decompression surgery is planned once the amount of forward protrusion of the eyes is stable for a minimum of six months, with optimum thyroid function and cessation of smoking in order to improve appearance and to restore the patient's appearance.

What does orbital decompression surgery involve?
The purpose of the surgery is to relieve the pressure in the orbit (eye socket) by increasing the available space for the orbital contents. This allows the eye to settle back into a more normal position within the eye socket.

Surgery is performed under general anaesthetic and typically involves an overnight stay after the operation such that the patient is discharged home the morning after surgery. The amount of surgery needed depends on the severity of disease and the amount of proptosis. Of the four 'walls' of the orbit, the medial (inner), lateral (outer) and inferior (floor) of the orbit can be decompressed and some fat can be removed from the orbit. Incisions are made within skin creases in the eyelids or within the conjunctiva on the inside of the eyelids in order to hide surgical scars.

What is the recovery like after orbital decompression?
Orbital decompression is a major operation. The skin incisions will heal within a couple of weeks but natural repair and healing of the deeper tissues can take a lot longer (many months). This can account for occasional deeper ache or discomfort during this healing phase.

Immediately after surgery, a firm dressing is applied over the eye(s). The dressing and a surgical drain, are both removed by a doctor the following morning on the ward. The patient is provided with antibiotic eye ointment, oral antibiotics and a tapering course of oral steroids before they go home.

Swelling and bruising of the eyelids usually develops in the week after surgery. It usually improves a lot within a couple of weeks but can take several months to settle completely.

Double vision may occur or worsen after decompression surgery. This may require subsequent surgery to re-align the eyes. Driving and working after surgery may be delayed for several weeks and this should be taken in to account when planning treatment.

What are the possible complications of orbital decompression?
Side effects may include: bleeding, bruising, infection, swelling (inflammation) and scarring. Specific side effects can include: double vision, a change in lid height or position, a decline in vision, and even loss of vision.

With lateral wall decompression, a sensation of eye movement with eating (known as masticatory oscillation) can rarely occur though this does not tend to require intervention.

With decompression over the orbital floor (this is undertaken in patients with more severe proptosis), there is often numbness over the upper cheek and upper front teeth and gums. This recovers completely in over 90% of patients. Full recovery of sensation can take up to one year.

All forms of orbital decompression surgery carry a risk of permanent loss of vision. This risk is in the region of 1 in 1000. In patients with visual loss before surgery, visual recovery may be incomplete. Because drainage of the air sinuses around the eye may be temporarily affected, nose-blowing, flying and scuba diving should be avoided for at least 3 weeks after surgery. Rarely surgery is required to improve sinus drainage after orbital decompression.

Self-help
In thyroid eye disease, the following measures may ease symptoms:

- Stop smoking
- Dark glasses (to soothe dry eyes and prevent discomfort caused by light – photophobia)
- Avoid dry dusty conditions if possible (these will aggravate dry eyes).
- Use ocular lubricants as prescribed by your doctor.
- Sleep a little bit propped up e.g. with 2 to 3 pillows at night to reduce puffiness around the eyes.
- Cool compresses in the mornings across closed eyelids can help to reduce puffiness around the eyes.
- Selenium supplements (from health food shops or in brazil nuts) have been shown to be beneficial.

Symptoms to report
If you have eye discomfort or ache, red eyes, reduced vision, worsening double vision or eye protrusion, you should contact Miss Mellington (via her secretary on 0121 277 0787), your GP or NHS Direct (0845 46 47 - 24 hours a day, 7 days a week) for advice, unless symptoms are severe in which case you should seek an urgent eye review at an Eye Casualty.

Further information

1. **Thyroid Eye Disease Charitable Trust**
   The Thyroid Eye Disease Charitable Trust is run by patients and clinicians. It provides information, care and support to those affected by thyroid disease.

   PO BOX 1928, Bristol, BS37 0AX, UK
   Website: http://www.tedct.co.uk
   Email: ted@tedct.co.uk
   Tel: 0844 800 8133

2. **British Thyroid Foundation (BTF)**
   The British Thyroid Foundation (BTF) supports people with thyroid disorders and is for anyone who wants to know more about thyroid disease in general.

   British Thyroid Foundation, 2nd Floor, 3 Devonshire Place, Harrogate, HG1 4AA, UK
   Website: www.btf-thyroid.org
   Email: info@btf-thyroid.org
   Tel: 01423 709707 or 01423 709448

3. **Thyroid Federation International (TFI)**
   Thyroid Federation International is an umbrella organisation for thyroid patient organisations all over the world.
   PO BOX 471, Bath ON K0H 1G0, Canada.
   Website: www.thyroid-fed.org
   Email: tfi@thyroid-fed.org

4. **EUGOGO- European Group of Graves Orbitopathy**
   Website: www.eugogo.eu