# Thyroid eye disease

The most common cause of chronic abducens nerve palsy.

Graves' eye disease, also known as thyroid eye disease, is an autoimmune condition in which immune cells attack the thyroid gland which responds by secreting an excess amount of thyroid hormone. As a result, the thyroid gland enlarges and excess hormones increase metabolism. The hypermetabolic state is characterized by fast pulse/heartbeat, palpitations, profuse sweating, high blood pressure, irritability, fatigue, weight loss, heat intolerance, and loss of hair and alterations in hair quality.

Pseudoabducens palsy: may be due to

a) thyroid eye disease: the most common cause of chronic VI palsy. Will have positive forced duction test (eye cannot be moved by the examiner).

The eyes are particularly vulnerable to Graves' eye disease, because the autoimmune attack often targets the eye muscles and connective tissue within the eye socket. This likely occurs because these tissues contain proteins that appear similar to the immune system as those of the thyroid gland. Ocular symptoms can range from mild to severe; but only 10-20% of patients have sight threatening disease. Another tissue that can also be involved in the immune attack of Graves' eye disease is the skin of the shins.

Graves' eye disease can also be present when the level of thyroid hormone in the blood is normal or low, depending on the degree of glandular stimulation caused by the immune attack and by the amount of thyroid gland destruction present at the time of diagnosis. However, most patients with eye symptoms have abnormal hormone levels that are accompanied by specific antibodies in the blood stream. The components of the disease affecting the thyroid gland and the eyes have common causes, but evolve separately and must each be treated separately.

Symptoms

Swelling (edema) of the eyelids and tissue around the eye

A constant stare

Eyelid retraction

Dry eyes or a sensation of grit or irritation to the eye

Watering and redness of the eyes

Sensitivity to light

A feeling of pressure in the eye sockets

Double vision

Loss of vision

The symptoms described above may not necessarily mean that a person has Graves' eye disease. However, if you experience these symptoms, contact your eye doctor for a complete examination to evaluate for this common eye condition.

Causes When the immune system attacks the muscles and other ocular tissues in the eye socket, the swelling and scarring resulting from the inflammation causes symptoms and signs noted above. In severe cases, the clear covering of the eye (cornea) may ulcerate, or the optic nerve may be damaged, either of which may result in a permanent loss of vision if not treated appropriately. The former is often due to a combination of the eyes bulging forward and scarring resulting in the eyelids retracting backward. The latter is due to thickened, inflamed and/or scarred muscles impinging on the optic nerve at the back of the socket.

In most patients who develop Graves' ophthalmopathy, the eyes bulge forward or the eyelid retracts to some degree. Many patients with mild to moderate Graves' ophthalmopathy will experience spontaneous improvement over the course of two to three years or will adapt to the abnormality. Severe ophthalmopathy will affect 10% of patients. It is caused by inflammation of the muscles, which causes them to swell. They can also become stiff (scarred), which interferes with movement of the eyes and causes double vision or impinges upon the optic nerve, causing loss of vision. In some patients, eye protrusion makes it difficult for the lids to close properly and the cornea becomes exposed and vulnerable. When the optic nerve is compromised, progressive and irreversible vision loss occurs. Rarely, orbital swelling may precipitate glaucoma that also affects the optic nerve.

Risk Factors Approximately one million Americans are diagnosed with Graves' eye disease each year. Women are five to six times more likely than men to get the disease. Cigarette smokers are at significantly increased risk to develop the disease, and when they do, often have more severe and prolonged activity that threatens vision.

Tests and Diagnosis If your doctor suspects you have an overactive thyroid gland, your thyroid function must first be evaluated and treated appropriately by an internist trained in doing so. Treatments include medications to suppress the production of hormone by the thyroid gland, radioactive iodine to eliminate hormone-producing cells, and surgery to remove the thyroid tissue. In most cases, replacement thyroid hormone is required following the natural course of the Graves' autoimmune attack on the thyroid gland or following effective treatment. Once your thyroid function is treated and returned to normal, the eye disease must be monitored as it often continues to progress. Eye involvement must be evaluated on a continuing basis by an ophthalmologist during the active phase of the disease and, if necessary, treated. Although symptoms often resolve on their own, activity, scarring, and visual loss not readily apparent to the patient may otherwise go unnoticed and cause permanent changes.

Treatment and Drugs Treatment for thyroid eye disease generally occurs in two phases. The first phase involves treating the active eye disease. This active period usually lasts two to three years and requires careful monitoring until stable. Treatment during the active phase of the disease focuses on preserving sight and the integrity of the cornea as well as providing treatment for double vision when it interferes with daily functioning and becomes bothersome.

Most patients experience relief from dry eyes by using artificial tears throughout the day and gels or ointments at night. Some patients also use eye covers at night or tape their eyes shut to keep them from becoming dry if the eyelids do not close properly. Dryness occurs because the lids are retracted and cannot blink properly, because the tear-producing glands have been affected by the autoimmune process and aren't functioning well, and/or because the forward bulging of the eyes prevents them from being completely covered by the lids. In some cases, acute swelling causing double vision or loss of vision may be treated for a limited time with prednisone. However, prednisone given for more than a few weeks at the dosages required to suppress the autoimmune inflammation always causes bothersome side-effects that may become severe. In patients who respond to prednisone, radiation therapy may be offered to reduce swelling, double vision, and, in severe cases, loss of vision. Most people get relief from their symptoms within two months of the radiation. However, radiation

treatment is only marginally effective at reducing these abnormalities and may cause ocular dryness. It can only be used at most twice in a person's lifetime and bears a slight risk for inducing tumors. Surgical decompression can also be used during the active phase, most often to relieve optic neuropathy. It is also helpful in reducing congestion, redness, pain, and ocular exposure.

Treatment during the remission phase that lasts indefinitely in most cases, involves correcting unacceptable permanent changes that persist after the ocular conditions of the active phase have stabilized. In the second phase, treatment of permanent changes may require surgery to correct double vision and reduce eyelid retraction. Surgery may be helpful in returning the eye to a normal position within the socket (orbital decompression).

It is important to stop smoking in order to reduce the severity, duration of activity, degree of scarring, and risk of optic nerve involvement, greatly improving the success of treating Graves' eye disease.

## Case series

The medical records of TED patients treated for strabismus from January 2005 to January 2016 were reviewed retrospectively for demographic and surgical data. The cross-sectional superior oblique area was compared to age-matched controls on high-resolution orbital computed tomography (CT) using a standardized protocol.

A total of 46 TED patients and 18 controls were included. The mean superior oblique cross-sectional area in TED subjects was 250% larger than in controls (22.88 ± 6.64 mm2 vs 9.32 ± 1.85 mm2. The mean cross-sectional area was >3 standard deviations from the mean of the control group in 96% of TED patients.

Superior oblique enlargement in TED may occur more frequently than generally recognized, challenging the notion that TED is primarily a disease of the rectus muscles [1].

1)
Del Porto L, Hinds AM, Raoof N, Barras C, Davagnanam I, Hancox J, Adams G. Superior oblique enlargement in thyroid eye disease. J AAPOS. 2019 Jul 22. pii: S1091-8531(19)30172-7. doi: 10.1016/j.jaapos.2019.04.010. [Epub ahead of print] PubMed PMID: 31344455.

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