Thyroid eye disease

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Thyroid eye disease is the most common cause of proptosis in adults, with women affected 3 to 10 times more frequently than men. On the other hand, the disease tends to be more severe in men. The mean age of presentation for Grave's thyroid disease is 41 years and for orbital disease it is 43.5 years. Thyroid eye disease is the result of autoimmune processes.

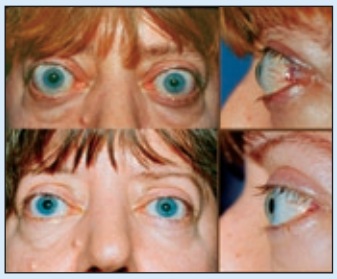
Pathophysiology

Both humoral and cell-mediated immune mechanisms have been implicated, resulting in extraocular muscle myositis, fibroblast proliferation, glycosaminoglycan overproduction, and orbital congestion. The hyperthyroidism of Graves' disease usually runs an independent course with stimulation of thyrotropin receptors on the thyroid cell plasma membrane by immunoglobulin.

Initial manifestations of thyroid eye disease include upper lid retraction and lid lag, due to increased sympathetic tone.
An inflammatory phase often follows and affects the orbital soft tissue to varying degrees (see table).

Graded Eye Signs – Grave's Disease

0 - No signs nor symptoms
1 - Only signs are upper eyelid retraction, lid lag, and stare
2 - Soft tissue signs and symptoms:
Resistance to retropulsion
Edema of conjunctiva and caruncle
Lacrimal gland enlargement
Injection over the horizontal rectus muscle insertions
Edema of eyelids
Fullness of the eyelids
3 - Proptosis
4 - Extraocular muscle involvement
5 - Corneal involvement secondary to exposure
6 - Sight loss secondary to optic nerve compression



■ *Eye signs improved by orbital surgery*

Proptosis reflects an increase in orbital content volume secondary to extraocular muscle enlargement, increased fat volume and accumulation of mucopolysaccharides that attract and bind water.

This process is accompanied and followed by round cell infiltration and fibrosis. The extraocular muscles involved in this fibrosis in order of frequency are: inferior, medial, superior, and lateral rectus. Patients usually present with vertical and/or horizontal diplopia associated with poor elevation and/or abduction of the eye.

The combination of hypotropia, eyelid retraction and proptosis predisposes to corneal exposure and ulceration.

The optic nerve may be compromised secondary to compression by the enlarged muscles at the orbital apex.

Thyroid eye disease can often be diagnosed clinically and if necessary, confirmed by the typical appearance of enlarged extraocular muscles with sparing of muscle tendons on CT (or MRI) of the orbit.

Serum T3, T4 and TRH assays are performed to establish thyroid gland activity (patients are often euthyroid). Thyroid antibodies demonstrate immunological abnormalities.

Management

The management of thyroid eye disease is largely independent of the management of Graves' disease.

Proptosis and corneal ulceration. Prednisone 100 mg po od x 1-2 days followed by orbital decompression surgery.

Acute disturbing diplopia with an inflamed eye. Prednisone 60-100 mg po od, which may be tapered slowly as the condition improves. Orbital radiation should be considered. If no improvement occurs within 10 days, then taper the patient off the steroids quickly.

Visual loss from optic neuropathy. Treat immediately. Options include prednisone 100 mg po qd, immunosuppressive agents, radiation therapy and posterior orbital decompression surgery.

When orbital findings stabilise, surgery of the orbit, extraocular muscles and eyelids can be undertaken.

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