Figure 1: 52 year old lady with severe bilateral symmetrical exophthalmos (proptosis) due to thyroid eye disease.

Figure 2: Patient after bilateral orbital decompression surgery.

Figure 3: Patient after lower eyelid retraction surgery.

Figure 4: Intraoperative photograph of right lower lid release retraction. Note arrow at higher lid margin.

Figure 5: 72 year old male patient with asymmetric proptosis due to thyroid eye disease. Note thickening of muscles greater on the left than on the right side, on axial CT scan to the left.

Thyroid EYE DISEASE

Thyroid eye disease is an autoimmune disease which affects the retro-bulbar tissues of the orbit. Dr. Raf Ghabrial As well as being disfiguring and sightthreatening, thyroid eye disease may cause proptosis (abnormal protrusion) of one or both eyes, along with a multitude of other ophthalmic signs and symptoms.

Epidemiology

The incidence of Graves' disease (the most common form of hyperthyroidism)

It can exist in the absence of a thyroid dysfunction, so called "euthyroid" status. Thus it can be considered as a separate clinical entity which often runs independent of its parent thyroid status.

Pathology

Thyroid eye disease can be explained by accumulation of tissue and fluid

"Thyroid eye disease can be explained by accumulation of tissue and fluid causing pressure on the orbit contents"

is approximately 12-20 people per 100,000 population. It occurs most commonly in middle-aged patients and the mean age of onset is 45 years, more commonly in women than in men. Smoking is associated with an increased risk of developing thyroid eye disease and a more severe form of the same.

Association with Thyroid Disease

Only less than five per cent of patients with thyroid disorder develop clinical thyroid eye disease, but careful examination may reveal up to 50 per cent of patients being affected in a sub-clinical manner or with blood tests for antibodies. Thyroid eye disease is frequently associated with hyperthyroidism and less commonly with hypothyroidism. causing pressure on the orbit contents. Most commonly affected tissues are the extraocular muscles; the inferior rectus muscle is infiltrated more frequently than the medial rectus muscle, less frequently involved are the lateral rectus and superior rectus muscle. Occasionally patients suffer infiltration of orbital fat and the muscles are relatively spare.

Clinical Presentation

Patients may have lid retraction and lid lag or more severe signs of diplopia and proptosis. The most feared manifestations are the sight-threatening complications including optic neuropathy and end-stage corneal exposure. All of these findings are explained by infiltration of the tissue and relative forward placement (proptosis/



exophthalmos) of the eye. Recently subtle changes in refraction including relative hypermetropia and astigmatic variation, explained by pressure on the globe from the enlarged muscles, have been reported by our group at Sydney University.

Investigation

Radiological examination with a CT scan is the investigation of choice (see Figure 5). Laboratory examinations for blood tests to show thyroid immune dysfunction can be useful but may not change the course of the disease if treated.

Treatment

Treatment of thyroid eye disease is undertaken when there is visionthreatening disease or when infiltration of the tissues around the eye cause ocular or cosmetic symptoms. This may be as simple supportive treatment such as ocular lubrication and sleeping on extra pillows to allow fluid to drain at night.

More likely, patients with severe disease may require immune treatment with medication such as steroids and other immunosuppressants (e.g. Azathioprine). Radiotherapy to the orbit has been traditionally used to help to decrease orbital inflammation. Recently this has been questioned with a double blind study where radiotherapy was applied to one orbit and sham radiotherapy to the other orbit. Minimal difference in each orbit outcome was detected at the end of the study.

Once the inflammation has been treated and the patient has been stable for some six months, surgical intervention may be considered. We adhere to a strict pathway whereby decompression of the orbits is undertaken prior to any strabismus or squint surgery. Only after the extraocular muscle strabismus surgery is performed can eyelid surgery be undertaken as the former procedures can have a direct affect on subsequent issues.

Orbital decompression surgery is traditionally performed by removing bone around the eye, the so-called "walls". The extraocular muscles while infiltrated can not be removed as they carry the all-important anterior ciliary artery to the anterior portion of the globe. To make room for these muscles, adjacent orbital bone is removed. Recent advances include scareless endoscopic surgery where bone can be removed from the nose; this allows rapid recovery and effective pressure release on the tissues. Some surgeons advocate removal of orbital fat in select cases.

Once the orbital volume has been removed then strabismus surgery may be performed. Usually a recession procedure of one of the shortened muscles is performed to effectively lengthen its course. An adjustment during the post-operative recovery phase can be beneficial to allow the patient to see single while the muscle is secured.

The final stage of surgical intervention is repair of the eyelid retraction (see Figures 3 and 4). This can be undertaken by lengthening the muscles of the eyelid aiming to minimise sclera show.

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