

Endoscopic orbital decompression for thyroid eye disease

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Abstract

Background Thyroid eye disease is a disorder of immune function resulting in extraocular myopathy and an increase in retrobulbar fat. These changes lead to the clinical features found in thyroid eye disease including proptosis, eyelid retraction, restrictive myopathy and sometimes compressive optic neuropathy. Orbital decompression is undertaken for optic nerve compression, exposure keratopathy or cosmesis. Previously this involved an external approach with the removal of the medial orbital wall along with the floor. An intranasal endoscopic approach with the removal of the medial orbital wall and part of the floor has recently been advocated as an alternative procedure.

Methods We present the results of 7 patients who underwent endoscopic orbital decompression. The effects on optic nerve function, proptosis and ocular motility were assessed. Symptoms before and after surgery were monitored.

Results The surgery was found to be successful in all cases with a reduction in proptosis and minimal effect on ocular motility. The surgery was also associated with a low post-operative morbidity.

Conclusion We suggest endoscopic orbital decompression is an effective and safe treatment for dysthyroid eye disease.

Key words Endoscopic, Dysthyroid eye disease

Thyroid eye disease is an autoimmune disorder in which there is lymphocytic infiltration of the extraocular muscles, and an accumulation of glycosaminoglycans in the orbital fat which leads to an increase in the volume of the orbital tissue. Depending on the severity, this results in orbital congestion, proptosis, lid retraction, restrictive myopathy with diplopia and in the worst cases corneal exposure and compressive optic neuropathy.¹

The clinical course is progressive over months to years. Some clinical signs persist after resolution due to cicatricial changes.

Treatment of thyroid eye disease consists of lubrication for corneal exposure, systemic immunosuppression, radiation therapy and

surgery. Systemic corticosteroids are useful in the acute phase of the disease. However treatment may need to be continued for a long time, and results in side effects. External beam radiation may be employed to limit the amount of proptosis and optic nerve compression. Orbital decompressive surgery is indicated if the above treatment modalities fail to control compressive optic neuropathy or severe corneal exposure. It is also used to minimise cosmetic deformity due to proptosis.

Surgical decompression involves removing one to four of the bony orbital walls and incising the orbital periosteum allowing the swollen orbital contents to prolapse into the adjacent spaces. This can be achieved either externally or using an endoscopic approach.

This paper reports on the results and complications of endoscopic decompression in such patients with thyroid orbitopathy.

Patients and methods

Seven patients underwent bilateral endoscopic orbital decompressions. There were 3 men and 4 women with a mean age of 37 (range 19 to 62) years. Indications for surgical decompression are listed in Table 1. Prior therapy included systemic immunosuppression and radiotherapy. Some patients went on to have either squint or oculoplastic surgery.

Preoperative assessment included a complete ophthalmological and orthoptic assessment including Hertel exophthalmometry and Hess charts to assess the degree of exophthalmos and oculomotility.

Otolaryngologic assessment included nasal endoscopy to identify any sinus or septal pathology. Imaging included either computerised tomography (CT) or magnetic resonance imaging (MRI). All operations were performed under general anaesthesia. All patients were followed at least 12 months post-operatively.

The aim is to remove the entire medial wall and part of the floor of the orbit so that the orbital contents can prolapse into the ethmoidal and maxillary cavities. A pre-operative nasal examination and coronal CT scan of the sinuses are performed to provide accurate details of the

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Fig. 1. Preoperative photograph of patient 1 shows severe exophthalmos and exposure keratopathy with bilateral ulceration.

variable ethmoidal anatomy and to identify any co-existing sinonasal pathology.

The operation is performed under general anaesthesia with the patient in a supine position. The nasal cavity is instilled with a mixture of 2 ml of 10% cocaine and 1 ml mixture of 1:1000 adrenaline solution followed by infiltration of the lateral nasal wall with a 1 ml mixture of 1:80 000 adrenaline and 2% xylocaine.

A standard endoscopic sphenoidectomy is performed and the medial wall of the orbit is carefully skeletonised and fully exposed as far back as the orbital apex. A generous middle meatal antrostomy is fashioned for access to the orbital floor. The thin bone of the medial orbital wall (lamina papyracea) is removed after careful separation from the periorbita with a Freer elevator.

Bony decompression of the orbital floor extends up to the infraorbital nerve canal to avoid damaging the nerve. Excessive decompression of the orbital floor may also lead to post-operative diplopia or aggravate a pre-existing diplopia¹ and increases the risk of recurrent maxillary sinusitis.² In practice, surgical access to the

orbital floor through the middle meatal opening is rather restricted and only the medial 0.5 to 1 cm of the bone is readily removed. In the presence of thick orbital floor bone, a bone cutting burr may first be used to thin bone down to facilitate removal with a Freer elevator. Care must be taken not to penetrate the periorbita prior to completing all the bone work as premature prolapse of orbital fat may obscure the surgical field.

Once the periorbita is fully exposed, multiple full thickness parallel linear incisions are made with a sickle knife in a posterior-anterior direction. This results in herniation of orbital fat into the ethmoidal and maxillary cavities. Gentle external compression of the globe allows identification of any undivided strands of periorbita which should be completely divided.

If decompression of the optic nerve is required, the thick bone over the orbital apex may first be thinned down with a drill and then removed with a Freer elevator. The aim is to decompress the nerve on its medial half from orbital apex.

Table 1. Patient details

Patient	Age	Sex	Presentation	Surgery	Surgical indications
1	33	F	1995	1995	Exposure keratopathy
2	19	M	1992	1996	Cosmesis
3	32	M	1991	1995	Cosmesis
4	61	F	1995	1995	Acute proptosis
5	37	F	1994	1995	Optic neuropathy
6	45	F	1992	1996	Cosmesis
7	34	M	1992	1995	Cosmesis



Fig. 2. Post-operative photograph of patient 1 5 months later following bilateral endoscopic decompressions demonstrates excellent reduction in proptosis with resolution of keratopathy.

The patient is maintained on antibiotics (Augmentin) for 5 days post-operatively and advised not to blow the nose for a week.

Results

There were no intra-operative or post-operative complications. All the patients except one were discharged within 24 hours. The other patient remained an extra day due to the effect of the general anaesthetic. The follow-up period was at least 12 months. Proptosis was reduced an average of 3.79 mm (range 2–7). There was no statistical correlation between the reduction in proptosis with age, sex, side of operation, surgical indication, preoperative proptosis, and duration of the disease process.

Four patients whose surgery was performed for cosmesis were pleased with their post-operative appearance. Two of these patients have had oculoplastic surgery. The patient whose surgery was performed for subluxation of the globe and acute proptosis experienced

no further episodes. The patient with compressive optic neuropathy had a post-operative improvement in visual acuity with no further loss in optic nerve function. In the seventh patient the exposure keratopathy improved post-operatively.

Two patients had preoperative diplopia, which in one patient increased post-operatively from 18 to 30 prism dioptres base out for near and from 20 to 35 prism dioptres base out for distance. There was minimal post-operative change in the second patient. Both patients went on to have squint surgery. The remaining five patients had no change in oculomotility, with two patients experienced a 'freeing of their eyes'. These results are summarised in Table 3.

Discussion

Thyroid eye disease can be divided into two broad stages. Initially there is an acute active inflammatory and congestive phase lasting 6–18 months. This stage is mediated by lymphocytes and fibroblasts and is partially responsive to immunosuppression and radiotherapy.¹ Corticosteroids are the most commonly used immunosuppressive agents, being particularly useful in patients with acute inflammatory orbital disease and optic neuropathy. Although these are improved with steroids, the problem recurs on cessation of therapy. There are many contraindications to, and complications of, steroid therapy. Corticosteroids have little effect on exophthalmos and not all patients respond to treatment. Immunosuppression can also be achieved with

Table 2. Treatment details of patients

Patient	Systemic steroids	Radiotherapy	Other surgery
1	Yes	No	Oculoplastic
2	Yes	Yes	No
3	Yes	Yes	Oculoplastic
4	No	No	No
5	Yes	Yes	Strabismus
6	Yes	No	No
7	No	No	Strabismus

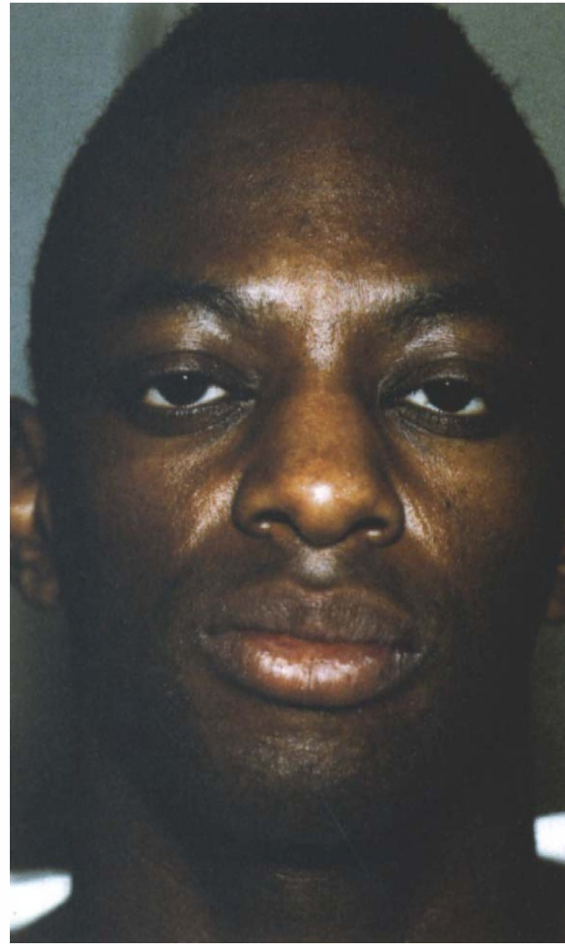
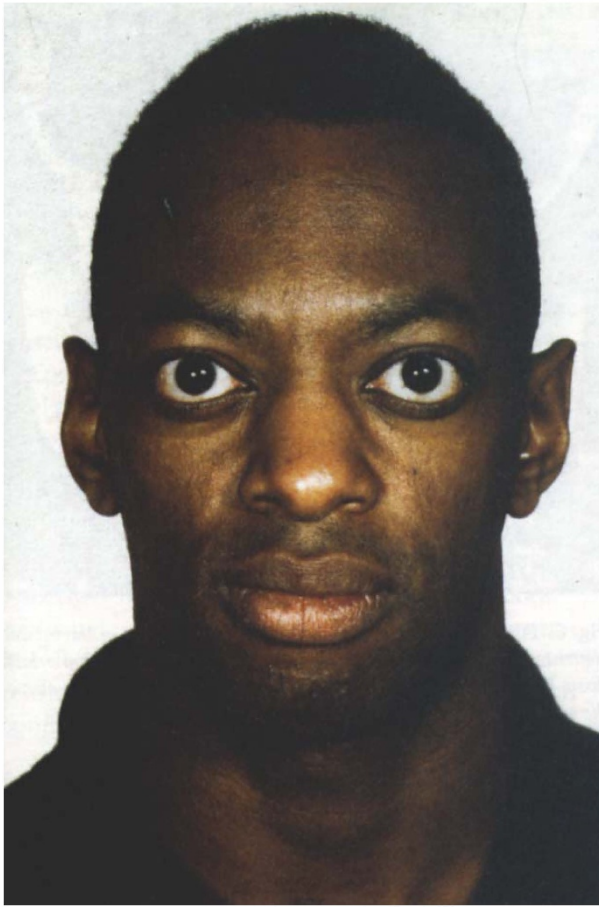


Fig. 3. Preoperative illustration of patient 3 shows marked bilateral exophthalmos.

Fig. 4. Post-operative illustration of patient 3 1 month following bilateral endoscopic decompressions demonstrates an excellent reduction in proptosis with improved cosmesis.

cyclosporin, methotrexate and azathioprine. All these agents can be associated with serious systemic complications and are not as effective as corticosteroids in controlling the inflammation.¹

The role of radiotherapy in the treatment of thyroid eye disease is still questioned. A dose of 20 Gy is recommended by most authors. Radiotherapy is associated with a significant improvement in subjective and objective signs of orbital congestion, in relief of compressive optic neuropathy, and fewer complications than high dose corticosteroids.³

The acute phase is followed by a chronic stable phase with hypertrophy and fibrosis of the extraocular muscles, lacrimal gland and orbital fat. These changes are usually permanent and unresponsive to either steroids or radiotherapy. Surgical treatment is required to treat this stage.⁴ Occasionally, a few patients require orbital decompression in the acute phase when visual acuity is threatened.

Unlike steroids and radiotherapy which try to control the local inflammatory process albeit temporarily, surgery permanently enlarges the orbital volume to

Table 3. A summary of post-operative changes

Name	Surgical indications	Change in proptosis	Change in ocular motility	Change in visual acuity
1	Exposure keratopathy	7 mm	No	Improvement
2	Cosmesis	2 mm	No	Nil
3	Cosmesis	4 mm	No	Nil
4	Acute proptosis	4 mm	No	Nil
5	Optic nerve	3.5 mm	No	Improvement
6	Compression cosmesis	3 mm	No	Nil
7	Cosmesis	3 mm	18-30 Δ BO (N) 20-35 Δ BO (D)	Nil

Δ = Prism
BO = Base out
(N) = Near
(D) = Distance

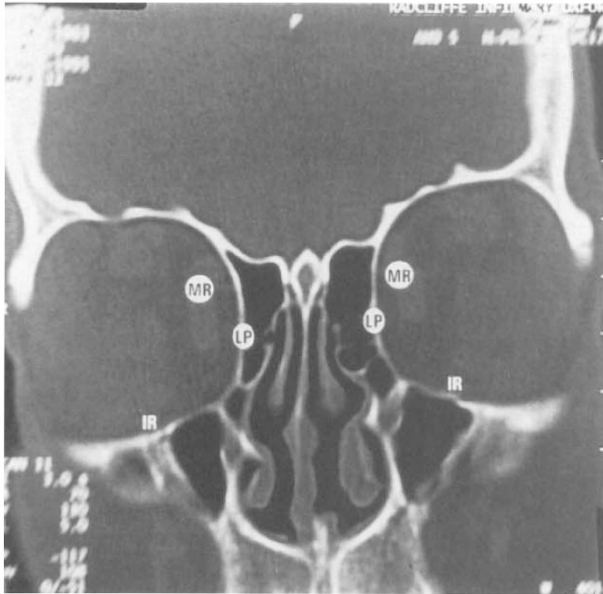


Fig. 5. Preoperative MRI scan. Preoperative coronal view shows congested orbital contents, with an enlarged medial rectus (MR) abutting the lamina papyracea (LP).

reduce the orbital pressure and to improve tissue fluid dynamics. Different surgical approaches have been employed without rigorous comparative evaluation. The timing and indications for surgery in relation to other treatments such as radiotherapy and systemic immunosuppression also require clarification.

Orbital decompression has traditionally been performed through a transantral approach, with the Walsh-Ogura transantral approach being the most commonly used procedure.² A maxillary antrostomy allows removal of the entire orbital floor with the preservation of the intraorbital nerve. The medial orbital wall is exposed and removed through a transantral ethmoidectomy. This approach avoids an external incision, but is associated with the morbidity of a Caldwell-Luc procedure.⁵ The post-operative morbidity includes considerable swelling and discomfort immediately following surgery and persistent numbness over the mandibular region following division of the anterior superior alveolar nerves. The Walsh-Ogura procedure achieves an average of 4 to 6 mm of reduction in proptosis. A larger reduction in proptosis can be achieved with the Kennerdell-Maroon four wall decompression.

With improved technology in fiberoptic instrumentation orbital structures can be safely and effectively operated on intranasally. Endoscopic dacryocystorhinostomy and orbital decompressions have been shown to be both effective and associated with a low morbidity.⁶⁻⁸

Endoscopic orbital surgery allows clear visualisation of orbital structures with no need for a skin incision. Some authors have suggested that severe septal deviation is a contraindication to endoscopic orbital surgery. We do not feel that this is the case as obstructing

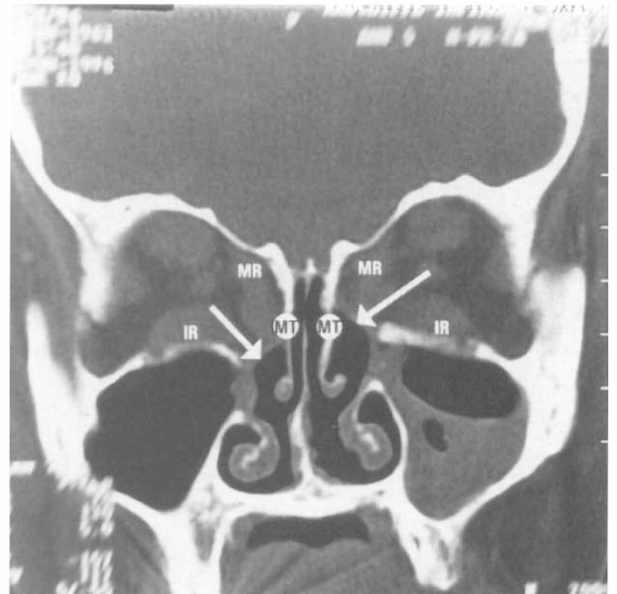


Fig. 6. Post-decompression MRI scan shows expansion of the orbital contents into the ethmoidal and maxillary sinuses. The medial orbital floors have been removed (arrows). The medial rectus now lies above the superior attachments of the middle turbinates (MT).

intranasal pathology is simply treated at the same time. Only 1 of our patients required a minor septoplasty at the time of surgery.

In our series of 14 orbits, we report a reduction in proptosis by an average of 3.79 mm, range 2 to 7 mm. This is in keeping with other reported series of endoscopic decompression.⁹ Kennedy *et al.* achieved a reduction in proptosis by an average of 4.7 mm, range 3 to 5 mm, in 5 orbits; however two of their procedures involved an exploratory Caldwell-Luc antrostomy with additional decompression of the orbital floor.⁸ Lund *et al.* achieved a similar reduction in proptosis irrespective of whether an external ethmoidectomy or an endoscopic approach was utilised.¹⁰ A greater reduction in proptosis can be achieved by a combined external lateral decompression. This technique achieves results comparable to those reported for a conventional Walsh-Ogura procedure while avoiding the morbidity of external ethmoidectomy or Caldwell-Luc antrostomy.

Our results show that endoscopic orbital decompression achieves a similar reduction in proptosis compared with a traditional external approach, and is associated with a lower incidence of post-operative complications. The endoscopic approach avoids post-operative infraorbital nerve anaesthesia as identification of the nerve is possible and it is not traumatised. As visualisation especially in the region of the ethmoid roof and orbital apex is better endoscopically compared with an external approach decompression of the optic nerve is achieved more safely by this approach. Furthermore, an external decompression may be followed by post-operative hypoglobus,¹¹ a complication not seen in our patients.

A worsening of extraocular muscle balance may follow orbital decompression with reported incidences

being as high as 69% following successful surgery.¹² McCord has highlighted the relatively low incidence of post-operative diplopia associated with a translid versus the transantral approach.¹³ In his series a 40% worsening of diplopia was associated with transantral compared with 5.6% incidence following the translid approach. In our series only 1 patient has a worsening of diplopia after surgery. Furthermore, none of the patients developed a significant A pattern, a complication thought to be caused by disturbance to the origin of the inferior oblique muscle.

Endoscopic decompression can be performed under local anaesthesia. In addition to a more rapid post-operative recovery, the use of local anaesthesia enables intraoperative monitoring of the patient's vision, reducing the likelihood of accidental optic nerve damage.

In conclusion, endoscopic orbital decompression is a safe and effective treatment for dysthyroid eye disease associated with lower post-operative complications compared with conventional external orbital surgery. The decision to enlarge the orbital space should be considered carefully with close co-operation between ophthalmologists and ENT surgeons.

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