CASE REPORT

Aqueous misdirection in thyroid eye disease

Devjyoti Tripathy,¹ Aparna Rao,² Aniruddha Banerjee,³ Debananda Padhy⁴¹Oculoplasty Services, LV Prasad Institute, Bhubaneswar, Odisha, India²Glaucoma Service, LV Prasad Eye Institute, Bhubaneswar, Odisha, India³Diagnostic service, LV Prasad Eye Institute, Bhubaneswar, Odisha, India⁴Glaucoma diagnostic Service, LV Prasad Eye Institute, Bhubaneswar**Correspondence to**
Dr Aparna Rao,
aparna@lvpei.org

Accepted 2 November 2014

SUMMARY

Secondary glaucoma in thyroid eye disease (TED) is attributed either to raised episcleral venous pressure or to mechanical compression induced trabecular meshwork damage by mucopolysaccharide deposition. While the mechanism for raised intraocular pressure (IOP) in TED is largely mechanical and is often evident during its active stage, our case presented with aqueous misdirection in the left eye during the inactive stage. Appropriate diagnostic tests should be ordered to diagnose cause of raised IOP, which would help determine appropriate treatment for the cause for secondary glaucoma in TED.

BACKGROUND

Causes of glaucoma in thyroid eye disease (TED) are multiple, though a majority result from mucopolysaccharide accumulation and mechanical compression of the trabecular meshwork. Other causes include secondary angle closure or raised episcleral venous pressure. Malignant glaucoma is a condition whereby anterior rotation of the ciliary proves results in aqueous misdirection causing raised intraocular pressure (IOP) postiridectomy. Although this condition is common in inflammatory conditions, its occurrence in TED is not known.

CASE PRESENTATION

A 58-year-old man was referred for a glaucoma consult for raised IOP in the left eye. On examination, his best corrected visual acuity (BCVA) was no

light perception and finger counting close to face, with inaccurate projection of rays in two quadrants in the right and left eye, respectively. Applanation IOP by Goldman applanation tonometry was 15 mm Hg in the right eye and 40 mm Hg in the left eye. The patient had been on oral antithyroid medication for Graves' disease off and on for the past 7 years. He had undergone cataract surgery in the right eye with loss of vision 2 years prior. A thyroid function test showed euthyroid status. The left eye had a proptosis of 9 mm on Hertel's exophthalmometry with eyelid retraction and ocular motility restriction. Clinical activity score¹ was two suggesting inactive TED.

INVESTIGATIONS

Imaging of the orbits showed thickened extraocular muscle bellies on the left side, suggestive of TED (figure 1A). Peripheral anterior chamber shallowing was seen in the right eye. A mid-dilated fixed pupil, axial and peripheral anterior chamber shallowing and an immature cataract were seen in the left eye. Gonioscopy showed closed angles in all quadrants in both eyes. Fundus evaluation revealed total optic atrophy in the right and advanced glaucomatous cupping with advanced visual field loss in the left eye, respectively.

DIFFERENTIAL DIAGNOSIS

Closed angles and advanced disc damage suggested missed primary angle closure glaucoma. On a provisional diagnosis of chronic angle closure

Figure 1 Composite illustrating (A) CT scan of the orbits at presentation depicting enlarged extraocular muscle bellies on the left side (asterisk); (B) slit-lamp picture of the left eye with a patent laser iridotomy (arrow) and a mid-dilated, fixed pupil with an immature cataract; (C) the axial shallowing (short arrow) and the peripheral shallowing (long arrow) of the anterior chamber; ultrasound biomicroscopy showing absence of the iridociliary sulcus (D) (arrow) with anterior rotation of the ciliary processes (E) (arrow).

To cite: Tripathy D, Rao A, Banerjee A, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-205824

Figure 2 Composite illustrating slit-lamp picture of the left eye with a functional bleb (arrow) superiorly 2 weeks after surgery (A) with deepening of the anterior chamber both axially (short arrow) as well as peripherally (long arrow) (B).

glaucoma in the left eye, a laser Nd:YAG iridotomy was performed followed by topical antiglaucoma medications (brimonidine and dorzolamide). A week later, the IOP in the left eye measured 38 mm Hg while on topical medication. The iridotomy was patent (figure 1B) but the axial and peripheral anterior chamber shallowing persisted (figure 1C). Ultrasound biomicroscopy (UBM) showed a loss of the iridociliary sulcus with anterior rotation of the ciliary processes (figure 1D, E) suggesting aqueous misdirection.

TREATMENT

Phacoemulsification with trabeculectomy and minimal pars plana vitrectomy with intraocular lens implantation was performed. The patient's thyroid profile remained euthyroid throughout the period.

OUTCOME AND FOLLOW-UP

Two weeks postoperatively, BCVA in the left eye was 20/80 with an IOP of 12 mm Hg. A functional bleb and a uniformly deep anterior chamber were evident (figure 2A, B). There was no recurrence of aqueous misdirection on topical cycloplegics at 1 year of follow-up.

DISCUSSION

Secondary glaucoma in TED is attributed to raised episcleral venous pressure or due to mechanical compression by mucopolysaccharide causing trabecular meshwork damage.¹⁻⁴ In our case, angle closure glaucoma was missed in the right eye while the left eye had aqueous misdirection confirmed on UBM despite euthyroid status, which has not been reported earlier, to the best of our knowledge.

Uveal effusions are known in angle closure glaucoma causing similar symptoms;⁵ yet no effusion was evident on UBM. We believe that chronic orbitopathy, oedematous tissues and vascular congestion in an eye with closed angles had caused sudden precipitation of aqueous misdirection with acute rise in IOP. Lack of evident active orbitopathy in this case ruled out inflammation and compressive optic neuropathy.

Although core vitrectomy is ideal, we could perform only minimal vitrectomy during the same procedure due to the hypotensive globe after trabeculectomy.⁶ Still, there was no recurrence following the surgery with deep anterior chambers. The difference in anterior chamber depth in both eyes and axial shallowing in the left eye with deepening after minimal vitrectomy ruled out chronic angle closure as the sole mechanism for raised IOP in our case. Possibly, chronic tissue oedema as a result of indolent inflammation or orbitopathy causes ciliary body swelling and forward rotation despite euthyroid status. Such possibilities should be kept in mind for appropriate clinical and/or surgical decisions in cases of TED with secondary glaucoma.

Patient's perspective

I was distressed that diagnosis of angle closure had been missed though I was on regular endocrinology follow-up, which resulted in blindness in one eye when I sought help for decreased vision. Timely management could have prevented me from losing my useful vision though surgery has helped restore some functional vision for my daily activities. I am now currently independent in my daily activities but still feel I have lost a lot of useful vision due to lack of early diagnosis of angle closure disease despite prior regular endocrinology follow-up.

Learning points

- ▶ Appropriate cause of secondary glaucoma should be sought with tailored investigations in thyroid eye disease.
- ▶ Proper eye examination is a must for all patients with thyroid eye disease to prevent blinding complications such as glaucoma, which may be missed by an endocrinologist.
- ▶ Anterior rotation of ciliary process and aqueous misdirection may be caused by low-grade chronic inflammation in thyroid eye disease, which may be diagnosed by ultrasound biomicroscopy.
- ▶ Limited vitrectomy and topical cycloplegics may be useful for resolution of the disease and also in preventing recurrence.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Manor RS, Kurz O, Lweitus Z. Intraocular pressure in endocrinological patients with exophthalmos. *Ophthalmologica* 1974;168:241-52.
- 2 Agapitos PJ, Hart IR. Long-term follow-up of ophthalmic Graves' disease. *Can Med Assoc J* 1987;136:369-72.
- 3 Goldberg I. Thyroid eye disease and glaucoma. *J Glaucoma* 2003;12:494-6.
- 4 Bartalena L, Pinchera A, Marcocci C. Management of Graves' ophthalmopathy: reality and perspectives. *Endocr Rev* 2000;21:168-99.
- 5 Kumar RS, Quek D, Lee KY, et al. Confirmation of the presence of uveal effusion in Asian eyes with primary angle closure glaucoma: an ultrasound biomicroscopy study. *Arch Ophthalmol* 2008;126:1647-51.
- 6 Debrouwer V, Stalmans P, Van Calster J, et al. Outcomes of different management options for malignant glaucoma: a retrospective study. *Graefes Arch Clin Exp Ophthalmol* 2012;250:131-41.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow