CASE REPORT

Post-strabismus surgery aqueous misdirection syndrome

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SUMMARY

A 24-year-old man was referred to the glaucoma clinic of our tertiary eye care centre in view of uncontrolled intraocular pressure (IOP) in the left eye despite maximal medication. The patient had undergone left eye cosmetic squint surgery 1 month before (medial rectus resection 9 mm and lateral rectus recession 12 mm) for congenital third nerve palsy. Post-squint surgery, the patient developed pain and was being managed symptomatically. However, 1 week later, he developed diffuse corneal oedema and severe pain, and was readmitted for management in the same hospital. He presented to our centre with an IOP of 16 mm Hg in the right eye and 58 mm Hg in the left eye. We made a diagnosis of left eye post-strabismus surgery aqueous misdirection syndrome, and performed left eye core vitrectomy with 360° goniosynechialysis and ultimately a trabeculectomy to reduce IOP.

BACKGROUND

Aqueous misdirection, or malignant glaucoma, is a rare type of secondary angle-closure glaucoma most commonly encountered after filtering surgery, but it can also occur spontaneously. It remains a difficult clinical problem in terms of diagnosis and management.¹ Early recognition and management may be sight saving. We report a rare case of post-strabismus surgery malignant glaucoma.

CASE PRESENTATION

A 24-year-old man was referred to our glaucoma clinic with uncontrolled intraocular pressure (IOP) on maximal antiglaucoma medication in the left eye (LE). The patient had undergone LE cosmetic squint surgery 1 month before (medial rectus resection 9 mm and lateral rectus recession 12 mm) for congenital third nerve palsy. Post-squint surgery, he developed pain and was being managed symptomatically in the same hospital. However, 1 week later, he developed diffuse corneal oedema. A very high IOP was noted and the patient was started on topical hypotensive medications (eye drop (e/d) timolol 0.5% and brimonidine 0.15%) and tablet acetazolamide 500 mg three times per day, and referred to our tertiary eye care centre. The patient presented to our centre 1 month after strabismus surgery with an IOP of 16 mm Hg in the right eye (RE) and 58 mm Hg in the LE. Best-corrected visual acuity was 20/20 in RE and light perception only, in LE. The right and left central corneal thicknesses were 539 and 695 µm, respectively. Ocular examination of the RE was unremarkable with a cup-disc ratio of 0.3:1 and a healthy neuroretinal

Figure 1 Clinical photograph showing corneal oedema, Descemet's folds and iridocorneal apposition.

rim along with open angles on gonioscopy. The LE had a residual exotropia and a mild lid oedema. The patient had a watery discharge and circumciliary congestion. Corneal oedema, Descemet's folds, epithelial bullae along with a large epithelial defect measuring 5×6 mm were noted ([figure 1](#)). The sclera showed thinning with uveal incarceration, 7 mm from the limbus ([figure 2](#)). The pupil was fixed and dilated (8 mm). Once the corneal oedema subsided and iris details were seen, no neovascularisation of the iris was observed. The peripheral anterior chamber (AC) was flat with iris adherent to the corneal endothelium (iridocorneal apposition); however, the central AC was deep and an anterior rotation of ciliary processes was noted on ultrasound biomicroscopy (UBM, [figure 3](#)). Owing to media haze, the fundus was hazily seen and revealed a vertically oval disc with a cup-disc ratio of 0.8:1 along with temporal disc pallor.

Figure 2 Clinical photograph showing scleral thinning with uveal tissue incarceration 7 mm from the limbus.

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Figure 3 Ultrasound biomicroscopy showing a flat peripheral anterior chamber (AC) (due to 360° iridocorneal apposition) with a deep central AC and an anterior rotation of ciliary processes.

INVESTIGATIONS

Ultrasonography (USG) was anechoic, with no evidence of haemorrhage or choroidal effusion. High-frequency UBM showed total iridocorneal apposition with a deep central AC of 3.58 mm depth and an anterior rotation of ciliary processes.

DIFFERENTIAL DIAGNOSIS

Suprachoroidal haemorrhage and choroidal effusion were the two primary differential diagnosis, which were ruled out after performing USG and UBM (described in investigations). There was no iris bombe suggestive of a pupillary block, in fact, the iris was adherent to the corneal endothelium and the central AC depth was greater than would be expected in a pupillary block.

TREATMENT

The patient was admitted at our centre and started on eye ointment 1% atropine three times per day and maximal hypotensive therapy (intravenous mannitol, tablet acetazolamide 500 mg once daily, syrup glycerol 30 mL three times per day, e/d timolol 0.5% two times per day and e/d brimonidine 0.15% two times per day). Topical steroids were also added to reduce the inflammation. The IOP on these medications lowered to 34 mm Hg. A LE core vitrectomy and 360° goniosynechialysis was performed to form the AC. The postoperative IOP over the first 3 days was 6–8 mm Hg. However, on the fourth and fifth day, the IOP in the LE was 29 and 49 mm Hg, respectively, and maximal hypotensive medications were restarted. As the IOP was persistently raised even on maximal tolerable medical therapy, a trabeculectomy with 0.04% mitomycin C (subconjunctival for 2 min) was performed in the LE.

OUTCOME AND FOLLOW-UP

Postoperatively, the IOP was 6 mm Hg and was maintained for the first 2 weeks. At 2 months follow-up post-trabeculectomy, the IOP was 18 mm Hg in the LE on two topical ocular hypotensive medications (e/d timolol 0.5% two times per day and e/d brimonidine 0.15% two times per day). In terms of visual acuity, the patient could appreciate hand movement close to the face. The visual acuity of the patient improved from light perception only to hand movements close to face due to resolution of the corneal oedema after IOP lowering. However, he had had poor visual acuity in his LE since childhood, due to strabismic amblyopia.

DISCUSSION

The term malignant glaucoma was coined by Von Graefe² in 1869. It has been described post-trabeculectomy, glaucoma drainage implant surgery, cataract surgery, laser iridotomy,

corneal transplantation, phakic intraocular lens implantation, cyclophotocoagulation and vitrectomy.³ However, there is no report in the literature to suggest it could be a complication of squint surgery.

There are various theories for malignant glaucoma such as the posterior pooling of aqueous or ciliolenticular block or the choroidal expansion theory.^{4–6} However, the exact mechanism still remains unclear.

The sequence of events postulated by us that lead to malignant glaucoma were: a scleral perforation during strabismus surgery resulted in a shallowing of AC, that then led to iridocorneal adhesions and also an anterior movement of the peripheral hyaloid, into apposition with the ciliary body, thereby rotating the ciliary processes anteriorly. This led to direct aqueous and vitreous filling the AC and posterior chamber with no exit through the trabecular meshwork. Since there was no aqueous egress through the trabecular outflow channels, this resulted in aqueous filling the AC and expansion of the AC and posterior chamber volume,⁷ and, subsequently, became a vicious cycle. However, unlike the classical aqueous misdirection syndrome described, our case had an anterior misdirection of aqueous that pushed the lens posteriorly instead of anteriorly, ultimately leading to a raised IOP with a deep AC. Hence, this was not a classical case of posterior aqueous misdirection but, instead, the vitreous and aqueous pooled into the AC and posterior chamber leading to the high IOP.

The incidence of scleral perforations during squint surgery varies from 0.2% to 7.8%.^{8,9} Many perforations after squint surgery in the young remain unrecognised because the two major complications, retinal detachment and endophthalmitis, are relatively infrequent.¹⁰ Retinal detachment generally does not occur either because the penetration is limited to the choroid, or because the vitreous, which is usually formed in a young patient, tamponades the retina.

As there is no reported complication of either malignant glaucoma or any other form of secondary glaucoma after strabismus surgery in the literature, to the best of our knowledge, our case report highlights the rare possibility of glaucoma as sequelae to an inadvertent scleral perforation during squint surgery. We believe the pathomechanism of this case is also rare and has never been reported so far.

Learning points

- Be aware of glaucoma as a possible though rare sequela to inadvertent scleral perforation during squint surgery.
- Ultrasound biomicroscopy can help in assessing the status of the ciliary body.
- Scleral perforation during strabismus surgery should be meticulously checked before closing of conjunctiva.

Competing interests None declared.

Patient consent Obtained.

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REFERENCES

- 1 Francis BA, Wong RM, Minckler DS. Slit-lamp needle revision for aqueous misdirection after trabeculectomy. *J Glaucoma* 2002;11:183–8.
- 2 Von Graefe A. Bietrage zur Pathologie, Therapie de Glaucoma. *Arch Ophthalmol* 1869;15:108.

- 3 Shaarawy T, Sherwood M, Hitchings R, et al. *Aqueous misdirection. Text Book of Glaucoma, Surgical Management*. Vol-2 1st edn. Saunders/Elsevier. 2009.
- 4 Shaffer RN. The role of vitreous detachment in aphakic and malignant glaucoma. *Trans Am Acad Ophthalmol Otolaryngol* 1954;58:217–31.
- 5 Weiss DI, Shaffer RN. Ciliary block (malignant) glaucoma. *Trans Am Acad Ophthalmol Otolaryngol* 1972;76:450–61.
- 6 Quigley HA, Friedman DS, Congdon NG. Possible mechanisms of primary angle-closure and malignant glaucoma. *J Glaucoma* 2003;12:167–80.
- 7 Epstein DL. The *malignant glaucoma syndrome*. *Chandler's and Grant's glaucoma*. Williams & Wilkins, 1997:285–303.
- 8 Morris RJ, Rosen PH, Fells P. Incidence of inadvertent globe perforation during strabismus surgery. *Br J Ophthalmol* 1990;74:490–3.
- 9 Dang Y, Racu C, Isenberg SJ. Scleral penetrations and perforations in strabismus surgery and associated risk factors. *J AAPOS* 2004;8:325–31.
- 10 Simon JW. Complications of strabismus surgery. *Curr Opin Ophthalmol* 2010;21:361–6.

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