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Treatment of aphakic malignant glaucoma using Nd:YAG laser posterior capsulotomy

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Malignant glaucoma is the term introduced in 1869 by von Graefe¹ to describe one of the more serious but rare complications of anterior segment surgery. It is best known following trabeculectomy but has been reported after a wide variety of anterior segment procedures.

It is notoriously refractory to medical treatment alone^{2,3} and surgical intervention has had only limited success.^{4,5} An additional treatment option in pseudophakic eyes or aphakic eyes with an intact posterior capsule is that of short pulsed Nd:YAG laser posterior capsulotomy which is minimally invasive, carries little risk and can re-establish forward flow of posteriorly misdirected aqueous through into the drainage angle of the anterior chamber.^{2,6} Successful use of Nd:YAG posterior capsulotomy can obviate the need for further surgical intervention.

A case is reported of malignant glaucoma developing post-trabeculectomy which persisted despite surgical treatment. Long term pressure control was established following Nd:YAG posterior capsulotomy.

Case report

A 69-year-old woman presented in 1987 with ocular hypertension and narrow drainage angles for which topical β blocker (timolol drops 0.25%) treatment was required the following year. Intraocular pressure (IOP) control was unsatisfactory and unresponsive to bilateral 360 degree argon laser trabeculoplasty. Adjunctive topical treatment using an adrenergic agonist (dipivefrin drops) did not improve IOP control (30 mm Hg bilaterally) and automated perimetry revealed an early arcuate scotoma in her right eye. A right trabeculectomy was performed in September 1992.

On the first postoperative day she had diffuse shallowing of the anterior chamber with an IOP

of 38 mm Hg and a diagnosis of malignant glaucoma was made. The following day a limited pars plana core vitrectomy was performed and the anterior chamber reformed internally through a surgical cyclodialysis. On the first postoperative day a deep anterior chamber was present with a normal IOP. Two days later a total hyphaema was present together with a peripherally flat anterior chamber and raised IOP. Maximum topical and systemic treatment reduced the IOP to 25 mm Hg. The hyphaema slowly resorbed over the following 2 weeks to about 30% of the corneal diameter, but the malignant glaucoma persisted despite full cycloplegia. At this stage she was referred for further management.

Her right IOP was 18 mm Hg on full medical treatment and her acuity 6/12 with a healthy disc. There was marked diffuse conjunctival and episcleral hyperaemia and the anterior chamber depth measured 1.6 mm centrally (Haag-Streit AC depth meter) but was peripherally closed and no drainage bleb was present overlying the trabeculectomy site. Over the following 4 days the IOP remained under 20 mm Hg on topical and systemic treatment and the inflammation settled on treatment with topical steroid although the anterior chamber remained peripherally flat.

B scan ultrasound showed ciliary body enlargement in the right eye but not in the unoperated left eye. The axial length of the right eye was difficult to measure accurately but that of the left eye was 21.1 mm. Her preoperative refraction was not hypermetropic and neither was there anisometropia in her reading prescription.

She was discharged and reviewed 2 weeks later when the anterior chamber was still flat with an IOP of 30 mm Hg and an optically clear zone was noted behind her lens, presumed to be seques-

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Figure 1 Flat anterior chamber with posterior capsule herniating through capsulorrhexis.

tered aqueous. Her corrected acuity remained good at 6/9.

She was then booked for further pars plana vitrectomy and extracapsular lens extraction without intraocular lens (IOL). At operation a corneal groove was cut and a 27 gauge anterior chamber paracentesis made tangentially in the peripheral cornea. A single port pars plana Ocutome vitrectomy was carried out and the anterior chamber inflated through the paracentesis with viscoelastic. The corneal section was completed followed by enlargement of the peripheral iridectomy to a broad iridectomy which enabled a large continuous tear needle capsulorrhexis to be fashioned. Following hydrodissection the nucleus was expressed and, after aspiration of the residual cortical lens matter, the section was closed with 10/0 nylon.

On the first postoperative day the anterior chamber had flattened and the vitreous was herniating the posterior capsule through the capsulorrhexis, forming a 'dome' whose apex was almost in contact with the corneal endothelium (Fig 1). The IOP was 10 mm Hg and there was no leak through the cataract section



Figure 2 Deep anterior chamber immediately following Nd:YAG posterior capsulotomy.

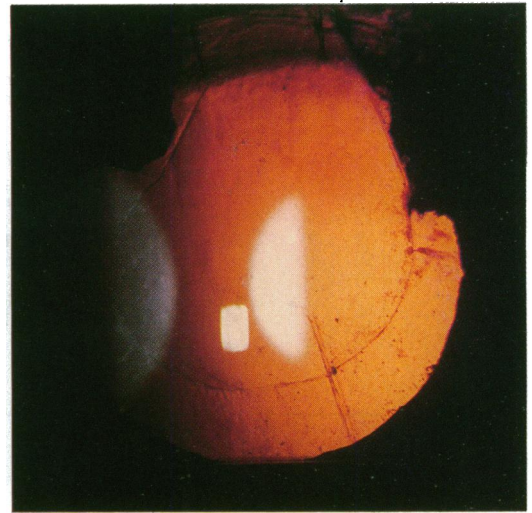


Figure 3 Retroillumination before Nd:YAG laser posterior capsulotomy.

and no conjunctival bleb. The early recurrence of malignant glaucoma was diagnosed. A Nd:YAG posterior capsulotomy combined with anterior vitreolysis was performed the following day resulting in an instantaneous forward gush of fluid accompanied by simultaneous deepening of the anterior chamber (Fig 2). Retroillumination highlights the small central laser capsulotomy (pre-treatment (Fig 3) and post-treatment (Fig 4)).

Over a 7 month follow up period the anterior chamber has remained deep and the IOP normal and stable in the absence of a trabeculotomy bleb and, surprisingly, no antiglaucoma medication. The normal IOP despite no treatment may well be due to the existence of an alternative outflow pathway via the surgical cyclodialysis. The corrected acuity is 6/9.

Comment

Von Graefe¹ introduced the term 'malignant glaucoma' in 1869 to describe raised intraocular pressure associated with a flat anterior chamber following uneventful surgery for angle closure

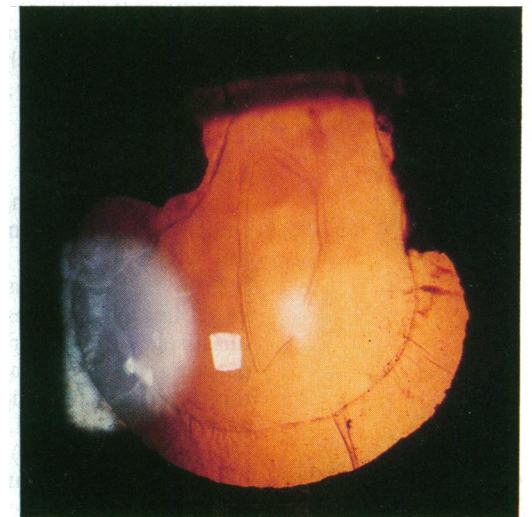


Figure 4 Retroillumination immediately following Nd:YAG laser posterior capsulotomy: the capsulotomy forms a central vertical ellipse.

glaucoma. It is now recognised to comprise the diagnostic triad of a diffusely flat anterior chamber, high intraocular pressure, and aqueous pooling that is sometimes visible in or in front of the anterior vitreous.

Malignant glaucoma is notoriously difficult to treat and carries a generally poor prognosis for long term control of intraocular pressure.

It is thought to involve the mechanisms of ciliolenticular block of aqueous flow leading to the misdirection of aqueous posteriorly into, or in front of, the vitreous gel leading to the characteristic diffuse shallowing of the anterior chamber accompanied by an often precipitous rise in intraocular pressure. Medical treatment alone is rarely successful in establishing control of the IOP.^{2,3} Pars plana vitrectomy has been used in the surgical management of malignant glaucoma with some definite but limited success in phakic as well as pseudophakic eyes.^{4,5}

However, when malignant glaucoma develops in pseudophakic eyes (with posterior chamber IOLs) there exists an additional treatment option

of Nd:YAG laser capsulotomy and/or vitreolysis^{2,6} which can be effective in arresting the spiralling rise in IOP by redirecting aqueous flow anteriorly thereby avoiding the need for acute surgical intervention with its inevitably higher morbidity. The same reasoning can be applied to aphakic eyes that have an intact posterior capsule, with similar chances of a successful outcome.

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Nasolacrimal obstruction and facial bone histopathology in craniodiaphyseal dysplasia

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Craniodiaphyseal dysplasia is a rare, severe, and progressive bone dysplasia characterised by thickening, distortion, and enlargement of the cranium and face.^{1,2} Epiphora and recurrent dacrycystitis can occur in this and other such bone diseases owing to nasolacrimal duct stenosis.^{3,4} The management of such a case provided an opportunity to obtain material for analysis, with the aim of gaining more information on the aetiology and possible treatment of this condition.

Case report

The patient was a female, aged 3 years in whom a diagnosis of craniodiaphyseal dysplasia had been

made soon after birth, from the typical clinical (Fig 1A) and radiological appearances (Fig 1B). A low calcium diet had been commenced and calcitriol therapy administered in an attempt to

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Figure 1 (A) Clinical appearance of a patient with craniodiaphyseal dysplasia. Note the paranasal bossing and hypertelorism, which has arisen as a result of the underlying bony abnormality. (B) Radiograph of the skull of the patient shown in Figure 1. Hyperostosis and sclerosis of the facial skeleton resulted in left nasolacrimal obstruction.



Fig 1A



Fig 1B