Figure 2 All vitreoretinal operations performed in Cologne in the years 1988 (n = 692) and 2003 (n = 813).

Comment

In summary, our data do not confirm the subjective impression of an improved rate of PVR in rhegmatogenous detachment in 2003 compared to 1988. The impression that PVR today is a decreasing problem may be explained by proportionally fewer interventions for PVR. The total number of operations and the number of new indications for surgery have grown, while ocular trauma and its PVR complication decreased.

Our results are supported by the SPR study,⁴ showing that primary vitrectomy did not reduce the risk of PVR over buckling. Our study is restricted by the limitations of a retrospective study. We also did not distinguish between phakic and pseudophakic eyes.

S Joeres, B Kirchhof, A M Jousen

Department of Vitreoretinal Surgery, Center for Ophthalmology, University of Cologne, Cologne, Germany

Correspondence to: Antonia M Jousen, MD, PhD, Department of Vitreoretinal Surgery, Center of Ophthalmology, Kerpener Strasse 62, 50931 Köln, Germany; jousena@aol.com

doi: 10.1136/bjo.2005.088856

Accepted for publication 1 February 2006

References

- Heimann H, Zou X, Jandek C, et al. Primary vitrectomy for rhegmatogenous retinal detachment: an analysis of 512 cases. *Graefes Arch Clin Exp Ophthalmol* 2005;26:1-10.
- Cardillo JA, Stout JT, LaBree L, et al. Post-traumatic proliferative vitreoretinopathy. The epidemiologic profile, onset, risk factors, and visual outcome. *Ophthalmology* 1997;104:1166-73.
- Kirchhof B. Strategies to influence PVR development. *Graefes Arch Clin Exp Ophthalmol* 2004;242:699-703.
- SPR Study Group. Oral presentation at the SOE Meeting Berlin, September 2005. (Publication forthcoming).

Pupillary block glaucoma in phakic perfluoropropane gas filled eye

Gas is widely used in vitreoretinal surgery. It is a valuable tool to treat difficult retinal detachments and severe cases of proliferative vitreous retinopathy.¹ Gas mediated pupillary block and iridocorneal apposition are known complications in aphakic eyes that underwent vitreoretinal surgery.²⁻⁴ We report a case of a phakic patient with an acute pupillary block glaucoma related to the migration of perfluoropropane (C_3F_8) into the anterior chamber. We are unaware of previous reports on this complication, and a Medline search found no literature on this topic.

Case report

A 46 year old man underwent a pars plana vitrectomy with 15% C_3F_8 (perfluoropropane) injection to his right eye for a macula on recurrent rhegmatogenous retinal detachment. There was no history of trauma. During discharge on the first postoperative day, the anterior chamber was deep and there were no signs of pseudoexfoliation, lental

dislocation, or zonule defects. Intraocular pressure (IOP) was 19 mm Hg. Five days later, the patient came to the outpatient department with acute pupillary block glaucoma secondary to a gas bubble between the iris and crystalline lens causing iridocorneal apposition (fig 1A). The IOP was 37 mm Hg. Topical and systemic antiglaucoma medications were started. The patient was maintained in a strict prone position. Two hours later, the gas bubble reduced in size because most of the bubble moved behind the crystalline lens (fig 1B). However, the IOP remained high and the iridocorneal apposition did not break down. An inferior Nd:YAG laser peripheral iridotomy at 6 o'clock was performed. It resulted in the immediate deepening of the anterior chamber and reduction in IOP to 16 mm Hg with no recurrence of the pupillary attack (fig 2). Six weeks after surgery, the anterior chamber was deep and the IOP was 17 mm Hg with no antiglaucoma medications.

Comment

Complete iridocorneal apposition is a known complication in aphakic eyes that undergo vitrectomy followed by intraocular gas injection.²⁻⁵ The incidence of this condition was estimated to be 3% in a retrospective study of 336 aphakic eyes.³ Buoyant forces from the bubble and pupillary block combine to push the iris forward onto the posterior surface of the cornea. This results in complete iridocorneal apposition. To the best of our knowledge, we report the first case of a phakic patient who developed an acute pupillary block glaucoma related to migration of C_3F_8 into the anterior chamber.

Zonulysis is the pathological change that allows a migration from the posterior into the anterior chamber. Such changes are usually seen after trauma or in cases of weak zonules such as in pseudoexfoliation. Our patient did not report any trauma nor did he have pseudoexfoliation. However, the superior part of the zonules might have been compromised by previous retinal surgery.

Initially, our patient maintained a good prone positioning and encountered no problems; however, as he did not keep to the position, this probably resulted in the pupillary block by migration of C_3F_8 into the anterior chamber, thus pushing the iris diaphragm anteriorly. In our patient, Nd:YAG laser iridotomy at 6 o'clock was sufficient to treat the pupillary block. A permanent iridocorneal apposition did not occur because of early recognition and intervention.

Figure 1 (A) Clinical photograph demonstrates C_3F_8 gas bubble blocking the pupil. Note the complete iridocorneal apposition. (B) Clinical photograph demonstrates the remnant of the gas bubble after 2 hours from strict face down position. Complete iridocorneal apposition still exists.

Figure 2 Clinical photograph demonstrates a formed anterior chamber after Nd:YAG-laser peripheral iridotomy (white arrow).

In conclusion, C_3F_8 migration into the anterior chamber, inducing a pupillary block glaucoma is a complication that has not been reported in phakic eyes. An early Nd:YAG laser iridotomy is a suitable way to reduce IOP in such cases.

S Al-Shahwan, F Ghadhfan

Glaucoma Division, Department of Ophthalmology, King Khaled Eye Specialist Hospital, Riyadh, Saudi Arabia

H Kalantan, A A Al-Torbak

Anterior Segment Division, Department of Ophthalmology, King Khaled Eye Specialist Hospital, Riyadh, Saudi Arabia

Correspondence to: Abdullah Al-Torbak, MD, FRCS, King Khaled Eye Specialist Hospital, PO Box 7191, Riyadh 11462, Saudi Arabia; atorbaq@health.net.sa

doi: 10.1136/bjo.2006.091918

Accepted for publication 5 February 2006

Did not receive any financial or material support to conduct this study.

The authors have no conflicts of interest or proprietary interest in any of the instruments or topics presented in this manuscript.

References

- 1 Chang S. Intraocular gases. In: Ryan SJ, Wilkinson CP, eds. *Retina*, 3rd ed. St Louis: Mosby, 2001:2417–61.
- 2 Han DP, Lewis H, Williams GA. Management of complete iridocorneal apposition after vitrectomy. *Am J Ophthalmol* 1987;103:108–9.
- 3 Konishi K, Kondo H, Oshima K. Iridocorneal apposition after vitrectomy and gas injection. *Retina* 2000;20:549–50.
- 4 Kumar A, Kedar S, Garodia VK, et al. Angle closure glaucoma following pupillary block in an aphakic perfluoropropane gas-filled eye. *Indian J Ophthalmol* 2002;50:220–1.
- 5 Michels RG, Wilkinson CP, Rice TA. Postoperative management. In: *Retinal detachment*. St Louis: Mosby, 1990:889–915.

Acute retinal pigment epitheliitis associated with intravenous bisphosphonate

Zoledronate is a newer bisphosphonate, primarily used for the treatment of tumour induced hypercalcaemia and skeletal related events in advanced bone malignancy. Bisphosphonates have been associated with a number of ocular side effects including conjunctivitis, episcleritis, scleritis, uveitis, and visual blurring.¹ We report a case of acute retinal pigment epitheliitis following the use of intravenous zoledronate. To our knowledge bisphosphonates have not

previously been associated with acute retinal pigment epitheliitis and could find no reference to it in computerised search utilising Medline and Pubmed.

Case report

A 47 year old glazier noted visual blurring and metamorphopsia within a day following intravenous infusion of zoledronate. One month after the infusion and onset of symptoms, his best corrected visual acuity was 6/5 in each eye. There was no vitritis or anterior uveitis. Fundus examination showed subtle clumps of hyperpigmentation surrounded by a paler halo of hypopigmentation in discrete clusters around both maculas at the level of the retinal pigment epithelium (RPE), more visible in the left eye (fig 1). The Amsler grid showed metamorphopsia corresponding to the area of RPE disturbance. The optic discs, retinal vessels, and surrounding retina were normal. Optical coherence tomography was normal. Fundus fluorescein angiography revealed an area of hypofluorescence corresponding to the area of hyperpigmentation, surrounded by an area of hyperfluorescence in the early phase and in the arteriovenous phase (fig 2), while fading in the late phase, with no leakage (fig 3). A detailed history, systems review, and comprehensive investigations were negative.

Comment

Acute retinal pigment epitheliitis (ARPE) or Krill's disease was first described by Krill and Deutman as a benign, acute self limiting inflammatory disorder of the RPE without systemic association, with reduced visual acuity, metamorphopsia, and subtle changes at the level of the RPE.² Clusters of lesions with hyperpigmented centres, surrounded by paler halos, at the posterior pole have been reported as early as 1 month after the onset of symptoms.³ These lesions become less

Figure 1 Colour fundus photograph (A) right eye, (B) left eye. These show subtle clumps of hyperpigmentation surrounded by a paler halo of hypopigmentation in discrete clusters around both maculas at the level of the retinal pigment epithelium, more visible in the left eye.

Figure 2 Fundus fluorescein angiogram, arteriovenous phase (A) right eye, (B) left eye, showing areas of hypofluorescence corresponding to the area of hyperpigmentation, surrounded by an area of hyperfluorescence.

distinct with time leaving an area of subtle pigment mottling. Although most recover over several months Chittum and Kalina describe one case with a persistent small scotoma.³

Acute retinal pigment epitheliitis is thought to represent areas of RPE inflammation with breakdown of the pigment epithelial barrier and subsequent leakage of serous

Figure 3 Fundus fluorescein angiogram, late phase (A) right eye, (B) left eye, illustrating fading hyperfluorescence with no leaking.