FHU has been described in patients with RP. Recently, Chowers *et al*¹ showed that patients with RP can develop autoimmune reactions to anterior chamber antigens which bear some resemblance to retinal antigens, leading to the clinical manifestation of FHU.

It is well recognised that autoimmune reactions to retinal S antigen, a rod outer segment protein, have been implicated in the pathogenesis of various forms of uveitis and hereditary retinal degeneration. In addition, another report has demonstrated that some patients with RP have circulating B cells reactive with the retinal antigens, especially S antigen.⁶ These suggest that the tendency for immune reactions in patients with RP could increase their susceptibility to develop FHU. It is important for clinicians to recognise that although it is extremely rare, bilateral FHU can be found in association with RP. This in turn may influence the management of complications such as low-grade uveitis in this select group of patients.

References

- 1 Chowers I, Zamir E, Banin E, Merin S. Retinitis pigmentosa associated with Fuchs' heterochromic uveitis. *Arch Ophthalmol* 2000; **118**: 800–802.
- 2 François J, Mastilovic B. L'hétérochromie de Fuchs associée aux hérédo-dégénérescences chorioétiniennes. Ann Oculist 1961; 194: 385–396.
- 3 van den Born LI, van Schooneveld MJ, de Jong PT, Bleeker-Wagemakers EM *et al.* Fuchs' heterochromic uveitis associated with retinitis pigmentosa in a father and son. *Br J Ophthalmol* 1994; **78**: 504–505.
- 4 Vuorre I, Saari M, Tiilikainen A, Rasanen O *et al*. Fuchs' heterochromic cyclitis associated with retinitis pigmentosa: a family study. *Can J Ophthalmol* 1979; **14**: 10–16.
- 5 Jay M. On the heredity of retinitis pigmentosa. Br J Ophthalmol 1982; 66: 405–416.
- 6 Reid DM, Campbell AM, Forrester JV. EB-virus transformed human lymphocytes from uveitis and retinitis pigmentosa patients secrete antibodies to retinal antigens. *J Clin Lab Immunol* 1988; 26: 107–111.

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Delayed acute angle closure after macular-hole surgery *Eye* (2003) **17,** 779–781. doi:10.1038/sj.eye.6700458

Intraocular pressure (IOP) elevation is a common complication of pars plana vitreous surgery.¹ Acute glaucoma may occur in the postoperative period secondary to angle closure caused by surgical adjuvants such as long-acting expansile gases, silicone oil, and perfluorocarbon liquids; by postoperative inflammation, and by oedema of the ciliary body and anterior rotation of ciliary processes.¹⁻⁴ Macular-hole surgery predisposes to angle closure because of prolonged prone positioning and the routine use of cycloplegic agents postoperatively. Since surgery to close a macular hole is a relatively recent innovation, to our knowledge only two cases of angleclosure glaucoma following this surgery have been recorded.⁵ We report two further cases of acute angleclosure glaucoma following macular-hole surgery in patients with axial hypermetropia, in whom neither symptoms nor signs suggestive of angle closure existed preoperatively.

Case reports

Two hypermetropic women in their seventh and eighth decades of life were referred with a diagnosis of a unilateral macular hole (detailed clinical characteristics are summarized in Table 1). They had no past ocular or medical history of note and gave no history suggestive of intermittent angle closure. After dilatation with guttae tropicamide 1% and guttae phenylephrine 2.5%, the IOP was found to be within the normal range, and they did not experience any subsequent symptoms of angle closure. Preoperative gonioscopic examination was not undertaken in these patients as there was no obvious abnormality of the anterior chamber depth. They underwent a routine vitrectomy with epiretinal membrane and internal limiting membrane peel (under indocyanine green visualisation), followed by 15% perfluoropropane gas injection.

Postoperatively, guttae dexamethasone 0.1% four times daily, guttae cyclopentolate 1% twice daily, and guttae chloramphenicol four times daily were prescribed, and they were instructed to posture face-down for 50 min every hour for 2 weeks. On the first postoperative day, the IOP was normal in both patients, with no evidence of pupillary block. The anterior chamber was formed and there was a 70–90% gas fill. At 1 week postoperatively, both patients developed an acutely painful red eye and reduced vision. Examination revealed an inflamed eye with corneal oedema and an IOP of 45 mmHg in the first

Clinical feature	Patient 1	Patient 2
Preoperative examinatio	n	
Age (years)	66	72
Sex	Female	Female
Refraction	+2.50 D BE	+3.0 D BE
Vision	6/5 RE	6/5 RE
	'Counting	6/36 LE
	fingers' LE	
IOP	18 mmHg RE,	18 mmHg BE
	17 mmHg LE	
Crystalline lens	Phakic, clear	Phakic, clear
Posterior segment	Stage 3 macular hole	Stage 4
		macular hole
Axial length	21.10 mm RE	21.95 mm RE
	20.99 mm LE	21.96 mm LE
First post-op day exami	nation	
IOP	22 mmHg LE	20 mmHg LE
Posterior segment	75% gas fill LE	90% gas fill LE
Examination at acute p	resentation (1 week postop	eratively)
Vision	Hand	Hand
	movements LE	movements LE
IOP	45 mmHg LE	70 mmHg LE
Anterior chamber	Shallow LE	Shallow LE
Gonioscopy	Angle closed LE	Angle closed LE
	Shaffer 1 RE	Shaffer 2 RE
Posterior segment	30% gas fill LE	75% gas fill LE
Examination 1 week pos	st-YAG iridotomy	
IOP (mmHg)	13	20
Iridotomies	Patent	Patent
Anterior Chamber	Formed	Formed

case and 70 mm Hg in the second. The anterior chamber was noted to be shallow, and gonioscopy confirmed that the angle was closed in the operated eye and narrow but open in the fellow eye. In one case there was 30% gas fill and in the other about 75%. Prone positioning was stopped, the IOP was controlled with medical therapy, and bilateral peripheral YAG laser iridotomies were performed. After 1 week, the patients were asymptomatic and the IOP was normal.

Comment

In a hypermetropic phakic eye with a short axial length, and a relatively large lens, prone positioning shifts the lens-iris diaphragm anteriorly, thus shallowing the anterior chamber and narrowing the angle. This is likely to happen after prolonged face-down posturing following macular-hole surgery, and the regular use of guttae cyclopentolate postoperatively provides another risk factor for acute angle closure. A 15%

perfluoropropane gas-air mixture is nonexpansile and is unlikely to have been responsible for the event as there was no gas-overfill, and the IOP was normal in both patients on the first postoperative day. Furthermore, by the time they presented with acute angle closure, the gas bubble was much smaller than in the immediate postoperative period. Resolution of the angle closure by a peripheral iridotomy rules out oedema of the ciliary body or anterior rotation of ciliary processes as being the primary causative factors.

It is evident from the above cases that the postoperative conditions following macular-hole surgery may provoke an attack of angle closure in an eye that on routine clinical evaluation may not be considered at high risk for spontaneous angle closure. All patients undergoing this surgery should be counselled about the risk of acute angle closure postoperatively. In addition, to decrease the possibility of postoperative pupillary block, gonioscopic examination should be undertaken in all hypermetropic patients who are undergoing macularhole surgery, and a prophylactic YAG laser iridotomy should be performed in those with narrow angles. This may not entirely reduce the risk of acute angle closure, as this complication has been described following macularhole surgery in an eye with a prophylactic YAG iridotomy.⁵ If the acute attack cannot be reversed then peripheral iridoplasty or goniosynechialysis could be considered.5 Combined phacoemulsification with lens implant and vitrectomy may also be an option and may be useful in some cases, as a significant proportion of these patients are likely to develop a cataract after the surgery.6,7

References

- 1 Han DP, Lewis H, Lambrou Jr FH, Mieler WF, Hartz A. Mechanism of intraocular pressure elevation after pars plana vitrectomy. Ophthalmology 1989; 96: 1357-1362.
- 2 Chang S, Lincoff HA, Coleman DJ, Fuchs W, Farber ME. Perfluorocarbon gases in vitreous surgery. Ophthalmology 1985; 92: 651-656.
- Alster Y, Ben-Nun Y, Loewenstein A, Lazar M. Pupillary block glaucoma due to residual perfluoro-decalin. Ophthalmic Surg Lasers 1996; 27(5): 395-396.
- 4 Lewis H, Han D, Williams GA. Management of fibrin pupillary-block after pars plana vitrectomy with intravitreal gas injection. Am J Ophthalmol 1987; 103: 180-182.
- Assalian A, Sebag M, Desjardins DC, Labelle PF. Successful goniosynechialysis for angle-closure glaucoma after vitreoretinal surgery. Am J Ophthalmol 2000; 130: 834-835.
- 6 Simcock PR, Scalia S. Phaco-vitrectomy for full-thickness macular holes. Acta Ophthalmol Scand 2000; 78: 684-686.
- 7 Hsuan JD, Brown NA, Bron AJ, Patel CK, Rosen PH. Posterior subcapsular and nuclear cataract after vitrectomy. J Cataract Refract Surg 2001; 27: 437-444.

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Sir,

Keratoacanthoma of the conjunctiva *Eye* (2003) **17**, 781–782. doi:10.1038/sj.eye.6700482

Cutaneous keratoacanthoma is relatively common, occurring most frequently in sun-exposed areas of Caucasians in middle life.¹ It is a rapidly growing squamo-proliferative lesion of symmetrical globular shape, ultimately developing an enlarging central keratin plug before resolving spontaneously to leave a scar. In contrast, mucosal keratoacanthoma is very rare. We report a case of conjunctival keratoacanthoma in a black patient, indistinguishable from squamous cell carcinoma clinically, which was treated with complete excision.

Case report

A 40-year-old man from Barbados was visiting relatives in this country and attended the eye emergency department with a sore and red right eye. He had noticed a lump on the nasal conjunctiva of his right eye for 2 months, which had gradually increased in size, and recently become inflamed and uncomfortable with a discharge over the preceding 2 weeks. There was no history of injury or previous ophthalmic surgery and he was systemically well.

Examination revealed a $4 \text{ mm} \times 6 \text{ mm}$ raised, soft, white, fleshy lesion on the nasal bulbar conjunctiva 1 mm from the limbus (Figure 1). The lump was mobile and there was contact bleeding from fine surface vessels. There was no lymphadenopathy and the remainder of the ophthalmic examination was normal. At the time the differential diagnosis included foreign body granuloma, infected pingueculum, and intraepithelial neoplasia/ carcinoma.

Cultures were prepared from conjunctival swabs and a topical antibiotic commenced, with no clinical improvement over the following week. The lesion was subsequently excised under local anaesthesia leaving bare sclera, and cryotherapy was applied to the



Figure 1 Anterior segment photograph showing raised, fleshy, bulbar conjunctival lesion.

conjunctival margins. The lesion was not adherent to underlying tissues.

The specimen was immediately immersed in 10% formalin, then bisected and paraffin-embedded. Histological examination demonstrated a hyperkeratotic, exophytic, and acanthotic lesion. The squamous epithelium was bulbous and well defined at the base with neutrophilic infiltrate and scattered microabscesses (Figure 2). The keratinocytes had eosinophilic/glassy cytoplasm and showed a moderate degree of atypia, with a very low mitotic rate (not exceeding three per 10 highpower fields). In the subepithelium, there was solar elastosis associated with a plasma cell-rich chronic inflammatory cell infiltrate. A diagnosis of conjunctival keratoacanthoma was made.

The conjunctiva healed and the patient was followedup for 2 months without evidence of recurrence before returning to Barbados.

Comment

Since Freeman in 1961, only a handful of cases of conjunctival keratoacanthoma have been documented, notably in white farm workers with heavy sun exposure,^{2,3} with only one previous case in a black patient.⁴ The documented cases have occurred on the bulbar conjunctiva, within the palpebral aperture and adjacent to the limbus, as in our case. These features highlight sunlight as an aetiology in these cases, and the finding of solar elastosis in the histology of this case supports this argument. Keratoacanthoma has, however, rarely affected the buccal mucosa,⁵ and a sun-related aetiology seems unlikely in this site.

Histologically, cutaneous keratoacanthoma is a heavily keratinized dermal squamous cell lesion, surrounded by acanthotic epithelium, often with an inflammatory