Eye (2006) 20, 1306–1333

www.nature.com/eye

CORRESPONDENCE

Sir,

**Sympathetic ophthalmia following severe fungal keratitis**

Sympathetic ophthalmia (SO) is thought to be an autoimmune reaction to retinal antigens exposed to conjuctival or orbital lymphatics. A study by the British Ophthalmic Surveillance Unit estimated the incidence of SO as 0.3/100 000, identifying 23 new cases in a 15 month period, with a history of ocular surgery, commonly retinal, or trauma in all cases.[1] One theory of pathogenesis suggests that concurrent infection of ocular tissue provokes an immune response involving exposed retinal antigens, thus inciting an autoimmune reaction.[2,3] SO is much more likely after traumatic globe penetration (0.19%) than intraocular surgery (0.007%),[4] and the higher incidence of concurrent infection may in part explain this difference. We present a case of SO developing after contralateral fungal infection, in the absence of penetrating trauma.

*Case report*

A 16-year-old male apprentice roofer sustained a corneal foreign body at work. He developed an aggressive Aspergillus fumigatus keratitis, which involved the limbus and required 8 weeks' intensive antifungal treatment (Figure 1). Three weeks later the sudden onset of pain heralded a central corneal perforation. Three days later the eye was eviscerated.

Eighteen days later he presented with pain, blurred vision, and photophobia in his fellow eye. Visual acuity was 6/12, with nongranulomatous anterior uveitis. Funduscopy revealed peripheral white retinal nodules (Figure 2). Histological examination of the evisceration specimen confirmed SO, with noncaseating granulomas, profound lymphocytic infiltration, numerous eosinophils in the choroid (Figure 3), and Dalen-Fuchs nodules. Treatment with high-dose oral and topical corticosteroids suppressed the intraocular inflammation. After 6 months the eye remains quiet, with a visual acuity of 6/4, using prednisolone 7.5 mg daily.

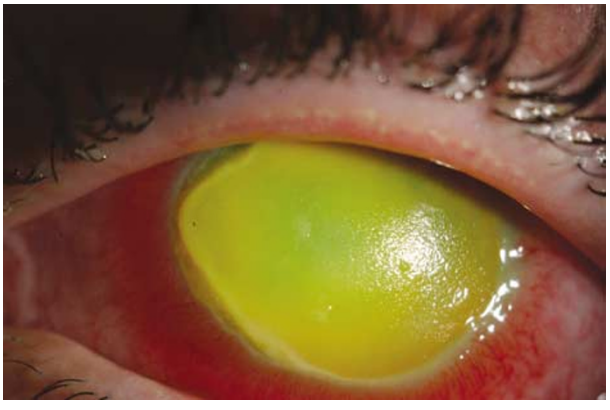

**Figure 1** Severe fungal keratitis in the left eye, involving the limbus.

**Comment**

development of SO in the absence of preceding trauma or surgery is very rare. In a series of 105 cases, Lubin *et al*[2] found that 6% followed spontaneous perforation of corneal ulcers and a further case with antecedent

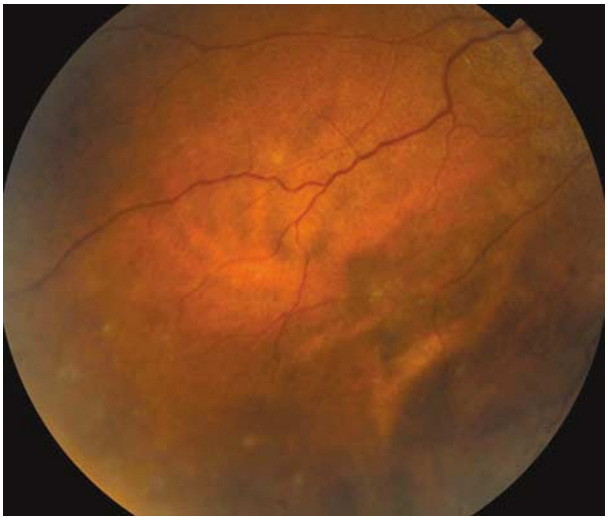

**Figure 2** The right fundal midperiphery, showing multifocal infiltrates and patchy mild retinal vasculitis.

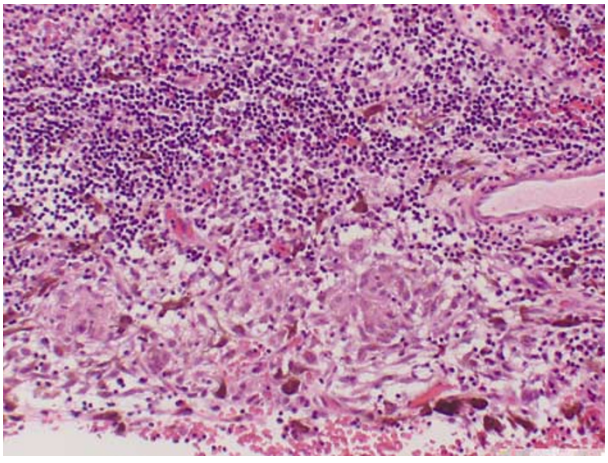

**Figure 3** Histology of left evisceration specimen. There is a profound lymphocytic infiltration of the choroid, and there are confluent noncaseating epithelioid granulomata. Eosinophils are numerous, and some epithelioid cells contain melanin granules (H&E $\times 10$ original magnification).

adherent leucoma has been described,[5] but these spontaneous perforations were precursor events. SO has been reported on several occasions following cyclodestructive procedures,[6] but almost all patients had previously undergone intraocular surgery. Malignant melanoma has been associated with SO, either primarily (albeit in association with spontaneous perforation[7]) or following irradiation and nonpenetrating surgery.[8] A blunt injury with hyphaema also led to SO.[9]

In our case, characteristic histological changes of SO were confirmed in the evisceration specimen. These changes, particularly the development of epithelioid granulomata, probably could not have occurred in the 3-day period between spontaneous perforation and evisceration, therefore preceded it.

We hypothesise that this chronic severe infection, with limbal involvement, allowed the diffusion of intraocular fungal antigens and proinflammatory mediators which allowed access through a disturbed blood-retinal barrier to expose retinal antigens and allow the development of SO. Fungal antigens may have played an adjuvant role in its development.

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Proprietary interests and funding: Nil

Prior Presentation: This case has been presented in poster format at the Fourth International Symposium on Vogt-Koyanagi-Harada Disease and Sympathetic Ophthalmia, Monaco, May 2005

*Eye* (2006) **20,** 1306–1307. doi:10.1038/sj.eye.6702156; published online 12 May 2006

Sir,

**Letter regarding correlation of retinal sensitivity measured with fundus related microperimetry to visual acuity and retinal thickness in eyes with diabetic macular oedema**

We like to congratulate Dr Okada and associates for their work on the microperimeter (MP 1). With the commercial unavailability of the SLO microperimter, MP 1 is the only alternative to study macular functions including light sensitivity threshold, fixation pattern, and stability.[1–3] Our initial study with the MP 1 on eyes with macular pathology revealed similar correlation of mean retinal sensitivity and visual acuity.[2] In another study, we found that the mean sensitivity of the macular area was approximately 18 dB in normal subject, which reduced with increasing age (unpublished data). 18 dB is higher than median sensitivity (15 dB) reported by the authors.

We would like to make certain comments regarding this study. In the methods section the testing conditions have not been described. This may affect the retinal sensitivity measured by the MP 1. If the test room is lighted, the retinal sensitivity measured could be lower than when measured in the darkroom with less interference of surrounding light during the test. The demographics of the patients have not been mentioned, especially the age. It is well known that the retinal sensitivity reduces with age in the normal subjects both by the SLO microperimeter and conventional perimetry. Whether the control normal subjects were age matched or not would affect the results and interpretation. The other reason the retinal sensitivity could be lower in this study compared to our data is the consequence of learning effect. Subjects tend to do better in subsequent field tests than the initial one due to the learning effect. In our

prospective study, we allowed every patient experience with machine in form of test stimuli before starting the test and we would only start the real test once the patient feels comfortable with the whole procedure. We feel lack of familiarity with the machine will affect the mean retinal sensitivity. The authors have not mentioned the initial level of sensitivity they used as the MP 1 allows the examiner to select this setting before test is initiated. This is important as in patients with diabetic macular oedema with mean sensitivity of 2 dB. If the test was started at 16 dB, then it would take a longer time to complete the test. The prolonged time could result in decreased patient cooperation parameters such as false-positives or fixation stability and these parameters have not been reported in this study. This data would be helpful in interpreting the reliability of the results.

The 4–2 strategy is faster but we believe when measuring the macular sensitivity 4-2-1 strategy is superior. We did not understand the rationale behind using the 12° cross for fixation. It would have helped significantly if the authors compared the retinal thickness at each quadrant surrounding the fovea and correlated the retinal sensitivity to thickness both in diabetic macular oedema and normal eyes.

We read this paper with great interest and would like to once again congratulate the authors on their important work in establishing anatomic and functional correlation in the diabetic macular oedema eyes.

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Financial Interest: None

*Eye* (2006) **20,** 1307–1308. doi:10.1038/sj.eye.6702157; published online 4 November 2005

Sir,

**Response to Shah and Chalam**

We appreciate the interest of Drs Shah and Chalam in our article and thank them for their comments. Our study was a pilot study to examine how the retinal sensitivity measured with the MP-1 correlates with other parameters, such as retinal thickness and visual acuity. From our experience, we feel that the testing conditions should be further modified especially for patients with poor visual acuity. In this study, the age of diabetic patients ranged from 25 to 76 years, and that of the controls from 42 to 76 years, as described in the Subjects and methods section. The reduced sensitivity may be due to the ages of the normal subjects. All tests were performed in a lighted room. In order to obtain more reliable data from patients with poor VA, we used a larger cross for fixation, and allow patients to learn the test. We also appreciate the other suggestions for further studies.

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*Eye* (2006) **20,** 1308. doi:10.1038/sj.eye.6702158; published online 4 November 2005

Sir,

**The subluxated lens: a patient's perspective**

There are several camera systems available to the ophthalmologist for documenting ocular conditions. However, it is difficult to document what the patient

experiences. We describe a case of subluxated lens due to Marfan's syndrome from the patient's point of view.

### *Case report*

In July 2003, a 60-year-old male was referred to the Vitreo-retinal Services of the Birmingham and Midland Eye Centre, Birmingham, UK for the management of subluxated lens in the right eye.

The patient complained of triple images when both eyes were open. The patient did not have any complaint with the right eye closed. Being a photographer, he was unable to do fine work with both eyes open. The best-corrected visual acuity was 5/60 and 6/6 in right and left eyes, respectively.

Slit-lamp examination of the right eye revealed an inferiorly subluxated lens with vitreous prolapse at the superior pupillary margin (Figure 1). The edge of the subluxated lens was close to the centre of pupil. There was iridodonesis, phacodonesis, and uneven deepening of the anterior chamber. The rest of the anterior segment examination was unremarkable. The left eye anterior examination was within normal limits. Intraocular pressure was 18 and 17 mmHg in right and left eyes, respectively. Fundus examination of both eyes was unremarkable. The patient underwent contact lens trial, but was dissatisfied. The patient underwent pars plana lensectomy, vitrectomy, and scleral-sutured lens implant for the right eye, and was very happy with the postoperative quality of vision.

The illustrations in this paper provided by the patient replicate the severely affected quality of his vision (Figure 2). He was able to create these images accurately, because he could compare with the normal image from his other eye (Figure 3). The photographic and computer skills of the patient played an important role in creating these images, which were prepared using Adobe photoshop software. (Full details of the exact computer process involved are available from the authors) (Figures 1–3).

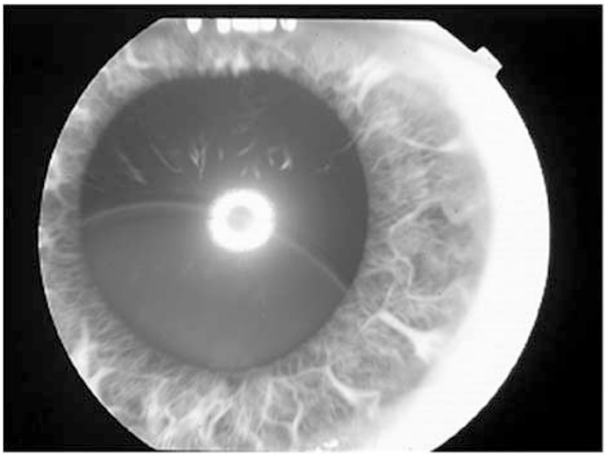

**Figure 1** Anterior segment showing subluxed lens

**Figure 2** As seen by eye with subluxated lens

**Figure 3** As seen by patient's normal eye

Previous articles have highlighted the experiences of patients during various ocular operations.[1,2] This paper illustrates the visual problems patients can have when the lens bisects the pupil. The severity of the image disturbance is much worse than we imagined from the clinical appearance and encouraged us to offer him surgery for his condition.

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*Eye* (2006) **20,** 1308–1310. doi:10.1038/sj.eye.6702161;
published online 13 January 2006

Sir,

**Trypan blue-associated retinal toxicity post complicated cataract surgery**

***Case report***

A 30-year-old Chinese female consulted for progressive blurring of vision of the right eye after sustaining a blunt injury 1 year prior. On examination, best-corrected visual acuity (BCVA) was 20/70. Pupillary responses were normal. There was an iridodialysis and subluxation of the cataractous lens. The posterior segment was unremarkable.

Trypan blue (0.06%) (VisionBlue®, DORC International, Netherlands)—enhanced phacoemulsification was performed using a temporal clear corneal approach. The anterior chamber was filled with sodium hyaluronate 3%-chondroitin sulphate 4% (Viscoat®, Alcon Ophthalmic, Fortworth, TX, USA). Trypan blue was injected directly over the anterior capsule, avoiding the area of zonulysis. However, the dye was noted to enter the vitreous cavity from the area without clinically evident zonulysis posterior to the corneal incision. Irrigation was immediately carried out with balanced salt solution to remove excess dye. The loss of red reflex made phacoemulsification and the visualization of the capsular bag very difficult. After a Cionni 1L capsular tension ring (Morcher® Cionni capsular tension ring, FCI-Ophthalmics Inc., MA, USA) was inserted and scleral fixated at the 2 O'clock position with 10–0 prolene under a scleral flap, an acrylic lens was implanted in the capsular bag. The iridodialysis was simultaneously repaired with 10–0 prolene. As there was no vitreous presentation, vitrectomy was not performed.

On the first postoperative day (POD), the visual acuity was 20/50. A grade 1 relative afferent pupillary defect (RAPD) was noted. The vitreous cavity had a moderate bluish hue that persisted until the fourth POD. (Figures 1 and 2) There was no macular oedema or other fundus lesions. Multifocal ERG (mfERG) showed reduced foveal P1 response without significant delay in implicit time (IT). Full field scotopic and photopic responses were subnormal without significant delays in IT.

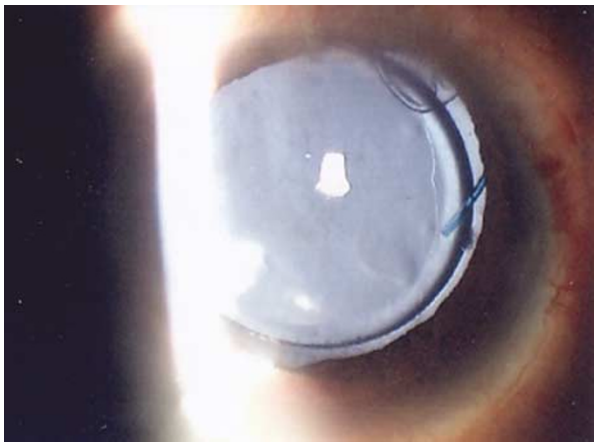

**Figure 1** Slit lamp examination on the first postoperative day shows a uniform bluish fundal glow.

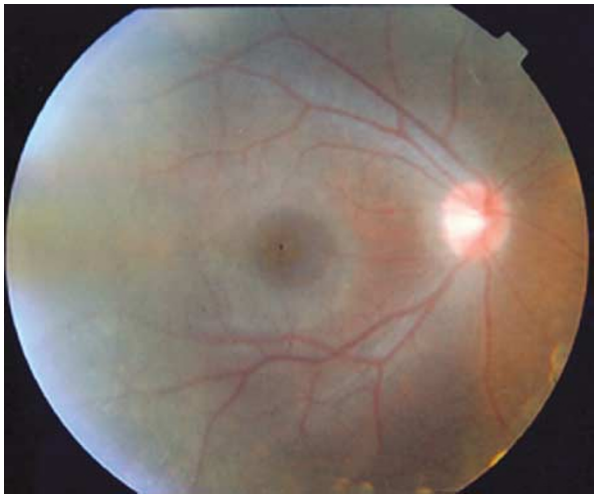

**Figure 2** Fundal examination shows a moderate bluish hue in the vitreous cavity.

At 1 month, BCVA was 20/20; the RAPD had resolved. Repeated testing showed normal photopic a-wave amplitudes and near normal photopic b-wave and 30 Hz flicker amplitudes.

**Comment**

Our patient presented with transient decrease in retinal responses post phacoemulsification, for which we suggest a multifactorial aetiology.

Inadvertent vitreous staining has been reported in cases with presumed zonular weakness post-trauma.[1,2]

In this case of post-traumatic cataract with subluxation, the dye was noted to enter the vitreous cavity through clinically evident and nonevident areas of zonular loss. Although Trypan blue at a concentration of 0.06% with exposure time of 2 min has not been found to cause retinal toxicity,[3–5] prolonged dye exposure has been reported to be toxic to the retina. In this case, the dye remained in the vitreous for 4 days thereby resulting in toxicity.

Photochemical damage from prolonged coaxial microscope light exposure could also explain the depressed mfERG foveal responses in our case. However, there were no fundus lesions typical of photochemical damage to support this diagnosis. Nevertheless, its role as a possible potentiating factor is considered.

Hence, the prolonged dye exposure, in addition to the extended surgical time, and hence, coaxial microscope light exposure, could have contributed to the transient retinal toxicity observed in our patient.

In complicated cases such as subluxated cataracts that may benefit from the use of Trypan blue, we recommend the following measures to minimize the risk of Trypan blue-associated toxicity to the retina: (1) Use lower concentrations of dye as previously described,[6,7] (2) Use dispersive ophthalmic viscoelastic devices (OVD) to seal the anterior chamber angles using the following techniques: (2a) Inject the dye onto the anterior lens surface under Viscoat®[7] (2b) the 3-step technique described by Marques *et al*,[8] or (2c) Akahoshi's soft shell stain technique.[9]

In conclusion, the use of capsular dyes is relatively safe for use in cataract surgery at low concentrations and brief exposure times. However, for complicated subluxated cataracts, it is necessary to exercise utmost caution to minimize outflow of dye into the vitreous cavity and prevent potential retinal toxicity.

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Proprietary Interests: The authors have no propietary interests in the drugs and equipment mentioned in this paper

Financial Support: No financial support was received for this paper

Presentation at Meetings: This paper has not been presented at any meeting

*Eye* (2006) **20,** 1310–1311. doi:10.1038/sj.eye.6702164; published online 18 November 2005

Sir,

**Effectiveness of emergency argon laser retinopexy performed by trainee doctors**

Dr Ghosh *et al*.[1] reported that a significant proportion of patients (24%) undergoing laser retinopexy required further interventions and the unfamiliarity of the trainees towards laser indirect ophthalmoscope had been attributed as the most important factor for retreatment. However, based on the data cited, we found it difficult to concur with authors' conclusion.

Authors had observed that 24 patients (24%) requiring retreatment and a significant proportion of them (13 patients) had inadequate coverage of the retinal break.[1] We believed that the technique in delivering laser was not the sole factor in determining the adequacy of laser

barrier. The other equally or even more important factor, namely the amount of any subretinal fluid (SRF) associated with the tears during initial presentation, had not been properly addressed in the article. It has been shown that amount of SRF carries significant bearing over the tissue reaction to laser and the overall completeness of the laser barriers.[2] Hence, the treatment success of laser indirect ophthalmoscope photocoagulation over slit-lamp-delivered laser system in complicated retinal tears relies on not only wider optical localization advantage but also the usual scleral indentation manoeuvre performed during laser delivery, through which the SRF can be displaced to facilitate laser absorption.[2] A proposed causality between surgeons' inexperience with laser technique and the proportion of retreatment without consideration of the patients factor (nature of the retinal breaks) at the same time is sheer.

Unless further information about the characteristics of the retinal tears treated by trainee ophthalmologists can be rendered, it may be difficult to reach authors' conclusion.

**Acknowledgements**

Financial and proprietary interest: Nil.
Financial support: Nil.

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*Eye* (2006) **20,** 1311–1312. doi:10.1038/sj.eye.6702167; published online 11 November 2005

Sir,

**Reply to Liu *et al***

Thank you for giving me the opportunity to reply to the letter by Liu *et al*. It raises a few questions, which I will clarify keeping in mind that it was a retrospective study:

(1) The trainees are taught to treat flat retinal tears and tears with a cuff of subretinal fluid (shallow SRF at the edges of tears only) with laser retinopexy.
(2) Review of our data has shown that of the 24 patients requiring retreatment, only three patients may have been outside the above criteria,
(3) In our conclusion, we had already pointed out that to improve treatment standards, patient selection and seeking vitreoretinal opinion in difficult cases is important.
(4) Although the failure of primary treatment for retinal tears is multifactorial, in our paper we have documented that inability to adequately treat/surround the retinal tears with laser retinopexy was the single most important factor in most of the patients requiring retreatment. This inadequacy was mainly due to the inability of the trainees in using indirect laser delivery system. An audit conducted of our trainees did confirm our belief that more supervised training of indirect laser treatment of trainees was essential.

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*Eye* (2006) **20,** 1312. doi:10.1038/sj.eye.6702168; published online 3 March 2006

Sir,

**Moraxella as a cause of necrotizing fasciitis of the eyelid**

Necrotizing fasciitis has received much interest in the media in recent years, due to its rapid progression, gruesome characteristics, and high mortality rate, estimated at 28% in a recent retrospective study.[1] Haemolytic streptococci, Staphylococcus aureus, *Escherichia coli*, pseudomonas, Enterobacter, Klebsiella,

Proteus, Bacteroides, Clostridium, and peptostreptococcus are among those species isolated from wound cultures and identified as causative, with streptococcus being the commonest at 62%.[1] While necrotizing fasciitis classically involves the trunk, groin, and lower limbs, primary involvement of the eyelids is a well-known entity.[2,3] We describe the first report of Moraxella species being the causative organism in a case of necrotizing fasciitis.

### *Case report*

A 50-year-old male presented with a 4-day history of left periorbital swelling, treated with oral flucloxacillin by his general practitioner. On examination, the left eye was swollen closed, erythematous, exquisitely tender, and hot to touch, but without a drainable or pointing abscess. Cervical lymphadenopathy, a pyrexia of 38.2°C, and tachycardia were noted. Blood tests revealed a raised C-reactive protein of 417, an ESR of 61, and a neutrophilia of $15.8 \times 10^9$/l. CT scan did not demonstrate any orbital involvement and excluded sinus disease as a cause. Preseptal cellulitis was diagnosed and intravenous benzylpenicillin and flucloxacillin were commenced; after 2 days without clinical improvement, oral metronidazole was added to the regime. At 4 days after his admission, after initial wound swabs were negative for microscopy and culture, the left eyelid began to develop areas of necrosis (Figure 1). At the same time, the eye was prised open enough to obtain a visual acuity of 6/9 and to establish that the conjunctiva was white and the cornea clear, thus reducing the likelihood of posterior spread of the infection. Surgical debridement took place immediately, down to healthy, bleeding tissue. Histology confirmed the diagnosis of necrotizing fasciitis, with the presence of inflammatory debris, necrotic tissue, and purulent exudates consistent with acute inflammation and necrosis. Culture of the debrided tissue grew *Moraxella catarrhalis*, and the antibiotic regime was altered to intravenous co-amoxyclav alone on discussion with the microbiologist. On day 11, 1 week after debridement, the patient was discharged home on oral co-amoxyclav, with a healthy, granulating wound. At 3 weeks post-operation, he has the upper lid hung up in down gaze, but no lagophthalmos on gentle eyelid closure, and no corneal staining (Figure 2).

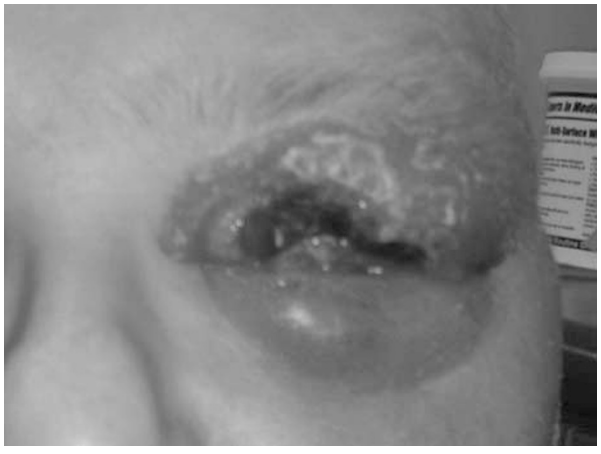

**Figure 1** Preoperative preseptal cellulitis with tissue necrosis.

### Comment

Moraxella species, a gram-negative, aerobic, oxidase-positive diplococcus, is a known commensal in the nasopharynx, and a common causative organism of otitis media, sinusitis, and laryngitis. While it has been linked in two cases to pre-septal cellulites,[4,5] no cases of necrotizing fasciitis as a result of infection by Moraxella have been reported in the literature. This may indicate an increase in the pathogenicity of the organism or, as is not uncommon in head and neck fasciitis, the presence of more than one infective agent, the other agent having been successfully treated with flucloxacillin and benzylpenicillin. Diagnosis of necrotizing fasciitis is based on the clinical presentation of pain, erythema, skin necrosis, and oedema, with subsequent histological findings of extensive fascial and subcutaneous tissue necrosis,[1] which the above case clearly demonstrated. Due to the increasing prevalence of B-lactamase producing Moraxella strains, explaining the poor response to initial antibiotic treatment, the recommended antibiotic regime is now co-amoxyclav or a cephalosporin (first, second or third generation) on recognition of, or when there is a suspicion of, Moraxella infection.

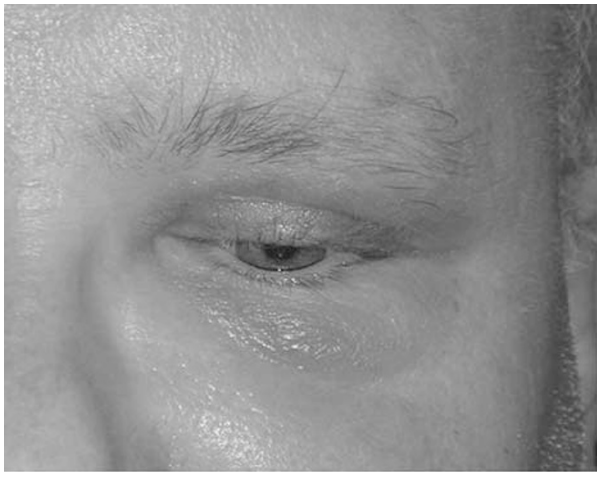

**Figure 2** 3 weeks postoperatively, showing the upper lid hung up in down gaze.

Emphasis on early surgical debridement of tissue at the first sign of necrosis, and regular dialogue with infectious diseases clinicians and microbiologists, particularly in those patients not responding to intravenous antibiotics, should ensure optimal management of this rare, but potentially life-threatening, condition.

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*Eye* (2006) **20,** 1312–1314. doi:10.1038/sj.eye.6702173; published online 18 November 2005

Sir,

## Microsporidia keratoconjunctivitis in a corneal graft

Ocular microsporidial infection has been reported to occur in two forms, a deep stromal keratitis in immunocompetent individuals or a bilateral superficial punctate epithelial keratitis in immunocompromised individuals.[1,2] We report a unique case of microsporidial epithelial keratoconjunctivitis occurring in the corneal graft of an individual who was locally immunocompromised.

### *Case report*

A 60-year-old male, who had undergone a repeat penetrating keratoplasty 6 months prior for a failed graft, following a transplantation for pseudophakic corneal oedema, presented with complaints of pain, redness, discharge, watering, and blurred vision of the left eye of 14 days duration. He was using prednisolone acetate eye drops twice daily. Visual acuity in the right and left eye was 20/20 and 20/100, respectively. The left eye had mild discharge with diffuse conjunctival congestion with multiple raised whitish confluent epithelial lesions on the temporal half of the graft (Figure 1a) and the underlying corneal stroma was clear. The anterior chamber was quiet. The iris had multiple areas of atrophy and the intraocular lens was in place. Clinically, microsporidial epithelial keratitis was suspected with a differential diagnosis of Thygeson's superficial punctate keratopathy and filamentary keratopathy. Both 10% potassium hydroxide-calcoflour white preparation (Figure 2a) and Gram stain of corneal scrapings showed plenty of

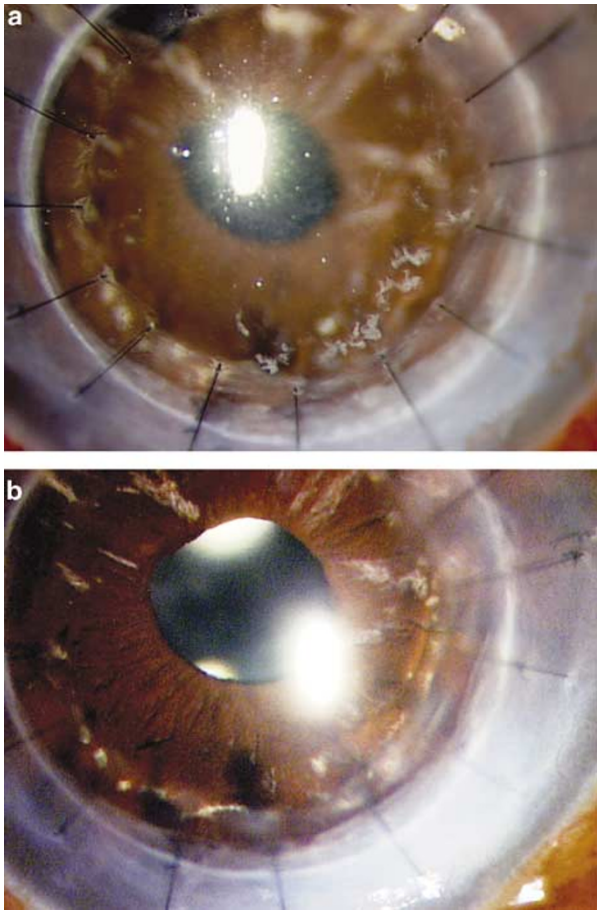


**Figure 1** (a) Left eye of patient at presentation showing multiple, whitish, confluent, elevated epithelial lesions. (b) Left eye of patient after 10 days of treatment showing complete resolution of epithelial lesions.

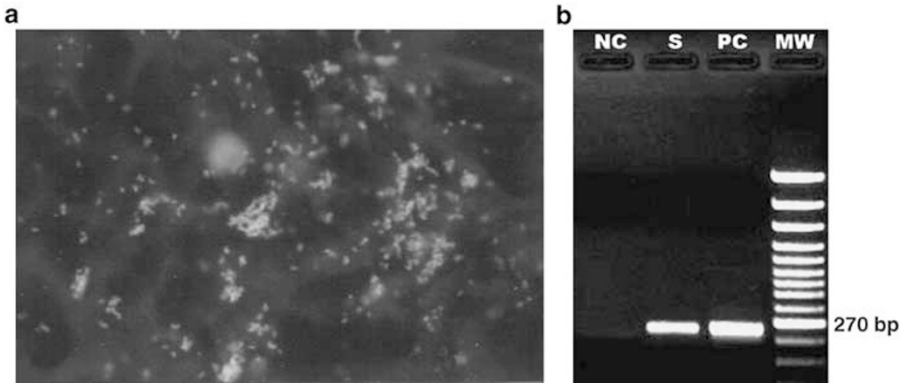


**Figure 2** (a) KOH/Calcofluor white stain of the corneal scraping showing oval fluorescing microsporidial spores ( × 500). (b) 1.5% Agarose gel electrophorogram showing the 270 base pair PCR product.

microsporidia spores, which were confirmed by 1% acid-fast stain. Polymerase chain reaction (PCR) for microsporidia was performed using primers capable of identifying several Enterocytozoon and Encephalitozoon species of microsporidia.[3] A single ~270 base pair fragment was observed on agarose gel electrophoresis and ethidium bromide staining of the PCR amplified patient sample (Figure 2b). Topical steroids were discontinued and he was treated with topical 0.3% ciprofloxacin eight times daily along with topical lubricants. After 10 days, all his lesions had disappeared (Figure 1b). The patient was seronegative for HIV by ELISA test.

Bilateral punctate epithelial keratopathy and conjunctivitis has been described in immunocompromised[1,2] and more recently in immunocompetent patients as well.[4,5] Previously described risk factors like trauma, contact lens wear, prior refractive surgery or exposure to contaminated water were absent in our patient. The only possible associated risk in this case was the use of topical steroids, leading to a localized immunosuppressed state, resulting in secondary infection by microsporidia. In our patient, diagnostic debridement probably debulked the epithelium of the load of organisms and hastened resolution. Contrary to belief that debridement worsens the infection by driving the organisms into the stroma; we found that debridement actually hastens resolution.[2]

## Comment

To the best of our knowledge, this is the first report of keratoconjunctivitis caused by microsporidia in a corneal graft. As a result of local immunosuppression, this infection can occur in patients who have been grafted, which has not previously been described. The differential diagnosis of microsporidial keratitis should be considered in this subset of patients presenting with typical features of multiple epithelial lesions in the cornea.

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*Eye* (2006) **20,** 1314–1315. doi:10.1038/sj.eye.6702178;
published online 25 November 2005

Sir,

**Scleral perforation following diode laser trans-scleral cyclophotocoagulation**

Diode laser trans-scleral cyclophotocoagulation (DLTSC) is an effective treatment in refractory glaucomas.[1–3] Complications, such as uveitis, pupillary distortion, conjunctival burns, hyphema, chronic hypotony, cystoid macular oedema, retinal detachment, and phthisis bulbi, have been reported.[1,4–8] Two cases of scleral perforation requiring suturing were described in the published literature.[9,10] We describe a case of scleral perforation during DLTSC in a patient with pre-existing scleral thinning, confirmed with ultrasound biomicroscopy. The leakage stopped after 1 day of patching without the need of suturing.

*Case report*

A 66-year-old Chinese woman had undergone limbal incision ECCE to both eyes 20 years ago with no intraocular lens implantation. Thereafter, she had been abusing unprescribed topical ophthalmic steroid for more than 20 years, unaware of its potential side effects. She had no other ophthalmic or medical history of note. On April 2004, she suffered a blunt trauma to her left eye while she bumped into a glass door with a resultant 3.5-mm full thickness dehiscence of the limbal cataract wound in the supero-temporal quadrant, with vitreous prolapse. There were peripheral corneal and scleral thinning from 10 to 2 o' clock positions, presumably related to her chronic steroid abuse.

Emergency repair of the corneal wound and anterior vitrectomy were carried out, followed by a repeat pars-plana vitrectomy and anterior vitrectomy 2 weeks later for persistent vitreous incarceration in the corneal wound.

Nevertheless, she suffered from subsequent secondary synechial angle closure glaucoma. Despite maximal antiglaucomatous medications, including four topical medications and oral acetazolamide 500 mg four times a day, her intraocular pressure eventually reached 48 mmHg. Gonioscopy examination revealed severe peripheral anterior synechiae $>270^{\circ}$ and a cup-to-disc ratio of 0.8. Visual acuity was 4/60 in the left eye at this stage.

DLTSC was performed under retrobulbar anaesthesia using a G-probe (IRIS Medical Instruments, Inc., Mountain View, CA, USA). A total of 14 laser spots each of 1500 mW, 2-s duration, were applied. A popping sound was heard at six points.

While applying the 14th laser application over the thinned sclera at 10 o' clock position, a gush of aqueous was noticed. Inspection under the operating microscope confirmed a round, 'punched out' full thickness perforation, 1.2 mm behind the limbus with leakage demonstrated with Siedel test (Figure 1). There were no vessels or pigment around the perforation site. There were no other conjunctival or scleral burns. The G probe was inspected and no black deposits at the tip were observed. The laser procedure was aborted. The eye was padded with antibiotic ointment and crepe bandage. Oral acetazolamide 500 mg was given four times daily to reduce aqueous flow through the perforation.

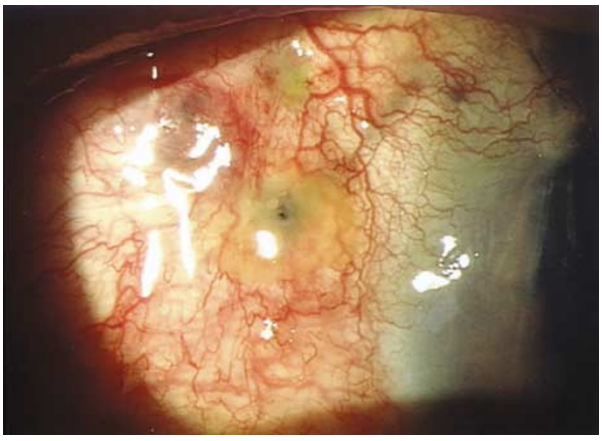

**Figure 1** A round, 'punched out' full thickness scleral perforation with leakage demonstrated with Siedel test.

On the next day, the leakage had stopped, possibly having been plugged by vitreous. The intraocular pressure was 9 mmHg. Topical prednisolone acetate 1% four times daily, topical chloramphenicol 0.5% four times daily, and topical atropine 1% twice daily were prescribed. During the subsequent 3 weeks, the scleral hole healed. Ultrasound biomicroscopy revealed a scleral thickness of 0.437 mm around the perforation site. In the ensuring 3 months, the intraocular pressure gradually increased to 40 mmHg despite antiglaucomatous agents. At the time of writing, the patient was pain-free, and she refused further intervention. Visual acuity was hand movement in the left eye at most recent follow-up. The right eye was aphakic with a best-corrected visual acuity of 1.0. The remote risk of sympathetic ophthalmitis and its symptoms were discussed, and the patient preferred observation at this stage.

**Comment**

Diode laser of 810 nm wavelength is an effective tool for treating refractory glaucomas.[1–3]

To the best of our knowledge, there were two reported cases of scleral perforation due to DLTSC in the published literature.[10,11] The case reported by Gaasterland and Pollack[9] was thought to be due to the

sharp edge of the probe cutting conjunctival vessels and causing bleeding. Thin adherent debris was then carbonized, allowing the laser tip temperature to rise and causing scleral perforation. The defect required suturing. This led to the redesigning of the laser probe tip. In our case, there was no such carbonized debris seen and we think it is unlikely to be the reason for our perforation.

Sabri and Vernon[10] reported a case of scleral perforation using the new contact G-probe. The defect required suturing with two 10-0 vicryl sutures. However, 1 week later, the scleral leak recurred and further suturing was needed.

In our case, with crepe bandage and oral acetazolamide for 1 day, we were able to stop the leakage. During subsequent follow-up, the scleral hole healed and was covered by intact conjunctival epithelium. This demonstrated that suturing may not always be necessary, especially when the perforation is small.

Pre-existing scleral thinning is a common risk factor in the two previously reported cases, and also in our patient. Hence, Sabri and Vernon[10] suggested the use of a lower laser power setting (50%), though there is no good proof that such a lower power could prevent perforation and is still as effective.

With heightened awareness of this complication, and appropriate management when it occurs, we believe the risk of scleral perforation and its consequences could be minimized.

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Financial and proprietary interest: Nil

*Eye* (2006) **20,** 1316–1317. doi:10.1038/sj.eye.6702179; published online 25 November 2005

Sir,

## Vitreous haemorrhage following cardiopulmonary resuscitation

Vitreous haemorrhage is known to occur by a wide variety of mechanisms. We present the case of a 27-year-old gentleman who developed a vitreous haemorrhage following cardiopulmonary resuscitation (CPR). We postulate that this occurred as a result of the chest compressions performed, through a mechanism similar to valsalva retinopathy. To our knowledge this is the first reported case of vitreous haemorrhage arising in this way.

*Case report*

A 27-year-old man presented to the accident and emergency department with sudden onset of severe retrosternal chest pain. There was no relevant past medical or ocular history and no previous history of chest pain. He was a cigarette smoker. Initial electrocardiography (ECG) showed ST segment elevation in the anterior chest leads. Shortly afterwards the patient collapsed on a trolley having had a ventricular fibrillation cardiac arrest. He was shocked into asystole and after 1 mg of adrenaline intravenously and CPR, he regained a perfusing rhythm. He was transferred for cardiac angiography, which showed 100% occlusion of his left anterior descending (LAD) coronary artery. The decision was taken not for thrombolysis but angioplasty instead. A proximal occlusion of the LAD was readily canalised and two stents were inserted. The patient was then transferred to the intensive care unit where he received intravenous dobutamine and noradrenaline for resuscitation of presumed stunned myocardium.

Following extubation 8 days later he complained of blurred vision in his right eye. He was subsequently reviewed in the ophthalmology department. Snellen visual acuities were 3/60 right eye and 6/4 left eye. He was found to have a dense vitreous haemorrhage and no fundal view in the right eye. Ultrasound revealed a flat retina. The haemorrhage failed to resolve and 3 months following myocardial infarction he underwent right vitrectomy. At surgery, dilatation of peripapillary vessels with adherent vitreous haemorrhage was noted, but no other retinal abnormalities. He made an uneventful recovery and postoperative visual acuity in his right eye was 6/6. Dilated fundoscopy since the surgery has not revealed any associated pathology.

**Comment**

This case describes a presumed iatrogenic vitreous haemorrhage. In the absence of contributing ocular pathology, the only causative factor we can identify is the cardiac compressions the patient received during his resuscitation.

Chest compressions during CPR are known to cause several complications, for example rib fractures, pulmonary haemorrhages and liver lacerations. However, ocular complications are more unusual. Chest compression by safety belt in automobile accidents is known to cause traumatic retinal angiopathy (Purtscher's disease) – even from a minor compression injury from a lap-shoulder belt[1] – and retinal detachment.[2] We also note the recent report by Chandra *et al*[3] of a case of Purtscher's retinopathy in one eye, and Valsalva retinopathy in the other, following a compressive chest injury.

The mechanism and efficacy of chest compressions as part of CPR have become better understood in recent years. There has been deliberation as to whether sternal compression generates a direct increase in arterial forward flow by cardiac compression, or whether it is the relaxation phase that encourages venous return through negative intrathoracic pressure. However, it is now generally thought that both mechanisms are acting to provide some cerebral perfusion, though the flow generated is insufficient to force vessel aneurysm or rupture. Babbs[4] used a mathematical model to show that even the most efficient compression technique would only generate a maximum of 3.1 l/min and 58 mmHg of systolic perfusion pressure. Pinming *et al*[5] confirmed this by performing transoesophageal echocardiography during closed-chest CPR, in cardiac arrest. Peak forward aortic flow was measured at $58.8 \pm 11.6$ cm/s; less than half of normal peak aortic flow.

Valsalva retinopathy is a well-described phenomenon. A preretinal haemorrhage in the macula area is the usual finding at presentation. The cause is thought to be a rupture of a retinal vessel, following physical exertion – usually a sudden and rapid rise in intrathoracic pressure (such as when coughing, heavy lifting or straining at stool). This is thought to cause a rise in the intravenous pressure, then a rise in retinal vessel intraluminal pressure, with consequent rupture of a retinal venule or capillary, perhaps at the site of a pre-existing vessel wall weakness.[6] Extension of the haemorrhage into the vitreous is rare, but is thought to occur if the force of the intrathoracic pressure rise is sufficiently high[7] or in the presence of a blood disorder such as sickle cell disease.[8]

In our case, we can rule out other causes of vitreous haemorrhage. There was no previous ocular pathology, no retinal pathology was identified during vitrectomy surgery and the patient had not been thrombolysed, or given any anticoagulant therapy. We therefore hypothesise that this patient's vitreous haemorrhage was due to the chest compressions received during CPR, via a mechanism similar to valsalva retinopathy.

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*Declaration*: No funding was received in the preparation of this manuscript. The work has not been presented at any meeting.

*Eye* (2006) **20,** 1317–1319. doi:10.1038/sj.eye.6702184; published online 18 November 2005

Sir,

## An unusual case of proptosis

We report a rare case of orbital xanthoma in a 38-year-old man presenting as droopy eyelid and proptosis. The only clue to the diagnosis was the presence of eyelid xanthelasma.

### *Case report*

A previously well 38-year-old Bangladeshi labourer presented with a 2-month history of worsening right ptosis, proptosis, and dull ache over the forehead (Figure 1). Examination revealed a right non-axial proptosis which measured 23 mm on Hertel's exophthalmometer (compared with 21 mm on the left). The right palpebral fissure measured 5 mm (compared with 7 mm on the left). The right upper eyelid had an area of xanthelasma in the nasal aspect; otherwise, it is not erythematous or tender to palpation. The ocular movement was normal except for dull ache and slight restriction on upgaze. The vision was normal in both eyes. An urgent CT scan revealed a mass which appeared to involve the superior rectus and levator superioris complex (Figure 2). A possible diagnosis of myositis was made and he was started on oral 100 mg diclofenac b.d. The blood tests for autoimmune antibodies, ESR, and thyroid function tests were normal. The only abnormal result was raised cholesterol level of 6.2 mmol/l. There was no clinical improvement after 2 weeks. A MRI scan was performed to elucidate the lesion. This showed the mass to be distinct from the superior rectus and appeared to involve only the levator superioris (Figure 3). An open biopsy through the skin crease revealed an uncapsulated orange coloured lesion between the orbicularis oculi and the orbital septum with infiltration of the levator aponeurosis (Figure 4). The lesion had a rubbery consistency and extended posteriorly beneath the orbital roof. The mass was removed *en bloc*. It had a uniform yellow surface

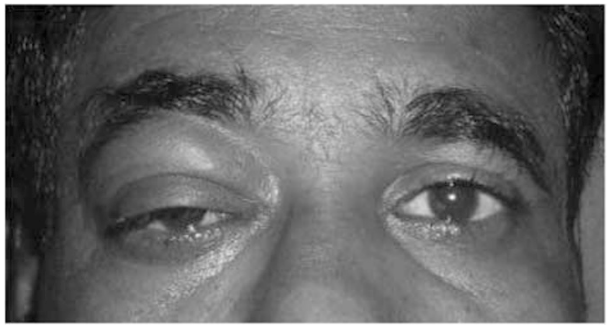

**Figure 1** A 38-year-old Bangladeshi labourer with right eye ptosis and proptosis.

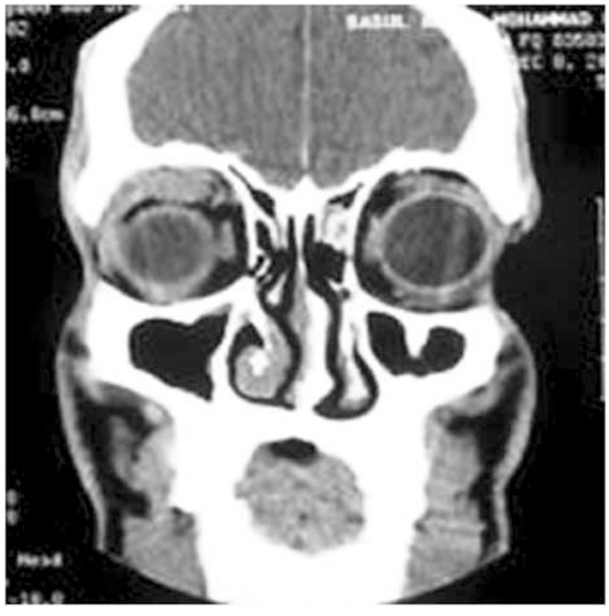

**Figure 2** A CT coronal scan of the orbit revealed a mass at the right eye superior rectus and levator superioris complex.

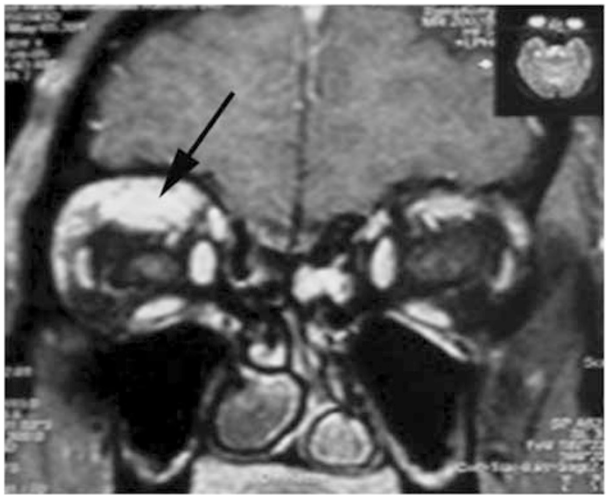

**Figure 3** A MRI coronal scan of the orbit showed the mass to be distinct from the right superior rectus and appear to involve only the right levator superioris.

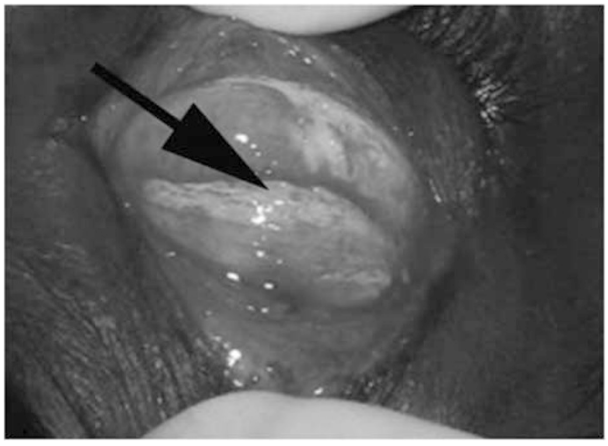

**Figure 4** An open biopsy through the skin crease of the right eye revealed an uncapsulated orange coloured lesion between the orbicularis oculi and the orbital septum with infiltration of the levator aponeurosis.

measuring $2.1 \times 1.5 \times 1.1$ cm. Postoperatively, there was a marked improvement of the proptosis. Histological report revealed a fibroadipose lesion made up of large number of macrophages with foamy cytoplasm and eccentric nuclei. There were no Touton's giant cells seen to suggest the diagnosis of xanthogranuloma. The final pathology diagnosis was orbital xanthoma (Figure 5).

## Comment

Xanthoma occurring within the orbit is extremely rare. There have been only a few reports in the literature.[1–3] They are typically found in the subcutaneous tissues

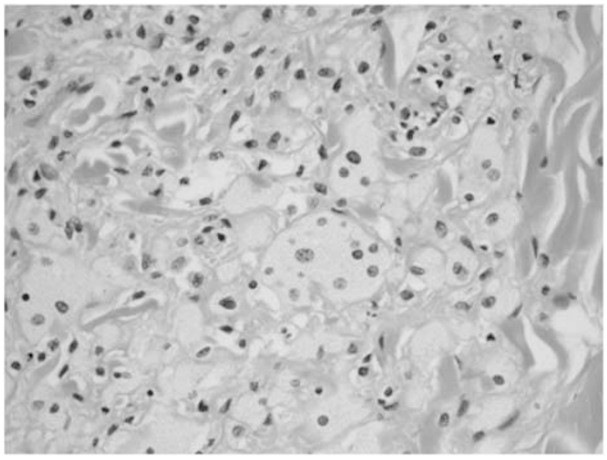

**Figure 5** Histology slide showing orbital xanthoma.

usually along the Achilles tendon and extensor tendons of the hand of patients with hyperlipidaemia. They are believed to develop as a result of leakage of lipids from the blood vessels into the surrounding tissue. The lipids were subsequently phagocytosed by macrophages. As the cholesterol is not degraded, the macrophages have foamy cytoplasm. Extracellular cholesterol can induce chronic inflammatory reaction which may account for the dull ache experienced by our patient. Giant cells similar to that seen in chalazion may occur in response to the inflammation and fibrosis may develop. The condition needs to be differentiated from two other conditions: xanthogranuloma and necrobiosis xanthoma. In the former, Touton's cells are found histologically and in the later the blood tests show dysproteinaemia.

On the CT and MRI scans, xanthoma could not be differentiated from other orbital benign tumours such as frontal mucocele, epidermoid cyst, dermoid cyst, haemangioma, meningioma, Langerhans cell histiocytoma, and fibrous dysplasia.[4] In cases of chronic inflammation, the clinical features may be confused with pseudotumour leading to unnecessary investigations for autoimmune diseases. Biopsy is the only certain mean of diagnosing orbital xanthoma.

In summary, as orbital xanthoma cannot be diagnosed with certainty either clinically or by radiological imaginations, this unusual condition should be included in the differential diagnosis of proptosis in patients with eyelid xanthelasma or hyperlipidaemia.

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Proprietary/Financial interest: None

*Eye* (2006) **20,** 1319–1321. doi:10.1038/sj.eye.6702185; published online 9 December 2005

Sir,

**Resolution of cystoid macular oedema after retinal detachment repair: is intravitreal triamcinolone useful?**

Poor functional visual outcome after successful retinal detachment (RD) repair is mainly owing to photoreceptor cell apoptosis.[1] Clinical appearance of the macula in the majority of these patients is normal, but a proportion have anatomical changes observable clinically.[2] More recently, ocular coherence tomography (OCT) has helped in revealing residual subfoveal fluid accumulation not visible clinically or on fluorescein angiography,[3] more frequent after scleral surgery.[4] OCT has also been helpful in showing postoperative cystoid macular oedema (CMO), responsive for the limited visual improvement in a small number of patients. The exact etiopathogenic mechanism of CMO after RD is unclear, but ocular inflammation may play a role, especially after the trauma of cryotherapy, scleral buckle, and subretinal fluid (SRF) drainage. CMO incidence, in phakic eyes, has been reported around 25–30% after cryotherapy and scleral explant,[5] and 8% after pneumatic retinopexy.[6] It has not been related to preoperative macular status or duration of the RD.[7] CMO spontaneous resolution has been described in up to 76% of cases,[5] within 2 years postoperatively. Other series, though, report a more bleak evolution of the condition.[8] Different treatments have been described, but none with great success. Topical and systemic non-steroidal anti-inflammatory drugs and steroids have been used. There are anecdotic reports of response to acetazolamide.[9]

A 78-year-old male presented to our Primary Care Department with complaints of floaters for the past week, and decreased central vision for the last 48 h. He underwent uneventful phacoemulsification and posterior chamber intraocular lens implant 3 years ago, but otherwise he had no other past ocular history. On examination, visual acuity was 6/36 and fundoscopy showed superotemporal bullous RD involving his macula, for which he underwent pars plana vitrectomy, cryoretinopexy, and 12% $C_3F_8$ gas endotamponade.

Immediate postoperative period was anodyne, with complete flattening of the retina. At 4 months after successful repair, though, best-corrected visual acuity (BCVA) was 6/18. Anterior segment was quiet with pseudophakia and fundoscopy revealed cystoid macular changes. OCT confirmed this clinical finding and also showed no presence of residual SRF (Figure 1). The patient was started on topical ketorolac and prednisolone, and 1 month later received 4 mg intravitreal triamcinolone acetonide (IVTA). OCT performed 2, 6, and 12 months later, still showed cystoid spaces in the macula and BCVA remained unchanged (Figure 2). At 15 months after IVTA, OCT showed total resolution of the CMO (Figure 3). Visual acuity, at this stage, had returned to pre-RD levels, with BCVA of 6/6.

There are several reports of successful treatment of uveitic, pseudophakic, and diabetic CMO with IVTA. However, no reference to CMO after RD treated with IVTA could be found in a PubMed literature search. There is a rationale for using steroids in its management, as increased prostaglandin levels in the retina and uvea and increased prostacyclin and thromboxane $A_2$ derivatives in the SRF have been reported.[10] All these mediators increase perifoveal microvascular permeability by disrupting capillary tight junctions,

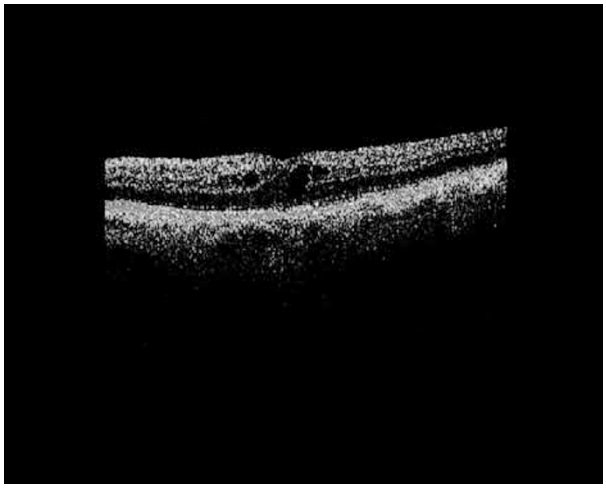

**Figure 1** OCT scan 4 months after successful retinal detachment repair, showing cystoid macular oedema.

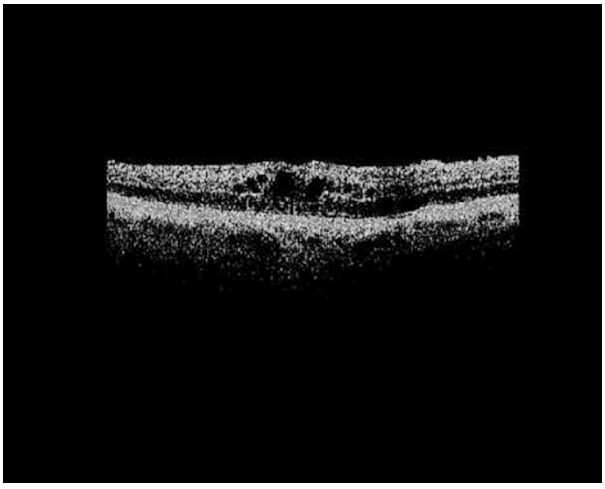

**Figure 2** At 2 months after intravitreal triamcinolone injection, OCT still reveals cystoid changes.

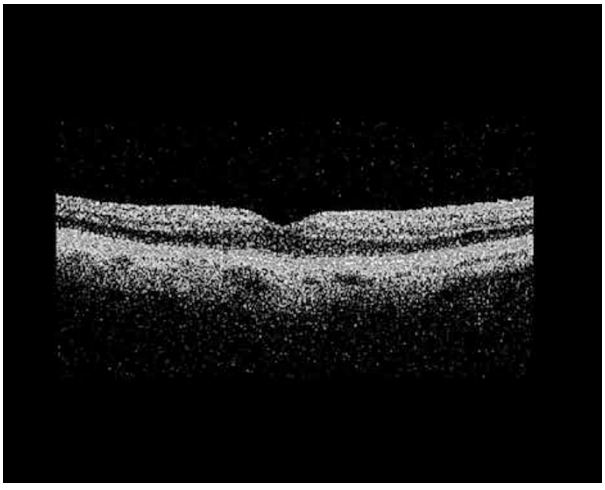

**Figure 3** Resolution of the cystoid oedema and restoration of normal foveal morphology, 15 months after IVTA.

producing the breakdown of the blood–retinal barrier (BRB), and subsequent oedema.[11] Steroids inhibit phospholipase $A_2$ and reduce levels of proinflammatory cytokines, increasing BRB function.[12]

The late visual improvement observed in our patient is unlikely to be explained by IVTA effect, with an expected action duration of 3 months or less in the vitrectomised eye.[13] With the current increase in use of IVTA for different eye conditions, many unresolving CMO after RD will be treated with IVTA. A randomised control trial (RCT) would have, therefore, its role in elucidating the usefulness of the drug in such cases. Potential side effects and complications of IVTA, such as endophthalmitis, cataract formation, and ocular hypertension, should be then weighed up in a treatment arm of a RCT for a condition that might, like in the case we present, resolve spontaneously in the long term.

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*Eye* (2006) **20,** 1321–1322. doi:10.1038/sj.eye.6702188;
published online 2 December 2005

Sir,

**Immediate argon peripheral iridoplasty (ALPI) as initial treatment phacomorphic glaucoma: a safe and cost-effective treatment?**

In their study of Argon Laser Peripheral Iridoplasty (ALPI) as an initial treatment for acute phacomorphic angle closure glaucoma, Tham *et al*[1] did not mention the number of patients they excluded from their trial and the reason for exclusion. Their recruitment rate of five patients per year may not be high enough to justify the cost of round-the-clock availability of facility and operator. This issue would be pertinent in developing countries where resources are more limited.

The authors additionally noted a 50% pressure increase in one of the 10 patients sampled; their suggested explanations imply that ALPI is somewhat unpredictable in its effects. Moreover, they could only postulate on how ALPI is able to reduce IOP as eight out of 10 patients had closed angles on gonioscopy. Their conclusion that ALPI is safe appears premature. Perhaps an *in vitro* model and ultrasound biomicroscopy would shed more light on the exact mechanism of ALPI in phacomorphic glaucoma.

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1 Tham CCY, Lai JSM, Poon ASY, Chan JCH, Lam SW, Chua JKH *et al*. Immediate argon laser peripheral iridoplasty (ALPI) as initial treatment for acute phacomorphic angle-closure (phacomorphic glaucoma) before cataract extraction: a preliminary study. *Eye* 2005; **19**: 778–783.

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The author has no financial or proprietary interest in the submitted correspondence

*Eye* (2006) **20,** 1323. doi:10.1038/sj.eye.6702190; published online 2 December 2005

Sir,

**Reply to S Thyagarajan**

We thank Dr Thyagarajan for his interest and comments on our paper.[1] We recruited 10 consecutive patients into the study which means that no patient was excluded during the period of study. Argon laser peripheral iridoplasty (ALPI) is an easily mastered procedure and requires the availability of an argon laser machine, which we think is widely used in most of the eye institutions. The issue may be pertinent in developing countries but our hospital is equipped with the argon laser machine mainly for the treatment of diabetic retinopathy and therefore, no extra cost is required for its use in the treatment of acute phacomorphic angle-closure.

One of our 10 patients had a 50% rise in the intraocular pressure (IOP) 15 min after ALPI. The IOP decreased gradually to below 25 mmHg in 4 h although systemic acetazolamide was given. If we consider the fact that the other nine patients had steady decrease in the IOP after ALPI, the failure of one case would not have implied an unpredictable IOP lowering effect of ALPI. Concerning the persistent angle closure in eight of our patients, we have admitted in the discussion that our study was unable to find out the exact IOP lowering mechanism of ALPI. Nevertheless, we concluded from the preliminary study that ALPI appeared to be safe and effective as the first-line treatment of acute phacomorphic angle-closure. We agree that *in vitro* study and randomized controlled clinical trial are needed to shed more light on it.

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1 Tham CC, Lai JS, Poon AS, Chan JC, Lam SW, Chua JK *et al*. Immediate argon laser peripheral iridoplasty (ALPI) as initial treatment for acute phacomorphic angle-closure (phacomorphic glaucoma) before cataract extraction: a preliminary study. *Eye* 2005; **19**: 778–783.

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*Eye* (2006) **20,** 1323–1324. doi:10.1038/sj.eye.6702191;
published online 2 December 2005

Sir,

**A case of cutaneous collision tumour: the importance of photographic documentation and large incisional biopsy**

An 87-year-old lady presented with a 1-year history of a left medial canthal lesion. There had been no recent change in its colour or size, nor associated bleeding or pruritis. She gave no history of previous skin lesions or excessive sun exposure. The lesion appeared as a firm pearly nodule $7 \times 8\,mm^2$ (Figure 1a) with small telangiectatic vessels on its surface and no associated pigmentation. It did not involve the punctum or canaliculi. This clinical appearance was suggestive of a basal cell carcinoma.

The lesion was photographed and an incision biopsy performed. Histopathological examination identified a cylindroma (Figure 1b). However, the preincisional clinical appearance of the lesion, confirmed by review of the photograph (Figure 1a), cast doubt over the biopsy result (postincisional Figure 1c appearance). A second incisional biopsy was thus performed, which revealed two separate pathologies (Figure 1d), a cylindroma and adjacent nodular basal cell carcinoma. The patient underwent Mohs' micrographic surgery and the defect reconstructed by direct closure.

## Comment

Contiguous or 'collision' tumours are an unusual entity. A retrospective study of 40 000 cutaneous biopsies found only 69 such examples.[1] The association of an adnexal tumour and a second neoplasm was found in only four patients, but none were contiguous with a BCC. In fact, this is a very rare association.

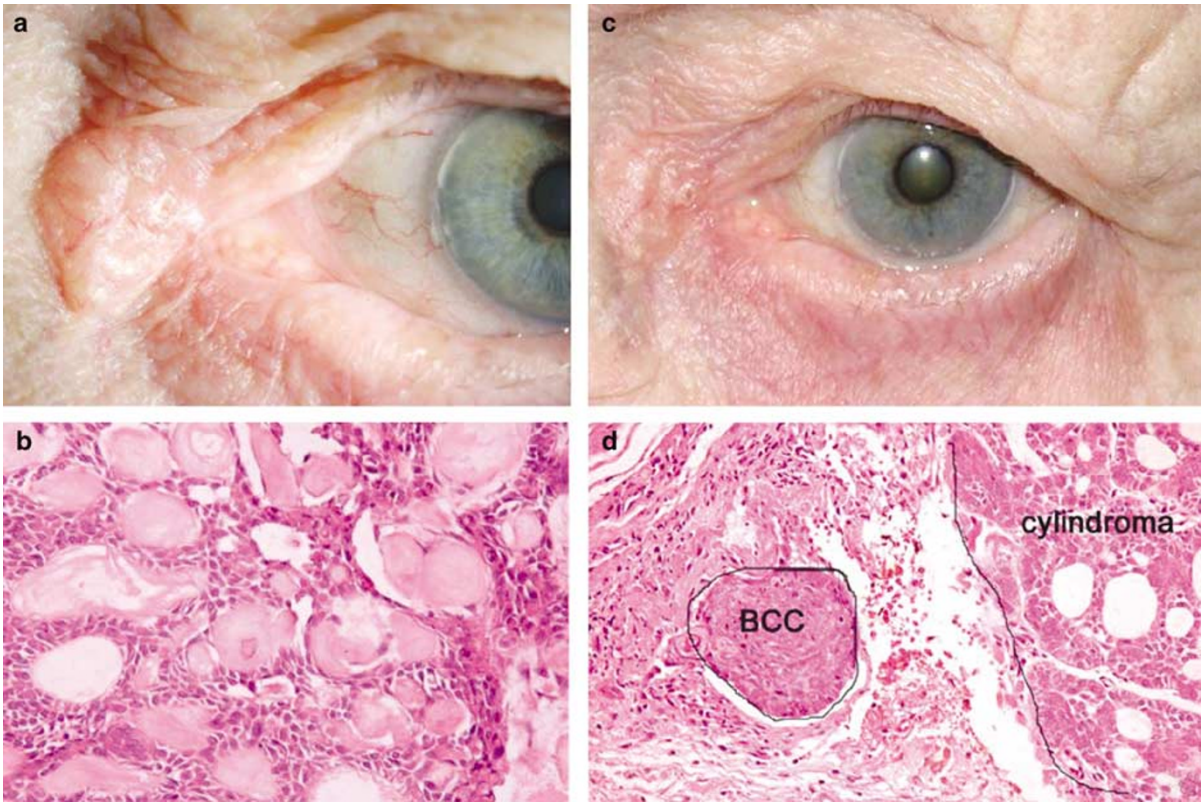


**Figure 1** (a) Preincisional photograph showing a lesion at the left medial canthus. (b) Haematoxylin and eosin stain of the first biopsy specimen. High power ( × 200). The lesion is composed of lobules of cells with large vesicular nuclei surrounded by thick eosinophilic material. There is widespread ductal differentiation. The appearances are consistent with those of a cylindroma. (c) Postincisional photograph of the lesion illustrating the change in the appearance of the lesion following the diagnostic biopsy. (d) Haematoxylin and eosin stain of the second biopsy specimen. High power ( × 200). Two different tumour types are seen. A focus of cylindroma is seen (right) juxtaposed with a focus of basal cell carcinoma (left). In this sample, the two lesions are intimately associated but appear separate.

Certain associations such as between cylindromas and apocrine cystadenoma are expected, as they are sweat gland proliferations. Similarly, basal cell and squamous cell carcinomas are malignant proliferations of keratinocytes and have similar histogenesis. However, most collision tumours occur by chance, and are not derived from similar cell lines nor share pathogenic mechanisms.

The coexistence of two or more neoplasms in a single cutaneous specimen is unusual and can be diagnostically misleading if only one of the two is discovered. Biopsy reports must always be questioned in the light of the clinical history and examination. Unless histopathological diagnoses are considered alongside the clinical appearance of the original lesion, which may be altered by surgery, the anomaly may not be questioned.

It is essential therefore that new lesions be photographically documented prior to any intervention. This will aid in the patient's future management particularly in situations where the patient is reviewed by a different clinician at subsequent visits. This objective tool is especially important in cases where the clinical appearance does not correlate well with histological findings. Performing a large incisional biopsy will also maximize the chance of identifying multiple lesions.

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Acknowledgement of any proprietary interests or research funding: Nil

*Eye* (2006) **20,** 1324–1325. doi:10.1038/sj.eye.6702192; published online 31 March 2006

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Sir,

## *Serratia marcescens* endophthalmitis secondary to pneumonia

A 56-year-old female was admitted to ITU with postoperative pneumonia secondary to *Serratia marcescens* treated with Imipenem 750 mg b.i.d. i.v. She underwent bowel resection for Crohn's disease 1 week prior to her pneumonia. A month later, she arrested and became comatosed despite resuscitation. She deteriorated, developing renal failure requiring haemofiltration. *S. marcescens* was grown from sputum and blood cultures and Teicoplanin 400 mg b.i.d. i.v. was started. After 24 h, she developed an acute right red eye.

On examination, there was an afferent pupillary defect, corneal oedema, and hypopyon. There was no fundal view (Figure 1). Examination of her left eye was unremarkable. A diagnosis of endogenous endophthalmitis was suspected and a vitreous tap performed with Ceftazidime 2.25 mg, Vancomycin 1 mg, and Amphotericin 5 $\mu$g given intravitreally. In addition, she was given hourly G Cefuroxime 5% and G Gentamicin 1.5%. *S. marcescens* sensitive to Ceftazidime was isolated from her vitreous and a repeated intravitreal injection of Ceftazidime and Vancomycin were given 72 h later. There was little ocular or systemic improvement and despite aggressive treatment she eventually died of multiple organ failure. An autopsy was declined.

Endogenous endophthalmitis (EE) accounts for 10% of all endophthalmitis.[1] Fungi are the most common causal pathogen[2] followed by bacteria.[1,3] Risk factors include systemic immunosuppression, sepsis, major surgery, indwelling catheters, and prolonged antibiotic therapy.[2] The overall prognosis is poor with useful vision preserved in only 40%, 6 and 7–15% patients die from septicaemia.[4,5]

Identifying the underlying cause is paramount. Conjunctival swabs poorly reflect intrinsic eye infection and vitreous tap/biopsy[6] should be performed and intravitreal antibiotics administered.

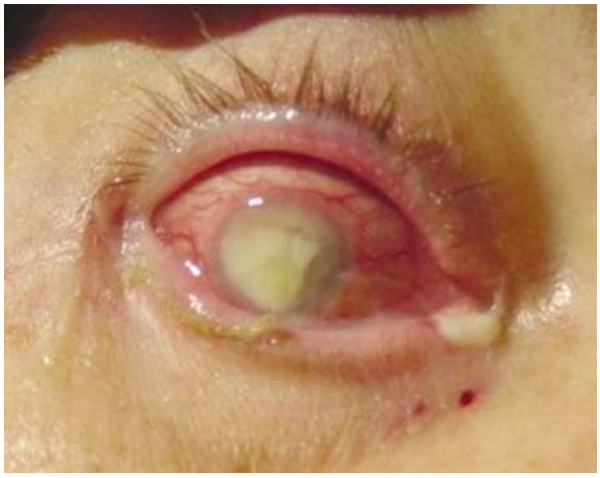

**Figure 1** Right eye of patient showing scleral injection, corneal oedema, and hypopyon.

*S. marcescens* is multiresistant Gram-negative bacillus that can produce a red pigment causing a pink hypopyon.[7] To our knowledge, this is the first reported case of *S. marcescens* pneumonia as a primary source for EE (the lung is the most common site for these pathogens[8]). Despite appropriate systemic and intravitreal antibiotics, the visual outcome was poor and the patient eventually died. As the incidence of EE (especially Gram-negative infections) appears to be rising,[3] then this aggressive organism may become a more common cause for this devastating condition.

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This has previously been presented at the Welsh Ophthalmic Forum, Camarthen, October 2003

*Eye* (2006) **20,** 1325–1326. doi:10.1038/sj.eye.6702194; published online 9 December 2005

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Sir,

**Tractional retinal break and rhegmatogenous retinal detachment consequent to branch retinal vein occlusion**

Traction on neovascular tissue causing tractional retinal breaks and consequent rhegmatogenous retinal detachment is an infrequent complication of branch retinal vein occlusion (BRVO). We report a case which was successfully managed with barrier laser photocoagulation.

### *Case report*

A 73-year-old man presented to ophthalmic casualty with history of sudden painless loss of vision in the left eye. There was no significant ophthalmic history and he suffered from hypertension. Visual acuities were 6/6 in right eye and hand movements in left eye. Anterior segment examination was normal; there was no fundus view due to dense vitreous haemorrhage. B-scan confirmed vitreous haemorrhage and flat retina.

Vitreous haemorrhage cleared 2 months later and visual acuity improved to 6/6 in the affected eye. Fundoscopy revealed a large posterior horseshoe retinal tear at 2 O'clock position with surrounding localised retinal detachment above the superotemporal retinal vessels. There was an avulsed neovascular frond attached to posterior hyaloid face and ghosting of superotemporal blood vessels suggesting an old BRVO (see Figure 1).

Barrier laser photocoagulation was performed surrounding the area of retinal detachment. After 6 months the patient's vision was stable with no progression of retinal detachment or development of further complications.

### Comment

BRVO may be associated with a number of complications including macular oedema, epiretinal membrane, retinal neovascularisation, vitreous haemorrhage, retinal breaks, and rhegmatogenous retinal detachment. Retinal breaks

**Figure 1** Left eye fundoscopy showing localized retinal detachment with horseshoe retinal tear at 2O'clock and an avulsed neovascular frond with ghosting of superotemporal blood vessels.

following BRVO are of two types—holes without traction and tears with vitreous traction with or without retinal neovascularisation.[1–3] Tears and retinal detachment due to traction on neovascular tissue are infrequent.

A strong association between BRVO with vitreous haemorrhage and posterior tractional retinal breaks was reported by Joondeph *et al*.[2] They reviewed 358 cases with BRVO and reported 1.6% incidence of posterior tractional breaks leading to rhegmatogenous retinal detachment in 0.6% of patients.[1] Kir *et al*[4] reported 3% incidence of retinal breaks and 1.3% of rhegmatogenous retinal detachment.

Retinal breaks are almost exclusively found in the distribution of the occluded vessel and often located at or posterior to equator.[4]

The most widely accepted hypothesis for pathogenesis of tractional retinal break in BRVO is vitreoretinal traction caused by vitreous contraction following retinal neovascularisation.[1–3] The influence of laser photocoagulation and/or vitreous haemorrhage resulting in retinal break formation remains controversial.[5]

Our case emphasises that vitreous haemorrhage in eyes with BRVO should alert the examiner to closely monitor the situation since traction on neovascular fronds may lead to retinal breaks and consequent retinal detachment.

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The authors have no proprietary interest in this work

*Eye* (2006) **20,** 1326–1327. doi:10.1038/sj.eye.6702195; published online 9 December 2005

Sir,

**Bilateral acute retinal necrosis syndrome following herpes simplex type 1 encephalitis**

Acute retinal necrosis syndrome (ARN) associated with herpes simplex viral encephalitis has been well described. Bilateral acute retinal necrosis (BARN) in this context is described but is extremely rare. We present a case of BARN following recent herpes simplex type 1 (HSV-1) encephalitis (HSE) in an immune competent patient.

*Case report*

A 59-year-old man presented to the ophthalmologist complaining of reduced visual acuity of 6/12 in his left eye. ARN was not recognised until he represented 2 weeks later with further decline in his left eye vision to hand movements and right eye vision reduced to 6/18. On recognition of BARN he was referred to our unit for further management. On arrival, he had bilateral florid, granulomatous uveitis and optic disc swelling. There was widespread bilateral retinal vasculitis and 360° confluent retinitis. In the left eye, the retinitis extended up to the disc nasally and there was widespread retinal detachment extending to the temporal arcades, Figure 1.

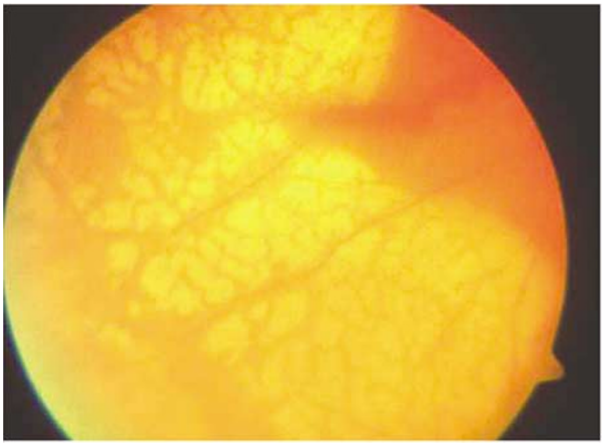

**Figure 1** Colour fundus photograph showing retinal necrosis in the periphery of the left eye.

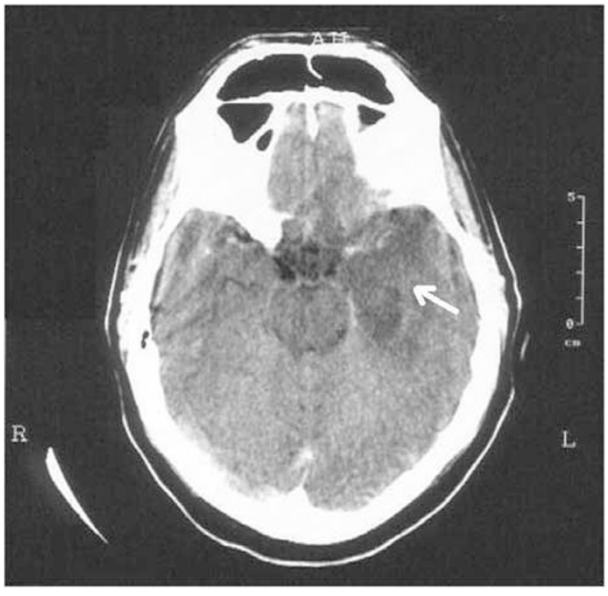


**Figure 2** CT scan showing low attenuation in the left temporal lobe.

At 1 month previously, the patient had been admitted with malaise, fever, and speech disturbance. CT imaging showed a low attenuation area in the left temporal lobe, Figure 2 and HSV-1 was detected on polymerase chain reaction (PCR) testing of a cerebrospinal fluid sample. He was treated with intravenous aciclovir 10 mg/kg TDS for 2 weeks and oral corticosteroid.

On admission to our unit, he was restarted on intravenous aciclovir 10 mg/kg TDS. He underwent left eye vitrectomy, 360° laser retinopexy, and silicone oil tamponade. The right eye also received prophylactic 360° laser retinopexy. Both eyes received intravitreal foscarnet, 2.4 mg in 0.1 ml. The patient was maintained on intravenous aciclovir for 14 days. Oral prednisolone was introduced after 48 h of intravenous aciclovir for his severe intraocular inflammation. PCR studies of the left vitreous sample were positive for HSV-1 and negative for other herpetic viruses. Lymphocyte surface markers, immunoglobulins and HIV testing were all normal or negative.

The patient was discharged on oral valaciclovir 1 g TDS in addition to a reducing dose of oral steroid. After 2 weeks, the retinal detachment in the left eye recurred in spite of silicone oil tamponade and he also developed a temporal, peripheral retinal detachment in the right eye. At 4 months, the retinal detachments are limited to the anterior edge of the laser barricade and do not extend across the temporal arcades in either eye. His vision is unchanged from discharge at 6/5 right eye, CFs left eye.

We plan to continue valaciclovir for at least 6 months.

**Comment**

ARN is a rapidly progressive retinal infection caused by herpes viruses. It usually occurs in immunocompetent patients. Although most cases are unilateral, bilateral disease has been reported in up to 35% of patients.[1] This figure is probably determined by early recognition and treatment of first eye involvement.[2] Acute retinal necrosis is a clinical diagnosis and standard diagnostic criteria have been published by Holland *et al*.[3] VZV and HSV-1 are associated with acute retinal necrosis in the older population while HSV-2 is the more common pathogen in under 25-year olds.[4]

We have found 20 cases of unilateral ARN associated with HSE in the absence of human immune deficiency virus in the literature in the last 20 years.[1,4–16] Pepose *et al*[15] reports a case of HSE 3 weeks following ARN. All others report ARN following HSE with variable time courses (mean of 6/12 years, median 5 years, range 10 days–20 years). BARN following HSE could only be found in seven case reports.[17–22] The majority of patients in the unilateral ARN group had no other factors which

might be associated with impaired host defence. In contrast, the reported cases of BARN following encephalitis all had some associated factors that may have reduced host defence. In two of the seven case reports, the initial encephalitis was either not recognised or there was a significant delay in treatment.[17] Two report association with high-dose intrathecal or pulsed intravenous steroid.[18,19] One case occurred during iatrogenic immunosuppression with chemotherapy agents[20] and a further case followed craniotomy and oral dexamethasone for a supra sellar lesion.[21] Finally, there was one case of HSV-2 in a neonate.[22] In our case, the only possible compromising factor was administration of dexamethasone over a 6-day period. However, this was given in addition to aciclovir and might be regarded as a standard part of his encephalitis therapy. In this respect, our patient is unique to others reported in the literature with BARN following HSE.

Prompt treatment of HSE is essential as there is a high mortality and morbidity. Early treatment may be important for limiting reactivation or neuronal translocation that could otherwise lead to further virus spread to other CNS sites including the eye. Standard antiviral treatment regimens do not usually extend beyond 14 days.[23] As ARN is potentially blinding with a retinal detachment rate of between 50 and 75%,[24] we question this short-term approach. In general, BARN tends to occur quite soon following encephalitis with most reported within 5 months and only one reported after 42 months.[18] We suggest that oral antiviral agents should be introduced after IV therapy for at least 6 months following HSE to reduce the risk of acute retinal necrosis. Oral valaciclovir, a prodrug of aciclovir has excellent bioavailability and is very well tolerated.[25] Patients with HSV encephalitis should be examined carefully for ocular disease. Patients who have associated factors that may affect host defence should be viewed with a high index of suspicion. Physicians should request immediate specialist review of any patient with HSE developing signs or symptoms of ocular inflammation. Thorough peripheral retinal examination is particularly advised for any patient presenting with uveitis and a previous history of HSE.

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*Eye* (2006) **20,** 1327–1330. doi:10.1038/sj.eye.6702196; published online 13 January 2006

Sir,

**Massive haemorrhagic retinal detachment after transpupillary thermotherapy for choroidal neovascularisation**

Transpupillary thermotherapy (TTT) has been successfully used for treatment of subfoveal choroidal neovascularisation (CNV).[1] Safety however remains a concern, given its narrow therapeutic window.[2] We report a haemorrhagic retinal detachment (HRD) complicating TTT for CNV.

### *Case report*

A 75-year-old diabetic, hypertensive, and cardiac man complained of poor vision OD for 2 months. Best-corrected visual acuity was 6/18 OD, and 6/6 OS. Anterior segment OU was unremarkable except bilateral pseudophakia. Fundus examination OD showed a large CNV with serous macular detachment and subretinal haemorrhage (Figure 1a). There was no evidence of age-related macular degeneration (AMD), or polypoidal choroidal vasculopathy (PCV). The left fundus was normal. Fundus fluorescein angiogram OD showed a predominantly occult CNV (Figure 1b). Indocyanine-green angiography ruled out PCV.

The options of observation, PDT or TTT were offered. With patient's informed consent and approval of the institutional review board, TTT was performed with a slit-lamp mounted 810-nm diode laser. Threshold power was determined with an inferonasal test spot. The lesion was treated with five 500–600 mW spots, each 3 mm, lasting 1 min. Power was reduced by 10% over the haemorrhage; increased similarly over serous detachment. No retinal blanching occurred.

The patient's vision suddenly dropped to hand motions 10 days post-TTT. Fundus examination revealed a massive posterior HRD (Figure 1c). Haematological investigations, cardiac, and carotid status were normal; he was not on anticoagulants. B-scan ultrasonography confirmed clinical findings; there was no suprachoroidal haemorrhage. Afterc 6 weeks, vision remained poor; fundus was not visible. Ultrasound revealed breakthrough vitreous haemorrhage, with reduced height of HRD (Figure 1d). The patient did not follow-up subsequently. When contacted a year later, he reported no change in visual status.

### Comment

AMD-related CNV may rarely bleed spontaneously at the stage of disciform scar, predisposed by systemic anticoagulants, hypertension, and cardiovascular disease.[3] Idiopathic CNV has a better prognosis; but older patients and larger lesions fare worse, irrespective of the baseline acuity.[4] In our patient, HRD was probably caused by TTT-induced vasodilatation, and subsequent closure,[2] resulting in dehiscence of choroidal vessels, weakened by age, hypertension, atherosclerosis, and diabetes. The use of multiple burns might also have contributed to HRD, by excessive thermal damage at the overlapping edges. However, we did not observe any visible retinal whitening, popping sound, or patient discomfort during or after TTT. Most patients with occult CNV have lesions larger than two disc areas,[5] and therefore require either multiple spots;[6] or large-spot TTT.[7,8] There is no evidence in the literature of the superiority of one technique over the other. Similar to

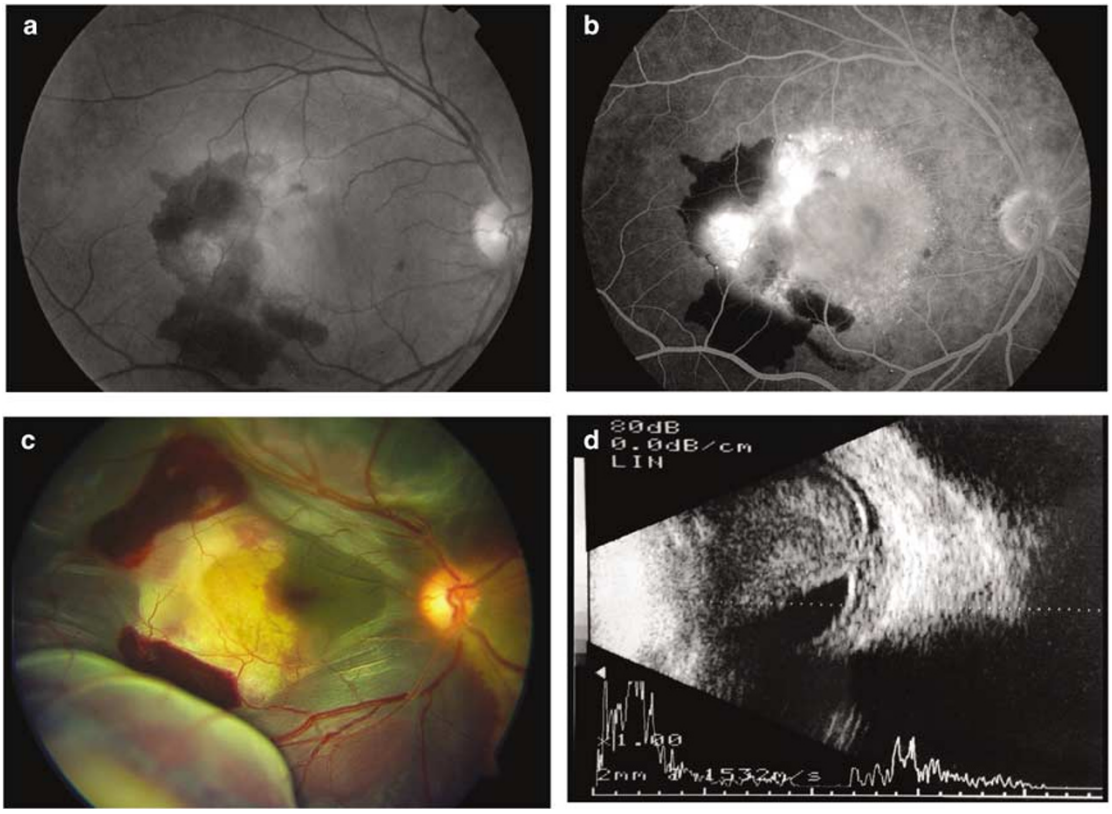


**Figure 1** (a) Fundus photograph OD showing a large area of choroidal neovascularisation (CNV) with serous macular detachment, and subretinal haemorrhage temporally. (b) Late-phase fluorescein angiogram reveals hyperfluorescence from the occult submacular CNV with a small temporal classic component; and blocked fluorescence temporally due to subretinal haemorrhage. (c) At 10 days after transpupillary thermotherapy (TTT), an extensive haemorrhagic retinal detachment was noticed, involving the posterior pole and temporal midperiphery, with organised subretinal blood temporal to fovea, and preretinal haemorrhage. (d) B-scan ultrasonography, 6 weeks after the complication, showing breakthrough vitreous haemorrhage with reduced height of the haemorrhagic detachment.

multiple spots, a large spot is also likely to deliver excess power by increased uptake in areas with subretinal blood/pigment, which we could avoid by titrating the power of individual burns.

We report a hitherto-undescribed complication of TTT. The older patients undergoing TTT for idiopathic CNV should be cautioned about the small risk of severe visual loss, especially when pretreatment acuity is good, and CNV is extensive.

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*Eye* (2006) **20,** 1330–1332. doi:10.1038/sj.eye.6702198;
published online 25 November 2005

Sir,

**Expanding role of local anaesthetia in vitreoretinal surgery**

We read with interest the paper by Costen *et al*,[1] regarding the expanding role of local anaesthesia (LA) in vitreoretinal (VR) surgery. The study involved 1003 patients undergoing VR surgery, of whom 920 (91.7%) had LA. They concluded that 'careful patient selection, together with the use of sedation when necessary, should ensure that the routine use of LA for VR surgery continues to become more acceptable to patients and medical staff alike'. Two of us have visited the Southampton Eye Unit and were very impressed with their VR service, but nevertheless we have some reservations about the conclusions of this study.

Every patient was under the care of a single consultant anaesthetist who works full time in ophthalmology. No doubt his considerable experience is responsible for the excellence of the blocks. However, this arrangement is impractical in many other hospitals. Anaesthetist staff may need to maintain skills in all areas of anaesthesia, if for no other reason than to carry out their on-call duties competently. Also, with less-experienced anaesthetist staff there will inevitably be a greater recourse to GA. Presumably all the LA surgery took place during routine surgical sessions. In other units when this is not possible, it would again increase the likelihood that a GA will be preferred.

The authors comment that 'in-patient beds are increasingly under pressure and hence general anaesthetic services are often stretched'. There is an implication here that the move to LA is in part driven by necessity rather than choice, and this may well be a factor in other units moving toward an increase in LA rates.

We note that top-up anaesthesia by sub-tenon's injection was required in 5% of cases overall, and sedation was used in 20.2%. In this unit we have tended to avoid sedation because of concerns that the patient may not always be alert enough to cooperate fully during surgery.

We are puzzled by the inclusion of patients undergoing retinopexy without vitrectomy (group 2 'retinopexies with or without vitrectomy') GA would not normally be considered for such patients. Their inclusion perhaps lessens the impact of the headline LA rate.

There is no doubt that many patients are better served with LA vitrectomies than GA, especially insulin-dependent diabetics and those in poor general health. However, we do not necessarily see it as desirable to strive for the high LA rate that is advocated in this study. After all, a GA offers a painless alternative to the LA block, and recovery these days is quick enough to make it perfectly feasible for day case surgery.[2] The greater degree of relaxation in teaching juniors, the avoidance of needles in close proximity to the eye, and the ability to treat the fellow eye, are obvious additional advantages of a GA.

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*Eye* (2006) **20,** 1322. doi:10.1038/sj.eye.6702199;
published online 2 December 2005

Sir,

**Reply to Gray *et al***

We note with interest the comments made in the letter from Gray *et al*, and thank the authors for their interest in our paper.

With reference to our use of anaesthetists, not all of our vitreoretinal cases have LA administered by one anaesthetist. The data we presented were indeed from one anaesthetist (ACW), but this does not represent our entire vitreoretinal workload. Other anaesthetists, both consultant and trainees, are assigned to the Eye theatres and as such have good exposure to ophthalmic anaesthesia.

In addition, much of our out of hours vitreoretinal work is also undertaken using LA, but these data are not included in this series. In cases where an untrained anaesthetist is present, the block is given by the surgeon and when no anaesthetist is available, sub-tenons anaesthesia is used.

With regard to our use of sedation, discussed in paragraph 4, we have not encountered any significant clinical difficulties or problems with patient cooperation and have found it to be extremely useful in some patients. We report high patient satisfaction rates in our paper[1] and in our opinion sedation is a useful adjunct in selected cases.

Ophthalmology is becoming an increasingly day-case oriented specialty. Many ophthalmic units face pressure over use of inpatient beds. While it is true that GA can be administered on a day-case basis, the absence of beds in which to recover patients adds pressure to the service and may result in elective cancellations. This is unfortunately a reality, and as such will of course be a factor driving a predominantly LA service. This apart, we find LA to be highly acceptable to both patients and staff alike.

Only 6% of patients (39/518) undergoing retinopexy (with or without vitrectomy) had GA.[1] This therefore has little effect on our reported LA rate, as suggested by Gray *et al*. Our previous work involved taking the opinion of the patient, who being recovered by the anaesthetist, had no preconceived surgical opinion on what does or does not hurt. We found that the laser and cryopexy were more important determinants of discomfort during vitrectomy than other aspects of the surgery, and so these were analysed as one group.[2] While we would agree with Gray *et al* that most retinopexy would not require GA, there are occasional anxious patients who have had failure of treatment at the slit-lamp, and for whom good anaesthesia is as vital as it is in a vitrectomy for retinal detachment.

In response to the comments in paragraph 6, by Gray *et al*, regarding patient comfort and training issues, we would like to draw attention to the findings in our paper. We noted high patient satisfaction rates, both with the anaesthetic injection and the procedure. We had no cases of globe perforation. Teaching cataract surgery under LA is an experience we have all been through and the principles, when applied to VR surgery are just the same. We have an active vitreoretinal teaching program for both specialist registrars and fellows. We have found LA to be perfectly acceptable for teaching, as many of the procedures reported in our series were performed by trainees.

With regard to examination of the fellow eye, we would agree with Gray *et al* that LA does pose a disadvantage here. What is not known is on how many occasions fellow eye treatment is needed, and whether or not it could easily be administered as an outpatient, or indeed the acceptability of another LA for the patient.

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*Eye* (2006) **20,** 1332–1333. doi:10.1038/sj.eye.6702200; published online 20 January 2006