Mixed infection (Pseudomonas and coagulase negative staphylococci) microbial keratitis associated with extended wear silicone hydrogel contact lens

Contact lens induced ulcerative keratitis is a serious complication which can be devastating for the patient if treatment is delayed. Extended wear is the commonest cause of microbial keratitis in contact lens wear.1 New extended wear silicone hydrogel contact lenses have higher oxygen transmissibility so that they can be worn continuously for 30 days. They can also be used as bandage contact lenses.

The risk of Pseudomonas microbial keratitis with overnight wear is significantly increased by contact lenses with low oxygen transmissibility.2 By virtue of high oxygen transmissibility, the silicone hydrogel contact lenses are thought to be associated with low risk of infectious keratitis.3,4 So far only four cases of microbial keratitis have been reported with their use.5 In spite of various claims of protection against serious microbial keratitis with pathogens such as P aeruginosa, we have recently across the first case of Pseudomonas keratitis in a patient wearing silicone hydrogel contact lenses.

Case report
A 23-year-old male patient presented with 1 day history of severe pain, ocular injection, photophobia, and reduced vision of right eye. He was wearing the day and night silicone hydrogel contact lenses, which was replaced once every 30 days (Ciba vision Focus day and night). He has been wearing these contact lenses for 7 months before the presentation.

Examination revealed a visual acuity of hand movement for the right eye and 6/5 for the left eye. The right eye had a central corneal ulceration of 3 mm in diameter surrounded by severe oedema and a 1 mm hypopyon. Cultures grew P aeruginosa and coagulase negative staphylococci both sensitive to ciprofloxacin and gentamicin. Topical ofloxacin and gentamicin were commenced with cyclopentolate. Unpreserved prednisolone eye drops (0.5%) were added after 1 week. Two weeks later, the epithelial defect points towards a multifactorial aetiology with central subepithelial corneal scar (fig 1). His vision improved to 6/18 unaided, 6/9 through the pinhole, 1 month after the admission.

Comment
The major barrier to prescribing a continuous wear contact lens is a perceived danger of microbial keratitis. Many factors are involved in the development of microbial keratitis and these include bacterial adherence to the lens surface, formation of bacterial glycoalyx on the lens, corneal hypoxia, deposits on the lens surface, and the effect of contact lens on closed eye environment.6 Silicone hydrogel contact lenses have high oxygen transmissibility and these lenses are colonised by similar numbers and type of micro-organisms compared with HEMA based materials.7 A number of studies have shown lower risk of infectious keratitis with new silicone hydrogel contact lenses.8

However, the use of silicone hydrogel contact lenses was associated with slightly higher levels of visible deposits,9 which may act as a risk factor for bacterial keratitis. As in our case young male patients were also considered a risk factor for contact lens induced microbial keratitis. Our experience suggests that extended wear silicone hydrogel contact lenses are not free of the risk of more serious microbial keratitis caused by P aeruginosa and coagulase negative staphylococci. With increasing popularity among optometrists and the use of silicone hydrogel contact lens as a bandage contact lens, such a serious complication cannot be ignored.

As suggested by other authors,10 our experience suggests that continuous wear and the use of an advanced image analysis system.11 Whichever method of count is used, precision depends primarily on good visualisation of the cell borders. It has long since been shown that, even under experimental conditions of perfect cell membrane visualisation using alizarin red, maximum precision ranges from +5% to −5%.12 Two commercial media are authorised by the Agence Francaise de Sécurité Sanitaire des Produits de Santé. They are very similar in composition, both being based on HEPES-buffered Iscove’s Modified Dulbecco’s medium containing sodium bicarbonate and 2% fetal bovine serum, with the same pH of 7.25 but the osmolality of Inosol (Bausch & Lomb, Chauvin-Opsia, Labège, France) is only 305 mosmol/kg (range 295–315) compared with 320 mosmol/kg (range 300–340) for CorneaPrepMax (Eurobio, Les Ulis, France). One has nevertheless acquired the reputation of allowing better visualisation of endothelialkeratitis in contact lens wear.7 Continuous wear of high oxygen transmissibility soft silicone hydrogel contact lenses is a randomised one-year study of 504 patients.8

Controlled study of the influence of storage medium type on endothelial assessment during corneal organ culture
Selection of corneal grafts in eye banks is mainly based on end-of-storage endothelial assessment, which consists of endothelial cell density (ECD) measurement and, to some extent, cell morphology. Below a certain ECD threshold, generally 2000 cells/mm², the cornea is deemed unfit for penetrating keratoplasty. Precise ECD measurement at the end of storage is thus a key issue for eye banks, and also for patients, because it influences the long-term survival of the graft.13

For long-term storage in organ culture, the most common method in Europe,14 endothelial observation is possible only by transmitted light microscopy. The endothelial cells are exposed to 0.9% sodium chloride or sometimes to 1.8% sucrose, with a small degree of osmotic cell shrinkage and dilatation of the intercellular spaces thus making individual cells visible. The cells can be counted manually, through a calibrated reticle or from a photograph, or using an automated image analysis system.15 Whichever method of count is used, precision depends primarily on good visualisation of the cell borders. It has long since been shown that, even under experimental conditions of perfect cell membrane visualisation using alizarin red, maximum precision ranges from +5% to −5%.16 Two commercial media are authorised by the Agence Francaise de Sécurité Sanitaire des Produits de Santé. They are very similar in composition, both being based on HEPES-buffered Iscove’s Modified Dulbecco’s medium containing sodium bicarbonate and 2% fetal bovine serum, with the same pH of 7.25 but the osmolality of Inosol (Bausch & Lomb, Chauvin-Opsia, Labège, France) is only 305 mosmol/kg (range 295–315) compared with 320 mosmol/kg (range 300–340) for CorneaPrepMax (Eurobio, Les Ulis, France). One has nevertheless acquired the reputation of allowing better visualisation of endothelial

References
4 Nilsson SEG. 7-Day extended wear and 30-day continuous wear of high oxygen transmissibility soft silicone hydrogel contact lenses. A randomised one-year study of 504 patients.9

Figure 1 Photograph taken 3 weeks after admission showing central corneal scar.
Table 1 Qualitative grading of the endothelial images

<table>
<thead>
<tr>
<th>Image quality</th>
<th>Score</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>2</td>
<td>Excellent view of cell borders; low background noise; cells visible on over 2/3 of image area</td>
</tr>
<tr>
<td>Average</td>
<td>1</td>
<td>Good view of cell borders; moderate background noise; cells visible on 1/3 to 2/3 of image area</td>
</tr>
<tr>
<td>Poor</td>
<td>0</td>
<td>Poor view of cell borders; high background noise; cells visible on less than 1/3 of image area</td>
</tr>
</tbody>
</table>

For the analyses in tri-image mode, overall quality was graded “good” if the three images obtained scores of 2/2/2 or 2/2/1, “average” if the scores were 2/1/1, 2/1/0, or 1/1/1, and “poor” if the scores were 1/1/0, 1/0/0, or 0/0/0.

The three images selected by the analyser were qualitatively assessed on a scale of three (Table 1) by three independent observers masked to storage medium. Discordant cases were reviewed.

The normality of the data distribution was tested using both the Lilliefors variant of the Kolmogorov-Smirnov test and Shapiro-Wilk normality test, with the cut off for non-normality set at p = 0.05. The quantitative variables (number of cells “reliably recognised”, ECD, touch up duration) were compared using a paired t test in the case of normal distribution, and otherwise by a non-parametric test (Wilcoxon). The image quality scores were compared by the χ² test in a 3x2 grid. p < 0.01 was deemed significant.

Table 2 Initial examination of paired corneas stored in medium A and B

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 30)</th>
<th>Group B (n = 30)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Image quality (good/average/poor, %)</td>
<td>56/27/17</td>
<td>43/30/27</td>
<td>0.526</td>
</tr>
<tr>
<td>Cells well recognised per se, “automatic” mode (n)</td>
<td>298 (68) 147–417/304</td>
<td>275 (89) 113–417/284</td>
<td>0.058</td>
</tr>
<tr>
<td>ECD, “automatic” mode (cells/mm²)</td>
<td>3267 (197) 2797–3672/3250</td>
<td>3248 (305) 2445–3753/3249</td>
<td>0.570</td>
</tr>
<tr>
<td>Cells counted, “touch up” mode (n)</td>
<td>5415 (126) 278–973/521</td>
<td>519 (259) 314–1777/519</td>
<td>0.579</td>
</tr>
<tr>
<td>ECD, “touch up” mode (cells/mm²)</td>
<td>3113 (341) 2343–4008/3107</td>
<td>3147 (391) 2461–3893/3190</td>
<td>0.533</td>
</tr>
<tr>
<td>Coefficient of variation of cell area, “touch up” mode (%)</td>
<td>29.1 (4.3) 21.2–47.2/28.1</td>
<td>29.1 (4.3) 21.7–49.6/29.1</td>
<td>0.986</td>
</tr>
<tr>
<td>Hexagonality, “touch up” mode (%)</td>
<td>53.9 (9.4) 36.0–75.0/53.2</td>
<td>51.6 (9.0) 34.8–69.6/51.9</td>
<td>0.212</td>
</tr>
<tr>
<td>Touch up duration (seconds)</td>
<td>343 (131) 129–560/334</td>
<td>406 (166) 171–721/324</td>
<td>0.006</td>
</tr>
</tbody>
</table>

Results were expressed as mean (standard deviation), minimum–maximum/median. The automatic analysis mode provides reliable results compared to the more time consuming touch up one. This allowed, in our eye bank, to decide in a few moments whether to continue with organ culture.
medium than in Inosol. The former facilitates ECD measurement at delivery, the main parameter of corneal quality of control. Our study shows that: (1) prolonged storage in CorneaPrep/Max caused no deterioration in image quality, unlike that with Inosol. Better visualisation of cell borders at delivery shortened touch up durations considerably, on average by three minutes per computerised analysis. This point should be particularly relevant for the eye banks which perform endothelial assessment only once, generally after 10–15 days of organ culture.3 (2) At the end of organ culture, the corneas stored in CorneaPrep/Max responded better to osmotic dilation of the intracellular spaces than those stored in Inosol. There was no such discrepancy at the start of storage. We may assume that this difference in behaviour is due to the increased osmolality of CorneaPrep/Max, which is 15 mosm/l greater than that of Inosol. During organ culture, the very gradual change of ionic content between the cells and/or of the intercellular junctions may increase water egress from the cells and thus promote dilation of the intracellular spaces in the presence of 0.9% sodium chloride. However, it is likely that over three days these changes did not have time to occur, which would explain the lack of initial differences in image quality. Further histological study could confirm the nature of these changes in the cells and/or their junctions but whatever their nature, they do not affect viability.

In 2001, the twenty one French eye banks stored on average 292 corneas (range 32–846).2 Because of their small size, the eye banks naturally prefer commercial organ culture media to their own preparations. The two media authorised for use in France are very similar in composition; neither is stored on average 292 corneas (range 32–846). Because of their small size, the eye banks naturally prefer commercial organ culture media to their own preparations. The two media authorised for use in France are very similar in composition; neither is

Table 3 Final examination of paired corneas stored in medium A and B

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A (n = 30)</th>
<th>Group B (n = 30)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Image quality</td>
<td>0.237</td>
<td>0.438</td>
<td>0.14</td>
</tr>
<tr>
<td>Cells well recognised per se, “automatic” mode</td>
<td>0.159</td>
<td>0.342</td>
<td>0.24</td>
</tr>
<tr>
<td>ECD, “automatic” mode (cells/mm²)</td>
<td>3.143 (159) 2829</td>
<td>3.453 (94) 3029</td>
<td>0.011</td>
</tr>
<tr>
<td>Cells counted, “touch up” mode</td>
<td>0.357 (72) 2.095</td>
<td>0.399 (79) 0.456</td>
<td>0.001</td>
</tr>
<tr>
<td>ECD, “touch up” mode (cells/mm²)</td>
<td>2.708 (130) 2.727</td>
<td>2.374 (130) 2.721</td>
<td>0.934</td>
</tr>
<tr>
<td>Overall cell loss, “touch up” mode</td>
<td>12.6 (9.3) 1.7</td>
<td>31.1 (10.8) 1.3</td>
<td>0.079</td>
</tr>
<tr>
<td>Coefficient of variation of cell area, “touch up” mode</td>
<td>28.4 (3.3) 23.7</td>
<td>27.2 (3.4) 21.7</td>
<td>0.098</td>
</tr>
<tr>
<td>Hexagonality, “touch up” mode</td>
<td>0.40/27.8</td>
<td>0.34/20.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Touch up duration (seconds)</td>
<td>531 (134) 361</td>
<td>388 (120) 226</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Results were expressed as mean (standard deviation), minimum–maximum/median. The automatic analysis was less relevant at delivery than at receipt (see Table 1), consequently, a touch up analysis should be recommended.

**References**


**Prospective case control study on genetic association of apolipoprotein e2 with intraocular pressure**

Glaucomas are a leading cause of blindness throughout the world. This group of diseases has a common characteristic: degeneration of the optic nerve that is usually associated with increased intraocular pressure (IOP). Increased IOP is one of the major risk factors for developing glaucomatous damage, whereby the loss of retinal ganglion cells is the typical pathological finding. However, the pathophysiology of pressure induced glaucomatous optic neuropathy remains unclear and is still a matter for debate. Genome scans have been performed to identify the genomic locations of glaucoma susceptibility genes. 2, 3

Apolipoprotein E (APOE), a lipid transporting protein produced in the liver and brain, is unique among apolipoproteins in that it has particular relevance to nervous tissue. It is involved in the mobilisation and redistribution of cholesterol in repair, growth, and maintenance of myelin and neuronal membranes during development or injury. Recently it has been shown that the APOE e4 allele is associated with elevated risk of normal tension glaucoma. 4, 5 The APOE e2 allele was shown to be significantly associated with an elevated risk of age related macular degeneration (AMD). 6

**Material and methods**

This prospective case control study included 32 controls (IOP <22 mm Hg, normal optic disc, normal visual field), 54 patients with ocular hypertension (OHT, IOP >21 mm Hg, normal optic disc, normal visual field), 96 patients with primary open angle glaucoma (POAG), 55 patients with preperimetric open angle glaucoma (pre-OAG, IOP >21 mm Hg, glaucomatous optic disc, normal visual field), and 41 patients with perimetric open angle glaucoma (OAG, IOP >21 mm Hg, glaucomatous optic disc, visual field defects). All individuals included in the study were unrelated, white, and had open anterior chamber angles, clear optic media, and a visual acuity of 20/25 or better. Exclusion criteria were all eye diseases other than glaucoma, diabetes mellitus, myopic refractive error exceeding −8 dipters, and visual acuity less than 0.7.

The study followed the tenets of the declaration of Helsinki for research involving human subjects and informed consent was obtained from all participants.

All control subjects and patients were thoroughly examined by optical biomicroscopy including slit lamp inspection, gonioscopy and ophthalmoscopy, and
Table 1 Distribution of APOE genotypes and allele frequency

<table>
<thead>
<tr>
<th>Frequency in APOE characteristic (n)</th>
<th>Mean age in years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal 32 (17)</td>
<td>54.8</td>
</tr>
<tr>
<td>OHT 54 (27)</td>
<td>53.3</td>
</tr>
<tr>
<td>Pre-OWG 55 (25)</td>
<td>54.4</td>
</tr>
<tr>
<td>OWG 41 (20)</td>
<td>56.3</td>
</tr>
<tr>
<td>Σ</td>
<td></td>
</tr>
<tr>
<td>Genotype</td>
<td></td>
</tr>
<tr>
<td>e2/e2</td>
<td>0</td>
</tr>
<tr>
<td>e2/e3</td>
<td>2</td>
</tr>
<tr>
<td>e2/e4</td>
<td>6</td>
</tr>
<tr>
<td>e2/e4</td>
<td>14</td>
</tr>
<tr>
<td>e2/e4</td>
<td>9</td>
</tr>
<tr>
<td>e2/e4</td>
<td>0</td>
</tr>
<tr>
<td>Allele frequency</td>
<td></td>
</tr>
<tr>
<td>e2</td>
<td>9</td>
</tr>
<tr>
<td>e3</td>
<td>40</td>
</tr>
<tr>
<td>e4</td>
<td>15</td>
</tr>
</tbody>
</table>

To conclude, we have shown a significant association between APOE and glaucoma and IOP. Quite recently it was argued that an IOP reduction of 1 mm Hg from baseline will decrease the risk of progression by about 10%. Although in need of confirmation, our data emphasize the role of APOE in regulation of IOP and may indicate that we have identified a susceptibility gene for glaucoma.

As a future perspective for the APOE alleles, analysis of a larger number of glaucoma patients—taking into account family history, age, and sex—will provide more detailed insight.

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Accepted for publication 7 June 2003

References

The safety of anterior chamber paracentesis in patients with uveitis

Anterior chamber (AC) paracentesis is a valuable procedure in the management of uveitis, particularly in diagnosing infective causes. It may also be used therapeutically to lower intraocular pressure and it provides samples for clinical research. Nevertheless, there have been isolated reports of AC paracentesis related serious complications, including endophthalmitis and corneal abscess. As the risk of trauma to the iris and lens are also major concerns, AC paracentesis is often used with reluctance.

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Although there are many studies on the analysis of aqueous humour obtained from AC paracentesis, our literature search showed only one publication on the safety of AC paracentesis. The purpose of this study was to describe a method of AC paracentesis that can be easily performed as an outpatient procedure with the patient sitting at the slit lamp.

Methods and results
A total of 70 patients (41 male, 29 female) aged 18–85 years (median 39 years) with various types of active uveitis attending the Birmingham and Midland Eye Centre underwent AC paracentesis. Fourteen paracenteses were performed for diagnostic purposes while the remainder for experimental analysis as part of another study. Patients with dilated pupils and undilated pupils were included. Local research ethics committee approval and informed consent was obtained.

Benoxinate 0.4% eye drops (minims) were instilled three times over a 3 minute period, followed by instillation of beta-blocker 5% anti-septic solution that had been drawn up into the empty benoxinate minium container. The patient was positioned at the slit lamp, the upper lid and eyelashes held out of the way by an assistant. No lid speculum was required.

Of the 70 paracenteses, 48 were performed using a 27 gauge needle attached to an insulin syringe, while the remaining 22 were performed using an aqueous pipette. Where a 27 gauge needle was used, this was inserted at the paralimbal clear cornea in a plane above and parallel to the iris with the bevel of the needle facing forward until the whole bevel penetrated the cornea (fig 1). Under direct vision, the sampler pulled the plunger of the syringe to aspirate the aqueous. The aqueous pipette (Visitec, Sarasota, FL, USA designed by O’Rourke) consists of a short 30 gauge needle mounted inside plastic tubing, which in turn is connected to a soft polyethylene suction-infusion bulb. The bulb was squeezed to create a vacuum and the needle inserted at the limbus as described above (fig 2). When pressure on the bulb was released, aqueous spontaneously filled the pipette. Using either method, the eye can be fixed with a pair of forceps at the opposite limbus, if necessary. After sampling an antibiotic drop was prescribed for three days. The whole procedure takes less than five minutes. All patients were re-examined 20 minutes after the procedure and 1–2 weeks later.

Two patients had an air bubble inadvertently injected into the AC. The visual acuity returned to normal at review in both cases. One patient developed an acute allergic conjunctivitis to betadine, which settled after treatment with prednisolone 0.1% eye drops. None of the 70 patients developed detectable corneal damage, lens changes, or endophthalmitis.

Comment
Various methods for performing AC paracentesis have been described. However, our literature search only identified one systematic report investigating the safety of AC paracentesis. This technique required the patient to lie supine under the microscope, needed insertion of a lid speculum and precincision of the cornea with a 15° micro sharp blade, and the aqueous was aspirated using a 27 gauge needle on a tuberculinc syringe. No serious complications were reported in 361 uveitis patients. A small hypophakia occurred in 3/72 (4.2%) patients examined 30 minutes after the paracentesis. The method described by O’Rourke using the aqueous pipette is relatively new and no systematic analysis of its safety profile has been published. Other methods for AC paracentesis include not using a syringe or a syringe with the plunger removed, thus avoiding any potential complications associated with aspirating with a plunger, but collecting the aqueous specimen may be technically difficult.

The cases of inadvertent injection of an air bubble into the AC both occurred using the aqueous pipette and was most likely caused by air trapped inside the bulb prior to inserting the pipette into the AC. We recommend ensuring the bulb is thoroughly depressed to evacuate all air before inserting the needle into the eye. Pressure on the bulb must also be maintained while the needle is being inserted, to avoid air entry.

Our study showed that performing AC paracentesis with the patient sitting at the slit lamp is safe using either the 27 gauge needle or the aqueous pipette. Precinision with a sharp blade and the use of a lid speculum is unnecessary.

Acknowledgements
We are grateful to Robert Harvey and Salaman Mirza for their help in this study.

References

Rapid recovery of night blindness due to obesity surgery after vitamin A repletion therapy

Night blindness is the most common and earliest symptom of vitamin A deficiency. The latter can be caused by general malnutrition, malabsorption of vitamin A, or impaired vitamin A metabolism due to liver disease.

Several surgical methods are currently used for the treatment of obesity. In the Scopinaro procedure, a biliopancreatic bypass is combined with a bypass of part of the small bowel, thus promoting intestinal malabsorption.

The fat soluble vitamin A can exist as retinol, its ester, and retinoic acid. It has several roles in ocular metabolism: it is essential for corneal and conjunctival epithelial cell RNA and glycoprotein synthesis, while retinal is the crucial chromophore which combines with both rod and cone opsins to form rhodopsin and activated cone opsins, which are essential for phototransduction.

Case report
A 39 year old man presented with a 6 month history of night blindness, progressing more rapidly in the past 2 weeks. Three years before he had undergone a partial gastrectomy and biliopancreatic derivation for morbid obesity (Scopinaro procedure). His mean body mass index (BMI) decreased from 30 kg/m² to 11 kg/m² 3 years later.

At presentation, visual acuity was 6/5 in both eyes with a spherical correction of +0.75 dioptres. Slit lamp examination and funduscopy were unremarkable in both eyes. Concentric narrowing in both eyes could be seen on Goldmann visual field (VF) analysis (fig 1A). Goldmann-Weeke dark adaptometry (DA)
showed a considerable decrease in sensitivity (fig 1B). Electro-oculography was subnormal before therapy with a lightPeak/darkTrough ratio of 166% for the right eye and 146% for the left eye (normal ratio >180%). ISCEV standard electroretinography showed only minimal residual scotopic responses in both eyes. The SRC of vitamin A before therapy was 14 μg/dl (normal range 30–80). Vitamin E levels (0.49 mg/dl; normal 0.5–1.8) and total protein levels were slightly subnormal. Vitamin B and vitamin D levels were normal.

Our patient was given 60 000 IU retinol/day and vitamin E 140 mg/day (Rovigon, Roche).

After only 3 days, partial normalisation of Goldmann VF’s occurred. After 3 days of vitamin A supplementation, scotopic ERG responses had already improved to one third of normal (fig 2). Subjectively, the patient reported a “sudden visual recovery” 3 days after initiation of therapy.

After 3 days of therapy the EOG Lp/Dt ratio returned to near normal.

Ten days after initiation of treatment, all ERG parameters returned to normal (fig 2). Complete normalisation of DA was also seen (fig 1B).

From day 36 Goldmann visual fields were considered to be normal. The ERG (fig 2) and EOG had completely normalised by then.

After 135 days of repletion therapy SRC of vitamin A was still only 26 μg/dl, while vitamin E levels returned to normal (0.6 mg/dl). Treatment was maintained.

Comment
Normal biliary secretion, fat absorption, dietary protein intake, and the presence of zinc are necessary for fat soluble vitamin absorption. Vitamin A has a major role in photoreceptor function because it combines, in the form of its 11-cis isomer, with photoreceptor opsins to form rhodopsin and activated cone opsins.

At presentation, the ERG in our patient showed a considerable decrease in rod and, to a lesser extent, in cone function.

After 3 days of vitamin A repletion a significant improvement in the scotopic responses was noted. All ERG responses normalised completely after only 10 days of therapy. This rapid recovery of all electrophysiological and clinical parameters indicates that vitamin A deficiency was still in the earlier stages. The lag between obesity surgery and symptoms can be attributed to the presence of considerable liver stores of vitamin A when surgery was performed.

Our patient was repleted with 15 times the recommended daily allowance (RDA) of retinol (RDA of retinol 4000 IU/day) and 12 times that of vitamin E (RDA of vitamin E 12 IU/day). Interestingly, vitamin E deficiency seems to decrease the amount of vitamin A which can be stored in the retina. Long term vitamin replacement therapy is essential after bilio-pancreatic derivation surgery of the Scopinaro type.

Only a limited number of reports have described cases of vitamin A deficiency following bowel surgery for obesity. In 1999 Smet et al described a case of night blindness and optic neuropathy after bilio-pancreatic bypass with normalisation of all electrophysiological parameters when retested after 10 months.

No cone dysfunction was reported. In all reports of vitamin A deficiency despite SRC well below those in our patient, the rod involvement is seen earlier and is more extensive than cone.

Figure 1  (A) Goldmann visual field analysis; although peripheral limits as tested with object V4 of Goldmann remained normal, only test object I4 was perceived more centrally, indicating loss of retinal sensitivity. On day 3 of therapy, the patient could already perceive test object I2 and I3 illustrating partial normalisation, while, unexpectedly, peripheral limits were more constricted. From day 36 Goldmann visual fields were considered to be normal. (B) Goldmann-Weekers dark adaptometry (DA) showed considerable decrease in sensitivity before repletion therapy; day 1 is at presentation, before treatment; considerable improvement seen on day 3, with complete normalisation on day 10.
ERG parameters were within normal limits, although amplitudes still increased up to day 22.

- Scotopic while b-waves were two thirds of normal in both eyes. The 30 Hz flicker responses were four fifths a-wave and 1/13 for b-wave in both eyes. Single flash cone responses still had normal a-waves, in the dark adapted eye were minimal, with amplitudes of approximately one quarter of normal for eyes. Amplitudes of oscillary potentials were only residual.

- Responses to a single bright white flash comparison at bottom. At presentation, only minimal residual scotopic responses were seen in both eyes. After 3 days of vitamin A supplementation, scotopic involvement.

In conclusion, our case proves that ERGs on day 1 (before treatment) and subsequent days as indicated; normal control for vitamin A from the retinal pigment epithelium.

In the early stages of vitamin A deficiency, the reasons for this remain obscure, although rods are known to be more dependent on availability of vitamin A from the retinal pigment epithelium. In conclusion, our case proves that refractive surgery of the Scopinaro type can induce vitamin A deficiency with progressive rod-cone dysfunction, as well as deficiencies of all fat soluble vitamins and low plasma proteins.

- PostScript 585

References


Incision-less frontalis suspension

Frontalis suspensions with alloplastic slings are well established. The thick eyebrow skin of infants is prone to scar formation. Forehead scars caused by frontalis suspension procedures can be problematic. We describe a technique of congenital ptosis surgery that avoids eyebrow incisions.

Surgical technique

This new procedure utilises a nylon monofilament suture for frontalis suspension. The nylon suture is passed in a circlage fashion via puncture wounds without making eyebrow incisions. Two puncture sites, approximately 10 mm apart, are marked 3 mm above the lash line centred over the area of desired maximal eyelid elevation. Another two puncture sites are marked above the eyebrow, appropriately in line with the lateral and medial canthus. The path of the circlage is marked out by joining the marked puncture sites. The eyelid and eyebrow are infiltrated with local anaesthetic with adrenaline (epinephrine). A Keith needle is dual threaded with a 4/0 Nylon and a 4/0 Vicryl suture. It is then passed from one eyelid puncture site towards the corresponding eyebrow exit site in a suborbicularis plane (fig 1, top left) with the globe protected by a lid guide. From this site, the needle is passed through the needle track to the adjacent eyebrow puncture site (fig 1, top right) and then down towards the remaining eyelid puncture site. At this point in the procedure, the ends of the Nylon and Vicryl sutures emerge through the two eyelid puncture sites. The two ends of the Vicryl.

Figure 2. ERGs on day 1 (before treatment) and subsequent days as indicated; normal control for comparison at bottom. At presentation, only minimal residual scotopic responses were seen in both eyes. Amplitudes of oscillatory potentials were only residual. Responses to a single bright white flash in the dark adapted eye were minimal, with amplitudes of approximately one quarter of normal for a-wave and 1/13 for b-wave in both eyes. Single flash cone responses still had normal a-waves, while b-waves were two thirds of normal in both eyes. The 30 Hz flicker responses were four fifths of normal in right eye and normal in left eye. After 3 days of vitamin A supplementation, scotopic responses improved to one third of normal in both eyes. Ten days after initiation of treatment, all ERG parameters were within normal limits, although amplitudes still increased up to day 22.
suture are then manoeuvred in a sawing fashion to create friction to release skin dimpling at the eyebrow exit sites (fig 1, bottom left). The Vicryl suture is then removed and the Nylon suture needle (SH needle) is passed from one eyelid puncture site to another via a deep, partial thickness tarsal passage with the eyelid everted to ensure no full thickness penetration (fig 1, bottom right). The two ends of the Nylon suture, exiting at one eyelid puncture site, are tied and the tension adjusted to achieve the desired lid elevation and contour. Occasionally, peaking of the eyelid occurs and can be managed by slightly enlarging the puncture site at the tight suture end with a Westcott scissors and gentle spreading to undermine the soft tissues around the suture. This undermining action helps to release the suture tension on the puncture site to smooth out the lid contour but should be done carefully to avoid cutting the suture. The puncture sites usually do not require closure.

Comment
We performed this surgery on three infants with visually significant congenital ptosis. The mean age and follow up period of the infants were 5.6 months and 6.9 months respectively. The visual axis was cleared in all patients as measured by an improvement of their margin reflex distance one (MRD1). The lid contour was good in all patients. An example is illustrated in figure 2. There were no intraoperative or postoperative complications. The eyelid puncture sites healed without visible scar.

This minimally invasive surgery is scarless and can be performed with little trauma to the orbicularis oculi muscle. We realise that the results of frontalis suspension using alloegenic material are not permanent and may be associated with late failures. However, this is a simple, safe, temporary measure that elevates the eyelid for visual development until the child is old enough for definitive surgery using autologous or banked tissues.

References

Spontaneous resolution of sixth nerve palsy with ipsilateral cavernous carotid dolichoectasia

A 73 year old man was evaluated for the sudden onset of binocular horizontal diplopia which was worse in left gaze and which began 1 day before initial examination. He also complained of a dull headache over his left brow. He had a medical history of hip and knee surgery and was taking no medications. He was a 50 pack a year smoker but had no other history of vascular disease, including hypertension and diabetes mellitus. He had no previous history of strabismus or eye muscle surgery. His ophthalmologist was concerned about giant cell arteritis (GCA) and ordered a Westergren erythrocyte sedimentation rate test, which was 15 mm in the first hour.

Additional history revealed that he had no jaw claudication, scalp tenderness, or other symptoms of GCA. Visual acuity was 20/25 in both eyes and his colour vision and confrontation visual fields were normal. His pupils were equal in size and briskly reactive without a relative afferent pupillary defect. A left abduction deficit was noted (fig 1) and, with alternate cover testing, there was a 10 prism dioptre esotropia in primary position and at near, which increased to 20 prism dioptries on left gaze and decreased to 2 prism dioptries in right gaze. He had slowed saccades of the left lateral rectus muscle. There was no evidence of ptosis or ocular motor synkinesis. The remainder of his cranial nerve and dilated fundus examination were normal. Magnetic resonance imaging (MRI) (fig 2) and magnetic resonance angiography (MRA) (fig 3) of the brain revealed a lateral course of the left cavernous carotid artery consistent with dolichoectasia.

Figure 1 (Top left) A Keith needle, threaded with a Nylon and a Vicryl suture, is passed from one eyelid puncture site to the corresponding eyebrow puncture site in a sub-orbicularis plane. (Top right) The Keith needle, loaded with the sutures, is passed from one eyebrow puncture site to the corresponding eyebrow puncture site in a sub-orbicularis plane. (Bottom left) The right eye. Postoperative picture of the patient after bilateral frontalis suspension using the described technique. Both the eyelids are adequately elevated with a satisfactory contour although the chin-up position is not totally ameliorated.

Figure 2 (Left) Preoperative picture of a 1 year old girl with bilateral congenital ptosis and a chin-up position. The child has bilateral poor levator function. (Right) Postoperative picture of the patient after bilateral frontalis suspension using the described technique. Both the eyelids are adequately elevated with a satisfactory contour although the chin-up position is not totally ameliorated.
Follow up examination 1 month later revealed no history of variability of the diplopia and no change in the ocular misalignment; however, over the next 2 months the patient reported a gradual improvement in symptoms. He returned 3 months after the initial onset of symptoms and his abduction deficit had resolved. There was no evidence of an ocular misalignment with alternate cover testing. Repeat MR/MRA showed no change in the calibre or position of the left cavernous carotid artery. He has reported no new symptoms with 1 month of additional follow up.

Comment
Dolichoectasia, or pathological enlargement, of the intracranial arteries is a finding rarely seen with neuroimaging or arteriography. Arteriosclerosis, with thinning of the media and defects in the internal elastic laminae of the vessel walls, is thought to predispose to progressive enlargement of the vessel lumen.\(^1\) Ectasia of the intracranial arteries is believed to cause symptoms because of compression of adjacent structures and/or ischaemia secondary to intraluminal thrombus formation and blockage of perforating vessels along the length of the dolichoectatic vessel.\(^2\)

Dolichoectasia of the cavernous carotid artery has been suggested as an infrequent cause of sixth nerve paresis. One in 23 patients with carotid ectasia (in a series of approximately 40,000 patients undergoing carotid arteriography) was found to have an acute sixth nerve palsy with "good recovery," although the clinical course was not specified.\(^3\) Ipsilateral dolichoectasia was noted in a 59 year old man with seven episodes of sixth nerve paresis, each lasting between 2–5 weeks.\(^4\) The authors did not provide an explanation for the mechanism of recurrence. A single patient with bilateral sixth nerve paresis was reported to have bilateral carotid dolichoectasia as the underlying cause.\(^5\) However, in the discussion the causal relation of the dolichoectasia, presumably from compression of the carotid artery, was called into question. In addition, dolichoectasia of the cavernous carotid artery has been noted in patients without ocular motor deficits.\(^6\)

This patient’s left sixth nerve paresis spontaneously resolved 3 months after the initial onset of symptoms. Despite the presence of ipsilateral cavernous carotid dolichoectasia, his clinical course is most consistent with that of a vasculopathic sixth nerve paresis. Whether the dolichoectasia was causative or an incidental finding is not clear in this patient. Arterial dissection in a previously ectatic vessel has been suggested as an explanation for the acute onset of symptoms in patients with dolichoectasia; however, no evidence of arterial dissection was seen in this patient’s MRI/MRA. Ischaemia of the vascular lumen of the sixth nerve, perhaps because of intraluminal thrombus formation, may have resulted in a vasculopathic sixth nerve palsy, but there was no evidence of thrombus formation on the MRI/MRA.

Because the causative mechanism in patients with persistent sixth nerve paresis from presumed dolichoectasia is not certain, treatment guidelines are not clear. Monocular occlusion and prism therapy may provide temporary or long lasting relief of diplopia. Neurosurgical intervention to relieve mechanical compression between the cavernous carotid artery is a difficult, potentially life threatening, procedure. Extracranial muscle surgery may correct the ocular misalignment, without treating the underlying mechanical compression, with uncertain long term benefit. Spontaneous resolution of the left sixth nerve palsy in this patient with ipsilateral carotid dolichoectasia suggests that a period of careful observation should precede plans for surgical correction of the ocular misalignment.

References

Intravitreal triamcinolone acetonide as treatment for extensive exudative retinal detachment
Coats’ disease or entities like Coats’ disease are characterised by a marked exudative retinal detachment with leakage of peripheral retinal vessels, pronounced subretinal deposition of lipids, and eventual progression to total retinal detachment. In some situations, iris neovascularisation can occur, suggesting an angiogenic component in the course of the disease. In view of the subretinal exudation from the leaking retinal vessels and the possibly neovascular aspect in the disease process, the purpose of this study was to evaluate whether intravitreal triamcinolone acetonide may be helpful in the treatment of Coats’ like diseases. Intravitreal triamcinolone acetonide has recently been shown to have a pronounced anti-edematous and possibly anti-angiogenic effect in diseases such as diffuse diabetic macular oedema, proliferative diabetic retinopathy, chronic pre-phthisical ocular hypotony, chronic uveitis, and persistent pseudophakic cystoid macular oedema.\(^1\)\(^2\)

Case report
The prospective clinical interventional case report included two patients who presented with sub-total exudative retinal detachment. A 39 year old female patient showed an extensive exudative retinal detachment extending from the temporal periphery of the fundus to the macular region. Diagnosed with Coats’ disease in her early teens, she had received multiple xenon arc coagulations as well as argon laser coagulations. Her visual acuity was 0.02. Intraretinal pressure measured 13 mm Hg. The second patient was a 75 year old woman presenting with almost total exudative retinal detachment with marked subretinal deposition of lipids. Visual acuity was 0.05. Intraretinal pressure measured 21 mm Hg.
Under topical anaesthesia, both patients received an intravitreal injection of 25 mg triamcinolone acetonide, which was transconjunctivally injected through the pars plana. Both patients were fully informed about the experimental character of the treatment and had given informed consent. The technique has already been described in detail.1 Follow-up after the injections were 2 years and 10 months, respectively.

After the injection, visual acuity remained unchanged, and intraocular pressure ranged between 10 and 15 mm Hg in the first patient. In the second patient, visual acuity eventually decreased to light perception after the injection. Intraocular pressure ranged between 19 and 25 mm Hg. In both patients, flare in the anterior chamber and in the vitreous cavity, as assessed by slit lamp biomicroscopy, decreased markedly. Upon ophthalmoscopy, the extent of exudative retinal detachment increased slightly, with subretinal strands being stronger and more visible.

Comment

Although intravitreal triamcinolone acetonide can markedly reduce retinal oedema in eyes with diffuse diabetic macular oedema and pseudophakic cystoid macular oedema, intravitreal triamcinolone acetonide was not pronouncedly helpful in reducing subretinal oedema and re-attaching the retina in the two patients presented in this study. This result was unexpected in view of the pre-sumed anti-inflammatory and anti-proliferative effect of steroids such as triamcinolone acetonide.1,2 It may be explained by a previous experimental study in which triamcinolone acetonide inhibited the proliferation of rabbit dermal and conjunctival fibroblasts in cell culture at 150 μg/ml, but paradoxically increased proliferation almost twofold at concentrations ranging from 1–30 μg/ml under identical culture conditions.3 As long as the individual responses on the speed of the progression of retinal pigment epithelium cells is unclear, intravitreal triamcinolone acetonide may thus cautiously be taken as adjunct treatment of marked exudative retinal detachment in eyes with Coats’ disease. A similar conclusion was drawn in a recent study on eyes with proliferative vitreoretinopathy, in which pars plana vitrectomy was combined with an intravitreal injection of 25 mg triamcinolone acetonide, and in which unexpectedly, the recurrence rate of proliferative vitreoretinopathy was not markedly diminished.4 Future randomised studies as well as investigations evaluating the effect of intravitreal steroids combined with other drugs such as 5-fluorouracil on the proliferation of retinal pigment epithelium cells and retinal detachment rate may be warranted.


Long term efficacy and safety of botulinum toxin A injection for crocodile tears syndrome

Gustatory lacrimation, also called crocodile tears syndrome (CTS), is an autonomous synkinesis in which patients tear excessively in response to salivary stimuli. It occurs most commonly in the setting of idiopathic or traumatic facial palsy and is thought to result from aberrant reinnervation of the lacrimal gland by salivary efferent fibres from either the seventh or ninth cranial nerve. Many patients tolerate CTS and require no intervention. For patients who cannot tolerate CTS, past treatments have included anti-cholinergic drugs, subtotal resection of the palpebral lobe of the lacrimal gland, and resection of the tarsal papillary process of the lower eyelid. Newer modalities that have been shown to be effective for a host of disorders characterised by involuntary muscle spasms, including blepharospasm, hemifacial spasm, and torticollis. Botulinum toxin A also has been used to treat a number of localised autonomic disorders, including axillary hyperhidrosis, palmar hyperhidrosis, and Frey syndrome. In 1996, Botterjordt et al reported the successful treatment of CTS by injection of botulinum toxin A directly into the lacrimal gland.3 Since then, there have been five reports of similar treatments, all of which were successful. All of these studies report complete or near complete resolution of the syndrome within a week with only infrequent, minor, and reversible complications. We now report a patient with CTS who has been successfully managed for 3 years without complications.

Case report

A 38 year old man presented in July of 2000 with a 6 month history of right sided tearing and hyperhidrosis of the auriculotemporal region when eating or when hungry. Fourteen months previously, he had undergone a total right parotidectomy for a mixed tumour of the parotid gland. Immediately after surgery, he had a complete right sided facial palsy and numbness of the right lower face. The facial palsy resolved completely 1 month later, but the facial numbness persisted. Eight months later, the patient began to experience increased tearing on the right after eating, most notable after eating minting. On examination, the patient had normal facial movement but decreased sensation to light touch in the region of the second division of the trigeminal nerve and spasms of the right lower lid on palpation. In addition, he perspired from the right side of the face and had tearing from the eye when hungry or upon eating. His ocular and neurologic examinations were otherwise unremarkable.

In light of the bothersome nature of CTS to this patient, we felt a trial of botulinum toxin A was warranted. Accordingly, after obtaining consent, we injected botulinum toxin A (Botox 2.5 U) transconjunctivally into the palpebral lobe of the right lacrimal gland under direct visualisation at the slit lamp biomicroscope without complications. The patient’s excess epiphora completely resolved within 5 days, and he remained asymptomatic for 11 months. He has subsequently undergone repeat injections of botulinum toxin A every 3–4 months and continues to be effective in controlling this patient’s CTS for 3 years without complications.

Comment

Several different groups have now reported a total of 12 cases of CTS treated with botulinum toxin A.6 All of the patients reported have had complete or near complete short term resolution of symptoms with doses of botulinum toxin A (Botox) ranging from 2.5–60 U. The higher doses seem to have additional benefit in terms of efficacy or duration. Injection of botulinum toxin A for CTS appears to be safe, although minor complications occasionally occur. Two of the patients injected transconjunctivally developed ptosis, one accompanied by a superior rectus palsy, whereas two others developed dryness of the injected eye. These complications resolved over several months. No cases of ptosis or other muscle weakness have been reported after transconjunctival injection, and our patient has had no complications with any of his injections over the last 3 years.

As with all new treatments, there are concerns about long term efficacy and safety. Botox has been found both safe and effective at the neuromuscular junction, but its long term effects on the peripheral autonomic system are unknown. The reports suggest that the repeated minor trauma of the injection could eventually impair lacrimal gland function. In light of these concerns, it is encouraging to be able to report that repeated injections of botulinum toxin A continue to be effective in controlling this patient’s CTS for 3 years without complication.

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Retinal arterial collapse pressure in eyes with retinal arterial occlusive diseases

Retinal arterial occlusions may be primarily or secondarily associated with low retinal arterial pressure. Based on previous ophthalmodynamometric studies, the purpose of the present study is to estimate the retinal vessel pressure in patients with central retinal artery or branch retinal artery occlusions and patients with amaurosis fugax.

Case report

This prospective clinical non-interventional comparative study included nine eyes of seven patients (mean age 68.8 (SD 13.7) years) with central retinal artery occlusion (n = 1 eye), branch retinal artery occlusion (n = 2), ischaemic ophthalmopathy (n = 2), or amaurosis fugax (n = 4). An age matched control group consisted of 27 eyes of 21 subjects attending the hospital because of cataract or refractive problems. After medical pupil dilatation, a conventional Goldmann contact lens fitted with a pressure sensor mounted into the holding ring was put onto the cornea. By slightly pressing the contact lens, pressure was applied onto the globe, and the pressure when the central retinal vein or artery started to pulsate was noted. The methods applied in the study adhered to the tenets of the declaration of Helsinki. The method has already been described in detail.

In the study group, central retinal artery collapse pressure measured 43.9 (SD 33.4) arbitrary units (AU) and was significantly (p = 0.004) lower than in the control group (78.0 (SD 19.2) AU) (fig 1). Within the study group, central retinal artery collapse pressure was lowest in the eye with central retinal artery occlusion, showing a pulse synchronous movement of the erythrocyte column in the vessel without applying any pressure onto the globe. In the two eyes with branch retinal artery occlusion, collapse pressure in the arterial branch lying in the oedematous part of the fundus measured 36.7 AU and 0 AU respectively. These values were significantly (p = 0.005) lower than the values obtained in the control group (fig 1). In the eyes with branch retinal artery occlusion, collapse pressure in the arterial branch in the non-oedematous part of the fundus was in the normal range (93.1 AU and 93.3 AU, respectively). In the patient suffering from ischaemic ophthalmopathy, central retinal artery collapse pressure was lower in the eye more severely affected than in the contralateral eye (14.7 AU v 51.7 AU). Both values were significantly (p = 0.02) lower than the values of the control group. In the eyes with amaurosis fugax, mean central retinal artery collapse pressure measured 73.0 (SD 15.4) AU which was not significantly (p = 0.55) different from central retinal artery collapse pressure in the control group (fig 1). Central retinal vein collapse pressure did not vary significantly between the study groups and the control group (8.8 (SD 12.2) AU v 6.1 (SD 8.4) AU; p = 0.54).

Comment

Central retinal artery collapse pressure as determined by the new ophthalmodynamometric technique was significantly lower in eyes with retinal artery occlusive diseases than in normal eyes (fig 1). Correspondingly, in the eyes with branch retinal artery occlusions, measurements were lower in the arterial branch affected by the occlusion than in the retinal artery branch with intact perfusion. As a corollary, in the patient suffering from ischaemic ophthalmopathy, the central retinal artery collapse pressure was lower in the eye more severely affected because of a complete stenosis of the carotid artery than in the contralateral eye. Interestingly, the eye with amaurosis fugax did not show significantly lower measurements than normal eyes. This agrees with previous studies using other ophthalmodynamometric techniques for evaluation of carotid artery perfusion. In conclusion, using a new ophthalmodynamometer with biometric observation of central retinal vessels during the examination, central retinal artery collapse pressure measurements were significantly lower in eyes with retinal arterial occlusive diseases than in normal eyes. Future studies may show whether determination of the central retinal artery collapse pressure in patients with increased risk for retinal arterial occlusions may be suitable to predict which patients have a higher risk for eventual retinal artery occlusion compared with other patients with a similar risk profile.

References


Modified self sealing sclerotomy for drainage of subretinal fluid during scleral buckling surgery

Drainage of subretinal fluid is probably the most dangerous step in scleral buckling surgery for uncomplicated retinal detachment. The most common complications include subretinal haemorrhage, retinal perforation, and vitreoretinal incarceration. Sclerotomy to drain subretinal fluid is traditionally made with a sharp knife, and the sclerotome, a modified self sealing sclerotomy technique for drainage of subretinal fluid during scleral buckling surgery.

Patients and methods

Twenty consecutive patients undergoing scleral buckling for primary rhegmatogenous retinal detachment from two vitrectomy surgery centres were enrolled in this prospective study. A scleral buckling procedure was performed using a circumferential scleral band (Mira 240, Mira, Waltham, MA, USA) sutured with the posterior border located 12 mm posterior to the limbus, and adding any necessary segmental spongs (Mira). Cryoretinopexy was performed using a CTU Ophthalmic Cryo Unit (Keeler, London, UK) to seal retinal tears. After surgery, sulfur hexafluoride (SF6) gas was used in all patients. The drainage site was chosen based on retinal elevation, as shown by intraoperative retinal examination with indirect

Figure 1 Box plots showing the retinal artery collapse pressure in the study group of patients with central retinal artery occlusion (CRAO), branch retinal artery occlusion, ischaemic ophthalmopathy, amaurosis fugax, and in the control group.

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ophthalmoscopy. A 3–4 mm half depth scleral incision was created perpendicular to the limbus using an angled bevel up blade (Alcon Laboratories, Fort Worth, TX, USA) with its sharp advancing edge directed perpendicular to the scleral surface (fig 1A). With a crescent knife, a 3 mm tunnel incision was then made to create a scleral flap parallel to the limbus (fig 1B). The scleral flap was retracted, and a 27 gauge needle was then used to perforate the scleral bed and choroid. (D) Subretinal fluid is expressed and dried with a cotton swab.

Results
All 20 patients had self sealing sclerotomies with its sharp advancing edge directed parallel to the limbus with its sharp advancing edge directed perpendicular to the scleral surface. The scleral flap is retracted, and a 27 gauge needle is then used to perforate the scleral bed and choroid. (D) Subretinal fluid is expressed and dried with a cotton swab. drainage was slow and gradual. No serious complications were associated with this technique. The results of the drainage procedures were recorded on surgical reports and reviewed. Drainage was slow and gradual. No serious complications were associated with this technique. The results of the drainage procedures were recorded on surgical reports and reviewed.

Comments
We describe the results and complications of 20 scleral buckling procedures for primary rhegmatogenous retinal detachments in which subretinal fluid was drained using a sutureless sclerotomy technique. A self sealing incision with a stepped wound construction is not new to ophthalmology. Cataract and vitreoretinal surgeons pioneered this technique as part of phacoemulsification cataract extraction and vitreous surgeries. Possible advantages to this type of incision include shortened operating time and reduced incidence of postoperative wound leak. This new way of constructing the sclerotomy for drainage has many advantages, and we propose it as an alternative to standard sclerotomy incision to drain subretinal fluid during scleral buckling surgery for uncomplicated retinal detachments.

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Accepted for publication 9 July 2003

Supported in part by the Fundación Arevalo-Coutinho para la Investigación en Oftalmología (FAOCO), Caracas, Venezuela.

The authors have no proprietary or financial interest in any products or techniques described in this article.

References


Conjunctival dendrite in a case of primary herpes simplex infection
Ocular involvement in primary herpes simplex infection is usually in the form of follicular conjunctivitis, blepharitis, and sometimes corneal involvement in the form of superficial punctuate keratitis, dendrite, or rarely geographical ulcer.1 We report a case of dendritiform lesion in the conjunctiva in a young girl with primary herpes simplex infection. To the best of our knowledge, conjunctival dendritiform lesion has not been reported before in primary herpes simplex infection.

Case report
A 20 year old girl presented to our outpatient department with complaints of redness and discomfort in her right eye of two days' duration. She gave a history of fever of one week's duration followed by appearance of vesicles at the right side angle of the mouth and on the right upper lid. Past ocular and systemic history was unremarkable.

Visual acuity was 6/6 unaided in both the eyes. There were vesicles at the angle of the mouth (fig 1A) and on the right upper lid. Slit lamp examination of the right eye with fluorescein staining revealed a dendritiform pattern of staining in the lower bulbar conjunctiva (fig 1). Cornea was clear and rest of the anterior segment was unremarkable. Left eye examination was unremarkable. Fundus examination in both the eyes was within normal limits. The patient was advised to use topical acyclovir 3% eye ointment five times a day and tablet acyclovir 400 mg five times a day.

On follow up after two days, there was superficial punctuate keratitis in the inferior half of the cornea in the right eye. The patient was asked to continue the same medication. One week later, the vesicles were absent and the conjunctiva and cornea were clear. The medication was discontinued.

Darouger et al, in a study of primary herpes simplex ocular infection, found 64% of the patients to be over fifteen years of age.4 Follicular conjunctivitis (7%), blepharoconjunctivitis (16%), and corneal dendritic ulcers (15%) were some of the lesions reported. Appearance of a dendritic ulcer on the conjunctiva, to the best of our knowledge, has not been reported in primary herpes simplex infection.

Dendritic lesions on histopathological study show that they are composed of round epithelial cells and variable sized syncitia containing bizarre shaped nuclei. The epithelial cells contain viral DNA.5 Recurrent infection with the virus in the form of epithelial keratitis commonly produces dendritic lesions on the cornea.1
Conjunctival dendrite is an interesting and apparently rarely reported lesion.

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Accepted for publication 7 July 2003

The authors have no proprietary interests in the products mentioned in the article.

References

Severe ocular trauma caused by an ostrich
The ostrich is a strange and harmless looking bird; however, in Africa attacks by ostriches on humans are not uncommon and sometimes result in death. We recently treated such a patient with an eye injury.

Case report
A 35 year old male patient presented with an injury sustained from being kicked in the face by an ostrich (Struthio camelus).

On examination the right eye was found to be normal but he had vision of bare light perception on the left with propitious of the globe and severe chemosis of the conjunctiva. Both upper and lower lids were avulsed medially. There was limitation in all positions of gaze which was more noticeable on attempted abduction (fig 1). The eye was soft and he had an oedematous cornea and a full hyphema. The posterior segment could not be visualised.

Computed tomography showed a blowout fracture of the left orbit (fig 2). There was a fracture of the left nasal bone with a comminuted lateral wall fracture. The medial orbital wall was also fractured with opacification of the left ethmoid sinus and herniation of the medial rectus into the sinus.

An intraocular haemorrhage as well as haemorrhage in the retrobulbar space was noted.

Under general anaesthesia, both the upper and lower lids were repaired and the hyphema was washed out. A posterior rupture was suspected clinically but the site of rupture could not be identified. The eye was subsequently eviscerated.

Comment
Ostriches usually inflict injury in one of two ways: the more serious injury is that of a slash or laceration, usually to the lower abdomen or limbs, caused by the ostrich kicking in a forward and downward motion with its powerful foot. The toennail of the ostrich is sharp and is used by the ostrich for protection against predators. The second type of injury is seen more commonly. This occurs when the ostrich uses its bony breast plate as a ram to knock the person to the ground. The ostrich then jumps upon the victim and, because an ostrich weighs 75–150 kg, this may cause contusion of the torso with rib fractures.

Our patient was bending while repairing a fence when he was kicked by an ostrich. He was struck in the face and sustained extensive facial trauma extending from his nasal bones to his orbital walls and ethmoidal sinus. The trauma also resulted in irreparable blunt trauma to the eye.

The injury caused was severe with no possibility of repair of the globe, and is the only documented case of an eye being lost due to injury by an ostrich.

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Accepted for publication 7 July 2003

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Swollen optic discs in a patient with the chromosome 22q11.2 deletion syndrome
The chromosome 22q11.2 deletion syndrome (22q11DS) encompasses velocardiofacial syndrome (VCFS), DiGeorge syndrome (DGS), and conotruncal anomaly face syndrome (CTFS) and is the result of a microdeletion of chromosome band 22q11.2. It is a relatively common genetic anomaly estimated to occur in approximately one in 4000 live births. The 22q11.2 deletion can arise de novo or can have an autosomal dominant
inheritance. The condition is thought to be due in part to abnormal development of the pharyngeal arch structures. Clinical findings are extensive and highly variable between patients. Prominent features include cardiac defects, cleft palate, dysmorphic facies, mal-descent of the thymus, hypoparathyroidism, immune deficiency and developmental delay. Ocular findings include hypertelorism, retinal vascular tortuosity, narrow palpebral fissures, small optic nerves, iris nodules, cataracts, and iris coloboma. We present a case of a boy who was found to have bilateral disc swelling that led to a diagnosis of 22q11DS.

Case report
A 14 year old boy presented to the accident and emergency department after having a generalised seizure. He had been admitted to another hospital, 2 days before this, with a sudden onset of collapse and subsequent respiratory arrest. At that time he was noted to have swollen optic discs and a head computed tomography scan done there was reported as normal. He had further seizures after admission to our hospital. Blood testing revealed low plasma calcium and high plasma phosphate levels. The patient had been complaining of back pain in recent months and his mother said that he had shrunken by a couple of centimetres over the past year. She also said that he had always been clumsy and he had been diagnosed as dyslexic at the age of 7. He had a history of behavioural problems which his family said often settled during holidays in the sun. A thoracic spine x ray revealed wedge-shaped fractures of three vertebral bodies. Further blood testing showed a relatively low parathormone level confirming the diagnosis of hypoparathyroidism. He was then referred to the ophthalmology department for assessment of his disc swelling.

He had been seen in the eye clinic a year before this presentation complaining of coloured lines in his field of vision. No abnormality was found and his discs on that occasion were noted to be normal. On examination this time he was noticed to have dysmorphic features, notably palpebral fissures slanting medially upwards, and abnormally formed low set ears. Visual acuities were 6/5 in both eyes, colour vision testing was unremarkable, and there was no relative afferent pupillary defect. There was no sign of cataract formation or any other abnormality of the anterior segments. Funduscopy revealed bilateral disc swelling with extensive peripapillary haemorrhages in both eyes (fig 1). The diagnosis of hypoparathyroidism combined with the patient’s abnormal facies raised the suspicion of a genetic disorder and blood was then sent for chromosomal analysis. His hypoparathyroidism was treated with vitamin D and calcium supplements and he responded well with his calcium reaching normal levels within a few days. Examination 6 weeks after his calcium had normalised showed most of the haemorrhages and disc swelling had cleared (fig 2). Results of chromosomal testing revealed a region 11.2 microdeletion of the long arm (q) of chromosome 22. Other members of his family are now also being tested.

Comment
Chromosome 22q11.2 deletion syndrome is one of the more common causes of congenital and childhood hypoparathyroidism which can lead to hypocalcaemia. Hypocalcaemia is a known cause of disc swelling, the mechanism of which is not known. Some patients with hypocalcaemic disc swelling have a loss of acuity and field typical of an optic neuropathy, while in others the features resemble papilloedema, with no significant visual loss. The swelling usually resolves after the calcium level returns to normal. 22q11DS is probably underdiagnosed. This case illustrates the importance of a correct and early diagnosis of this relatively common genetic disorder so that treatment can begin in an effort to prevent further medical and developmental complications. The highly variable clinical features require a high level of awareness of the condition across several different disciplines. Patients, especially children, presenting with swollen optic discs and who have normal imaging studies of the brain should have a calcium level checked. If abnormal and it is found to be due to hypoparathyroidism then chromosomal analysis should be considered, especially if other parts of the history or examination raise the suspicion of a genetic disorder.

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Accepted for publication 16 July 2003

References

The correlation of phenylephrine 1% with hydroxyamphetamine 1% in Horner’s syndrome
Pharmacological testing in Horner’s syndrome involves the use of cocaine to confirm the diagnosis and hydroxyamphetamine to localise the lesion to the post-ganglionic (third order) or non-postganglionic neuron. However, hydroxyamphetamine bromide 1% (Paraldehyde) is not always readily available to the ophthalmologist. An alternative drug for localising the site of the lesion is phenylephrine 1% which can easily be prepared by dilution of stronger concentrations (2.5% or 10%) and which is almost universally available in most ophthalmologists’ offices. Because of the principle of denervation supersensitivity, a Horner’s syndrome produced by a lesion interrupting the postganglionic fibres should dilate the pupil when phenylephrine 1% is placed in the conjunctival sac. The pupil

![Figure 1](https://example.com/figure1.png)

**Figure 1** Shows bilateral disc swelling with extensive peripapillary haemorrhages in both eyes.

![Figure 2](https://example.com/figure2.png)

**Figure 2** Examination 6 weeks later showed most of the haemorrhages and disc swelling had cleared.

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Table 1  Mean change in pupillary diameter in patients with Horner’s syndrome

<table>
<thead>
<tr>
<th></th>
<th>Baseline pupil</th>
<th>Change in pupil diameter following</th>
<th>Change in pupil diameter following</th>
<th>Change in pupil diameter following</th>
<th>Change in pupil diameter following</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Cocaine 10% (mm)</td>
<td>Hydrosuxyamphetamine 1% (mm)</td>
<td>Phenylephrine 1% (mm)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal pupil</td>
<td>Normal pupil</td>
<td>Normal pupil</td>
<td>Normal pupil</td>
<td>Normal pupil</td>
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<tr>
<td></td>
<td>Horner’s pupil</td>
<td>1.5</td>
<td>2.1</td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.25</td>
<td>2.25</td>
<td>0.25</td>
<td></td>
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<td></td>
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<td>3.0</td>
<td>2.5</td>
<td>0.2</td>
<td></td>
</tr>
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<td></td>
<td></td>
<td>0.5</td>
<td>2.9</td>
<td>0.5</td>
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</table>

The law of denervation supersensitivity states that an organ deprived of its normal innervation becomes more sensitive to the chemical transmitter normally released from those nerves. Thompson and Mensher documented supersensitivity of the iris dilator to phenylephrine 1% in one patient. They further tested 13 patients but used a 10% concentration, which dilates the normal pupil. They determined that the affected pupil of the three patients with post-ganglionic lesions dilated sooner and more vigorously than the unaffected pupil. Ramsay tested 14 patients with phenylephrine 1% and found in patients with Horner’s syndrome that 71% of pupils were supersensitive. However, the responses of the post-ganglionic and non-post-ganglionic Horner’s syndrome were not reported separately. Other studies have reported on the use of phenylephrine 10% and epinephrine 1%, and adrenaline (epinephrine) 0.1% in the pharmacological testing of Horner’s, but none has investigated the efficacy of phenylephrine 1% in identifying post-ganglionic Horner’s lesion. Our study shows that phenylephrine dilates the post-ganglionic Horner’s pupil, but not the non-post-ganglionic or normal pupil. We found the sensitivity of 81% and a specificity of 100%. Hydrosuxyamphetamine 1% has been shown to have a sensitivity of 93% and specificity of 83%. Only one out of 11 patients with Horner’s syndrome in our series had a lesion, which localised to the post-ganglionic neuron with hydrosuxyamphetamine 1%, but not with phenylephrine 1%. Phenylephrine 1% has some limitations:

1. It does not dilate the normal pupil. If neither pupil dilates with phenylephrine 1% it could be either because the lesion is non-post-ganglionic or the drops are ineffective.
2. The degree of supersensitivity is determined by the extent of denervation. A partial post-ganglionic lesion may be difficult to distinguish from a preganglionic lesion; in both conditions the iris may dilate minimally. In our study there was only one patient with a preganglionic lesion.
3. Tests for supersensitivity may also be subject to false positive errors owing to the variations in penetration of the drug. The drug should be placed strictly on intact corneas so that the same dose reaches each iris.
4. Supersensitivity of the iris dilator increases with age. Phenylephrine sensitivity of the iris increases by 0.23 mm per decade after age 20.

In summary, we here report the first series of patients with Horner’s syndrome, which compared the pupillary response of phenylephrine 1% to hydrosuxyamphetamine 1%. Phenylephrine 1% correlates well with the results of hydrosuxyamphetamine 1% in localising the lesion to the post-ganglionic neuron and is a reliable alternative to hydrosuxyamphetamine 1% should pharmacological testing be desired and hydrosuxyamphetamine 1% not be available.

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Accepted for publication 25 July 2003

References

Tetraspannin protein KAI1 expression in retinoblastoma

KAI1/CD82 is a metastasis suppressor gene located on human chromosome 11p11.2. It is a member of the structurally distinct family of cell surface glycoprotein, transmembrane 4-protein superfamily. KAI1 was initially isolated as a gene that suppressed metastasis of rat prostate tumour cells. KAI1 is down-regulated in several types of human malignancies. The purpose of this study was to investigate the expression of KAI1 in retinoblastoma and to correlate clinicopathologically.

Methods
There were 30 archival specimens of retinoblastomas from 2800 to 2002. There were 12 tumours with no invasion of choroid, or optic nerve and 18 tumours with invasion of choroid/optic nerve, and one tumour which had metastasised to the submandibular region. There were six well differentiated tumours, six moderately differentiated tumours, and 18 poorly differentiated tumours (table 1). Immunohistochemical
staining was performed using a sensitive labelled streptavidin biotin (LSAB kit, Dako) on tumours using monoclonal antibodies for tetraspanin KAI1 (C33, Novacastra) and for proliferation index Ki-67 (Clone MIB-1, Dako, Denmark) after antigen retrieval.

The immunohistochemical details are given in table 1. KAI1 expression was seen in the control lymphoid follicle of the tonsil (fig 1A). Intense KAI1 positivity with more than 80% positivity was seen in all 12/12 tumours with no invasion (fig 1B). Among the 18/18 tumours with invasion, KAI1 was decreased in all 18. The invading front of the tumour had less KAI1 than the tumour at the central portion. Retinoblastomas with focal and diffuse invasion of choroid had negative immunoreactivity (fig 1C). Tumours at the post-laminar and surgical end of optic nerve invaded no invasion (fig 1B). Among the 18/18 tumours with invasion, KAI1 was decreased in all 18. The invading front of the tumour had less KAI1 than the tumour at the central portion. Retinoblastomas with focal and diffuse invasion of choroid had negative immunoreactivity (fig 1C). Tumours at the post-laminar and surgical end of optic nerve invaded.

<table>
<thead>
<tr>
<th>No</th>
<th>Age (years)/sex</th>
<th>Clinical pathological parameters</th>
<th>Chemotherapy</th>
<th>KAI1 immunoactivity</th>
<th>% Cells stained</th>
<th>Stain intensity</th>
<th>% Cells stained</th>
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<td>40</td>
<td>37</td>
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<tr>
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<td>No</td>
<td>90</td>
<td>3+</td>
<td>40</td>
<td>3+</td>
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<tr>
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<td>1/F</td>
<td>RE, well diff</td>
<td>No</td>
<td>90</td>
<td>3+</td>
<td>63</td>
<td>5+</td>
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<tr>
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<td>50</td>
<td>2+</td>
<td>40</td>
<td>3+</td>
</tr>
<tr>
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<td>8/M</td>
<td>LE, poor diff</td>
<td>No</td>
<td>90</td>
<td>3+</td>
<td>40</td>
<td>3+</td>
</tr>
<tr>
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<td>80</td>
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<td>60</td>
<td>4+</td>
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<td>95</td>
<td>3+</td>
<td>49</td>
<td>5+</td>
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<td>5/F</td>
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<td>45</td>
<td>2+</td>
<td>50</td>
<td>3+</td>
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<td>9</td>
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<td>3+</td>
<td>40</td>
<td>4+</td>
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<tr>
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<td>85</td>
<td>3+</td>
<td>70</td>
<td>6+</td>
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<tr>
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<td>2+</td>
<td>66</td>
<td>5+</td>
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<td>85</td>
<td>3+</td>
<td>50</td>
<td>4+</td>
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<tr>
<td>13</td>
<td>7/F</td>
<td>LE, mod diff, No choroidal</td>
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<td>1</td>
<td>0</td>
<td>56</td>
<td>80</td>
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<tr>
<td>14</td>
<td>1/M</td>
<td>RE, poor diff, Pre laminar</td>
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<td>20</td>
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<td>60</td>
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<td>Postop chemo 4</td>
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<td>0</td>
<td>70</td>
<td>5+</td>
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<tr>
<td>16</td>
<td>4/M</td>
<td>Both eyes, LE, poor diff</td>
<td>Postop chemo 4</td>
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<td>6+</td>
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<td>40</td>
<td>2+</td>
<td>64</td>
<td>6+</td>
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<tr>
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<td>No</td>
<td>20</td>
<td>1+</td>
<td>66</td>
<td>5+</td>
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<tr>
<td>19</td>
<td>1.1 M/F</td>
<td>RE, poor diff, diffuse choroidal invasion and post laminar optic nerve invasion</td>
<td>Postop chemo 6</td>
<td>1</td>
<td>0</td>
<td>80</td>
<td>6+</td>
</tr>
<tr>
<td>20</td>
<td>3/M</td>
<td>LE, poor diff, post laminar</td>
<td>Postop chemo 2</td>
<td>10</td>
<td>1+</td>
<td>60</td>
<td>6+</td>
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<tr>
<td>21</td>
<td>4/F</td>
<td>RE, mod, focal choroidal invasion</td>
<td>No</td>
<td>10</td>
<td>1+</td>
<td>69</td>
<td>6+</td>
</tr>
<tr>
<td>22</td>
<td>2/F</td>
<td>RE, poor diff, post laminar</td>
<td>No</td>
<td>0</td>
<td>0</td>
<td>70</td>
<td>6+</td>
</tr>
<tr>
<td>23</td>
<td>2.5/F</td>
<td>RE, poor diff, diffuse</td>
<td>No</td>
<td>10</td>
<td>1+</td>
<td>60</td>
<td>6+</td>
</tr>
<tr>
<td>24</td>
<td>3/M</td>
<td>RE, poor diff</td>
<td>No</td>
<td>1</td>
<td>0</td>
<td>70</td>
<td>6+</td>
</tr>
<tr>
<td>25</td>
<td>5/F</td>
<td>RE, diffuse choroidal invasion, surgical end of ON invaded</td>
<td>Postop chemo 6</td>
<td>1</td>
<td>0</td>
<td>60</td>
<td>6+</td>
</tr>
<tr>
<td>26</td>
<td>5/F</td>
<td>Both eyes, (RE) well diff, pre laminar, focal choroidal invasion</td>
<td>No</td>
<td>10</td>
<td>1+</td>
<td>75</td>
<td>6+</td>
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<tr>
<td>27</td>
<td>2/F</td>
<td>Both eyes, (LE) poor diff, diffuse choroidal invasion</td>
<td>No</td>
<td>10</td>
<td>1+</td>
<td>60</td>
<td>6+</td>
</tr>
<tr>
<td>28</td>
<td>2/M</td>
<td>RE, poor post, laminar</td>
<td>No</td>
<td>1</td>
<td>0</td>
<td>80</td>
<td>6+</td>
</tr>
<tr>
<td>29</td>
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<td>Both eyes, (RE), well diff, lamina cribrosa</td>
<td>Preop and postop chemo</td>
<td>10</td>
<td>1+</td>
<td>60</td>
<td>6+</td>
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<tr>
<td>30</td>
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<td>Postop chemo 1</td>
<td>0</td>
<td>1</td>
<td>60</td>
<td>6+</td>
</tr>
</tbody>
</table>

diff = differentiated; Postop = postoperative; chemo = chemotherapy.

**Table 1** Clinicopathological and immunohistochemical features of the retinoblastomas used in the study

**Results**

The immunohistochemical details are given in table 1. KAI1 expression was seen in the control lymphoid follicle of the tonsil (fig 1A). Intense KAI1 positivity with more than 80% positivity was seen in all 12/12 tumours with no invasion (fig 1B). Among the 18/18 tumours with invasion, KAI1 was decreased in all 18. The invading front of the tumour had less KAI1 than the tumour at the central portion. Retinoblastomas with focal and diffuse invasion of choroid had negative KAI1 immunoreactivity. Tumours with pre-laminar optic nerve invasion had weak KAI1 immunoreactivity (fig 1C). Tumours at the post-laminar and surgical end of the optic nerve (inset, fig 1C) and at the metastatic site (fig 1D) had negative KAI1 immunoreactivity. Negative KAI1 reactivity was seen in 50% (9/18) of poorly differentiated retinoblastomas. Statistically significant correlation was observed between KAI1 expression and Ki-67 labelling index in the whole study group (p<0.001). No statistically significant correlation was observed between KAI1 expression and differentiation. Significant statistical correlation was observed when KAI1 expression was compared with tumour invasion (p<0.001).

**Comment**

Retinoblastoma joins a growing list of cancers in which downregulation of KAI1 is associated with tumour progression. In our study KAI1 was identified by the monoclonal antibody CD33. It was originally shown as inhibitory to syncytium formation induced by human T cell leukaemia virus type I, and this specific inhibition to syncytium formation induced by some human T cell line by this antibody was strongly associated with altered glycosylation of cell surface antigen, suggesting that the C33 antigen—that is, KAI1, might have a possible role in the cell to cell adhesion mechanism.

Thus, KAI1 may link to the cell surface molecules, such as integrins, cadherin, and other TM4SF members, and loss of KAI1 function may have a significant role in the progression of retinoblastoma. The mechanism of KAI1 downregulation is not known. The 5′ promoter region of the gene contains a CpG island, raising the possibility of gene silencing by promoter methylation. Thus, biologically, our findings suggest a potential implication of KAI1 in tumour progression and these molecules may provide novel insights into tumour progression in retinoblastoma.

**Acknowledgement**

Financial support: Indian Council Of Medical Research, India.

**References**

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Bilateral ocular surface squamous neoplasia: a clinicopathological case report

Ocular surface squamous neoplasia (OSSN) was first described by Lee and Hirst as an umbrella term that encompasses intraepithelial and invasive squamous cell carcinoma of the conjunctiva and cornea. The incidence of OSSN ranges from 0.02 to 3.5 per 100 000 population and varies geographically, with greater frequency near the equator. Generally, it is a slow growing tumour that rarely metastasises, but is capable of causing extensive local tissue destruction. Bilateral OSSN is extremely rare and offers a unique opportunity to study the biological characteristics of bilateral OSSN of the conjunctiva. The following case report describes the clinical presentation, histopathology, and immunohistochemical evaluation of tumour proliferation markers of a patient diagnosed with bilateral OSSN.

Case report

An 86 year old white woman was referred to the Doheny Eye Institute because of redness in her right eye that had developed over a period of several months. She had undergone a mastectomy in 1954. She had no history of ocular trauma, toxin exposure, or tobacco use. Her brother and sister died from liver cancer. An ophthalmic examination revealed a visual acuity of 20/100 in each eye. Ectropion and indurated lower eyelid margins were present bilaterally with no loss of cilia. A closer examination revealed a thickened epithelium that lined the palpebral conjunctiva and cul de sac of the right eye (fig 1A). The left lower palpebral conjunctiva showed similar changes. However, there was a focal nodule on the inferior bulbar conjunctiva (fig 1B).

The patient underwent a biopsy of the right palpebral conjunctiva. Histopathological examination of the specimen revealed...
acanthotic epithelium with squamous metaplasia, occasional dyskeratotic cells, parakeratosis, and hyperkeratosis. Multiple abnormal mitotic figures were present. The basement membrane was intact, and a diagnosis of conjunctival squamous cell carcinoma in situ was diagnosed. A subsequent biopsy of the left conjunctiva revealed full thickness squamous metaplasia of the epithelium with acanthotic and marked dysplastic changes. Multiple abnormal mitotic figures were seen and the basement membrane was also intact, with an extensive chronic inflammatory cell infiltrate in the stroma (fig 1C). These findings were also consistent with a diagnosis of squamous cell carcinoma in situ of the conjunctiva.

Immunohistochemical analysis (Dako, Carpinteria, CA, USA) revealed that neoplastic cells were positive for pankeratin, human papillomavirus (HPV) (fig 1D), and Ki-67 (MI B-1) in both specimens. Moreover, both biopsies indicated the presence of a few bcl-2 positive cells. The right eye biopsy was p53 positive and the left was p53 negative. The immunohistochemically positive cells were counted by methods described previously. Table 1 summarises the immunohistochemical findings of both biopsies. Because of the patient’s fragile health, surgical intervention was deferred and she was treated with topical mitomycin C in both eyes. At the follow up examination 13 months after the biopsy, a mass was found in the right lower palpebral conjunctiva. A second conjunctival biopsy was performed and she was treated with topical mitomycin C in both eyes. At the follow up examination 13 months after the biopsy, a mass was found in the right lower palpebral conjunctiva.

Comment

The aetiology of bilateral OSSN remains unclear. Causative factors that are believed to contribute to the development of unilateral OSSN include ultraviolet light exposure, ocular trauma, predisposing genetic factors, and infection with HPV. Previous reports have provided convincing evidence of an association with HPV type 16 in some cases of bilateral conjunctival dysplasia. It has been postulated that the interaction between HPV and ultraviolet light may have a role in the development of HPV related tumours in patients who are exposed to the sun. However, both conjunctival lesions in the present case were located in areas that were not exposed to sunlight, suggesting a possibility that HPV infection in both eyes may have led to the development of bilateral OSSN. Conjunctival OSSN has been described as a slow growing, well differentiated tumour of low grade malignancy that responds to local excision and rarely metastasises. Immunohistochemical studies of bilateral lesions allow comparison of the proliferative potency in both eyes, and offer a unique opportunity to study some biological aspects of bilateral tumours under the same environmental conditions at the same point in time. In recent years p53, bcl-2, and MI B-1 have been used as markers of proliferative potency. The p53 gene is a common cellular target in human carcinogenesis and is thought to have an important role in cellular proliferation. In contrast with the wild type p53, mutants of the p53 gene produce an abnormal protein with a long half life and are thus immunohistochemically detectable. Also, p53 has been reported to be a prognostic marker in several tumours. Bcl-2 is a proto-oncogene that is thought to have a role in oncogenesis by inhibiting programmed cell death and preserving cells from p53 induced apoptosis. However, the mutant p53 protein also induces apoptosis and decreases the expression of bcl-2 proteins. Mahomed et al suggested that the interplay between the effects of the increased mutant p53 proteins and the absence of bcl-2 expression in tumorigenesis may promote clonal expansion, leading to progressively increased genomic instability. The synergy of the presence of mutant p53 and absence of bcl-2 in the present case might have allowed the progression of the tumour in the right conjunctiva.

Ki-67 is a nuclear antigen expressed in all stages of the cell cycle except the resting stage. MI B-1 is a monoclonal antibody that recognises the Ki-67 antigen, which is a marker of cellular proliferation and reported to be a prognostic factor for various cancers. The high immunoreactivity of MI B-1 in conjunctival OSSN is usually associated with highly aggressive tumour growth. Our results demonstrate a higher immunoreactivity of MI B-1 in the right conjunctival specimen. These findings indicate that the right conjunctival specimen is more aggressive than the left, and is consistent with this patient’s clinical course.

In conclusion, this case represents a rare example of conjunctival pathology: OSSN as a bilateral tumour. To our knowledge, this is also the first report that compares the right and left biopsies of conjunctival OSSN by immunohistochemical analysis of potential oncogenic factors. Enhanced expression of MI B-1 and presence of mutant p53 protein in the absence of bcl-2 may contribute to the aggressive biology of OSSN.

Acknowledgements

This work was supported in part by NEI core grant EY03040 and by an unrestricted grant from Research to Prevent Blindness Inc, New York, USA.

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Accepted for publication 1 August 2003

References


Employing endoscopic guidance for placement of a black diaphragm aniridia intraocular lens following destructive Acanthamoeba sclerokeratitis

Anterior segment reconstruction can be particularly challenging when anatomic landmarks are lost. We describe a case of destructive Acanthamoeba sclerokeratitis resulting in aniridia, aphakia, loss of limbal architecture, and corneal opacification that was approached surgically with penetrating keratoplasty and placement of a black diaphragm aniridia intraocular lens under endoscopic guidance.

Case report

Our patient, a 48 year old female contact lens wearer, was diagnosed with Acanthamoeba keratitis in June 2000. Before our evaluation, she had been treated with tobramycin and...
dexamethasone ointment, topical trifluridine, oral acyclovir, oral prednisone, and topical prednisolone acetate 1%. We diagnosed Acanthamoeba keratitis and began aggressive treatment with polyhexamethylene biguanide, chloridehexide, and oral clotrimazole. By January 2001, she was culture negative, but had developed necrotising sclerokeratitis with limbal involvement, dense corneal opacification, and descemetocele formation. Urgent penetrating limbal keratoplasty was performed. Upon placement of the lid speculum, spontaneous perforation of the cornea occurred with prolapse of the lens and necrotic iris. We performed a 12.5 mm diameter keratomileusis resection, removed residual lens and necrotic iris, and performed anterior vitrectomy. A 13.0 mm keratomileusis graft was placed and covered with an amniotic membrane graft. (fig 1)

Eighteen months later, the patient had negative cultures, a quiet eye, an opaque corneal graft, controlled intraocular pressure, and counting fingers vision with projection to all four quadrants. However, the patient complained of severe glare and light sensitivity. Soft contact lens wear was unsuccessful because of irregular postsurgical topography. After extensive discussion, this highly motivated patient elected to pursue further anterior segment reconstructive surgery to address the aniridia, aphakia in the absence of capsular support, and corneal opacity.

Penetrating keratoplasty and implantation of a sulcus fixated Morcher 67F black diaphragm polymethylmethacrylate lens was planned. External landmarks for transscleral suture fixation had been lost due to infectious necrosis and the large keratolimbal graft. Instead, suture placement was guided by an ocular endoscope (URAM E2 MicroProbe Laser System, EndoOptiks, New Jersey, USA). Following excision of an 8.0 mm diameter corneal button, a 10-0 prolene suture on an STC-6 needle (Ethicon Inc, New Jersey, USA) was passed externally under a scleral flap and viewed internally via the endoscope as it entered the ciliary sulcus. The suture was passed through the lens fixation loop. A 25 gauge needle was passed externally into the ciliary sulcus under endoscopic visualisation, the STC-6 needle was passed into its bore, and the complex guided out of the eye. This process was repeated for the opposing haptic, the sutures were tied, and an 8.0 mm donor button was placed. In the early postoperative period, the intraocular lens was positioned without obvious decentration or tilt, and the patient reported substantial improvement in her glare symptoms.

Figure 1  Endoscopic view of the STC-6 needle as it enters the ciliary sulcus.

Comment
A black diaphragm intraocular lens design allows simultaneous treatment of aniridia and aphakia. The Morcher 67F has a 13.5 mm length, 10 mm diameter optic, and a 5 mm central clear zone. Precise haptic capture in the ciliary sulcus is necessary to minimise risks of haptic-optic crowding, mechanical irritation, and tilt or decentration of a small optic zone. Unfortunately, lens decentration and tilt is commonly observed following transscleral fixation of lenses. This can be attributed to suboptimal haptic position following blind passage of fixation sutures. Althaus and Sundamcher have described the usefulness of direct endoscopic visualisation in eyes undergoing transscleral suture lens fixation. In our patient, accurate lens position was critical, and the risk of lens malposition high, given her unfavourable anatomy. Our experience confirms that endoscopic visualisation is valuable for the placement of transscleral lens fixation sutures, particularly when surgical landmarks are lost and when mild lens malposition might adversely affect the surgical outcome.

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Accepted for publication 4 August 2003

A video clip of this procedure is available on the BJO website (www.bjophthalmol.com)

References

MAILBOX

Variant CJD and tonometry
We read with interest the paper by Lim et al. The authors interestingly showed the importance of tonometer head wiping in reducing corneal epithelial cell count and the significance of damaged prism surfaces in trapping debris. They hypothesised the possible role of variant Creutzfeldt-Jakob disease (vCJD) transmission from cornea epithelial cells present on the tonometer surface. The question is really one of what constitutes the infectious dose of vCJD for this mode of transmission, and this is currently unknown.

There has been one definite, one probable, and two possible cases of CJD through corneal transplantation but one can hardly compare the prion load in a full thickness corneal graft with a mean epithelial cell count of 910 (after wiping or wiping). Two of the four cases of transmission had multiple graft procedures.

The evidence from animal studies on CJD infected corneal transmission is also variable. Herzberg transplanted CJD infected corneas onto two Capuchin monkeys; both remained disease free for up to 4 years. Manuelidis et al showed transmission of CJD when infected corneas were placed directly into the anterior chamber of uninfected guinea pigs but did not show transmission of CJD after penetrating keratoplasty. Tateishi injected emulsified CJD infected cornea into the brains of six mice and only one developed characteristic changes after 2.8 years.

These studies certainly suggest that an intraocular/intracerebral delivery must be needed for transmission but even then, the inocula that produced disease after intracerebral inoculation only irregularly transmit disease after peripheral (intracameral) inoculation. In addition, host genetic factors (homozygosity at codon 129) will also determine the risk of susceptibility. There is also convincing evidence that the agent strain and host genotype will determine whether ocular involvement even occurs in experimental rodent models of scrapie.

More recently, western blot analysis on eyes of patients that had been infected with sporadic CJD and vCJD confirmed earlier results (also in human eyes) that PrPSc could only be detected in the retina and not in cornea or sclera. From the evidence, the risk of transmission from tonometry would suggest this to be more theoretical than practical, and may be reduced further—as the authors suggest—by adequate wiping or regular replacement of tonometer prisms. The question of risk can only be truly resolved by transmission studies using primary human diseased eye tissue.

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Accepted for publication 10 July 2003

References
Intravitreal triamcinolone acetonide for exudative age-related macular degeneration

We read the article by Jonas et al. on intravitreal triamcinolone injections for exudative age-related macular degeneration with interest. The paper stated that visual acuity increased significantly (p < 0.001) from 0.16 (SD 0.11) to a maximum of 0.23 (0.17). The authors therefore picked the best from one of up to 10 postoperative visual acuity measurements and compared it with a single preoperative visual acuity measurement. This is misleading the reader regarding the true effectiveness of the treatment.

The Photocoagulation Study Group found that the differences in between two repeated tests were one line or more in 13% of cases and the differences were greatest in patients with visual acuity of 20/100 or worse. By taking up to 10 postoperative measurements, Jonas et al. greatly increased the chances of a positive result. The difference between mean pre-injection 0.16 (20/125 or 6/36) and mean postoperative 0.23 (20/87 or 6/26) was less than one line on the Snellen chart.

Their table 1 gave the mean visual acuity pre-injection and at various time intervals post-injection. At 1 and 2 months, the p values were 0.04. It was unclear whether the p values were one or two tailed but both were described as not significant (NS) in table 1. Multiple significance testing at each of a number of time points is generally not recommended—if it is done, some kind of adjustment to the p values is needed. Looking at the results presented in table 1, readers might conclude that triamcinolone had a transient and doubtful beneficial effect on the visual acuity.

The authors go on to further analyse the results into improvements of three or six or more lines. The vision was tested on a Snellen chart which has irregular steps. Three or six lines do not therefore represent a constant change in visual angle (as in a logMAR chart) and therefore the analysis was confusing.

Variations in intraocular pressure of 5 or 6 mm Hg occur diurnally in normal individuals as well as glaucomatous patients. We believe that this change in IOP may affect the intraocular pressure, the comparison of the baseline with the highest value (p < 0.001) was misleading as was the comparison of the highest value with that at 7 months (p > 0.001). Of more interest might be the number of patients who had very high levels (the range extended to 64 mm Hg) and whether these very high intraocular pressures responded to treatment.

The authors’ experience in using triamcinolone is well recognised. We congratulate them on an otherwise excellent piece of work.

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Accepted for publication 17 July 2003

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Authors’ reply

We thank Wong and colleagues very much for their interest in our study. We agree with them that comparing a single preoperative measurement with the best out of a series of postoperative measurements gives a tendency towards a falsely high increase in visual acuity after the triamcinolone injection. That there was an increase in visual acuity after the injection in some patients, however, may have been demonstrated in table 1 giving the visual acuity measurements before and after the application of triamcinolone acetonide. At 1 and 2 months, the difference from the preoperative values was significant with a p value of 0.04. Unfortunately, the values are described as non-significant in what is a typographical error in the manuscript. We regret this error. The authors agree with Wong and colleagues, that the effect of triamcinolone acetonide is temporary, and that repeated injections may be necessary. In some patients, repeated intravitreal injections of triamcinolone acetone di were associated with repeated increase in visual acuity.

We also agree with Wong and coworkers that the preoperative intraocular pressure measurement with the highest value during the follow up again falsely increased the number of ocular hypertensive subjects after the injection. In this matter, however, it was not in favour of the treatment since it did not significantly increase the number of patients with unwanted side effects. Concerning the number of patients with very high intraocular pressures after the injection, a preliminary analysis shows that about 1% of the eyes injected will need filtering surgery (own data).

The authors would like to thank Wong and colleagues for constructively commenting on the study. In the study, we did the authors, that the intravitreal injection of triamcinolone acetone di treatment for exudative macular degeneration is still in clinical evaluation and that its therapeutic effect has not been proved so far. This may be even more important in a recent randomised study using 4 mg triamcinolone acetone di did not demonstrate an effect on the risk of loss of visual acuity during the first year of the study in eyes with classic choroidal neovascularisation. A significant anti-angiogenic effect, however, was found 3 months after treatment. In view of the anti-angiogenic, anti-inflammatory, and anti-endothelial effects of intravitreal triamcinolone, further randomised and different trials using different doses of triamcinolone acetone di eyes with different types of subfoveal neovascularisation may be warranted.

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Accepted for publication 17 July 2003

Viscosurgery in diabetic vitrectomy

Grigorian and colleagues recently recounted their experience of using viscosurgery to remove epiretinal membranes (ERMs) from eyes with proliferative diabetic retinopathy (PDR). They concluded that viscosurgery (‘viscoseduction’ (injection of Healon between the fibrovascular proliferations and the retina) is safe and is equally as effective as its non-use. On the contrary, their study shows that viscoseduction is not cost effective (because of the costs of both the viscoelastic and the extra operating time), and confirms that the technique is inherently unsafe in PDR.

The use of Healon to aid dissection of fibrovascular ERMs during vitrectomy was introduced by Jonas et al.18 but was not widely adopted. Viscoselastic material squirting out from under the ERMs was “messy” and led to the formulation (in the 1990s) of yellow tinted Healon to aid its visualisation and simplify its removal and then Healon GV (viscosity 10 times that of Healon) for adherent diabetic ERMs. In 1984, we began undertaking “viscodelamination” in diabetic vitrectomy. This technique was primarily directed at stripping the posterior vitreous cortex. Viscodelimination was especially useful in combined tractional and rhegmatogenous retinal detachments (CTRD) with very limited non-rhegmatogenous posterior vitreous detachment (PVD) present. Because of the prohibitive cost of Healon, methylcellulose 1% was injected in the majority of eyes.

To summarise our experience, stripping of the PVM usually proceeded uneventfully during slow pressurisation of the closed retrohyaloid compartment by viscoelastic, as did separation of any loosely adherent, sparsely vascularised ERMs which were contained within the peripheral vitreous cortex. In well photocogulated eyes, the separation
et al
Grigorian
operations. (It is acknowledged that
compared with three in 26 conventional
34 viscodissections in eyes in the range C4-6
there were 10 iatrogenic posterior breaks in
similar ("") complexity. Thus,
firmly in groups of eyes with pathology of
outcome compared with four in 89 eyes having
induced in 65 eyes undergoing viscodissec-
tion compared with three in 58 eyes
predicted in theory, but again unpredictable
pressure during the surgery. However, as was
had a detached retina 6 months after
vitreous haemorrhage). Six of 30 eyes (20%) was used, this was not the case in eyes with
CTRD group compared with three of 58 eyes
seven of 43 eyes (16%) in the viscosurgery
outcomes. After 6 months of follow up, for
appear to have been reflected in the ultimate
membrane.

The intraoperative problems
amount of traction necessary to dissect a
"" does not account for the degree of adhe-
"" rectus contracture" is currently printed as
"Compensatory head tilt away from side of
fixing eye." It should have been printed as
"Compensatory head tilt toward the side of
the fixing eye."

Elevation
the perpendicular hydraulic forces necessary
fibroglial membranes were sometimes
peeling the ERMs. Furthermore, recurrent
disrupted the retina ahead of, and instead of,
the hydraulic tension sometimes
outgrowths from the retina, ERM retinal
disrupted. Avulsion generally occurred at
the ERM and the retina tended to be
sometimes continued posteriorly, culminat-
ing in a complete PVD. In the case of more
adherent fibrovascular ERMs, their "visco-
Elevation" sometimes occurred through
stretching of the vascular and glistening pegs
classified the ERMs to the retina. The PHM
and ERMs could then be removed en bloc
using the suction cutter. However, instead of
stretching, the vascular connections between
the ERM and the retina tended to be
disrupted. Avulsion generally occurred at
the point of greatest weakness at the origins
of neovascular outgrowths from the retinal
veins. Although correlating with ERM vascu-
larity and with the density of neovascular
outgrowths from the retina, ERM retinal
adherence was unpredictable, and bleeding
was ultimately an inevitable consequence of
the perpendicular hydraulic forces necessary
to effect peeling of more adherent ERMs.
Fortunately, the bleeding from side punctures
in the retinal veins was constrained by the
viscoelastic (so called "haemorrhagic con-
finement") and a high ambient intraocular
pressure during the surgery. However, as was
predictable in theory, but again unpredictable
in practice, the hydraulic tension sometimes
disrupted the retina ahead of, and instead of,
peeling the ERMs. Furthermore, recurrent
fibroglial membranes were sometimes
observed later even in eyes where viscodela-
mination had proceeded uneventfully. This
has been attributed to the difficulty in
completely removing viscoelastic from the
retinal surface, with preretinal retention of
growth factors. Not for nothing is one
viscoelastic mixture marketed as Viscoat.
We had discontinued viscosurgery in PDR
by 1988 in favour of purely mechanical
methods that minimise ERM elevation.
Fifteen years on and Grigorian and collea-
gues have clearly come to a very different
conclusion from ours despite reporting a
considerable excess of iatrogenic posterior
retinal breaks during, and recurrent detach-
ment after, viscosurgery. By back calculation
from their assiduously collected data, it
appears that 20 posterior breaks were
induced in 65 eyes undergoing viscodissec-
tion compared with four in 89 eyes having
conventional surgery. This trend was con-
firmed in groups of eyes with pathology of
similar ("relatively high") complexity. Thus,
there were 10 iatrogenic posterior breaks in
34 viscodissections in eyes in the range C4-6
compared with three in 26 conventional
operations. (It is acknowledged that
Grigorian et al state that the complexity score
"does not account for the degree of adhe-
sion," neither was it "a good predictor of
the amount of traction necessary to dissect a
membrane." ) The intraoperative problems
appear to have been reflected in the ultimate
outcomes. After 6 months of follow up, for
example, a detached retina was evident in
seven of 43 eyes (16%) in the viscosurgery
group compared with three of 58 eyes
(5%) undergoing conventional surgery.
Furthermore, although eyes with CTRD
seemed to fare well whether or not Healon
was used, this was not the case in eyes with
tractional detachments (with or without
vitreous haemorrhage). Six of 30 eyes (20%) had a detached retina 6 months after
viscodissection compared with only two of
37 eyes (5%) after conventional surgery.
Indeed, most of the data favoured conven-
tional surgery. Lower viscosity Healon was
proposed as a future means of reducing the
frequency of iatrogenic breaks, but this is
unlikely to be helpful in their attempt to
achieve the impossible—that is, a worth-
while increase in the case success of ERM removal without an unacceptable
added risk of retinal haemorrhage, tears,
and scarring. Better by far would be to avoid
viscoelasticst altogether.
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Accepted for publication 21 July 2003

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In the article by Brodsky et al in the February
issue (Br J Ophthalmol 2004;88:268–272), a
portion of the text within fig 2 was incor-
rectly labelled. The label under ‘‘Superior
rectus contracture’’ is currently printed as
‘‘Compensatory head tilt away from side of
fixing eye.’’ It should have been printed as
‘‘Compensatory head tilt toward the side of
the fixing eye.’’
Potential diagnostic dilemmas using the multifocal electroretinogram in intermittent exotropia

Multifocal electroretinography (mfERG) is a valuable technique in assessing macular function in retinal disease objectively as it provides spatial information. Altered responses give an estimate of the extent of central retinal dysfunction. Fixation is known to be an important technical factor in mfERG recording. We present findings in a patient with asymptomatic intermittent exotropia that reinforce the importance of adequate consideration of potential fixation errors.

Case report

The patient was a 52 year old man with maternally inherited diabetes and deafness (MIDD) consequent upon a mitochondrial DNA nucleotide A3243G point mutation, and examined as part of a series of patients with MIDD. Visual acuity was 20/20 (ETDRS chart) bilaterally. Fundi showed symmetrical bilateral irregular patches of retinal pigment epithelium atrophy at the posterior poles. The foveas were normal.

Macular function was assessed initially by mfERG recorded binocularly with a stimulus size of 61 hexagons using the RETI-scan System (Roland Consult, Wiesbaden, Germany). The patient fixated on the centre of a large diagonal cross, centred over the central hexagon, at a viewing distance of 33 cm. Pupils were dilated. Refractive errors were corrected with −6.25 dioptres (D) right eye and −6.25 spherical dioptres combined with −0.75 cylindrical dioptres at 5° left eye. Additional +3D were given for a viewing distance of 33 cm. Each recording session consisted of eight trials over about 20 minutes.

Upon binocular recording, changes reflecting the retinal dystrophy were visible in the right eye trace array outside the central hexagon. The normal foveal response was consistent both with normal foveal function and central fixation throughout testing (fig 1A). Amplitude reduction was observed in many left eye traces with an additional ‘off centre’ peak also visible in three dimensional plot (fig 1B). These findings are not suggestive of MIDD. The mfERG was repeated monocularly. The left eye findings now showed a normal central response and alterations in parafoveal function consistent with MIDD (fig 1A, B).

Subsequent orthoptic examination revealed a near type intermittent exotropia with poor motor fusion and additional microtropia. The latent deviation of the left eye was 2 prism dioptries base-in at 6 metres and 18 prism dioptries base-in at 33 cm. A small vertical height component was demonstrated on the Hess chart. Stereopsis was subnormal.

Comment

Patients with intermittent exotropia can be completely controlled having binocular vision or may have a manifest exotropia. Under binocular mfERG stimulation, the left eye presumably fixated in exotropia at times of fusional decompensation, and the stimulus pattern shifted by the extent of the squint deviation. At times of positive binocular vision the fixation was located almost centrally. The fixation was slightly shifted because of the microtropia (fig 1B) which was not detected by direct observation.

Examiners should always be aware that not only retinal disease can affect the mfERG. Asymptomatic strabismus is a reason for fixation instability and represents a potential dilemma in the interpretation of binocular mfERGs. Even with direct observation a small intermittent strabismus may be not be detected. This could result in a broadened central peak rather than the double peak seen in our patient and thus be mistaken for macular dysfunction. This would be potentially disastrous in a patient with optic nerve disease where the mfERG should be normal.

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doi: 10.1136/bjo.2003.040584

Accepted for publication 31 January 2004
Furosemide is a potent diuretic which is an anthranilic acid derivative. Chemically, it is 4-chloro-N-furfuryl-5-sulfamoylanthranilic acid. Furosemide is indicated for the treatment of oedema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome.

Here I report a case of a diabetic patient, with nephrotic syndrome, who experienced marked improvement in diabetic macular oedema after systemic treatment with furosemide.

**Case report**

A 41 year old woman with type II insulin dependent diabetes mellitus was referred for decrease in vision in both eyes over the past 2 months. Besides the diabetes, her past medical history was positive for irregular menstrual cycle and gastroparesis. The patient had also noticed a gain in weight of about 30 lb (13.5 kg) over the same period of time, from 154 lb (69.3 kg) to 196 lb (88.2 kg). She was treated with insulin for the diabetes and Regulin Forte for the nephrotic syndrome. She was referred for the diabetes and Regulin Forte for the nephrotic syndrome. She was referred for the diabetes and Regulin Forte for the nephrotic syndrome.

The patient lost 30 lb (13.5 kg) and she had improvement in her vision. In 3 weeks the patient lost 30 lb (13.5 kg) and she had returned to her usual weight of 154 lb (69.3 kg). Three weeks later her vision had improved to 20/80 in both eyes. On fundus examination there was marked improvement in the macular oedema in both eyes. OCT examination confirmed the partial resolution of the macular oedema. The central retinal thickness measured by OCT was 763 μm in the right eye and 722 μm in the left eye (fig 2A–B). The patient was offered grid laser treatment for the macular oedema, but she did not feel well and she refused the treatment. A follow up appointment was arranged for 3 weeks’ time.

The following day the patient presented to the emergency room complaining of nausea and weakness. She was admitted to the hospital with a diagnosis of nephrotic syndrome and fluid overload. Her albuminuria level was 350 mg/l (normal value <12 mg/l).

The patient was treated with systemic furosemide 40 mg twice a day for 2 weeks. A few days after starting the treatment with furosemide she began to lose weight. She also noticed an improvement in her vision. In 3 weeks the patient lost 30 lb (13.5 kg) and she had returned to her usual weight of 154 lb (69.3 kg). Three weeks later her vision had improved to 20/80 in both eyes. On fundus examination there was marked improvement in the macular oedema in both eyes. OCT examination confirmed the partial resolution of the macular oedema. The central retinal thickness measured by OCT was 250 μm in the right eye and 218 μm in the left eye (fig 2C, D).

**Comment**

Diabetic macular oedema is characterised by hyperpermeability of retinal blood vessels and subsequent formation of hard exudates and macular oedema, the degree of which can be estimated by measurement of retinal thickness. The severity and progression of diabetic macular oedema has been associated with the presence of nephrotic syndrome and to the degree of proteinuria.\(^\text{1-7}\) In a recent...
retina fluid collection is secondary to a defect in a patient where the subneurosensory osemide could worsen the presence of a leakage as evaluated by urinary albumin thickness as evaluated OCT, and glomerular et al demonstrated the pre-

The exact mechanism of the action of furosemide in resolving the macular oedema. The oedema thickness is unknown. Tsuboi et al. demonstrated that furosemide inhibits primarily the absorption of sodium and chloride not only in the proximal and distal tubules but also from abnormal salt and water retention. Furosemide is used in the treatment of fluid overload experienced by patients with nephrotic syndrome because it is a potent and other lipids, and oedema. The oedema results not only from the hypo-osmolar state caused by the loss of plasma proteins, but also from abnormal salt and water retention.

Nephrotic syndrome is characterised by massive proteinuria, which leads to hypoproteinaemia/hypoalbuminemia, hyperlipidaemia with elevated cholesterol, triglycerides and lipid. The study Knudsen et al demonstrated the presence of an association between retinal thickening as evaluated OCT, and glomerular leakage as evaluated by urinary albumin excretion rate in type II diabetic patients with diabetic macular oedema.7

Furosemide is used in the treatment of fluid overload experienced by patients with nephrotic syndrome because it is a potent and rapid-acting diuretic. It has been demonstrated that furosemide inhibits primarily the absorption of sodium and chloride not only in the proximal and distal tubules but also in the loop of Henle.

I reported what is, to my knowledge, the first case of marked improvement of diabetic macular oedema after systemic treatment with furosemide documented by OCT examination. I think that the fluid overload secondary to the nephrotic syndrome was the main cause of the worsening of the macular oedema in this patient, and that the intensive treatment with furosemide was responsible for reducing the amount of fluid overload and resolving the macular oedema. The exact mechanism of the action of furosemide in resolving the macular oedema is unknown. Tsuboi et al demonstrated that furosemide inhibits fluid absorption across retinal pigment epithelium (RPE) in an experimental model of retinal detachment in monkeys.8 According to their study furosemide could worsen the presence of a neurosensory macular detachment especially in a patient where the subneurosensory retina fluid collection is secondary to a defect in RPE pump.9 However, the FA study of this patient at presentation demonstrated multiple retinal microaneurysms at the posterior pole of both eyes and diffuse intraretinal microvascular leakage of dye in the late phase of the study. The FA study was consistent with the appearance of diffuse diabetic macular oedema. In such cases the intraretinal and subneurosensory retina leakage of dye is secondary to a microvascular retinal defect and not to a failure in the RPE pump. I think that restoration of more normal oncotic pressure within the retinal vasculature was the underlying reason for the response to the drug of the diabetic macular oedema.

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doi: 10.1136/bjo.2004.042580

Accepted for publication 17 March 2004

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Vision loss as a complication of gamma knife radiosurgery for trigeminal neuralgia

Gamma knife radiosurgery has been found useful for treatment of trigeminal neuralgia (TN).1–4 Although it is generally safe and well tolerated by most patients, adverse effects have been reported.1–4 Potential ocular complications include “dry eye” and “corneal numbness.”5–6 We describe a case of vision loss that occurred 9 months after gamma knife radiosurgery for TN.

Case report
A 68 year old man presented in September 2003 with 3 weeks of fluctuating blurred vision in the right eye. The blurring began 3 weeks earlier and had been preceded by complete numbness of the right side of his face for 1 week.

The patient’s medical history was remarkable for right sided TN that began in 1998, predominantly involving the V2 dermatome. It had been managed medically at first but eventually became incapacitating and led to hospital admission. In December 2002, he had undergone gamma knife radiosurgery (Leksell Gamma Knife; Elekta Inc, Norcross, GA, USA). The dose matrix grid was 0.6 mm and the right trigeminal root entry zone was targeted tangential to the brainstem. The prescription dose was 40 Gy to the 50% isodose line. A single run with a single shot (treatment time, 29.25 minutes) used a 4 mm collimator. Within days, the patient had clinically significant improvement in symptoms, was pain free, and required no pain medication.

The patient also had a history of chronic renal failure requiring dialysis three times weekly, hypertension, a myocardial disorder, prostate cancer, and hyperlipidaemia. In 1998, he had resection of a left frontal lobe meningioma. His ocular history included bilateral cataract surgery but no history of the posterior or herpes simplex.

On initial examination, his vision measured 20/25 right eye and 20/25 left eye. Slit lamp examination of the right cornea revealed a fine punctate epitheliopathy (fig 1A). Corneal sensation, tested with a Cochet-Bonnet aesthesiometer (Luneau Ophthalmologie, Chartres Cedex, France), was absent even at a 5 mm filament length, both subjectively and by blink reflex. Sensation in the left cornea was present at a 60 mm filament length.

The patient’s vision gradually declined despite treatment with preservative free artificial tears and placement of a punctal plug in the right lower lid. Ten weeks after presentation, his vision measured 20/200 right eye and 20/25 left eye. Slit lamp examination revealed severe epithelial keratopathy in the right eye (fig 1B). Fourteen months after radiosurgery, 12 weighted magnetic resonance imaging showed increased signal intensity in the anterior aspect of the right fifth nerve (fig 2).

A punctal plug was placed in the right upper lid. The patient began using topical serum tears four times daily. Partial tarsorrhaphy was discussed with the patient but

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Figure 2 OCT examination of the right eye (A) and of the left eye (B) demonstrates marked thickening of the macula and large, intraretinal, fluid filled, cystoid spaces. Note the presence of a serous elevation of the neurosensory retina in the right eye. (C, D) OCT examination of the right eye (C) and of the left eye (D) of the same patient after systemic treatment with furosemide for 2 weeks demonstrates marked improvement in the macular oedema. There is almost complete resolution of the intraretinal cystoid spaces in both eyes. There is persistence of a small, shallow, subfoveal, serous, neurosensory detachment in the right eye.

A
B
C
D

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Our patient had a vision loss to 20/200 associated with the onset of right sided facial numbness 10 months after low dose (40 Gy) gamma knife radiosurgery for TN. Although high dose radiosurgery (90 Gy) is a known risk factor for complications with gamma knife radiosurgery, the low dose our patient received has not been associated with such complications. Patients undergoing gamma knife radiosurgery for TN should be warned of this potential complication and should be evaluated preoperatively and postoperatively by an ophthalmologist.

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doi: 10.1136/bjo.2004.045773
Accepted for publication 16 March 2004

Figure 1 (A) Neurotrophic punctate epithelial keratopathy in the right eye at presentation. (B) Advanced neurotrophic keratopathy 10 weeks after initial presentation.

Figure 2 T2 weighted magnetic resonance imaging shows increased signal intensity in anterior aspect of right fifth nerve.

decided. During the next 6 weeks, the corneal surface improved, and at last follow up the patient’s vision was 20/60.

Comment
Gamma knife radiosurgery is an effective treatment for TN with few complications.3,4 Pollock et al6 noted an increased incidence of “trigeminal dysesthesia” and “corneal numbness” after high doses (90 Gy) of gamma knife radiation. In an animal model, a 100 Gy dose caused nerve necrosis.7 Matsuda et al8 identified a “dry eye complication” of epithelial keratopathy after gamma knife radiosurgery for TN. Despite these documented ocular side effects, no cases of vision loss have been reported.4

Neurotrophic keratopathy has been recognised in patients with herpes simplex, herpes zoster, and after laser in situ keratomileusis (LASIK) surgery.9,10 Mild neurotrophic keratopathy may be manifested as a punctate epithelial keratopathy. In severe cases, corneal decompensation can lead to severe vision loss.

Combined aspirin and clopidogrel in cataract surgical patients: a new risk factor for ocular haemorrhage?
Clopidogrel (Plavix, Bristol-Myers Squibb/Sanofi) is a thienopyridine with antiplatelet aggregation pathways.11 Both aspirin and clopidogrel have established benefits in the secondary prevention of fatal and non-fatal coronary and cerebrovascular events.12 The CURE study has concluded that combining low dose aspirin and clopidogrel in patients with acute coronary syndromes results in additional improvement in outcome over aspirin alone.13 We can therefore expect increasing numbers of ophthalmic patients who have been started on this combined treatment (“COM”).

Departmental concerns were raised by experience with a 76 year old normotensive patient who was on COM. He developed progressive zonal dialysis from unexpected vitreous pressure during standard phacoemulsification. An intracapsular extraction and anterior vitrectomy were required. An iridectomy led to extensive intraoperative hyphaema and vitreous haemorrhage. Postoperative ultrasound confirmed no evidence of choroidal haemorrhage, and the vitreous blood cleared within 3 months to produce 6/6 Snellen acuity with aphakic contact lens correction.

There is a lack of adequate data on the risk of surgery associated ocular bleeding with COM. Clopidogrel taken alone causes less gastrointestinal haemorrhage than aspirin, but has an otherwise similar risk profile to aspirin.9,10

Post-marketing surveillance of clopidogrel has recorded “conjunctival, ocular, and retinal haemorrhage.” Further information regarding these events, and whether they occurred during ocular surgery, was not available at the time of writing (Nicola Murphy, Sanofi Pharmacovigilance Department, personal communication February 2004). A detailed Medline literature search has produced no relevant ophthalmic case experiences.

The CURE study found higher “major and minor” bleeding rates in patients taking COM compared with aspirin alone, but showed no increase in life threatening or intracranial haemorrhage, and does not record ocular haemorrhage. Of note is the significant increase in major bleeding events recorded in COM patients undergoing coronary artery bypass graft surgery where the clopidogrel was stopped less than 5 days before the procedure.12

A February 2004 telephonic survey of nursing staff running cataract pre-assessment clinics at 15 ophthalmic units across the United Kingdom indicated variable approaches to such agents. Individual consultants at three units stopped clopidogrel preoperatively. Twelve units continued clopidogrel; none had specific policy for patients on aspirin and clopidogrel. The related and relevant issue of anaesthetists’ approach to regional ocular anaesthesia in patients on such treatment was not addressed.

Translating the CURE study results into a “number needed to treat,” approximately 47 patients with acute coronary syndrome would require treatment for 9 months with aspirin and clopidogrel to prevent one cardiovascular death, non-fatal myocardial infarction, or stroke.13 Stopping clopidogrel for a short period is therefore unlikely to make a material difference to the vascular event risk for an individual.

In summary, there is an increased risk of systemic bleeding associated with COM compared to aspirin alone. The degree of perioperative bleeding risk with elective eye surgery is still undefined. Our departmental policy has been changed to stopping clopidogrel for 1 week in patients on combination treatment given for cataract surgery, and to use a similar approach to that normally
employed for patients taking aspirin in those on clopidogrel alone. Other departments’ experience with this increasingly used anti-platelet agent would be valued.

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doi: 10.1136/bjo.2004.045997
Accepted for publication 11 March 2004

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The role of corticosteroids in fungal keratitis: a different view
Fungal infections of the cornea continue to be an important cause of ocular morbidity.¹ This report describes a situation which occurs in clinical practice in patients with misdiagnosed fungal ulcers who are treated with a combination of topical steroids and antibiotics drops. A common strategy when these patients are finally diagnosed with fungal keratitis is to switch to antifungal agents and discontinue the corticosteroids. We have recently seen two patients with fungal keratitis who demonstrated severe inflammation and corneal necrosis after the abrupt discontinuation of corticosteroids.

Case 1
A 32 year old woman with a corneal transplant in her right eye was referred for evaluation of an unresponsive corneal ulcer in her transplant. The patient had been treated with a combination of moxifloxacin 0.5% drops hourly and prednisolone 1% drops four times per day. On initial examination there was a diffuse corneal infiltrate consistent with the presence of an endothelial plaque and hypopyon. We performed confocal microscopy which showed hyphae characteristic of a fungal infection. After cultures were taken we modified the therapeutic regimen to flucncola

Case 2
A 13 year old girl who was a soft contact lens wearer was referred for evaluation of a corneal ulcer. The patient had been treated for 2 weeks with cefazolin 50 mg/ml and tobramycin 0.3% on an hourly basis, prednisolone 1% five times per day, and ketocnazole 400 mg by mouth. On initial examination there was a diffuse corneal infiltration consistent with the presence of an endothelial plaque and hypopyon. The patient had been treated with a discontinue the steroid drops. The patient showed signs of worsening during the next 2 days; the cornea perforated and an emergency keratoplasty was performed.

Comment
The analysis of the previous cases suggests that in patients with fungal keratitis who previously received topical corticosteroids, the abrupt cessation of these agents is likely to lead to an acute inflammatory reaction and even perforation. The proper use of corticosteroids in the treatment of fungal corneal infections continues to be debated among experts.³ The controversy arises because there are two goals in the treatment of corneal infection that are inherently incompatible: (a) to rid the affected tissue of the replicating microorganisms causing the infection, and (b) to limit the degree of structural damage caused by the infectious process.³ We recommend a gradual tapering of the corticosteroids in these cases which allows for the antifungal agents to act, and the host immune mechanisms to take control of the inflammatory response. However, clinical application in patients should be determined individually in all cases.

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doi: 10.1136/bjo.2004.047423
Accepted 31 March 2004

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Authors’ qualifications and the BJO
It is not often that journal policy is dictated by input from the readership. A notable exception to this probably occurred in the ANZ Journal of Surgery in 2002, following a letter to the editor in 2001 addressing authors’ qualifications.¹ In it we pointed out that journal aspirations to international recognition and increased circulation may be enhanced by having the authors’ qualifications consistently published. We indicated that the qualifications of one’s immediate superiors, including departmental heads where one may have trained overseas, can be recognised. The educational progress of one’s colleagues—for instance, a clinician’s higher qualifications (for example, PhD) may be determined. We pointed out that the reader can determine whether the author is in effect a qualified ophthalmologist, a resident, or still a medical student. In some parts of the world, the rivalry between optometrists and ophthalmologists may be highlighted by one group publishing in the other’s journal. Thus, qualifications may be used to discriminate between the two groups. Where the qualification discriminates between physicians and surgeons, this too can be recognised. In these days of enhanced medicolegal confrontation, a medical practitioner’s viewpoint can be differentiated from that of a lawyer.

Finally, we pointed out that if author qualifications are designated, the reader may be quite sure that the article was not written by the medical records librarian, let alone the hospital trolley boy in a moment of inspiration.

We have observed that in recent issues of the BJO, there appears to be an inconsistent approach to appending qualifications. Only the corresponding author is liable to be given a qualification; the first author usually goes without. For example in volume 88 number 5 (May 2004), in the perspective, only the corresponding author, Azuara-Blanco writing on cannabinoids and glaucoma received a qualification. In the extended reports, only Miyamoto on oil droplets in rabbits, Shaarawy on day one intraocular pressure, Orgul on blood flow in glaucoma, and Probst on fibronectin in diabetes received recognition. We pointed out that the authors of all the other extended reports missed out. In other words, in this issue of the journal, only one third of the corresponding authors, let alone the co-authors of extended reports had qualifications. No one in the letters section was designated with a qualification. None of the three editorial writers received a qualification. We are left wondering as to whether Professor König, writing on the cost effectiveness of treatment for ambylopia, was a paediatric ophthalmologist branching out into community medicine, a medical politician, a health economist, a statistician, or a psychologist having a degree in medicine. Whatever he is, he reached a reassuring conclusion in his article, that amblyopia therapy is “likely to be very cost effective.”³ We also do not know whether Schwab, writing about the “Halcyon days,” with the university affiliation of UC Davis, was an artist, the university photographer, an ornithologist, an anthropologist, or a Greek mycologist. His article on postoperative leak in trabeculectomy, Henderson can be recognised as a surgeon by the British appellation “Mr.” We thus presume he has an FRCS or an FRCOphth, but we don’t really know.

Our point is made. As we demonstrated in our original article,¹ 89.5% of the 19 journals regularly read by us use author qualifications.
A journal of the integrity, breadth, and currency of the BJO should, in our view, append author qualifications in 2004.

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doi: 10.1136/bjo.2004.047068
Accepted for publication 17 June 2004

MAILBOX

Mohs surgery: efficient and effective

We read with interest the report of Hsuan et al.1 The authors present a case series of 55 patients with basal cell carcinoma on the eyelids. There are no details regarding the size or histological subtypes of basal cell carcinoma in the results and therefore it is difficult to assess the applicability of the results to other groups of patients who may have more or less severe basal cell carcinoma. The authors make several generalisations regarding Mohs surgery that we believe are unsubstantiated and we wish to take the opportunity to clarify a few points.

The essence of Mohs micrographic surgery is 100% histological frozen section margin control. There is no other technique that enables 100% margin examination, including the authors’ bread loaf section technique. Mohs micrographic surgery has 99% 5-year cure rates for basal cell carcinoma because of the thorough margin examination. In distinction, standard bread loaf section technique examines approximately 0.1% of the surgical margin, with an increased potential to miss infiltrative tumour extensions. Because the bread loaf technique is least likely to accurately detect a positive margin, many surgeons employ a tangential peripheral section analysis as a means of obtaining more thorough examination of the margin.

Mohs micrographic surgery has another advantage, which is true tissue sparing. The margin of normal skin removed during Mohs micrographic surgery may be as little as 0.5 mm. When operating on the eyelid, I must be the difference between sacrifice and preservation of a critical structure (that is, punctum). The authors sacrificed 2 mm on both sides of the skin cancer, which in some cases may have resulted in up to 3 mm of unnecessary skin removal. This could result in more complicated reconstruction for patients.

The authors state that their patients were happy to have multiple operative sessions. For patients undergoing Mohs micrographic surgery, complete tumour removal is accomplishment in one session, with reconstruction performed on the same day as tumour extirpation. The inconvenience to patients associated with staged re-excision after 48 hours of histological examination and then a final stage reconstruction 48 hours after the last histological sample is taken should not be underestimated. Patients in general are pleased with their care based primarily with their interaction with the physicians. However, we doubt that any patient would choose three surgical interventions over 5 days rather than one surgical intervention with 100% margin control in 1 day.

The authors state that Mohs surgery is “too expensive.” This statement is unsubstantiated. In a cost analysis by Cook and Zitelli,2 Mohs surgery was found to be similar in cost to excisional surgery and less expensive than frozen section analysis. With three potential operative encounters, the cost of staged excision of basal cell carcinoma in the United States would exceed that for Mohs micrographic surgery with reconstruction on the same day. It is also important to note that the pathological charges are included in the Mohs surgery fee, as the Mohs surgeon functions as both the surgeon and pathologist. Therefore, pathology charges generated for multiple staged re-excisions must be included in any calculation of cost associated with staged excision.

The authors characterised Mohs surgery as “laborious.” I would argue that one doctor performing a very efficient tissue sparing operation all in a matter of 2–4 hours, a typical duration for Mohs surgery and reconstruction, with the pathology included within that time frame and fee, is both cost efficient and labour efficient. Mohs surgery has been especially designed for accuracy, tissue sparing, convenience, cost efficiency, and labour efficiency.

Mohs surgeons are expert in the complete removal of complex skin cancers, particularly on the central facial area. Mohs surgeons work closely with our colleagues in oculoplastic surgery in the United States to coordinate expert reconstruction of the resultant defects. In places where Mohs surgery is less available, close communication between the surgeon and pathologist, and tangential vertical margin processing may offer a reasonable therapeutic option, although one that is more inconvenient, costly, and laborious for patients and physicians alike.

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doi: 10.1136/bjo.2004.047068
Accepted 19 March 2004

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Macular infarction after intravitreal amikacin: authors’ reply

We thank Doff et al for their useful and expert opinion.3 The choice of which agent to use to empirically treat Gram negative organisms implicated in endophthalmitis remains controversial. As amikacin has been proved to cause macular infarction, we think one should look at viable alternatives. Ceftazidime is already in widespread use in the United Kingdom and appears not only to have an excellent safety profile but also good clinical effect. Unfortunately, until we have proper in vivo and in vitro “head to head” comparison studies, it is difficult to know which is the more efficacious agent. As far as synergism is concerned, vancomycin and ceftazidime are usually not tested together because vancomycin acts on Gram positive organisms and ceftazidime is used primarily for Gram negative infections. However, there is one study that reported synergy between vancomycin and ceftazidime against Gram positive organisms.4

The study by Kwok and colleagues raises a concern that ceftazidime precipitation, as assessed by in vitro studies, may affect its action in vivo.5 The authors of our study have noticed temporary precipitants in vivo without apparent alteration of clinical effect (AR). Previous animal models do show that ceftazidime reaches intravitreal minimal inhibitory concentrations for Gram negative microbes after a single intravitreal injection.6 Perhaps assay at the time of repeat injection, non-invasive confocal Raman spectroscopy of the anterior chamber, or further animal models may provide additional insight into ceftazidime pharmacokinetics and the phenomenon of ceftazidime precipitation so as to guide future therapeutic choice. Ultimately the decision lies with the treating surgeon, who should be aware of both the efficacy and safety profiles of the agents available. We still believe, with the evidence presented in our article,7 that ceftazidime currently represents the best agent for the treatment of Gram negative microbes in endophthalmitis.

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doi: 10.1136/bjo.2004.042838
Accepted 22 January 2004

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LASIK in children?

O’Keeffe and Nolan report on LASIK surgery in five children with unilateral high myopia who were presumed to have amblyopia.8 One subject had bilateral high myopia.

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Optic nerve hypoplasia is associated with high myopia. In addition, anisometropic myopia is a common sequela of retinopathy of prematurity. Thinning of the sclera without posterior staphyloma formation has long been known to be associated with high myopia. Best corrected visual acuity in these patients is often limited by associated retinal and chorioretinal pathology.

None of the treated eyes obtained acuity better than 6/15. This limited outcome following refractive surgery may be because optical enlargement of the retinal image rather than enhanced neurosensory function.

In the three children who were less than 3 years old improved literacy, familiarity with the test procedure, and the Hawthorn effect were certainly important factors in their assumed improvement. The absolute lack of progress in one child was a probable manifestation of pre-existing retinal pathology rather than non-compliance with patching."

The authors advocate increased use of LASIK to thin the corneas of highly myopic children who already have profound reductions in scleral thickness. “From a clinical viewpoint, optic nerve hypoplasia should be carefully looked for in all patients with unilateral bilateral high myopia and visual loss.” It may well be more appropriate to improve the quality of retinal and optic nerve evaluations before performing irreversible surgical procedures with unknown long term consequences for these abnormal eyes.

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doi: 10.1136/bjo.2004.042853
Accepted for publication 22 January 2004

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NOTICES

4th International Congress on Autoimmunity
The 4th International Congress on Autoimmunity will take place 3–7 November 2004 in Budapest, Hungary. The deadline for the receipt of abstracts is 20 June 2004. Further details: Kenes International Global Congress Organisers and Association Management Services, 17 Rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland (tel: +41 22 908 0488; fax: +41 22 732 2850; email: autoimm04@kenes.com; website: www.kenes.com/autoimm04).<ref>

XVI International Congress for Eye Research

Ophthalmic Anesthesia Society
The 18th Annual Meeting of the Ophthalmic Anesthesia Society will be held on 1 – 3 October 2004 in Chicago, USA. For further details: Ophthalmic Anesthesia Society (OAS), 793-A Foothill Blvd, PMB #119, San Luis Obispo, CA 93405 USA (tel: 001 805 534 0300; fax: 001 805 534 9030; email: info@eyeanesthesia.org; website: www.eyeanesthesia.org).

Glaucoma Society Silver Jubilee Meeting 2004
The Silver Jubilee Meeting and Dinner for the Glaucoma Society will be held on 3 December 2004 at the Royal College of Physicians in Regents Park, London. The meeting will take place between 8.30am and 5pm and the dinner will be held between 6.30pm and 10pm. For further information, please contact: Janet Flowers, Administrator, 29 Quarry Hill, Grays, Essex, RM17 5BT (tel: 01375 383172; e-mail: glaucos@ukrice.freeserve.co.uk).

Amsterdam Retina Debate
The Amsterdam Retina Debate will be held on 10 December 2004 at the Academic Medical Centre, Amsterdam, The Netherlands. For further information, please contact: Nicolaas Tulp Institute; tel: +31 20 566 8585; fax: +31 20 696 3228; email: retinadebate@amc.uva.nl

Sunshine Fund for Blind Children
The Royal National Institute of the Blind are permanently in great need of new, used, foreign, British and all other kinds of postage stamps. The stamps are sold to raise money for children in need of specially adapted toys and everyday gadgets, holidays, books for parents and the any other needs of blind and partially sighted children throughout the UK. Please send stamps (British and foreign stamps should be sent in separate envelopes) to the following address: RNIB, PO Box 6198, Leighton Buzzard, LU7 9XT.

Prestigious Helen Keller Foundation prize awarded to one of London’s most eminent ophthalmologists
Professor Alan Bird, Institute of Ophthalmology, University College London and Consultant Ophthalmologist at Moorfields Eye Hospital NHS Trust, has been awarded one of ophthalmology’s most prestigious prizes, the Helen Keller Prize for Vision Research. The prize was created in 1994 by the Helen Keller Foundation for Research and Education, based in the USA, and honours the scientists and researchers working in the field of blindness and visual loss. Professor Bird is one of the world’s leading experts on age related macular degeneration (AMD), inherited macular degeneration and Retinitis Pigmentosa (RP), and has led research into the identification of the genes which cause retinal degeneration. As well as his scientific research, Professor Bird also continues to treat patients at regular clinics at Moorfields Eye Hospital. Further information on Moorfields is available at: www.moorfields.nhs.uk. Further information about the Helen Keller Foundation is available at www.helenkellerfoundation.org. Further information on the Institute of Ophthalmology is available at www.ucl.ac.uk/ioo.

Sophie sees sight saving projects in Tanzania with VISION 2020
HRH The Countess of Wessex has recently returned from a trip to Tanzania in her role as Patron of VISION 2020: The Right to Sight. Throughout the trip The Countess met with representatives of and visited projects supported by VISION 2020 Partners, including Sight Savers International (SSI), Christian Blind Mission (CBM), International Eye Foundation, International Trachoma Initiative (ITI), Helen Keller International (HKI), International Centre for Eye Education (ICEE), the SEVA Foundation and the Kilimanjaro Centre for Community Ophthalmology (KCCO). VISION 2020: The Right to Sight is a global initiative of the International Agency for the Prevention of Blindness (IAPB) and the World Health Organization (WHO), with a coalition of international Non-Governmental organisations. VISION 2020 aims to eliminate unnecessary blindness and partial or total blindness in all age groups, particularly the millions of needlessly blind, The Right to Sight. For further information, please visit www.v2020.org.

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Solitary CD30+ anaplastic large cell lymphoma of the eyelid showing regression

CD30+ anaplastic large cell lymphoma (ALCL) belongs to the group of T cell non-Hodgkin’s lymphomas. The primary cutaneous variant of ALCL usually presents as a solitary, cutaneous, or subcutaneous reddish violet lesion, which can be superficially ulcerated. We present the case of a solitary CD30+ ALCL of the eyelid showing regression.

Case report
A 39 year old man presented with a 4 week history of a progressive painless ulcerating nodule on the right upper eyelid, unresponsive to oral fluocxacillin. He was systemically well and denied recent foreign travel or contact with animals.

A 17 mm diameter ulcer with rolled margins and serosanguinous exudate was evident over the right upper eyelid (fig 1). His margins and serosanguinous exudate was evident over the right upper eyelid (fig 1). His.

Histology of the biopsy taken from the lid ulcer margin showed epidermal necrosis associated with ulceration (fig 2). The ulcer base showed haemorrhagic granulation tissue infiltrated by a mixture of lymphocytes, plasma cells, neutrophils, and eosinophils.

There were also ill defined groups of large blast cells showing enlarged and pleomorphic nuclei and high mitotic activity. The immunohistochemical staining showed these cells to be of T cell lymphoid lineage. Many of the large blast cells were CD30 positive but negative for ALK-1 protein. The features were of a CD30 positive anaplastic large cell lymphoma (ALK negative).

Treatment options such as surgery and radiotherapy were discussed with the patient but as the lesion remained stable over a 10 day period, a conservative approach was agreed. A moderately potent topical corticosteroid (mometasone furoate 0.1% cream) was applied to the lesion once daily. When followed up 8 weeks later the ulcer had completely healed without scarring (fig 3).

Eighteen months has elapsed since presentation. There has been no recurrence of his disease, and he remains in good health.

Comment
ALCL represents a group of large cell lymphomas. They consist of a proliferation of predominantly large lymphoid cells with strong expression of the cytokine receptor CD30 (≥75%). Using molecular and clinical criteria, three entities have been identified: primary systemic anaplastic lymphoma kinase (ALK)+ ALCL, primary systemic ALK– ALCL, and primary cutaneous ALCL.

Primary cutaneous ALCL arise de novo in the skin, commonly on the head and neck of older patients with a median age of 60 years and a male/female ratio of 3:2. Most patients present with solitary, asymptomatic nodules, which can be superficially ulcerated. Primary cutaneous ALCL has a more favourable prognosis than systemic ALCL, with an 5 year survival of approximately 90%. Partial or complete spontaneous regression can be observed in up to 25% of patients with primary cutaneous ALCL, accounting for the previous designation of “regressing atypical histiocytosis.” Treatment of localised lesions usually includes excision with or without radiation. However, patients with disseminated skin disease may benefit from systemic polychemotherapy.1 In our patient the lesion had resolved within 3 months of initial appearance. The application of a moderate potent topical steroid might have contributed to the regression of the ulcer.

We present a case of a primary cutaneous ALCL of the eyelid showing regression. Ophthalmologists should be aware of this sometimes self regressing entity and an expectant policy might be indicated in non-progressing tumours, thus avoiding potentially mutilating surgery or radiotherapy.

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doi: 10.1136/bjo.2004.042804
Accepted for publication 14 July 2004

References
Choroidal translocation with a pedicle following excision of a type 1 choroidal neovascular membrane

Excision of type 1 choroidal neovascular membranes (CNVM) in age related macular degeneration (AMD) have a poor visual outcome because of loss of retinal pigment epithelium (RPE). Simple replacement of the RPE may not work because the relation with Bruch's membrane and choroidal complex is disturbed. Creating a free graft of these three layers detaches the choroid from its blood supply. Hence, restoration of these three layers to the subfoveal position while maintaining a connection to the adjacent choroidal blood supply is desirable.

Case report

A 74 year old woman presented with a 3 month history of a left central scotoma and visual acuity (VA) of counting fingers (CF). Clinical examination and fluorescein fundus angiography (FFA) confirmed a type 1 subfoveal CNVM. The fellow eye was 20/30 with scattered soft drusen. Pars plana vitrectomy (PPV) and excision of the CNVM were performed as described previously. Atrophic choroidal vessels underlying the CNVM were not removed. A retinotomy was formed temporal to the fovea and vertical scissors inserted into the subretinal space. The RPE, Bruch's membrane, and choroid were incised en bloc in the area temporal to the site of the CNVM to create a graft on a pedicle. The graft was manipulated to a subfoveal position. The pedicle and graft were equally sized to maximally exploit the rich choroidal vasculature and maintain continuity to the choroidal circulation. We were unable to predetermine the position of choroidal vessels as indocyanine green angiography (ICG) was unavailable to us at the time of surgery. Surprisingly, little bleeding occurred and was easily controlled by increasing the infusion height. The patient required two subsequent operations for a rhegmatogenous retinal detachment with grade B proliferative vitreoretinopathy. The retina was flattened after inferior retinectomy and silicone oil insertion.

At review 4 years following initial surgery her vision was CF with a central scotoma on Goldman field testing. The area of translocated RPE, Bruch's membrane, and choroid was visible beneath the fovea with bare sclera demarcating its original site (fig 1A). At 4 years following surgery there was no recurrence of the CNVM on FFA (fig 1B) and ICG angiography demonstrated that the graft and pedicle were vascularised (fig 2).

Comment

Excision of type 1 CNVMs has a poor prognosis because of loss of RPE and atrophy of the choroid. Restoration of the normal anatomical relation between the retinal receptors and the underlying structures is essential for visual recovery. Retinal translocation with strabismic surgery for the movement of the retina to healthy RPE is prolonged and hazardous. Transplantation of homologous RPE cells alone to a subfoveal position has met with varied success. Aylward et al reported no visual improvement after transplantation of an autologous free graft, with fibrosis of the grafts at 10 months, perhaps because of loss of blood supply. Late revascularisation of some grafts has been reported at 1 year.

There was no visual improvement in our patient as she had a retinal detachment and additional procedures. We thought that the rich and redundant blood supply of the choroid allowed some freedom in the choice of graft harvest site. As proof of principle we have demonstrated that a choroidal/RPE graft with a pedicle is a feasible surgical technique, resulting in a sustained and vascularised graft. This technique is simpler than time consuming retinal translocation and does therefore merit further investigation.

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doi: 10.1136/bjo.2004.050716
Accepted for publication 4 August 2004

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Intravitreal triamcinolone acetonide and central serous chorioretinopathy

Intravitreal injections of triamcinolone acetonide have increasingly been performed as treatment for intraocular diseases with intraretinal oedema and with subfoveal fluid accumulation, such as diffuse diabetic macular oedema, persistent pseudophakic cystoid macular oedema, central retinal vein occlusion, and exudative age related macular degeneration. In view of the widening spectrum of indications for intravitreal triamcinolone acetonide injections, it was the purpose of this study to evaluate whether intravitreal triamcinolone acetonide injections may be useful as treatment of longstanding central serous chorioretinopathy.

Case report

A 50 year old patient presented with a decrease in visual acuity to 1/20 in his right eye because of longstanding central serous chorioretinopathy. Six years earlier, visual acuity had started to deteriorate, and had remained at 1/20 for the past 2 years. Fluorescein angiograms showed a mottled appearance of the retinal pigment epithelium close to the foveola, and a leakage of dye in the late phase of the angiogram. There was
no clear smoke stalk phenomenon (fig 1). In optical coherence tomography, the central retina was detached. Despite intensive topical treatment with prednisolone acetate eye drops and oral intake of carbamylhydrase inhibitors, the morphological appearance of the fovea and visual acuity remained unchanged. Under topical anaesthesia, the patient received an intravitreal application of 20–25 mg of triamcinolone acetonide, which was transconjunctivally injected through the pars plana into the centre of the vitreous cavity. The technique has already been described in detail. The patient was fully informed about the experimental character of the treatment and had signed an informed consent. After the injection, all topical and systemic medication for his macular disorder was stopped.

Within the first 5 months after the injection, fluorescein angiograms and optical coherent tomograms did not show any marked changes in the macula (fig 2). Correspondingly, visual acuity remained at 1/20. Intraocular pressure increased up to levels of 30 mm Hg and was reduced to the normal values by topical application of a carbonic anhydrase inhibitor. Thirteen months after the injection, the fovea was still slightly detached. Visual acuity remained at 1/20.

The clinical course suggests that in this eye with longstanding central serous chorioretinopathy an intravitreal injection of a high dosage of triamcinolone acetonide was not accompanied by a fast resolution of the subfoveal fluid and an increase in visual acuity. For more than 5 months after the injection, the fovea remained clearly detached. The partial resorption of the subfoveal fluid 13 months after the injection may not have necessarily been caused by intravitreal triamcinolone but may be explained by the natural course of the disease. The report agrees with other investigations in which patients with central serous chorioretinopathy did not markedly benefit from systemic steroid treatment. This single case report, therefore, does not favour the use of intravitreal triamcinolone acetonide for this treatment.

Vascular occlusion in serpiginous choroidopathy

Serpiginous choroidopathy (SC) is a rare disease inducing a permanent loss of vision, caused by a progressive destruction of the retinal pigment epithelium and choriocapillaris. Until now, no articular or systemic predisposing factors have been reported. SC usually affects both eyes and occurs in patients between the fourth and sixth decade, without any sex or race predilection. Clinically, deep cream-coloured lesions develop in the peripapillary region and then along the retinal vessels, centrifugally, inducing an atrophy of the retina. Other lesions may develop, isolated, in the posterior segment. The anterior segment is typically quiet; nevertheless, a mild anterior uveitis and/or vitritis have been observed. The course of the disease results in successive attacks and recurrences inducing permanent retinal atrophic changes and subsequently an irreversible loss of vision. Choroidal neovascularisation may occasionally develop. No specific diagnostic tests are available such that the diagnosis of SC is mostly clinical.

Case report

A 30 year old Indian man presented with a history of painless progressive visual loss affecting the right eye. No other ophthalmological or systemic complaints were present. His medical history was unremarkable.

Ophthalmological examination revealed a visual acuity of 20/50 in the right eye and 20/20 in the left eye without a correction in both eyes. Anterior segment examination revealed a mild inflammation with fine keratic precipitates on the inferior part of the right corneal endothelium. Intra-ocular pressure was 10 mm Hg in both eyes. Fundus examination of the right eye disclosed a moderate vitreous inflammation (cells: +) and multiple deep cream choroidal lesions around the optic disc and along the superior and inferior retinal (temporal and nasal) vessels (fig 1). Our differential diagnosis was a white dot syndrome (APMPE, SC), an infection (tuberculosis), or a sarcoidosis. We decided to hospitalise the patient.

A clinical examination revealed an erythrocyte sedimentation rate of 8 mm in the first hour (normal range 1–12), and a normal white blood count. Immunoglobulin electrophoresis, quantitative immunoglobulin levels, CD4/CD8 lymphocytes count, C3-C4 and CH50 examination were within the normal range. Tests for connective tissue disorders were negative and serum angiotensin converting enzyme was discretely elevated (74 U/L normal range = 18–55) with a normal lysozyme level. Infectious serologies (toxoplasmosis, Borrelia burgdorferi, Treponema pallidum, HIV, herpesvirus, Leptospira, Bartonella, rickettsiosis, brucellosis) were within the normal limits. An anterior chamber tap (polymerase chain reaction for herpes simplex virus (HSV) 1, HSV2, varicella zoster virus, cytomegatovirus, Epstein-Barr virus, toxoplasmosis, Mycobacterium tuberculosis) was negative. A lumbar puncture was normal (proteins 0.31 g/l, white cells 3x10^6/l, lymphocytes 74%), without oligoclonal bands on electrophoresis. PPD skin test was positive (15 mm) but chest x ray was normal. We have to consider that the patient has had a BCG vaccine in his childhood. The patient was HLA-B27 and A-29 negative but HLA B-7 positive. The initial clinical examination was completed by a neurological and a dermatological examination which were normal. A magnetic resonance image cerebral scan was normal.

The patient was given a course of methylprednisolone intravenously (4x250 mg/day for 5 days) followed by oral prednisolone (1 mg/kg) at tapering doses, and aciclovir (3x10 mg/kg), intravenously for 10 days. We covered the patient with rifampicin, isoniazide, ethambutol, pyrazinamide, and B6 vitamin. Topical steroids and mydriatics were administered.
increased frequency of HLA-B7 in this affection. The patient was treated with steroids, acciclovir, and anti-tuberculosis treatment.

A regression of the inflammation in the right eye was noted as well as a “cicatrization” of the choroidal lesions, which appeared as multiple geographic areas of atrophy of both the retina and pigmented epithelium between areas of normal retina. Our suspected diagnosis was a SC.

After 3 weeks, the patient developed the same lesions in the left eye with an occlusion of the superior temporal vein (fig 2). At that time the patient was on prednisone 40 mg/day and anti-TB treatment. A complete clinical examination was done again, but still all results were within the normal limits. The same treatment was introduced (methylprednisolone, intravenous acciclovir).

As the relapse occurred under steroid therapy (prednisone 40 mg/day), the administration of anti-TB treatment was continued too. No secondary effects were noted. We followed the patient during 12 months; his visual acuity returned to 20/20, without a correction, in both eyes. The anterior segment was normal. Posterior segment examination disclosed permanent geographic choriorretinal atrophic lesions along the vessels in both eyes, confirming the diagnosis of SC.

Comment

SC induces a progressive loss of the retinal pigment epithelium and choriocapillaris. The cause of this disorder is still under investigation but some studies suggest that an inflammatory or a vascular factor are involved in the pathogenesis. Histopathological studies have shown the presence of extensive lymphocyte choroidal infiltrates but in eyes chronically affected. The distribution of the lesions and their angiographic features (fluorescein and green indocyanine) may suggest a choroidal occlusion. Genetic studies demonstrated an increased frequency of HLA-B7 in this affection, our patient was positive. SC affects not only white people but also oriental and blacks people. There is no sex predilection and the patients are middle aged when the diagnosis is made. The patient described in this report was 30 year old which is uncharacteristic. The aetiology remains unknown although recently tubercular seriginous-like choroiditis has been reported.

Clinically, inflammatory signs may be noted both in segments (anterior uveitis, vitritis) along with the classic whitish choroidal lesions in the acute stage. The most frequent ocular complication of SC is sub-retinal neovascularisation which affects 13% to 20% of the eyes. Retinal vasculitis is also observed.

Gupta et al reported a case of SC with a branch vein occlusion in the acute phase. Our patient developed a vein occlusion while under treatment (steroids, acciclovir, and anti-TB therapy) which is rare and was never reported to our knowledge. Haemorrhages are sometimes observed in inflammatory diseases (Behcet’s syndrome, sarcoidosis) or in infectious posterior uveitis (syphilis, viral infections, toxoplasmosis).

This case demonstrates that SC can affect young patients and that HLA-B7 can be found in Indian patients with SC. The clinical features (vascular occlusion in this case) and the development of new lesions while under treatment let us suspect that the cause of this disease is still not clear.

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doi: 10.1136/bjo.2004.052126
Accepted for publication 27 August 2004

References


Persistent acanthamoeba keratitis in a non-contact lens wearer following exposure to bird seed dust

Acanthamoeba keratitis is a serious and vision threatening disease. It is commonly associated with contact lens wear (up to 93%). Early diagnosis and treatment are essential to improve the visual outcome. Devastating ocular damage can be attributed to various factors such as misdiagnosis, incorrect treatment, excessive topical steroid before diagnosis, and resistance.

Acanthamoeba keratitis in non-contact lens wearers is rare and poses a diagnostic challenge. We present a case of acanthamoeba keratitis in a non-contact lens wearer following accidental exposure to bird seed dust. The strain of acanthamoeba obtained from this patient appeared to show in vitro sensitivity to propamidine (MIC, 3.125 μg/ml) and chlorhexidine (MIC, 6.25 μg/ml) but unfortunately there was no retained debris at the time of presentation. Initially he was treated as a case of viral keratitis with topical acciclovir and steroid. Although there was an early improvement, the keratitis relapsed after 2 weeks. At that stage a typical ring infiltrate suggestive of acanthamoeba keratitis developed and epithelial culture grew Acanthamoeba polyphaga. He was started on intensive treatment with PHMB, Brolene, and neomycin. His symptoms improved and his visual acuity recovered to 6/9 over a period of 3 weeks. Topical steroids were then added. The antimicrobial treatment was given for 2–3 months and withdrawn gradually over next 4–6 weeks after complete resolution. But following complete cessation of all drops he developed a recurrence with positive cultures. We restarted the intensive treatment with PHMB and chlorhexidine but the clinical response was poor. Pain was severe with intense limbal inflammation and signs of scleritis.

A corneal biopsy was performed which showed persistence of infection. Resistance to PHMB (minimum inhibitory concentration (MIC), 3.125 μg/ml) and chlorhexidine (MIC, 6.25 μg/ml) was demonstrated in the culture obtained from the biopsy. The strain showed in vitro sensitivity to propamidine (MIC, 3.9 μg/ml). A change of treatment to topical propamidine isethionate 0.1% (Brolene) and neomycin led to a rapid response with a decrease in symptoms. Six months after initial diagnosis he is comfortable on maintenance treatment with propamidine isethionate 0.1% and neomycin, but unfortunately he has developed a dense central corneal scar (fig 1) and vision of hand movements.

Comment

Acanthamoeba keratitis not related to contact lens wear has been reported before and risk factors include trauma, dirty water splash, and exposure to leaf juice. Exposure to bird seed dust has to our knowledge not been reported previously as a known risk factor. Unfortunately, an attempt to culture acanthamoeba from the actual bird seeds and tray was unsuccessful. The second
uncommon feature in our case is the demonstration of in vitro resistance of this strain of acanthamoeba to two of the modern first line acanthamoeba drugs (PHMB and chlorhexidine) while showing a good sensitivity to propamidine.

This is contrary to what has been reported by other authors. We are unable to say whether resistance developed during treatment or was pre-existent, as sensitivity profiles of the earlier isolates were not obtained. This patient’s initial good clinical response was achieved with a combination of PHMB and propamidine with the latter tapered early during the course of treatment, indicating at least partial in vivo sensitivity to PHMB in the earlier stages. A poor association between in vivo and in vitro resistance has been described for biguanides, but this case shows that in vitro MIC can be useful information in the management of persistent acanthamoeba keratitis.

Other authors have stressed the need for long term treatment and this case also underscores the importance of prolonged effective antimicrobial treatment in order to prevent recurrences.

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doi: 10.1136/bjo.2004.047670
Accepted for publication 26 July 2004

References

Brinzolamide induced reversible corneal decompensation
Topical carbonic anhydrase inhibitors (CAIs) such as brinzolamide 1% (Azopt; Alcon Laboratories, Fort Worth, TX, USA) attenuate bicarbonate efflux, and this may lead to corneal oedema.

To our knowledge, this is the first report of complete resolution of corneal oedema after cessation of topical brinzolamide 1%.

Case reports
A 57 year old African-American man with primary open angle glaucoma (POAG) presented with painless blurry vision left eye 1 hour after instilling brinzolamide 1% in both eyes. He had been on brinzolamide 1% both eyes twice daily, brimonidine tartrate 0.2% (Alphagan) in both eyes twice daily, and latanoprost 0.005% (Xalatan) in both eyes once at night for 2 years. On presentation, best corrected visual acuity (BCVA) was 20/25 right eye and 20/50 left eye. The left eye had mild corneal oedema, Descemet’s folds, and whitish fleck-like debris on the corneal endothelium (fig 1A). Intraocular pressures (IOPs) were 15 mm Hg and 16 mm Hg. The brinzolamide 1% in both eyes was discontinued. Timolol maleate 0.5% (Timoptic) in both eyes twice daily and topical prednisolone acetate 1% (Pred Forte) left eye four times daily were started. By 1 week follow up, the cornea was clear (fig 1B). Specular microscopy revealed endothelial cell counts (ECC) of 1355 cells/mm² right eye and 648 cells/mm² left eye with enlarged pleomorphic endothelial cells left eye (fig 2). Central corneal thickness (CCT) was measured as 512 μm right eye and 505 μm left eye.

A 77 year old white man, who had had cataract extraction 46 years earlier and subsequent aphakia right eye, had been followed for open angle glaucoma in both eyes for 25 years. He was on timolol maleate 0.5% in both eyes twice daily, latanoprost 0.005% in both eyes once at night, and pilocarpine hydrochloride 4% gel (Pilopine Gel HS) in both eyes once at night. His visual acuities were hand movement right eye and counting fingers at 1 foot left eye. An IOP of 19 mm Hg right eye and 10 mm Hg left eye necessitated the addition of brinzolamide 1% twice daily right eye. Both corneas were clear at that time. Fifteen months after starting brinzolamide 1%, there was moderate corneal oedema right eye. Brinzolamide 1% was discontinued. Over 3 months, the corneal oedema in the right eye gradually resolved. The patient later needed trabeculectomy with mitomycin C right eye because of medically uncontrolled IOP.

Comment
The Merck Worldwide Adverse Experience System database for dorzolamide 2% includes so any corneal effect from a similar medication should indeed be potentially reversible. Dorzolamide’s peak concentration in rabbit corneas is also reached 1 hour after dosing, and the half life in the cornea is approximately 2 hours. This is consistent with case 1 having blurry vision 1 hour after instilling brinzolamide 1%.

Patients with primary open angle glaucoma or ocular hypertension and a baseline ECC of greater than 1500 cells/mm² have an average 3.6% decrease in ECC after a year of dorzolamide 2% three times daily. This endothelial loss is much higher than the 0.6% annual rate seen in normal subjects.

Topical CAIs can cause corneal oedema in compromised corneas—for example, those
with cornea guttata, but the oedema is reversible if identified early. Before initiating brinzolamide 1%, an ECC may be needed for high risk patients (that is, monocular, previous ocular surgery, corneal disease, etc.). Because of the potentially irreversible nature of the corneal decompensation, topical CAIs may be relatively contraindicated in patients with significant corneal disease.

Although dorzolamide and brinzolamide are both topical CAIs, their chemical formulas are different, and a side effect that is associated with dorzolamide may not necessarily be assumed to be associated with brinzolamide. It is important to be aware that brinzolamide can also potentially cause corneal oedema.

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doi: 10.1136/bjo.2004.049544
Accepted for publication 25 July 2004

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An unusual cause of acquired horizontal diplopia in a young adult
Cysticercosis is caused by infection from the larval form of Taenia solium, is endemic to regions with poor sanitation. Human cysticercosis more commonly affects the central nervous system, with less common involvement of ocular tissues. Myocysticercosis is a subset of orbital cysticercosis and is considered a rare entity.

Case report
A 19-year-old Nepalese housewife presented with left sided headache that had been present for 1 year. She had been treated for migraine headache in another hospital. Visual disturbance revealed orthoptia in primary gaze with Snellen acuity of 6/6 bilaterally. Both the Humphrey visual fields and the colour vision testing were normal in both eyes. There was no relative afferent pupillary defects detected. Ocular motility testing revealed left abduction deficit with the resulting horizontal diplopia. Hertel’s exophthalmometer reading revealed no proptosis. Her fundi examination were both normal and there were no optic disc swelling.

Magnetic resonance image (MRI) (brain/orbit) with gadolinium contrast (fig 1) showed a cystic enhancing mass measuring 1.2 cm × 0.6 cm adjacent to and including left medial rectus muscle, sparing the muscle tendon. Further assessment with B-scan ultrasonography (fig 2) revealed an intra-muscular cyst within the left medial rectus muscle located within mid-orbit.

Full blood count found no eosinophilia; systemic cysticercosis involvement was excluded by negative radiological findings (chest x-ray and computed tomography (CT) of brain and abdomen were all normal).

The patient was prescribed treatment with albendazole 15 mg/kg per day for 8 days. Unfortunately, she had intolerable side effects (nausea, vomiting, and distressing nocturnal left eye pain) to the medication which she used for only 3 days. She was reluctant to continue with albendazole. Her symptoms settled after a short course of oral analgesics.

She has remained asymptomatic. Repeated Hess and diplopia charts B-scan ultrasonography re-evaluation at 6 months did not reveal any cysts in the muscle and her ocular motility had returned to normal.

Comment
The finding of “scœla” within the intramuscular cyst and her status of Nepalese native lend strongly to a diagnosis of myocysticercosis. Enzyme linked immunosorbsent assay (ELISA) to detect the antibody to cysticercosis was unavailable in Singapore. A positive test may lend support to the diagnosis but a negative ELISA result does not rule out the diagnosis. Owing to the largely isolated and relatively mild infection of myocysticercosis, the sensitivity of ELISA is low. For the same reason, the absence of peripheral eosinophilia in this case is not surprising, consistent with the finding in literature.

None of the cases in a large series of orbital hydatid cysts were found within an extracocular muscle, hence making this diagnosis unlikely. The location within an extracocular muscle accounted for only 1.1% to 4.1% of the total reported cases of cysticercosis. Statistically, medial rectus is the most commonly involved extraocular muscle; although any of them can be involved. As a general rule, the restriction of extraocular movements is greatest in the direction opposite to the involved muscle, as in this case (fig 2).

Surgical excision of an extraocular muscle cyst had been described. However the potential risk of damage to adjacent tissue and adhesion from surgical exploration should not be taken lightly, particularly when effective medical therapy is available.

Stool tests should be done for all the members of the family to detect asymptomatic carrier because the treatment with systemic antihelmintic treatment is highly effective. It also serve to break the life cycle of the parasite.

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doi: 10.1136/bjo.2004.052258
Accepted for publication 1 July 2004

Figure 1 T1 axial MRI scan showing left intramuscular cyst with “scœla” within.

Figure 2 Left medial rectus intramuscular cystic lesion on B-scan (pretreatment).

References
Use of scanning laser ophthalmoscopy in visual conversion reaction

Visual conversion reaction is a psychosomatic anomaly that manifests as reduced visual acuity (VA) and visual field defects. Scanning laser ophthalmoscopy (SLO) can detect a scotoma and VA under direct fundus observation. However, there have been no reports of patients diagnosed with visual conversion reaction using SLO. We report a patient with visual conversion reaction using SLO.

Case report
A 20 year old woman presented with a sudden bilateral loss of vision. She reported being under severe stress at work. The best corrected visual acuity (BCVA) was counting fingers in both eyes. The external eye examination and pupillary responses were normal in both eyes. Conventional ophthalmoscopy, funduscopy, and fluorescein angiography were unremarkable. The visual fields were constricted to within 5° of fixation using Goldmann perimetry (fig 1A). The results of magnetic resonance imaging, computer tomography of the brain and orbits, visual evoked potentials, and electroretinography were unremarkable. A general medical examination showed no abnormalities. There were no scotomas (based on Goldmann size III stimulus on the retina), and the stability of fixation was central and stable using SLO microperimetry in both eyes (fig 1B). The VA using SLO was 20/200 in both eyes. We followed this patient for 10 months, and she consistently demonstrated impaired VA and visual field defects. She ultimately retired from the workforce.

Ten months later, the BCVA was 20/20 both eyes. The visual fields in both eyes using Goldmann perimetry were normal (fig 2A). There were no scotomas, and the stability of fixation was central and stable in both eyes using SLO microperimetry (fig 2B). The VA using SLO was 20/20 in both eyes. We diagnosed visual conversion reaction in this case.

Comment
This is the first report of a patient with visual conversion reaction using SLO. In this case, the BCVA was counting fingers in both eyes at the first visit. However, the VA using SLO was 20/200 both eyes and better than the conventional examination. The visual fields were constricted in both eyes to within 5° of fixation using Goldmann perimetry. However, there were no scotomas in either eye using SLO microperimetry. The distinction between the VA and visual fields between the conventional and SLO examinations was demonstrated over the 10 month follow up period. Ten months after the initial examination, the VA and visual fields were normal in both eyes by both conventional and SLO examinations. There was no distinction between them during the recovery period. Van de Velde reported that SLO results were comparable with those obtained during a conventional examination in normal subjects. The distinction between the VA and visual field between the conventional and SLO examinations may help in the diagnosis of patients with visual conversion reaction. Future clinical studies of several cases of visual conversion reaction using SLO are needed.

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doi: 10.1136/bjo.2004.052282
Accepted for publication 13 July 2004

References
The severe acute respiratory syndrome coronavirus in tears

We welcome the article by Loon et al.1 Earlier, we published our finding of the SARS coronavirus in specimens collected by the novel technique of conjunctiva upper respiratory tract irrigation (CURTI), but not in paired nose and throat swabs, very early in the course of the disease.2 In designing CURTI, we considered safety to medical personnel and, also, finding a method that samples all three portals of entry for upper respiratory viral pathogens—the eyes, nose, and mouth. Loon et al’s findings complement our study by showing that SARS coronavirus can indeed be found in tears.

While we agree with their conclusion that the ability to isolate the virus early in the course is important, we do not think that the eyes are important organs that propagate the virus, other than to ophthalmologists and to unwary close contacts. For instance, the eyes cannot generate infectious aerosol. Rather, we feel that the eyes are important portals of entry and have not been given sufficient attention—witness medical personnel in full personal protection gear and N95 masks but without watertight goggles, and sometimes without splashguards. We also think that employing the services of ophthalmologists for the purpose of collecting tear specimens for the diagnosis of SARS is difficult to achieve in most medical environments. On the other hand, our method of CURTI is entirely self help, deployable in quarantine locations, and avoids unnecessary contact between an infectious source and susceptible individuals.

The finding of SARS CoV in tears raises several additional questions:

(1) How does the virus end up in the tear? Was it the result of direct inoculation at the time of infection into permissive conjunctival epithelial cells, either by hand or aerosol, or was it the result of secretion from a lacrimal gland infected haematogenously? The lacrimal glands are not very different anatomically from the salivary glands. Yet saliva has been shown to be a poor specimen for the laboratory diagnosis of SARS.4

(2) Was there any evidence of conjunctivitis, lacrimitis, or evidence of infection of the globe or nasolacrimal sac?

(3) Is there any means or advantage in sampling the nasolacrimal sac, to which the tear drains, and could the nasolacrimal duct system be itself a hiding place for the SARS coronavirus during the incubation period?

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doi: 10.1136/bjo.2004.054130
Accepted for publication 14 July 2004

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Comments on using fibrin glue in pterygium surgery

I read with great interest the article by Karonyi and coworkers, who evaluated a new technique for pterygium surgery using a fibrin tissue adhesive (Tissuel Duo Quick). In their randomised trial the authors concluded that using the glue instead of sutures caused less postoperative pain and shortened the surgical time. Nevertheless, the timing of the randomisation is not clearly stated in their report.

Whether or not the surgeon knew the patient’s group (sutures or fibrin glue) at the end of surgery?

In their report the authors did not mention the risk of infection associated with the use of fibrin glue. However, fibrin glue is a heat-sensitive product, so it cannot be Tisseel. In the article by Hino et al, the specific trade name of the fibrin sealant is not given, but the text states that the sealant in question is treated by dry heat to inactivate viruses—Tisseel is vapour heated, so it cannot be Tisseel. In the article by Kawamura et al, the product is Beriplast (Aventis Behring). Nevertheless, we will pay attention to this matter in the future. We have not been able to find any report of HPV B19 infection in more than 700 procedures since 1999. Any clinical infection with HPV B19 has not yet been detected in our patients.

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doi: 10.1136/bjo.2004.056648
Accepted for publication 19 August 2004

Authors’ response

We thank Dr Alvarenga for his comment on our article about using fibrin glue for pterygium surgery.

We write in the “Material and methods” section that only the thickened and keratinised portion of the conjunctiva was excised and the graft was prepared to have the same size as the nasal conjunctiva. Regardless of which group the patient was randomised to, the knowledge of the surgery method did not influence the results or conclusions of the study. It is also impossible to randomise the patients without the surgeon’s knowledge, because the grafts were handled somewhat differently in the different methods described.

The fibrin adhesives in the references mentioned by Alvarenga are clearly not Tissuel Duo Quick (Baxter). In the article by Hino et al, the specific trade name of the fibrin sealant is not given, but the text states that the sealant in question is treated by dry heat to inactivate viruses—Tisseel is vapour heated, so it cannot be Tisseel. In the article by Kawamura et al, the product is Beriplast (Aventis Behring). Nevertheless, we will pay attention to this matter in the future. We have not been able to find any report of HPV B19 infection when fibrin glue was used in minor surgery. To date we have used the fibrin glue in more than 700 eye procedures since 1999. Any clinical infection with HPV B19 has not yet been detected in our patients.

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doi: 10.1136/bjo.2004.056648
Accepted for publication 19 August 2004

Korean kindergarten vision screen programme

Lim et al report a large scale effort to mix home plus health centre acuity screening in preschool children.1 We are very encouraged by the work of Lim et al, particularly concerning the frequency of ocular symptoms in the Korean preschooler, the number of children who were not dismissed from specified follow up (presumed amblyopia risk), and the inclusion of a simple, home administered test which over 97% of children were able to pass. It is of high merit that parents were carefully instructed to place tissue and tape over the non-tested eye,

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though this does not preclude seeking if the parent is not paying particular attention. Positive answers to the parental questionnaire were not very specific for eye disease and therefore could greatly increase societal cost if used as a screening method. We have a few points of clarification for these authors: How was the home acuity test initially validated? The number of children who passed their home exam have gold standard confirmatory exams from which false negative and true negative rates could be estimated? The positive predictive value estimates utilise gold standard exam criteria that need further definition and/or standardisation. (1) It is not clear whether amblyopia was diagnosed at multiple eye clinics and by general or paediatric ophthalmologists, it is not clear what criteria are used to define amblyopia, and the criteria to be included as a “significant” cycloplegic refractive error vastly overestimates risk factors compared to a recently published attempt to standardise reporting of vision screening research. We would urge the authors to perform additional calculations on the breakdown of gold standard exam “significant” refractive errors and better define how amblyopia was diagnosed.

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doi: 10.1136/bjo.2004.056663
Accepted for publication 19 August 2004

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IVF and retinoblastoma
I read with great interest the letter published in the BJ o by Lee et al.1 It reports on the first child born after in vitro fertilisation (IVF) and harbouring a unilateral retinoblastoma in the United States. However, it should be noted that this reported child is the eighth documented child (not the sixth as mentioned by the authors). The first child ever observed was reported by our group in 2001. He had a unilateral disease.2 In 2002, a second child with bilateral disease was documented in the Netherlands.3 In 2003, five additional cases were reported from the Netherlands (two with bilateral disease and three with unilateral disease). In this paper, an estimated relative risk of 4.9 to 7.2 for an IVF born child in the Netherlands to develop retinoblastoma was surmised.4

The issue of the possible association of assisted reproductive techniques (ART) with an increased risk of retinoblastoma has raised great concern worldwide. The interest of this association is highlighted by the fact that the expression of retinoblastoma in childhood is influenced by epigenetics—a regulatory mechanism not involving DNA sequence which could be affected by the various ART techniques.

In recent years, tens of thousands of children were born after ART. However, not one single case of retinoblastoma was observed until 2001. The possible reasons for this phenomenon were discussed.5 Awareness regarding the occurrence of retinoblastoma in ART born children sparked by our original observation of the first case in 2001 has probably been a trigger for the unveiling of additional cases. Therefore, more cases are to be expected in the near future.

Whether the increased number of observed cases indicates that ART born babies have a higher risk of developing retinoblastoma remains to be carefully investigated. None the less, a thorough prospective assessment of the possible association between ART and retinoblastoma is mandatory. Ongoing multicentre and multinational control studies will hopefully provide the needed answers to this “thorny” but most crucial aspect of ART. Till then, accurate accounting of previous observations is, of course, a key factor for a better insight into these issues.

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doi: 10.1136/bjo.2004.056739
Accepted for publication 20 August 2004

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Call for papers for the 5th annual meeting of the BOPSS to be held on 15 and 16 May 2005 at The Belfry, Birmingham. The abstract submission deadline is 4 February 2005, and abstracts can be submitted online at www.bopss.org.

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This will take place on 5-8 October 2005 in Vilamoura, Portugal. Further details: Christy Lacroix, EVER Secretary, Kapucijnenoever 33, B-3000 Leuven, Belgium (tel: +32 (0)16 233 849; fax: +32 (0)16 234 097; email: ever@skynet.be).

CORRECTIONS
In the paper titled Prospective case control study on genetic association of apolipoprotein ε2 with intraocular pressure (Br J Ophthalmol 2004;88:581-2), the authors have been listed incorrectly. The correct listing is A Junemann, S Bleich, U Reulbach, K Henkel, N Wakili, G Beck, B Rautenstrauss, C Mardin, G O H Naumann, A Reis, J Kornhuber. The journal apologises for this error.

In the letter titled Bilateral decompression retinopathy after orbital decompression surgery (Br J Ophthalmol 2004;88:1605-6), the authors have been listed incorrectly. The correct listing is G J Ben Simon, R A Goldberg, J D McCann. The journal apologises for this error.