Case of acute zonal occult outer retinopathy with altitudinal hemianopsia

Since the clinical entity of acute zonal occult outer retinopathy (AZOOR) was initially proposed, it has been noted that the visual loss may be misattributed to lesions in the optic nerve or central nervous system. Even with a likely visual field defect for those diseases—an afferent pupillary defect and reduced subjective central flicker fusion threshold—clinicians should be always aware of the possibility of AZOOR.

Case report
A 32 year old woman noticed a large scotoma in her right eye. She was examined by an ophthalmologist who found her corrected visual acuity to be 0.4 in the right eye and 1.0 in the left eye. She also had an afferent pupillary defect in the right eye. Goldmann perimetry showed a superior altitudinal hemianopic defect in the right eye (fig 1). Subjective central flicker fusion threshold was reduced in the right eye (18 Hz) and normal in the left eye (35 Hz). Ophthalmoscopic examination, fluorescein angiography, blood screening, and computed tomography were normal. Suspecting ischaemic optic neuropathy (ION), a 5 day course of intravenous sucirrin hydrocortisone, 100 mg/day, was used but this treatment was not effective. The patient was then referred to us for further examination. Full field rod and cone electroretinograms (ERGs) were reduced in her right eye (about 50% of those in the left eye). Multifocal ERGs (mfERGs) recorded with the VERIS Science 4.0 system (Electro-Diagnostic Imaging, San Mateo, CA, USA) revealed reduced responses in areas corresponding to the visual field defect (fig 2). These findings led us to presume the diagnosis to be AZOOR. While we have followed her for approximately a year, no retinal finding has been observed and the visual defect has not changed.

Figure 1. Goldmann visual field of the right eye showing superior altitudinal hemianopic depression including the blind spot. The field for the left eye was normal (not shown).

Figure 2. (Upper panel) Trace array of multifocal ERGs (mfERGs) of the right eye showing marked depression in the superior retinal areas. Waveforms that are close to ‘extinguished’ within the lesion are one of the characteristics of mfERGs in AZOOR. Recorded with: length of m-sequence, $2^{14}$; base rate, 75 Hz; flash intensity, 2.67 cd/m$^2$; band pass filter, 10-300 Hz; and artefact removal procedure, one iteration (0-80 ms on the first order kernel). (Lower panel) The 3D topographic plot translated from the trace array shown above showing functional distribution of the retina corresponding to the visual field defect.

Comment
Although altitudinal hemianopsia is found in many optic nerve disorders—for example, as meningioma, optic neuritis, ION, sinuses or intracranial artery disorders, and congenital abnormalities of the optic nerve head, this type of field defect had been rarely reported in AZOOR. The visual field abnormality in AZOOR varies from case to case, but central or paracentral scotoma is most common. When the clinical entity of AZOOR was initially proposed, the visual loss was attributed to retrobulbar neuritis, a pituitary adenoma, or other intracranial lesions during the early stages of this disease. Normal fundus and fluorescein angiographic findings with dense scotoma led ophthalmologists to suspect optic nerve or intracranial diseases. In addition, the afferent pupillary defect and disc swelling in AZOOR were very misleading. Our patient had a superior altitudinal hemianopic defect, an afferent pupillary defect, the reduced subjective central flicker fusion threshold and no disc swelling, and thus could be differentiated from ION or posterior ION (PION) without optic disc involvement. PIION is an unusual subset of ION and frequently related to arteritis. ERG observations may be critical for the differential diagnosis of cases with visual field defect without observable retinal lesions that are usually attributed to optic nerve or intracranial diseases. While the full field ERG may be sufficient to make the diagnosis, it may be critical for the differential diagnosis of cases with visual field defect which strongly reinforce the diagnosis of AZOOR.

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References

Congenital optic nerve head pit associated with reduced retinal nerve fibre thickness at the papillomacular bundle

Congenital pits of the optic nerve head result from an imperfect closure of the superior edge of the embryonic fissure. An unequal growth on both sides causes a delayed closure of the fissure at approximately 5 weeks of gestation. Optic pits appear as crater-like...
Histological sections of optic pits define defects in the lamina cribrosa associated with rudimentary retinal tissue, resembling pigmented tissue and aberrant nerve fibres. These anomalous papillomacular nerve fibre bundles may be less resistant, predisposing this sector to spontaneous schisis-like retinal detachments during later life.1

We present a young patient with a unilaterial optic pit and a clinically significant temporal nerve fibre loss. In vivo measurements by optical coherent tomography (OCT) determined the thickness of RNFL at the side of the pit and the corresponding papillomacular bundle.

Case report
A 27 year old white woman presented with a 9 month history of blurred vision; her best corrected visual acuity was 20/20 right eye and 20/25 left eye. On Goldmann perimetry in both eyes, there were no visual field defects, arcuate or paracentral scotomas. On slit lamp examination the anterior segment appeared normal in both eyes. Fundus biomicroscopy of the left eye revealed a large optic nerve head with a grey oval pit at the temporal margin and a brownish rim at the temporal side. The papillomacular bundle appeared to be darker, extending from the edge of the optic nerve to the macula, compared to the superior and inferior quadrant corresponding with severe RNFL loss according to the semiquantitative assessment of Niessen et al1 (Fig 1). Fundus examination of the right eye was unremarkable.

Linear OCT disclosed a significantly thickened RNFL in the superior quadrant (Fig 2A) and thinned RNFL at the temporal quadrant of the optic nerve in the left eye. There were no signs of a schisis-like retinal detachment (Fig 2B). Circular OCT demonstrated a significantly reduced thickness of the RNFL in all quadrants but predominantly in the temporal (90 μm) quadrant (Fig 2C).

Comment
The oval depression of the optic nerve head in optic pits may relate to an enlarged optic nerve head, an incomplete closure of the embryonic fissure and a reduced RNFL. The size of the optic disc was significantly larger when compared to the mean size (1.76 mm) in normal eyes, reducing the mean RNFL density.2 In addition, an incomplete closure of the embryonic fissure seems to prevent a proper fusion of the temporal optic nerve. OCT confirmed both a reduced mean RNFL and significant loss especially in the papillomacular bundle. Whereas normal subjects have a mean RNFL of 153 μm and 126 μm in the temporal quadrant,3 our patient with an optic pit had a mean RNFL of 115 μm and 90 μm in the temporal quadrant. Glaucomatous eyes with a mean RNFL below 103 μm frequently develop visual field defects,4 whereas in our patient with a mean RNFL of 115 μm none became apparent.

OCT precisely measured the retinal thickness with micrometer scale and provided additional evidence for pronounced reduced thickness of the RNFL in the temporal quadrant of the optic pit. Fundus photogra-phy confirmed the enlarged optic disc and temporal RNFL damage. The imperfect closure and lack of papillomacular nerve fibre bundles represent a “locus minoris resistenciae” in optic pits, the development of a spontaneous schisis-like detachment during ageing.

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References
Ocular ischaemic syndrome in thyroid eye disease, confirmed using magnetic resonance angiography

Ocular ischaemic syndrome (OIS) is most commonly caused by severe ipsilateral carotid artery stenosis. Occasionally it is caused by ophthalmic artery stenosis. Features commonly observed are iris neovascularisation, angle neovascularisation, rubecob ductal glaucoma, and iritis. In the posterior segment common signs are narrowing of the retinal arterioles, mid-peripheral retinal haemorrhages, optic disc pallor or neovascularisation and, rarely, retinal neovascularisation. Fluorescein angiography characteristically demonstrates delayed filling of the retinal circulation and occasionally patchy filling of the choroidal circulation is also observed. To the best of our knowledge OIS has not previously been described in thyroid eye disease. We report a case of OIS in thyroid eye disease confirmed by magnetic resonance angiography (MRA) and treated by orbital decompression.

Case report

A 48 year old woman with known thyroid eye disease presented with a 4 week history of pain, redness, and reduced visual acuity in her right eye. Eight years previously she had undergone bilateral three wall orbital decompression for severe corneal exposure. On examination visual acuity was counting fingers in the right eye and 6/9 in the left. There was bilateral lid retraction and mild generalised restriction of eye movements. There was bilateral proptosis measuring 24 mm in the right eye and 23 mm in the left (Keeler exophthalmometer). A right relative afferent pupillary defect was present. Gonioscopy showed an open, grade 2 (Fig 1), and moderate anterior chamber angle neovascularisation, rubeotic glaucoma, and iritis. In the posterior segment common signs were present in the angle. Fundal examination was limited by the corneal oedema but no specific abnormality was identified. Examination of the left eye was normal.

Fluorescein angiography showed delayed filling of the retinal vasculature in the right eye relative to the left. Computed tomography scans of the orbits showed previous bilateral three wall orbital decompression and diffuse enlargement of extraocular muscles. Carotid duplex ultrasound examination was normal. An MRA of the orbits demonstrated that blood flow in the right ophthalmic artery was reduced. Blood flow in the left ophthalmic artery was normal (Fig 2).

The patient was admitted and treated with intravenous mannitol and acetazolamide and topical apraclonidine 0.5% and betaxolol 0.5% but intraocular pressure remained elevated at 29 mm Hg. A further right orbital decompression was performed (where the lateral orbital wall was removed as far posteriorly as the anterior wall of the middle cranial fossa and superiorly to the floor of the anterior cranial fossa). Postoperatively the right prop-tosis measured 21 mm, the relative afferent pupillary defect resolved and the intraocular pressure was controlled (<20 mm Hg) with oral acetazolamide and topical apraclonidine 0.5% and betaxolol 0.5%. The corneal oedema resolved and the visual acuity gradually improved to 6/9. At the 3 month follow up postoperatively the rubecob ductal iris vessels had regressed. An MRA performed 4 months postoperatively demonstrated normal blood flow in both ophthalmic arteries (Fig 3).

Comment

Imaging methods available for evaluating the ophthalmic artery include duplex ultrasonography and cerebral angiography. Duplex ultrasonography is a non-invasive technique that gives quantitative information about flow; however, it requires an experienced operator and it is not always possible to positively identify the ophthalmic artery. Cerebral angiography is an invasive techni-que with the inherent risk of embolisation and stroke. MRA is a relatively new technique and has not previously been used to investigate disturbances of blood flow in the ophthalmic artery. It is non-invasive and does not require the level of technical experience required for Doppler studies. MRA detects blood flow at a defined velocity. In this case 25 mm/s was chosen as it has been shown in many studies using Doppler ultrasound to be the mean ophthalmic artery blood flow. The absence of signal from the right ophthalmic artery in the preoperative MRA demonstrates that at no stage during the cardiac cycle was blood flowing at this velocity in the artery. The images shown in Figures 2 and 3 are composites of all slices taken through the orbits. Hence it is not possible that one of the ophthalmic arteries could have been missed as a result of the orientation of any one particular slice.

Various abnormalities of the orbital circulation have been reported in thyroid ophthal-mopathy. Blood flow in the superior ophthalmic vein has been shown to be reduced, or even reversed in some patients. Increased central retinal artery, ophthalmic artery, and retinal blood flow have also been demonstrated. Ischaemia of the optic nerve head has been postulated to have a role in the development of optic neuropathy in some patients with thyroid ophthalmopathy. However, to the best of our knowledge ophthalmic artery obstruction as a result of thyroid eye disease has not previously been described.

In summary, this case demonstrates for the first time, the ocular ischaemic syndrome as a result of ophthalmic artery obstruction in thyroid eye disease. Furthermore, it demonstrates the usefulness of MR imaging in evaluation of the ophthalmic artery.

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References

Sirolone oil in diabetic vitrectomy

Castellarin and colleagues’ recount their recent experience of infusing silicone oil in a small series of patients with advanced diabetic eye disease, either during primary vitrectomy (12 eyes) or after earlier surgery had failed (11 eyes). They compare their vitrectomy (12 eyes) or after earlier surgery with previous reports and conclude had failed (11 eyes). They compare their vitrectomy (12 eyes) or after earlier surgery with previous reports and conclude.

The fibroglial epiretinal proliferation (that is, retinal attachment being well, the silicone oil could then be retrieved it by clicking on “eletters” submit a response. Providing it isn’t libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on “read eletters” on our homepage.

Silicone oil in diabetic vitrectomy


If you have a burning desire to respond to a paper published in BJO, why not make use of our eletters option? Go to our website (www.bjophthalmol.com), find the paper that interests you, click on “full text” and send your response by email by clicking on “eletters submit a response”. Providing it isn’t libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on “read eletters” on our homepage.

The editors will decide, as before, whether to also publish the eletter as a mailbox in a future paper issue.

References


Table 1 Surgical indications

<table>
<thead>
<tr>
<th>Indications</th>
<th>Eyes with ≤ 6 months' follow up</th>
<th>All operated eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRD+VH</td>
<td>7/17 (41.2%)</td>
<td>1/23 (43.5%)</td>
</tr>
<tr>
<td>TRD</td>
<td>3/17 (17.6%)</td>
<td>4/23 (17.4%)</td>
</tr>
<tr>
<td>TRD</td>
<td>2/17 (11.8%)</td>
<td>2/23 (8.7%)</td>
</tr>
<tr>
<td>TRD+FS</td>
<td>1/17 (5.9%)</td>
<td>2/23 (8.7%)</td>
</tr>
<tr>
<td>TRD+NVG</td>
<td>1/17 (5.9%)</td>
<td>1/23 (4.3%)</td>
</tr>
<tr>
<td>TRD+VH+FS</td>
<td>1/17 (5.9%)</td>
<td>1/23 (4.3%)</td>
</tr>
<tr>
<td>TRD+FS+NVG</td>
<td>1/17 (5.9%)</td>
<td>1/23 (4.3%)</td>
</tr>
</tbody>
</table>

*TRD = traction retinal detachment; VH = vitreous haemorrhage; TRD = traction rhegmatogenous retinal detachment; FS = fibrinoid syndrome; NVG = neovascular glaucoma.

Table 2 Anatomical results of pars plana vitrectomy (PPV) membrane peeling, and silicone oil infusion (SOI)

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Retinal reattachment rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eyes with ≥ 6 months' follow up</td>
<td>Overall</td>
</tr>
<tr>
<td>PPV+SOI as primary intervention</td>
<td>12/17 (71%)</td>
</tr>
<tr>
<td>PPV+SOI for failed previous vitrectomy All eyes</td>
<td>7/8 (88%)</td>
</tr>
<tr>
<td>Eyes with &lt; 6 months' follow up</td>
<td>Overall</td>
</tr>
<tr>
<td>PPV+SOI as primary intervention</td>
<td>5/9 (56%)</td>
</tr>
<tr>
<td>PPV+SOI for failed previous vitrectomy All eyes</td>
<td>9/12 (75%)</td>
</tr>
</tbody>
</table>

Table 3 Characteristics of eyes undergoing repeat pars plana vitrectomy

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total (n=17)</th>
<th>Failed first PPV (n=9)</th>
<th>Failed primary SOI (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative best corrected visual acuity</td>
<td>20/400</td>
<td>&lt;20/400</td>
<td>20/400</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>48 mm Hg</td>
<td>23 mm Hg</td>
<td>25 mm Hg</td>
</tr>
<tr>
<td>Presence of NVG</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Retinal detachment</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Successful PPV</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Successful SOI</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Retinal reattachment</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

The anatomical results of surgery among eyes with at least 6 months' follow up are quite similar to those of the entire cohort although a lower reattachment rate occurred among eyes undergoing surgery after having failed previous vitrectomy (Table 2). After a single operation with pars plana vitrectomy (PPV) with membrane peeling and SOI, the retina was fully attached in 12 (71%) of 17 eyes with at least 6 months' follow up. In the eight eyes that were operated for the first time with primary SOI, the retina was attached in seven (88%) eyes. In the nine eyes that underwent SOI after previously failed vitrectomy, the retina was attached in five (56%). The difference in the reattachment rate between these two groups was not statistically significant (Fisher's exact test, p = 0.18) as was the case in the cohort of 23 eyes.

Among eyes with at least 6 months' follow up, three of the five eyes with recurrent detachment underwent repeat PPV and SOI. The retina was reattached in two (40%) eyes. The third eye developed hyphema despite complete retinal attachment five months after SOI and underwent repeat surgery with additional SOI and epipapillary dissection but became phthisical. The remaining two patients declined additional surgery. Therefore, with multiple operations employing PPV and SOI, the final anatomical success rate at last follow up was 14/17 (82%), which is close to the final anatomical success rate (20/23 (87%)) reported previously.

With longer follow up, visual results were not as good as we reported initially (Table 3). Among eyes with at least 6 months' follow up (and as noted in our initial report), two (9%) had no light perception at last follow up (neither underwent scleral buckling). Both eyes had previous history of light perception. Loss of light perception was due to retinal and optic nerve ischaemia in each case. Preoperatively, the visual acuity of all patients ranged from light perception to less than counting fingers at 1 foot. Six months or more after surgery, three (18%) of 17 eyes had vision greater than or equal to 5/200. Among the entire cohort of 23 eyes, five (22%) had vision greater than or equal to 5/200.

Rubecous iridis was present preoperatively in seven (30%) of 23 eyes, three of which had neovascular glaucoma (NVG). The rate of NVG regression among eyes with at least 6 months' follow up was the same as in the entire cohort (Table 4). The rate of rubecous iridis regression was greater than we reported initially (that is, 3/7 (43%)) because of continued regression of rubecous during the longer period of follow up (Table 4). Of the three eyes with NVG, one eye underwent Baerveldt valve placement with normalisation of the postoperative intraocular pressure. Five months after surgery, however, the eye developed hyphema. Subsequently, the valve was removed, and the eye underwent additional SOI. As noted above, however, the eye became phthisical. As reported initially, the NVG regressed after surgery in the second eye, and the third eye had NVG regression but no light perception postoperatively because of ischaemia. As noted in our initial report, only one eye developed de novo rubecous iridis. Among the 17 eyes with at least 6 months' follow up, six (35%) had intraocular pressure <5 mm Hg. In our initial report, five (23%) of 23 eyes had intraocular pressure <5 mm Hg. Three of these six eyes had persistent retinal detachment, which we presume to be the cause of the hypotony. Among eyes with at least 6 months' follow up, the remaining 11 (65%) had intraocular pressure ranging from 6–48 mm Hg, with four patients taking anti-glaucoma medications.

Intraoperative complications were not different among eyes with at least 6 months' follow up versus the entire cohort of 23 eyes (Table 5). Postoperative complications differed in that there was an increased prevalence of cataract, hypotony, and silicone oil in the anterior chamber over time, which is not surprising.

McLeod suggests that in addition to using data from eyes with at least 6 months' follow up, one should use the status of the fellow eye to judge surgical success. At the time of surgery, all fellow eyes had proliferative diabetic retinopathy (in addition to other vision threatening conditions) and had undergone full panretinal photocoagulation (Table 6). Among patients with traction retinal detachment in the fellow eye, two underwent fellow eye surgery and one patient refused surgery (Table 6). Six (29%) of 21 fellow eyes were pseudophakic. Among the six fellow eyes with visual acuity <20/400, two had no light perception. Among the 21 patients we reported, the severity of disease in the fellow eye was such that two patients underwent PPV+SOI bilaterally, and results from both pairs of eyes were...
owing to the severe nature of the proliferative retinal detachments in all these cases, silicone oil was needed in all operated eyes. Diabetic retinopathy was the underlying condition in which we operated (Table 1). Ten eyes (43.5%) had recurrent vitreous haemorrhage, and silicone oil was used to maintain media clarity postoperatively.

We felt that silicone oil might foster recurrent retinal detachment and posterior retinal breaks and retinectomies. McLeod asks, ‘What is the appropriate use of silicone oil in the diabetic eye in the modern era?’ He suggests that in cases where breaks can be managed with gas tamponade and silicone oil can be reversed with retinal reattachment and laser photocoagulation, the use of silicone might be ‘gratuitous.’ McLeod suggests that appropriate uses of silicone might include patients with posturing difficulties or patients in whom there is a need for early visual rehabilitation.

We recognise that the conclusions of our study are limited because it is a non-randomised retrospective study without a control group. Thus, we cannot identify the ‘virtuous’ indications for the use of silicone oil in the setting of severe proliferative diabetic retinopathy based on these data. Without the use of silicone oil, for example, rubeosis iridis might have regressed, and the retina might have remained attached in eyes exhibiting the fibrinoid syndrome. None the less, the data from our study are consistent with the notion that silicone oil is an acceptable and useful tool in the management of eyes with severe complications of proliferative diabetic retinopathy. Our experience suggests, but does not prove, that silicone oil tamponade improves the prognosis in some otherwise unsalvageable cases. Among 11 (48%) of 23 eyes in this series, silicone oil was used initially at the time of repeat vitreous surgery. Short term retinal reattachment was achieved in eight (73%) of these eyes. Among these 11 eyes, nine had follow up of at least 6 months, and retinal reattachment was maintained in five (56%) of these nine with a single operation. Overall, 10 eyes failed initial PPV with or without SOI, underwent repeat PPV and SOI, and had at least 6 months’ follow up. Retinal reattachment without phthisis was achieved in seven (70%) of these eyes. Since the initial vitrectomies employed modern surgical techniques, these results indicate that even in the modern surgical era, use of silicone oil can improve anatomical (and functional) outcome in selected cases.

Table 3 Visual outcome

<table>
<thead>
<tr>
<th>Visual outcome</th>
<th>Eyes with &gt;6 months’ follow up</th>
<th>All eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved</td>
<td>4/17 (23.5%)</td>
<td>10/23 (43.5%)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>10/17 (58.9%)</td>
<td>3/23 (13.0%)</td>
</tr>
<tr>
<td>Worse</td>
<td>3/17 (17.6%)</td>
<td>10/23 (43.5%)</td>
</tr>
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</table>

Table 4 Regression of rubeosis iridis and neovascular glaucoma

<table>
<thead>
<tr>
<th></th>
<th>Eyes with &gt;6 months’ follow up</th>
<th>All eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rubeosis iridis regression</td>
<td>5/7 (71%)</td>
<td>5/7 (71%)</td>
</tr>
<tr>
<td>Neovascular glaucoma regression</td>
<td>2/3 (67%)</td>
<td>2/3 (67%)</td>
</tr>
</tbody>
</table>

Table 5 Prevalence of complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Eyes with &gt;6 months’ follow up</th>
<th>All eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraoperative retinal break</td>
<td>3/17 (18%)</td>
<td>4/23 (17%)</td>
</tr>
<tr>
<td>Silicone oil in anterior chamber</td>
<td>3/17 (18%)</td>
<td>3/23 (13%)</td>
</tr>
<tr>
<td>Cataract</td>
<td>3/17 (18%)</td>
<td>3/23 (13%)</td>
</tr>
<tr>
<td>Hypotony with attached retina</td>
<td>3/17 (18%)</td>
<td>3/23 (13%)</td>
</tr>
<tr>
<td>Fibrinoid reaction</td>
<td>1/17 (6%)</td>
<td>1/23 (4%)</td>
</tr>
<tr>
<td>New onset rubeosis iridis</td>
<td>1/17 (6%)</td>
<td>1/23 (4%)</td>
</tr>
<tr>
<td>Keratopathy</td>
<td>1/17 (6%)</td>
<td>2/23 (9%)</td>
</tr>
</tbody>
</table>

Table 6 Condition of fellow eye

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proliferative diabetic retinopathy</td>
<td>21/21 (100%)</td>
</tr>
<tr>
<td>Status post panretinal photoagulation</td>
<td>21/21 (100%)</td>
</tr>
<tr>
<td>Status post vitrectomy</td>
<td>10/21 (48%)</td>
</tr>
<tr>
<td>Clinically significant macular oedema</td>
<td>3/21 (14%)</td>
</tr>
<tr>
<td>Traction retinal detachment</td>
<td>3/21 (14%)</td>
</tr>
<tr>
<td>Rubeosis iridis</td>
<td>2/21 (10%)</td>
</tr>
<tr>
<td>Neovascular glaucoma</td>
<td>2/21 (10%)</td>
</tr>
<tr>
<td>Central retinal vein occlusion</td>
<td>1/21 (5%)</td>
</tr>
<tr>
<td>Macular pucker</td>
<td>1/21 (5%)</td>
</tr>
<tr>
<td>Primary open angle glaucoma</td>
<td>1/21 (5%)</td>
</tr>
<tr>
<td>Visual acuity</td>
<td></td>
</tr>
<tr>
<td>20/20–20/50</td>
<td>6/21 (29%)</td>
</tr>
<tr>
<td>20/60–20/100</td>
<td>7/21 (33%)</td>
</tr>
<tr>
<td>20/200–20/400</td>
<td>2/21 (10%)</td>
</tr>
<tr>
<td>Worse than 20/400</td>
<td>6/21 (29%)</td>
</tr>
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</table>

References


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Primary scleral buckle placement during repair of posterior segment open globe injuries

We read with interest the recent article by Arroyo and associates.1 They are to be commended on a very interesting study to compare the visual and anatomical outcomes of patients undergoing primary or secondary scleral buckle placement during posterior segment open globe repair with matched control patients who did not undergo primary scleral buckle placement.

Prophylactic scleral buckle of posterior segment open globe injuries has been a controversial topic in ophthalmology. The value of scleral buckling to support peripheral and especially inferotemporal breaks is rarely disputed. However, the utility of using an encircling buckle in the absence of retinal breaks remains controversial.

The benefits of primary scleral buckle placement are that it is technically easy and there is no scarring between the wound and overlying capsule and conjunctiva. However, there are some important considerations against primary scleral buckle such as the perforating injury subsequent rhegmatogenous retinal detachment (RD) is often not directly related to the site of the posterior exit wound but develops secondary to a new retinal break in the vitreous base region within 2 clock hours of the scleral wound.1 In addition, it is usually difficult to place a buckle over the exit wound and involves potential high morbidity (especially in the hands of an inexperienced doctor who usually receives the patient in the emergency room (at least in Venezuela)).

To counter subsequent traction at the vitreous base, a vitrectomy may be just as effective as a prophylactic scleral buckle, avoiding the associated morbidity.1 If retinal incarceration occurs through the wound, secondary reconstruction must almost always be performed anyway, typically involving a scleral buckle and vitrectomy 10–14 days after the injury (when inflammation is under control, and the intraocular anatomical status has been assessed adequately).1

We believe that the results of the study by Arroyo and associates contribute to the understanding of the role of prophylactic primary scleral buckle in the treatment of posterior segment open globe injuries. Their impressive results suggest that the benefits of placing a prophylactic primary scleral buckle may outweigh the risks involved. A multicentre randomised clinical trial is desirable to confirm their results.

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References

NOTICES

Guidelines

Helping the blind and visually impaired

The latest issue of Community Eye Health (No 45) discusses help for the blind, with an editorial by Sir John Wall of the Royal National Institute for the Blind on the rights of blind people. For further information please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email: Anita.Shahi@khtm.ac.uk; website: www.jceh.co.uk). Annual subscription (4 issues) UK£28/US$45. Free to developing country applicants.

Second Sight

Second Sight, a UK based charity whose aim is to eliminate the backlog of cataract blind in India by the year 2020 and to establish strong links between Indian and British ophthalmologists, is regularly sending volunteers to India. Details can be found on the charity’s website (www.secondsight.org.uk) or by contacting Dr Lucy Mathen (lucymathen@yahoo.com).

Specific Eye Conditions (SPECs)

SPECs is a not for profit organisation which acts as an umbrella organisation for support groups of any conditions or syndromes with an integral eye disorder. SPECs represents over 50 different organisations related to eye disorders ranging from conditions that are relatively common to very rare syndromes. The website acts as a portal providing access to support groups’ own websites. The SPECs website provides a valuable resource for professionals and may also be of interest to people with a visual impairment or who are blind. For further details about SPECs contact: Kay Kirkpatrick, SPECs Development Officer (tel: +44 (0)1180 532438; email: k@eyeconditions.org.uk; website: www.eyeconditions.org.uk).

The British Retinitis Pigmentosa Society

British Retinitis Pigmentosa Society (BRPS) was formed in 1975 to bring together people with retinitis pigmentosa and their families. The principle aims of the BRPS are to raise funds to support the programme of medical research into an eventual cure for this hereditary disease, and through the BRPS welfare service, help members and their families cope with the everyday concerns caused by retinitis pigmentosa. Part of the welfare service is the telephone help line (+44 (0)1280 860 363) for any queries relating to retinitis pigmentosa, especially for those recently diagnosed with retinitis pigmentosa (tel: +44 (0)1180 821 354; email: lynda@brps.demon.co.uk; website: www.brps.demon.co.uk).

Surgical Eye Expeditions International

Volunteer ophthalmologists in active surgical practice are needed to participate in short term, sight restoring eye surgery clinics around the world. Contact: Harry S Brown, Surgical Eye Expeditions International, 27 East De La Guerra, C-2, Santa Barbara, CA 93101-9858, USA (tel: +805 963 3303; fax: +805 965 3364; email: hsbrown.md@cox.net or see: http://www.specis.org/). For further information please contact: SPECS Development Officer (tel: +44 (0)1280 821 334; email: sppedinfo@ukonline.co.uk; website: www.specis.org/)

Rise in organ transplant numbers

According to UK Transplant, the UK has seen the highest number of organ transplants in six years. Last year (1 April 2002 to 31 March 2003) 2777 patients had their lives saved or dramatically improved through the generosity of 1064 donors. This equated to a 6% increase compared to the previous 12 months (1 April 2001 to 31 March 2002). Furthermore during 2002–3, the highest number of people benefited from a cornea transplant for five years (1997–98) and 240 more people had their sight restored than the previous year. For further information see
Elimination of avoidable blindness

The 56th World Health Assembly (WHA) considered the report on the elimination of avoidable blindness (doc A56/26) and urged Member States to: (1) Commit themselves to supporting the Global Initiative for the Elimination of Avoidable Blindness by setting up a national Vision 2020 plan by 2005; (2) Establish a national coordinating committee for Vision 2020, or a national blindness prevention committee to help implement the plan; (3) Implement the plan by 2007; (4) Include effective monitoring and evaluation of the plan with the aim of showing a reduction in the magnitude of avoidable blindness by 2010; (5) To support the mobilisation of resources for eliminating avoidable blindness. The WHA also urged the Director-General to maintain and strengthen WHO’s collaboration with Member States and the partners of the Global Initiative for the Elimination of Avoidable Blindness as well as aid in the coordination and support of national capability.

Ophthalmic Anesthesia Society (OAS)—17th Scientific Meeting

The 17th Scientific Meeting of the Ophthalmic Anesthesia Society (OAS) will be held 3-5 October 2003 at the Westin Michigan Avenue Chicago, Chicago, USA. Programme co-chairs: Marc Allen Feldman MD MHS and Steven T Charles MD. The CME joint sponsor is the Cleveland Clinic Foundation; CME hours are pending. Fees for OAS members are $300; non-members $475; students $50. Further details: OAS, 793-A Foothill Blvd, PMB 119, San Luis Obispo, CA 93405 USA (tel: +1 805 534 0300; fax: +1 805 534 9030; email: info@eyeanaesthesia.org; website: www.eyeanaesthesia.org).

Glaucoma Society 24th Annual Meeting and Dinner

The Glaucoma Society 24th Annual Meeting and Dinner will take place on 20 November 2003, from 8:30 am to 5:00 pm at The Royal College of Physicians, London, UK. Further details: Ms Janet Flowers (email: glausoc@upei.eire.freeserve.co.uk).

Detachment Course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding the Annual Meeting of Iranian Society of Ophthalmology

The detachment course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding Annual Meeting of Iranian Society of Ophthalmology will be held on 29-30 November 2003 and 1-4 December 2003 respectively, at the Razi Conference Center, Hemmat Hyw, Tehran, Iran. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Brenningerbau, 72076 Tuebingen, Germany (tel: +49 7071 295209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Masheyekhi, Dr Siamak Moradian, Dept of Ophthalmology, Labbafinejad Medical Center, Pasdaran Ave, Boostan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbafii@hotmail.com).

5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT)

The 5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT) will take place 11-14 March 2004, in Monte Carlo, Monaco. Please visit our website for details of the scientific programme, registration, and accommodation. To receive a copy of the Call for Abstracts and registration brochure please submit your full mailing details to http://www.kenes.com/isopt/interest.htm. Further details: ISOPT Secretariat (website: www.kenes.com/isopt).

XVth Meeting of the International Neuro-Ophthalmology Society


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: autoim04@kenes.com; website: www.kenes.com/autoim2004).
Tears and conjunctival scrapings for coronavirus in patients with SARS

Severe acute respiratory syndrome (SARS) was first recognised in Guangdong Province in China and later in Hong Kong in March 2003. Within a matter of weeks, the outbreak has evolved to become a global health threat and almost 30 countries have been afflicted with the novel coronavirus strain (SARS-CoV). SARS is a highly contagious potentially lethal disease. The main route of transmission is by respiratory droplets, although the virus has also been isolated in stool and in urine. Tears, being one of the body fluids, may potentially harbour the coronavirus. The presence of viruses in these body fluids may affect our precaution practices and sites of sampling for diagnostic tests.

Case series

A prospective interventional case series study was conducted on the identification of the SARS-CoV virus in tear secretions and conjunctival cells of patients with confirmed SARS. Approval was obtained from the ethics committee of the Chinese University of Hong Kong. Consecutive patients with probable SARS in the Prince of Wales Hospital, Hong Kong, during the epidemic period from April to May 2003 were recruited. Other than the routine samples of nasopharyngeal, mouthwash and stool, tear swab and conjunctival scraping were taken randomly from one eye of all recruited patients.

The tear swab was taken by putting a sterile cottonwool stick into the deep lower fornix of each patient’s eye after a single drop of topical anaesthetic agent (1% amethocaine eye drops) was applied. Conjunctival scraping was performed at the lower palpebral conjunctiva with a bent tip of a sterile 23 gauge needle. All ocular samples were collected by a single ophthalmologist with personal protective equipment recommended by the infection control unit of the hospital. Particular care was taken not to contaminate the samples.

The samples were analysed by virus culture and RT-PCR. The SARS-CoV specific primers CO R1 (sense) 5’ CAC GTT TTC TAC AGG TTA GCT AAC GA 3’ and CO R2 (antisense) 5’ AAA TGT TTA CGC AGG TAA GCG TAA AA 3’ were used to detect the presence of SARS-CoV RNA. All the patients were further categorised as confirmed SARS with a seroconversion or fourfold increase in antibody titre. The antibody against coronavirus was detected by indirect immunofluorescent technique based on Vero cells infected with SARS-CoV isolated from a patient with SARS.

A total of 20 probable SARS patients were recruited and 17 were later confirmed with paired convalescent sera. Among the confirmed cases, the mean age was 40.5 (SD 8.8) years and 12 (70.6%) female. They were recruited during the first (n = 6, 35.3%), second (n = 8, 47.1%), and third (n = 3, 17.6%) weeks of their diseases. Five (29.4%) of the 17 patients were positive for SARS-CoV by PCR with the samples from nasopharynx or stool (table 1). In all tear and conjunctival scraping samples, no SARS-CoV virus could be detected by RT-PCR or isolated by viral culture. Apart from two patients having mild and self limiting conjunctival bleeding after scrapings, no other ophthalmic complication was reported.

Comment

The routes of transmission of SARS other than respiratory droplets and stool are still enigmatic. In fact, tears have been reported to convey the virus and its genetic material were only present for a brief period of the disease, and the samples were not collected at the right time. Thirdly, the virus might not be present in tears at all.

The limitations of this study are the relatively small sample size and only the fact that one sample of tear swab and conjunctival scraping were taken from each patient. We cannot totally exclude the presence of virus in the tear secretion, but it is clear that conjunctival swabs and conjunctival scrapings are not useful samples for confirming or excluding the diagnosis.


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Conflict of interest: Nil.

References


Table 1 Samples for RT-PCR in probable and confirmed SARS

<table>
<thead>
<tr>
<th>Probable SARS (n = 20)</th>
<th>Positive convalescent antibody, confirmed case (n = 17)</th>
<th>Negative convalescent antibody, excluded cases (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of patient with positive RT-PCR (%)</td>
<td>No of patient with positive RT-PCR (%)</td>
<td></td>
</tr>
<tr>
<td>Nasopharyngeal aspirate and stool</td>
<td>5 (29.4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Tear swab</td>
<td>0 (0) *p = 0.0444</td>
<td>0 (0) *p = 0.0444</td>
</tr>
<tr>
<td>Conjunctival scraping</td>
<td>0 (0) *p = 0.0444</td>
<td>0 (0) *p = 0.0444</td>
</tr>
</tbody>
</table>

*Fisher’s exact test, two tail.
Paediatric oculomotor nerve palsies are rare lesions. The most frequently cited mechanism is perinatal injury to the peripheral third nerve, although they may be due to congenital absence of the nerve and/or nucleus and be accompanied by neurologic deficits. Septo-optic dysplasia consists of optic hypoplasia, mid-brain malformations, and hypothalamohypophyseal dysfunction. We present three children with congenital third nerve palsy and septo-optic dysplasia.

**Case reports**

We report on three children with bilateral optic nerve hypoplasia, with visual function from light projection to 0.05 and nystagmus. Magnetic resonance imaging revealed absent septum pellucidum, thinning of corpus callosum, and posterior pituitary ectopia in two cases and infundibular hypoplasia in the third case. They had anterior pituitary hormone deficiency—growth hormone, adrenocorticotropic hormone, and hypothyroidism in case one and two, with additional diabetes insipidus in the third case. There were no other associated brain or ocular anomalies. These children had no positive history of perinatal trauma, drugs, or toxic agents. There was no history of parental consanguinity.

Case 1: unilateral, left, pupil sparing third nerve palsy, with fixed exodeviation of 45 prism dioptres and hypotropia of 30 prism dioptres, no aberrant regeneration, no ptosis. Case 2: fixing with the paretic left eye (visual acuity 0.05 both eyes) demonstrated an elevation deficit of −3, adduction deficit of −2 on this side but good depression. There was no ptosis, anisocoria, or aberrant regeneration of the oculomotor nerve.

The third case was brought to our attention immediately after birth with bilateral third nerve palsy and pupil involvement on the left side (figs 1 and 2). Orthoptic examination revealed bilateral defective medial gaze and elevation and defective depression on the left side. In course of the first year of life pupillary reaction recovered without aberrant regeneration. At the age of 12 years the child died during febrile illness.

**Comment**

Our three children with septo-optic dysplasia had unilateral congenital third nerve palsy in two cases and bilateral palsy in one case.

![Figure 1 Bilateral third nerve palsy with pupillary involvement left side, age 3 months.](image1)

There was no ptosis and no involvement of the contralateral superior rectus muscle in the two patients with unilateral nerve disturbance, indicative perhaps of peripheral nerve defect. A possible explanation for the lack of aberrant regeneration may be due to extreme atrophy or even absence of the third nerve. The child with bilateral third nerve palsy (case 3) had postnatal pupillary involvement in one eye, with regeneration within 1 year. Peripheral nerve damage as well as nuclear defects may have been responsible.

Previous reports in the literature showed that several kinds of brain damage could result in congenital oculomotor palsies, such as brainstem infarction, cerebellar and midbrain hypoplasia, absence of basal ganglia, etc. Two theories have been proposed regarding the pathogenesis of septo-optic dysplasia. As all affected components arise from different tissues and processes at different times, developmental anomaly or dysplasia makes little embryological sense. Genetic causes are exceptional. A vascular disruptive sequence similar to porencephaly, possibly involving the proximal trunk of the anterior cerebral artery is discussed by Lubinsky. Our findings of congenital third nerve palsy in de Morsier syndrome do not support this hypothesis, as the third cranial nerve and its nuclei are not within the territorial distribution of the anterior cerebral artery and there were no additional defects in the median and paramedian areas of the frontal lobes. The partial palsy in case two, with some amount of adduction, elevation, and depression and the recovery of the pupillary involvement in case three implicate probably prenatal traumatic, infectious or toxic insults, thus supporting the theory of secondary degeneration.

**Figure 2 Optic hypoplasia.**

Penetrating eye injury from rear view mirrors

Penetrating eye injuries are a common complication of severe motor vehicle accidents. The majority of cases accompany facial laceration when the head of a front seat occupant passes forward, and then back, through the broken windshield. The frequency of such injuries has been greatly reduced since legislation was introduced to make the wearing of seatbelts for all occupants and the use of laminated windshield and glass compulsory. Glass entering the car through an open side window from a broken external rear view mirror is an uncommon but potentially preventable cause of severe ocular injury. First reported in 1990 by Keenan there have been two subsequent reports from Australia. We report two further cases and highlight deficiencies in legislation regarding the manufacture of external rear view mirrors.

**Case reports**

**Case 1**

A 17 year old man was driving a 1987 registered Fiat Uno when the driver’s external rear view mirror struck an oncoming van. The mirror broke and a fragment of glass passed through the open side window and hit his left eye. He sustained a corneal and scleral laceration but the lens and iris were not damaged. Following primary repair he made an uneventful postoperative recovery and retained a corrected visual acuity of 0.9 with spectacles.

**Case 2**

A 22 year old woman passenger of a 2002 registered Peugeot 206 was struck in the right eye when the side mirror struck the mirror of parked car. The mirror was broken and a fragment of glass entered her right eye through the open passenger window. She sustained a large corneal and scleral laceration with loss of the majority of the iris and damage to the lens. She underwent primary repair of the laceration with lens aspiration and anterior vitrectomy. She subsequently required a second vitrectomy with gas injection for vitreous gel incarceration and a retinal tear. She regained a visual acuity of 0.24 with a contact lens correction, but further improvement was prevented by corneal scar.

**References**


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Comment
Broken rear view mirrors have previously been identified as a cause of severe ocular injuries.1,2 The two cases we describe occurred in similar circumstances and both patients sustained penetrating eye injuries caused by glass from the broken external rear view mirror entering the car through an open window. The external rear view mirrors project beyond the body of the car and in both cases the mirror was broken following a collision with another vehicle in which the wing mirrors were clipped. No other vehicle damage occurred.

All exterior rear view mirrors fitted to cars in the United Kingdom have to comply with the European Community Directive 71/127/EEC which was last amended in 1988 (Directive 88/321).3 This requires mirrors designed for use in cars to undergo a "pendulum test" using a 7 kg weight on a pendulum of 1 metre length released at a 60 degree angle from the vertical to represent the impact of a head hitting the mirror. The test is performed with the weight hitting both the reflecting surface side and repeated on the opposite surface. If, at the end of this test, the glass of the mirror breaks, any fragments should adhere to the back of the protective housing. However, the directive further specifies that:

1. Partial unsticking of the glass is permitted provided that this does not exceed 2.4 mm either side of the cracks. It is acceptable for small shards to detach themselves from the surface of the glass at the point of impact.
2. The reflecting surface shall be made of safety glass.

It is therefore permissible for small splinters of glass to become detached from the surface of the glass at the point of impact, which is the case for any automobile safety glass. However, in both our cases the fragments appear to have been released from the mirror by spalling following a relatively high velocity impact on the reverse of the mirror housing. The broken glass fragments had sufficient momentum to pass through the open window of the car and penetrate the eye. The test described above simulates an object striking the mirror at a velocity of 3.1 m s⁻¹, compared to a velocity of 13.4 m s⁻¹ when the mirror of a car travelling at 30 miles per hour hits a stationary object. This velocity would be considerably higher if the car mirror clipped an oncoming car.

These cases highlight that there is a continuing risk of severe eye injury following an impact on the back surface of an external rear view mirror. Current legislation is based on a test that does not replicate the most probable circumstance of impact or the associated risk to the eye. The test is based on the effect of a low velocity impact, as opposed to the high velocity impact that would result from two cars passing in opposing directions. Replacing the glass with plastic would be an option to reduce the risk, although current plastic surfaces do not have the resistance to abrasion of glass which could result in scratches causing distracting light scatter. Avoiding driving with the side windows down, or repositioning the external mirrors are further options.

References

Coats’ disease in a vegetarian female

Coats’ disease is an idiopathic condition characterised by telangiectatic and aneurysmal retinal vessels changes with intraretinal and subretinal exudates.1,2 Here we report one case of a young female with an unusual manifestation of Coats’ disease.

Case report
A 14 year old female was referred for loss of vision in the left eye of a few weeks’ duration. The referring ophthalmologist thought the patient had a chorioretinal inflammatory process with secondary exudative retinal detachment and had recommended a complete medical examination for choriorioretinitis.

Visual acuity was 20/20 right eye and 20/400 left eye. Anterior segment examination was normal in both eyes. There were no cells or flare in the anterior chamber and in the anterior vitreous in either eye. Posterior fundus examination of the right eye was normal. In the left eye there was an exudative, neurosensory macular detachment and intraretinal lipid exudation temporally (fig 1A), leading to an area of microvascular changes in the temporal periphery. A serous, dependent neurosensory detachment with intraretinal lipid exudation was present as well. Fluorescein angiography study of the left eye confirmed the presence of marked microvascular changes in the peripheral retina (fig 1B). There was capillary non-perfusion, dilation and beading of the larger vessels, aneurysmal and telangiectasia formation. There were also telangiectatic vascular changes and intraretinal leakage of fluorescein dye throughout the peripheral fundus. The patient was diagnosed with Coats’ disease, even if the clinical presentation was somewhat unusual for Coats’ disease, given the absence of massive intraretinal and subretinal lipid exudation. A complete medical examination inclusive of PPD, chest x ray, lime titre, Bartonella titre, FTA-ABS, VDRL, ACE level, CBC with differential, was negative. The patient had been an absolute vegetarian from birth; her cholesterol level was 116, and her triglyceride level was 83.

Scatter laser photoacoagulation was applied around the area of telangiectatic retinal changes and in the temporal retina. Six months later the vision was improved to 20/100. There was partial resolution of the exudative, neurosensory retinal detachment and more lipid deposition in the macula. There was also new occurrence of preretal neovascularisation and vitreous haemorrhage (fig 2A, B). Further scatter photoacoagulation was applied to the temporal retina. One year after the original presentation the vision in the left eye had improved to 20/40. There was complete resolution of the exudative neurosensory detachment both in the macula and in the inferior periphery. The lipid exudation in the macula had mostly reabsorbed. There was also total regression of the preretal neovascularisation in the temporal periphery (fig 2C, D). There was also complete resolution of the microvascular telangiectatic changes and intraretinal leakage both in the temporal retina, which had been treated with scattered laser photoacoagulation and in the nasal retina, which had received no treatment.

Comment
This is an unusual manifestation of Coats’ disease because of the limited intraretinal lipid exudation.1 A possibility is that the reduced lipid exudation was related to the fact that the patient was totally vegetarian with very low cholesterol and triglyceride levels.

Figure 1 (A) Composite clinical picture of the left eye demonstrated the presence of serous, neurosensory macular detachment, and mild lipid exudation. A well localised area of microvascular retinal changes was present in the temporal periphery. There was also a dependent, exudative, neurosensory detachment and mild lipid exudation throughout the fundus. (B) Composite fluorescein angiography photographs of the same eye confirmed the presence of vascular changes consistent with Coats’ disease. There was capillary non-perfusion, microaneurysm and telangiectasia formation, beading and irregularity of the larger vessel walls. Intraretinal vascular leakage was present throughout the fundus.

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www.bjophthalmol.com
levels. The development of preretinal neovascularisation in the temporal periphery was also unusual. We thought that it was secondary to retinal ischaemia and not to a break in Bruch’s membrane after laser photocoagulation. We also found interesting the fact that the scatter photocoagulation of the temporal periphery resulted in regression of the microvascular telangiectatic changes both in the area that had received laser treatment and in the nasal periphery that had received no treatment. As expected, scatter laser photocoagulation resulted also in reduction of the lipid exudates, similar to what has been described in diabetic retinopathy.

Case report

A 10 year old African-American girl developed sudden painless loss of vision in both eyes over 2 days. The child was an inpatient admitted for examination of a multisystem disorder, affecting her pulmonary, gastrointestinal, muscular, and renal systems. On examination the best corrected vision was hand movement in both eyes. Confrontation visual fields were full bilaterally. Pupils were normal, without an afferent papillary defect. Anterior segment examination was completely normal. Posterior segment examination revealed massive retinal oedema in the papillomacular bundle extending into the peripapillary region bilaterally (fig 1). There was pronounced macular oedema and subging in the venous system. There were extensive scattered intraretinal haemorrhages bilaterally.

Fluorescein angiogram demonstrated bilateral blocking defect caused by retinal opacification and haemorrhages and non-perfusion in the macular region (fig 2). There was also pruning and non-perfusion of both small retinal capillaries and choroidal vessels. The retinal and arterioles venules demonstrated staining and leakage. Both optic discs stained with fluorescein.

The patient had patchy pneumonitis and asthma, hypertension, acute renal failure, elevated liver enzymes, and eosinophilia of 40%. Renal biopsy showed non-specific glomerular inflammation, but no eosinophilic infiltration or granulomata. A magnetic resonance image of the liver showed no involvement is rare. We describe a case of CSS in a child that resulted in bilateral optic neuropathy with vasculitis and multiple branch retinal artery occlusions affecting the macula.

References


Churg-Strauss syndrome in a child: retina and optic nerve findings

Allergic granulomatosis and angitis, also known as Churg-Strauss syndrome (CSS) is predominantly a disease of adults. Ocular involvement is rare. We describe a case of CSS in a child that resulted in bilateral optic neuropathy with vasculitis and multiple branch retinal artery occlusions affecting the macula.

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References

parenchymal damage. An infectious examination was negative for tuberculosis, Lyme, cat scratch disease, syphilis, HIV, cytomegalovirus, Epstein-Barr virus, toxoplasmosis, or toxocariasis. An oncological examination was negative. ANA, anti-dsDNA, ANCA, complement levels C3, C4, and CH50 were all normal. The diagnosis of Churg-Strauss syndrome was made based on three of six criteria advanced by the American College of Rheumatology, which include asthma, hyperesinophilia of >10%, non-fixed pulmonary infiltrates.

The patient was started on high dose intravenous steroids (2 mg/kg/day) and cyclophosphamide. Intravitreal triamcinolone acetamide injection was performed in the right eye with improvement of macular oedema. However, the vision remained no better than 20/400 in both eyes at 1 month.

Comment

CSS is a systemic granulomatous necrotising vasculitis affecting small to medium-sized vessels. Takanashi et al classified ocular manifestations into two groups: pseudo-tumour-type and ischaemic vasculitis-type. Pure ischaemic vasculitis-type manifestations were associated with a sudden onset loss of vision, a quiet looking eye, lack of computed tomography (CT) abnormalities, and a positive ANCA. In contrast, pseudotumour-like manifestations involved a chronic onset, a red eye with improvement of macular oedema, however, the vision remained no better than 20/400 in both eyes at 1 month.

References


Metastatic oesophageal carcinoma presenting as a lacrimal gland tumour

A 64 year old man was referred to the Department of Ophthalmology (Southern General Hospital) with 4 month history of painless swelling of the left side of the left orbit. There was no diplopia. His general health appeared reasonably good. Examination showed fullness in the region of the lacrimal gland associated with ecchymosis (fig 1). Visual acuity was 20/20 in each eye. Direct coronal computerised tomography scan of the orbits (fig 2) showed the presence of an extensive ill defined mass lying in the anterior third of the orbit and displacing the globe medially and slightly downwards. The superior and lateral rectus muscles could not be seen separate from the mass anteriorly although they were defined posteriorly. There was no evidence of perineural spread. The radiologist concluded that the appearance is consistent with pericranial gland tumour but lymphoma and a secondary tumour should also be considered.

Biopsy of the orbital mass was initially considered to be a poorly differentiated adenocarcinoma of the lacrimal gland (reported by Professor W R Lee, Western Infirmary, Glasgow).

On further questioning (systemic review) the patient subsequently volunteered a history of dysphagia. In view of this history, barium meal examination was performed and a 4 cm shouldered stricture with associated mucosal irregularity was identified (fig 3).

An endoscopic biopsy of the oesophageal tumour revealed an infiltrating poorly differentiated squamous cell carcinoma with a histological pattern almost identical to that of the orbital tumour (fig 4, reported by Dr Richard Morton, Southern General Hospital).

In light of this development, the tumour obtained from the orbital biopsy was reviewed and the diagnosis of secondary deposits of a poorly differentiated metastatic squamous cell carcinoma of the oesophagus was made (fig 5, reported by Professor W R Lee, Western Infirmary, Glasgow).

The oesophageal stenosis was dilated and a stent inserted. He was treated with palliative radiotherapy to the oesophagus and the left orbit, but he died 3 months later of generalised metastatic disease.

Comment

At the time of initial examination, there was very little to indicate that the patient had metastatic disease. The most likely diagnosis in this patient therefore was a primary malignant tumour of the lacrimal gland with...
orbital extension. He was admitted for investigations and treatment. After the findings of the CT scan, it was deemed unlikely that the tumour could be excised. Accordingly the appropriate management was to perform a biopsy initially. This was done 2 weeks following his initial presentation. After the pathological report of a poorly differentiated tumour was made and sent to the orbit, it was recognized that the diagnosis of a silent carcinoma of the middle orbit, 

Vitreoretinal abnormalities in the Conradi-Hunermann form of chondrodysplasia punctata

Autosomal dominant chondrodysplasia punctata or Conradi-Hunermann disease is a rare disorder with variable expressivity. It is characterized by dysplastic skeletal changes with premature punctate epiphysical and paravertebral calcification, associated with moderate growth deficiency, scoliosis, limb asymmetry, flexion contractures of the hips, knees and elbows, talipes equinovarus, short neck, frontal bossing, nasal bone hypoplasia with characteristic “koala” facies and dystrophic changes in hair and skin. Among the ophthalmic features reported, cataracts are by far the most common, occurring in about 20% of cases of Conradi-Hunermann disease compared with a much higher incidence (>66%) in the more severe (and usually lethal) autosomal recessive and X linked dominant forms the disease. In addition, optic atrophy, microphthalmos, iris hypoplasia, and Axenfeld-Rieger syndrome have been described.

Case report

We describe unusual vitreoretinal abnormalities in a 28 year old woman with Conradi-Hunermann disease. Her original diagnosis had been made in childhood based on clinical and radiological grounds, and she had undergone numerous cosmetic and reconstructive procedures on her nose, jaw, and lower limbs, as well as right cataract surgery at the age of 18 years for cortical lens opacity. Refraction showed low hyperopia and her left eye, also affected by cataract, had been considered amblyopic. She presented to us with a short history of photopsia and floaters in the left eye, but with no change to her corrected Snellen visual acuities of 6/12 right and 6/36 left.

Anterior segment examination revealed lower lid distichiasis and quiet pseudophakia on the right, and peripheral cortical lens opacities and a small pupil on the left. Her intraocular pressures were normal. Funduscopy showed healthy discs and maculae, with temporal dragging of vessels on the right (as had been noted many years previously). No posterior vitreous detachment was evident in either eye, but a number of unusual vitreoretinal tractional complexes were present, more marked on the left, with underlying retinal pigment epithelium disturbance (fig 1). The fundal view on the left was impaired by the cortical cataract and small pupil.

Strands of condensed vitreous emanated from each complex, pulling partial and full thickness operculae. The right eye showed flatter complexes, which appeared more stable, with less traction. In the left eye the precise nature of some of the tractional complexes was difficult to ascertain because of the cortical cataract and small pupil; it was uncertain whether they represented tractional schisis or full thickness combined tractional and rhegmatogenous lesions. A

References


Possible tractional schisis with inner leaf break and absent RPE changes

Dense vitreous condensations pulling tractional complexes

Localised tractional retinal detachments with underlying RPE changes and breaks in thinned retina

Figure 1 Vitreoretinal abnormalities in chondrodysplasia punctata. Retinal drawing with photographic insets of the left fundus, illustrating the nature and distribution of the vitreoretinal tractional complexes described. Vitreous condensations emanating from each complex can be seen, but the partial and full thickness operculae are difficult to appreciate in the photographs because of cortical cataract and small pupil. The right fundus is not shown here but had similar lesions, only less florid.

A group of three such lesions was located just temporal to the macula in the left eye. Given the unstable appearance and symptomatic nature of the left eye, the tractional lesions associated with retinal breaks were treated by argon laser retinopexy. One of the three temporal tractional complexes was un treatable with laser because of the cortical lens opacity.

Two weeks later the patient noticed a nasal visual field disturbance associated with photopsia in the left eye. She had developed a partial posterior vitreous detachment with a temporal rhegmatogenous retinal detachment transecting the macula, secondary to breaks associated with the temporal tractional complexes.

She subsequently underwent left phacoemulsification, intraocular lens implantation, pars plana vitrectomy, encirclement, endolaser, cryotherapy, and C3F8 gas injection. During the surgery the condensed vitreous was treated to be very adherent to the surgical eye. Thereafter the retinal breaks opened up within the complexes and also in a few areas of apparently “normal” retina. Postoperatively the retina was flat, but she developed raised intraocular pressure associated with a fibrous anterior uveitis and iris bombe—this settled with topical therapy and Nd:YAG laser peripheral iridotomy. Six weeks later an inferotemporal retinal detachment secondary to proliferative vitreoretinopathy occurred. This was treated successfully by membrane peel, endolaser, and gas tamponade. Her retina has remained flat since, with a Snellen visual acuity of 6/36 in this left amblyopic eye.

Comment
We describe previously unreported vitreoretinal abnormalities in a patient with Conradi-Hunermann disease. The pathogenesis of these lesions is not understood, but it may not be surprising that yet another skeletal dysplasia is associated with vitreoretinal changes. Identification of the underlying genetic defect in this particular variant of chondrodysplasia punctata may provide insight into the development of both bone and vitreous. We hope that better reporting of vitreoretinal features in Conradi-Hunermann syndrome will help improve understanding and management of this condition.

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References

“Imploding antrum” or silent sinus syndrome following nasotracheal intubation

Silent sinus syndrome is a condition in which chronic asymptomatic maxillary sinus disease presents with spontaneous unilateral enophthalmos and hypoglobus. Patients have inferior displacement of the orbit due to a downward bowing of the orbital floor and atelectasis of the maxillary antrum. Although the underlying maxillary sinus pathology is “silent” it has been suggested recently that “imploding antrum” more closely describes the acute nature of maxillary sinus collapse that occurs in the majority of cases. We present a typical case that appears to have been related to previous nasotracheal intubation.

Case report
A 27 year old woman was referred to ophthalmology outpatients complaining that her left eye was lower than the right and that when she looked down, she could see more of her left cheek than on the other side. She had started to notice this following an admission to hospital 6 months previously with viral meningitis. Since then, she had also experienced a “heavy blocked” feeling in the left side of her face together with some left nasal congestion. There was no history of acute sinusitis or chronic rhinosinuitis and, in particular, no history of trauma or surgery to the face—although she had had a nasotracheal intubation for a routine general anaesthetic 1 year earlier.

On examination, she had normal visual acuity and extraocular movements, but was found to have 2 mm of enophthalmos and hypoglobus. On subsequent review in the ENT clinic, nasendoscopy revealed a left deviated nasal septum with obstruction...
of the middle meatus, while the middle turbinate could not be visualised. Magnetic resonance imaging clearly showed depression of the left orbit with thinning of the orbital floor and a marked reduction in maxillary sinus volume (fig 1). The maxillary antrum was opacified and the middle turbinate severely lateralisated. The ethmoid, frontal, and sphenoid sinuses were clear.

She underwent septoplasty and functional endoscopic sinus surgery (FESS) during which a left middle meatal antrostomy was performed. Biopsies from the antrum demonstrated polypoid fibrous and mucoid material with no viable cellular tissue. Bacterial and fungal cultures were negative.

At follow up she had reduced hypoglobus of the left eye, an improvement in the left nasal airway and resolution of facial pressure. Orbital floor reconstruction was not required.

Comment

Silent sinus syndrome generally occurs in the third to fifth decade, shows equal sex distribution, and presents with anything from profound depression of the enophthalmos. Other symptoms can include transient vertical diplopia, upper lid retraction, lagophthalmos, malar depression, and facial nerve palsy. By definition, there is no history of acute or chronic sinusitis and no previous facial surgery or trauma. The amount of enophthalmos varies from 2–5 mm and visual acuity and extraocular movements are usually normal. Much speculation exists about the cause of silent sinus syndrome. The most popular theory of pathogenesis is that osteoelastic obstruction results in reduced aeration of the antrum, causing negative sinus pressure and atrophy. In the most recent review, Rose et al found that, as well as the orbital floor being drawn downwards, there was abnormal concavities in the medial and posterolateral walls of all the maxilla that could be assessed on CT scanning. They also describe an iatrogenic version of the disease occurring after orbital decompression. These relatively acute changes have led them to use the term “imploding antrum syndrome.”

It is possible in our case that the patient’s previous nasotracheal intubation caused sufficient local damage as to occlude the osteoelastic complex and create the conditions necessary for development of the syndrome. This supports the hypoventilation theory and also highlights the importance of careful history taking in establishing risk factors for this increasingly recognised condition.

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No association of p53 codon 72 and p21 codon 31 polymorphisms in Taiwan Chinese patients with pterygium

After abnormal expression of the p53 gene being found in epithelium, pterygium is now considered to be a result of uncontrolled cell proliferation, like a tumour. The p53 gene is a tumour suppressor gene, whose function is mediated by stimulation of p21 (Waf-1) gene, another tumour suppressor gene, to control cell cycle and prevent tumour formation. Mutations in either p53 or p21 are detected in many tumour cells, and polymorphisms of p53 codon 72 or p21 codon 31 were found to be associated with many tumours.

Because of the abnormal expression of the p53 gene in pterygium epithelium, there is evidence that hereditary factors may have a role in the development of pterygium. It is logical to suspect the correlation between pterygium formation and p53 and p21 polymorphisms.

In this study, p53 codon 72 and p21 codon 31 polymorphisms were evaluated in order to understand whether these two polymorphisms are associated with increased susceptibility for pterygium.

Patients and methods

A total of 128 pterygium patients (71 men and 57 women) were enrolled in the study with ages ranging from 35 to 90 years (mean 64.6 years). One hundred and three volunteers aged 55 years or more without pterygium were enrolled as the control group. There were 64 men and 39 women in the control group (age range from 50 to 83 years with an average of 64.2 years).

The genomic DNA was prepared from peripheral blood. For p53, the primer Pro 72 was designed for p53 codon 72 in proline form and Arg 72 for arginine form, according to the procedure described by Storey et al. For p21, the primer for codon 31 was designed from codon 1 start (5'-CTCTTTCTAACTCTCTGGGAG-3') to codon 91 (5'-CTCTT CCAACCTCATCCC GG-3'), according to the procedure described by Li et al. The PCR products from the same individual were mixed together and 10 µl of this solution were loaded into 3% agarose gel containing ethidium bromide for electrophoresis.

Results

There were no significant differences between both groups in age and sex. The frequency of the genotype of p53 codon 72 and p21 codon 31 polymorphisms in the pterygium group and control group is shown in table 1. There were no significant differences between both groups. The frequency of the alleles for the p53 codon 72 and p21 codon 31 between pterygium and control groups was not statistically different (table 2).

Analysis of combination p53 codon 72 and p21 codon 31 polymorphisms, there was also no significant difference between both groups (table 3).

Comment

Weinstein et al suggest the cause of p53 mutation in pterygium may be ultraviolet radiation or be hereditary. Detorakis et al proposed a “two hit” model for DNA abnormalities in pterygium. The first hit could be either inherited or incurred by ultraviolet radiation, and the second hit could be caused either by solar light or by viral infection. Though the hereditary factor was proposed to have a role in pterygium formation, there were few studies to clarify this proposition. In this study, we try to investigate the hereditary factor of pterygium by single nucleotide polymorphism (SNP) marker. Single nucleotide polymorphisms are the most abundant types of DNA sequence variation in the human genome, and the SNP marker has provided a new method for identification of complex gene associated diseases such as tumour.

The p53 codon 72 and p21 codon 31 polymorphisms are two of the most important SNP markers for tumour susceptibility identification. However, there are no significant differences between the pterygium and control group in our study. We suggest the p53 codon 72 and p21 codon 31 polymorphisms maybe cannot

### Table 1 Distribution of p53 codon 72 and p21 codon 31 polymorphisms in the pterygium and control group

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Pterygium (n=128)</th>
<th>Control (n=103)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>p53GG</td>
<td>84 (65.6)</td>
<td>53 (51.5)</td>
<td>0.50</td>
</tr>
<tr>
<td>p53GC</td>
<td>39 (30.5)</td>
<td>52 (49.5)</td>
<td>0.65</td>
</tr>
<tr>
<td>p53CC</td>
<td>5 (3.9)</td>
<td>16 (15.5)</td>
<td>0.01</td>
</tr>
<tr>
<td>p21AA</td>
<td>24 (18.8)</td>
<td>14 (13.6)</td>
<td>0.40</td>
</tr>
<tr>
<td>p21AC</td>
<td>76 (59.4)</td>
<td>68 (66.0)</td>
<td>0.40</td>
</tr>
<tr>
<td>p21CC</td>
<td>28 (21.9)</td>
<td>19 (18.4)</td>
<td>0.20</td>
</tr>
</tbody>
</table>

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Table 2 Allelic frequencies for p53 codon 72 and p21 codon 31 polymorphisms in the pterygium and control group

<table>
<thead>
<tr>
<th>Allele</th>
<th>Pterygium (n = 128) (%)</th>
<th>Control (n = 103) (%)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>p53</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allele G (arginine)</td>
<td>144 (56.3)</td>
<td>118 (57.3)</td>
<td>0.82</td>
</tr>
<tr>
<td>Allele C (proline)</td>
<td>112 (43.8)</td>
<td>88 (42.7)</td>
<td></td>
</tr>
<tr>
<td>p21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allele A (arginine)</td>
<td>124 (48.4)</td>
<td>96 (46.6%)</td>
<td>0.69</td>
</tr>
<tr>
<td>Allele C (serine)</td>
<td>132 (51.6)</td>
<td>110 (53.4%)</td>
<td></td>
</tr>
</tbody>
</table>

Table 3 Distribution of combination p53 codon 72 and p21 codon 31 polymorphisms in the pterygium and control group

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Pterygium (n = 128) (%)</th>
<th>Control (n = 103) (%)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>p53/p21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GG/AA</td>
<td>9 (7.0)</td>
<td>3 (2.9)</td>
<td>0.84</td>
</tr>
<tr>
<td>GG/AC</td>
<td>29 (22.7)</td>
<td>25 (24.3)</td>
<td></td>
</tr>
<tr>
<td>GG/CC</td>
<td>4 (3.1)</td>
<td>4 (3.9)</td>
<td></td>
</tr>
<tr>
<td>GC/AA</td>
<td>10 (7.8)</td>
<td>9 (8.7)</td>
<td></td>
</tr>
<tr>
<td>GC/AC</td>
<td>31 (24.2)</td>
<td>32 (31.1)</td>
<td></td>
</tr>
<tr>
<td>GC/CC</td>
<td>19 (14.8)</td>
<td>13 (12.6)</td>
<td></td>
</tr>
<tr>
<td>CC/AA</td>
<td>5 (3.9)</td>
<td>2 (1.9)</td>
<td></td>
</tr>
<tr>
<td>CC/AC</td>
<td>16 (12.5)</td>
<td>11 (10.7)</td>
<td></td>
</tr>
<tr>
<td>CC/CC</td>
<td>5 (3.9)</td>
<td>4 (3.9)</td>
<td></td>
</tr>
</tbody>
</table>

become useful genetic markers for pterygium susceptibility. This could be the basis of future surveys.


BOOK REVIEWS

The Ophthalmology Examinations Review


Preparing for postgraduate examinations is now a nearly universal event for ophthalmologists in training. In many countries it will become a part of a practising ophthalmologist’s professional life as well. Preparation books for these types of examination are increasingly being published. Dr Tien Yin Wong has written a book to prepare ophthalmologists for postgraduate examinations. Granted this book is addressed primarily to those who take the British style examinations but it could be easily used by trainees in countries where exams are slightly different. This book is organised in a clear fashion with specific topics being outlined in a bullet point fashion. Important information is highlighted in bold print and the table of contents and index are both well organised and detailed enough that information can be found quickly and almost flawlessly. The topics covered include all of the classic subspecialty areas of ophthalmology and some additional miscellaneous areas including lasers in ophthalmology and epidemiology, public health, and research methods. The author also attempts to prioritise examination material with a system of stars that are meant to rate the likelihood that the material under review is commonly used in testing circumstances. This prioritisation attempt is perhaps the least successful part of the book. Overall, however, Dr Wong includes an enormous amount of information in just over 400 pages, and has done so in an attractive way. The book is easy to read with outline forms and lots of highlighted information in box or table presentations. While there has been an obvious attempt to make this book concise and to the point this reader still finds it slightly unnerving that no references are included. However, this is a book that is meant to prepare one for tests and not to provide detailed reference information. It easily and successfully fulfils this role.

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How to Write a Paper. 3rd ed.


This multi-authored book is a must for both aspiring authors and those who think they can already write. The book is very easy to read, informative, and amusing. Why all should read it is because it informs of the processes involved in publication, the role of editors and assessors and, of course, publishers; thus, the book increases the understanding of what happens behind the scenes. Other chapters are equally important, presenting a common theme—how to captivate the reader. The book assists in helping authors write abstracts, introductions, best ways to present results, and how to generate informative discussion. In all chapters the approach emphasises brevity and how to get the audience interested from the very beginning. There is even a chapter on titles! Remember, as the book also says, most readers do not read the whole paper, if you are lucky they may read just the title. If you want your work to be read make it short and interesting from the very beginning. This book will help authors achieve their desired result.

A D Dick

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Pediatric ophthalmology and strabismus. 2nd ed

Pediatric ophthalmology is a large and developing subject and a new comprehensive, up to date text is much to be welcomed. In this revised edition of a major textbook, the editors and contributing authors have made great efforts to include many recent and important pieces of research. Despite this, there is a very practical flavour to most of the chapters; a nice touch is the inclusion of several practically useful tables (guidelines for planning strabismus surgery) and graphs of normal adnexal dimensions just within the covers.

Three new chapters been added since the first edition; “Visual electrophysiology in children,” “Strabismus surgery,” and “Ocular disorders with systemic manifestations.” The first is a masterly and highly readable summary of the usefulness of electrophysiology in children and is a fitting tribute to one of the authors, Dr Tony Kriss, who sadly died soon after its completion. The chapter on strabismus surgery by Kenneth Wright contains elegant descriptions, beautifully illustrated, of a range of surgical techniques used in strabismus surgery. Finally, there is a unique account by Dr Maya Eibschitz-Tsimhoni, of the ocular manifestations of inherited disease. She includes a glossary of terms used to describe dysmorphic features, an alphabetical thesaursus of syndromes with prominent ocular features and their many alternative names and a clear description of the general and ocular findings in each, together with the genetic locus where known, or inheritance pattern. Generally, the neuro-ophthalmological and systemic or developmental problems are dealt with particularly well, but all of the chapters are comprehensive and well illustrated with clinical pictures and clear diagrams where appropriate.

I have rarely enjoyed reading a textbook as much as this one. My only complaint is of its bulk and the fact that apparently there is no CD version. This textbook is certainly useful enough for one to take it to clinics regularly but it is difficult to do this if attending outreach clinics, especially if carrying a laptop computer, an indirect ophthalmoscope, and a retinoscope as well. I would therefore warmly recommend this book to all ophthalmologists who see children but I would also beseech the publishers to think about a more portable medium for this potentially extremely useful text.

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NOTICES

Low vision care
The latest issue of Community Eye Health (No 49) deals with the problems and management of low vision. For further information please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email: Anita.Shah@lshtm.ac.uk; online edition: www.jceh.co.uk). Annual subscription (4 issues) £22/US$45. Free to developing country applicants.

Elimination of avoidable blindness
The 56th World Health Assembly (WHA) considered the report on the elimination of avoidable blindness (doc A56/26) and urged Member States to: (1) Commit themselves to supporting the Global Initiative for the Elimination of Avoidable Blindness by setting up a national Vision 2020 plan by 2005; (2) Establish a national coordinating committee for Vision 2020, or a national blindness prevention committee to help implement the plan; (3) Implement the plan by 2007; (4) Include effective monitoring and evaluation of the plan with the aim of showing a reduction in the magnitude of avoidable blindness by 2010; (5) To support the mobilisation of resources for eliminating avoidable blindness. The WHA also urged the Director-General to maintain and strengthen WHO’s collaboration with Member States and the partners of the Global Initiative for the Elimination of Avoidable Blindness as well as aid in the coordination and support of national capability.

XVth Meeting of the International Neuro-Ophthalmology Society

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: autoim04@kenes.com; website: www.kenes.com/autoim04).

XVI International Congress for Eye Research

Optometry giving sight launches new website
Optometry Giving Sight (OGS) is delighted to announce the launch of its official website www.givingsight.org which will go live on Friday 21 May 2004. Further to VISION 2020’s recent announcement that HRH The Countess of Wessex will be showing her support for the ground-breaking campaign OGS at a luncheon she will be attending at the Mansion House on Monday 17 May, the launch of the website is another step forward in the progress of this exciting new project.

OGS is a unique collaboration of the International Agency for the Prevention of Blindness (IAPB), World Optometry Foundation (WOF) and the International Centre for Eyecare Education (ICEE). The purpose of the OGS project is to generate new income primarily from the Optometrists, Optical Dispensers, Optometric Practices, Opticians and optical outlets throughout the world, for the purposes of VISION 2020: The Right to Sight, including the delivery of eye and vision care; the development of human resources; and the improvement of infrastructure needed to improve eye care services around the world. Attached is the web launch press release, OGS poster and a photograph with caption. For further information, please contact: Isabel Ganier (tel: +44 7879 824 400; e-mail: iganier@v2020.org; website: www.givingsight.org) and for further information on ’VISION 2020: The Right to Sight’ please visit www.v2020.org.

CORRECTION
In the letter by Shortt et al (Br J Ophthal 2003;87:1302), the image presented in fig 3 was the incorrect figure. The correct figure is shown below. The journal apologises for the error.

Figure 3 Postoperative MRA. Normal blood flow is present in both ophthalmic arteries (arrows).