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, though the virus has also been isolated in body fluids, may potentially harbour the virus and its genetic material were only detected by RT-PCR or isolated by viral culture. Apart from two patients having mild pneumonia: a prospective study. Lancet 2003;361:1767–72.


Congenital third nerve palsy in septo-optic dysplasia

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 neurological deficits. Septo-optic dysplasia consists of optic hypoplasia, mid-brain malformations, and hypothalamosyphophysical function. 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 2 dioptres, no aberrant regeneration, no ptosis.

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 brain-stem 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 Moris 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.

Reference:

Comment

Broken rear view mirrors have previously been identified as a cause of severe ocular injuries.1 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).1 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 unstickling 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 released 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 ms⁻¹, compared to a velocity of 13.4 ms⁻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, opposed to the high velocity impact that on the effect of a low velocity impact, as associated risk to the eye. The test is based on a test that does not replicate the most rear view mirror. Current legislation is based on a test that does not replicate the most.
 levels. The development of preretal neovascularisation in the temporal periphery was also unusual. We thought that it was secondary to retinal ischaemia and not a break in Bruch’s membrane after laser photoablation. We also found interesting the fact that the scatter photoablation 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 photoablation resulted also in reduction of the lipid exudates, similar to what has been described in diabetic retinopathy.

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 opacities 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.

## Case report

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## References


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

Allergic granulomatosi and angiitis, also known as Churg-Strauss syndrome (CSS) is predominantly a disease of adults. 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.

## Figure 1

Colour fundus photographs of the right (A) and left (B) eyes, showing retinal opacification and intraretinal haemorrhages in the macular and peripapillary regions.
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,1 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 acetate 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.6 classified ocular manifestations into two groups: pseudo-tumour-type and ischaemic vasculitis-type.4,5 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.3 In contrast, pseudotumour-like manifestations involved a chronic onset, a red eye, orbital abnormalities on CT, and a negative ANCA.3 Takanashi et al advised that patients with positive ANCA CSS, who are visually asymptomatic, be carefully examined and treated prophylactically with high dose steroids in order to prevent potentially irreversible visual loss from the ischaemic-type ocular manifestations.4 The devastating loss of vision in this child with ANCA negative CSS suggests that all patients with CSS should be considered for prophylactic high dose corticosteroids, regardless of their ANCA status.

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Data accumulation is in conformity with all US federal and state laws and is in adherence to the tenets of the Declaration of Helsinki.

doi: 10.1136/bjo.2003.039859

Accepted for publication 8 December 2003

Financial support: None.

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

Figure 1 The appearance of the patient at the time of presentation.

Figure 2 Fluorescein angiogram (recirculation phase) showing extensive macular non-perfusion and blocking defect from retinal opacification and haemorrhage. There is vasculitis-type leakage from the posterior pole retinal vessels (both arteries and veins). Both optic discs stain with fluorescein.

Figure 3 The computed tomography scan shows an irregular tumour mass in the lacrimal fossa and in the superior part of the orbit. Note the erosion of the orbital bone.
The histological appearance of the squamous carcinoma within dense fibrous tissue in the orbital biopsy (magnification ×400, haematoxylin and eosin).

Figure 5: The histological appearance of the poorly differentiated squamous carcinoma within dense fibrous tissue in the orbital biopsy (magnification ×400, haematoxylin and eosin).

the presence of metastasis suspected, the symptoms of dysphagia led to further investigation and demonstration of an oesophageal primary carcinoma. (The oesophageal biopsy was done 10 days after the orbital biopsy).

Orbital extension. He was admitted for 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 the occurrence of the orbital tumour.

As far as we know, our case is the first report of a silent carcinoma of the middle third of the oesophagus presenting as a lacrimal gland neoplasm and the case reiterates the importance of careful history taking and thorough preoperative assessment.

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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 characterised by dysplastic skeletal changes with premature punctate epiphysial 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 ocular 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 of 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 photopia 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. Fundoscopy 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
a Snellen visual acuity of 6/36 in this left eye. Her retina has remained flat since, with no further retinal detachment occurring. This was treated successfully by pars plana vitrectomy, encirclement, endoanlaser emulsification, intraocular lens implantation, and Nd:YAG laser peripheral iridotomy. Six weeks later an inferotemporal redetachment and Nd:YAG laser peripheral iridotomy. Six weeks later an inferotemporal redetachment associated with a fibrinous anterior uveitis and resulted in raised intraocular pressure associated with the temporal tractional complexes; multiple retinal breaks associated with the temporal tractional complexes. She subsequently underwent left phacoemulsification, intraocular lens implantation, pars plana vitrectomy, enucleation, endolaser, cryotherapy, and C3F8 gas injection. During the surgery the condensed vitreous was found to be very adherent to the retina, and the vitreous was not able to be easily removed from the retina. Multiple 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 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 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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doi: 10.1136/bjo.2003.038638
Accepted for publication 8 December 2003

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 sinus. 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 outpatient clinic 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 rhinosinusitis 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 2 mm of hypoglobus. On subsequent review in the ENT clinic, nasendoscopy revealed a left deviated nasal septum with obstruction and the face—although she had had a nasotracheal intubation for a routine general anaesthetic 1 year earlier.

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 sinus. 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.
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 ethmoidal, 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 polyloid fibrinous 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 2–5 mm enophthalmos varies from 2–5 mm. Though the hereditary factor was considered to be a result of uncontrolled cell proliferation, like a tumour.1

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.1 Mutations in either p53 or p21 are detected in many tumour cells,1,2 and polymorphisms of p53 codon 72 or 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 no significant difference between both groups (table 3).

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 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.1 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.9

The p53 codon 72 and p21 codon 31 polymorphisms are two of the most important SNP markers for tumour susceptibility. 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

References


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>
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</thead>
<tbody>
<tr>
<td>p53</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GG</td>
<td>42(32.8)</td>
<td>32(31.1)</td>
<td>0.65</td>
</tr>
<tr>
<td>GC</td>
<td>60(47.9)</td>
<td>54(52.4)</td>
<td></td>
</tr>
<tr>
<td>CC</td>
<td>20(16.3)</td>
<td>17(16.5)</td>
<td></td>
</tr>
<tr>
<td>p21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>24(18.8)</td>
<td>24(18.8)</td>
<td>0.50</td>
</tr>
<tr>
<td>AC</td>
<td>56(44.6)</td>
<td>56(54.3)</td>
<td></td>
</tr>
<tr>
<td>CC</td>
<td>28(22.6)</td>
<td>23(22.2)</td>
<td></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>
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<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>
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<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>
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<tbody>
<tr>
<td>p53/p21</td>
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<tr>
<td>GG/AA</td>
<td>9 (7.0)</td>
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<td>GG/AC</td>
<td>29 (22.7)</td>
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<tr>
<td>GG/CC</td>
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<td>16 (12.5)</td>
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<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.

References
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 have 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 alphabeticthesaurus of syndromes with prominent ocular features and their many alternative names and a clear description of the general and oculo-facial features of 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.jehc.co.uk). Annual subscription (4 issues) UK £28/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 similar 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 18-22 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
The XVI International Congress for Eye Research will be held on 29 August-3 September 2004 in Sydney, Australia. For further information, please contact: icr2004@tourhosts.com.au (website: www.tourhosts.com.au/icr2004).

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 Gandier (tel: +44 7879 424 400; e-mail: igander@c2020.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 Opthalmol 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).