Wegener's granulomatosis

Wegener’s granulomatosis (WG) is a systemic granulomatous inflammatory disease of unknown origin. It occurs at any age, with the peak incidence in the third and fourth decades. The classic diagnostic triad of WG is necrotising granuloma of the upper or lower respiratory tract, vasculitis, and nephritis. Identifying a raised ANCA titre is virtually diagnostic of the disease, especially with the classic granular cytoplasmic staining pattern of neutrophils (C-ANCA). This disease is usually fatal if untreated; however, early diagnosis and instigation of immunosuppressants such as cyclophosphamide can induce long term remission.

Case report

A 79 year old woman presented with a 2 week history of a painful, red left eye. She had mild arthritis and was also noted to be profoundly deaf, requiring hand written notes for communication. There was no history of sinus problems.

Her best corrected visual acuities were 6/18 in the right eye and 6/12 in the left eye. The left peripheral supratemporal cornea showed three areas of 80% corneal thinning, 1×1 mm in size, with an overlying epithelial defect (Fig 1). There was adjacent conjunctival injection and moderate anterior chamber cellular activity. The other eye was normal with no signs of inflammation. Examination of her hands showed no obvious rheumatoid abnormalities. Initial investigations performed were full blood count, electrolytes, C reactive protein, erythrocyte sedimentation rate (ESR), and rheumatoid factor. Corneal scraping and cultures were also undertaken to exclude an infective cause. The only significant test result was a raised ESR of 94, with other tests being within the normal range. A provisional diagnosis was made of rheumatoid corneal melt and treatment was commenced with topical dexamethasone 0.1%, non-stered hourly and oral prednisolone at 40 mg/day.

After 5 days of steroid treatment, the corneal thinning was resolving and the conjunctival injection settling (Fig 2). Also by the fifth day, the patient’s hearing had dramatically improved. She could now conduct a conversation at normal volume. On further questioning, she said her hearing had deteriorated only over the past few months and had ascribed it to “natural” old age deterioration. This suggested a link between her corneal changes and hearing loss and she was investigated further for Wegener’s granulomatosis (WG). Her serum antineutrophil cytoplasmic antibodies (cANCA) titre was found to be raised (≥ 160). Rheumatoid factor was negative.

Oral cyclophosphamide was commenced on diagnosis of WG and the oral prednisolone slowly tapered. Her ocular inflammation, hearing, and arthritis continued to improve on treatment and at the 2 month follow up, there was minimal peripheral corneal thinning and no inflammation. Best corrected visual acuity in the left eye remained at 6/12. Examination by an otolaryngologist found mild hearing impairment and no significant sinus pathology. Rheumatological review found only mild arthropathy.

Comment

The majority of the ocular involvement in WG is caused by focal vasculitis, involving the anterior and/or posterior segment of the eye and corneal melting has been reported in 11–16% of patients with WG.1-4 Profound sensorineural deafness is a less common feature, occurring in about 6% of patients on presentation.5-6 It occurs either secondary to inflammation of the cochlear vessels or serious oitis media.7 Other important systemic features include pyrexia, weight loss, recurrent epistaxis, sinus discharge, haemoptysis, peripheral neuropathy, cerebral vasculitis, and renal failure (major cause of death). Fortunately in this case, the patient had the limited form of WG, with no renal involvement.

This patient presented with an uncommon combination of corneal melt and profound deafness. As deafness is a relatively common problem among the elderly patient population it may be overlooked as an important symptom, especially if ocular pathology is the main feature at presentation. It was not until the patient’s hearing improved on treatment with oral prednisolone that its significance became apparent. WG is a rare condition and early recognition and treatment are essential as the disease carries a significant risk of serious complications. A full systemic history in patients with corneal melt is important in formulating the correct diagnosis and thereby expediting the appropriate treatment.

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References

1 Von der Woude FJ. Anticytoplasmic antibodies in Wegener’s granulomatosis. Lancet 1985;2:48

Pigmentary retinopathy, macular oedema, and abnormal ERG with mitotane treatment

Adrenocortical carcinoma is a rare tumour with a poor prognosis. Mitotane (o,p’-DDD), a chemotherapy drug that suppresses the adrenal cortex and modifies peripheral steroid metabolism has been reported to cause ocular side effects including visual blurring, diplopia, cataract, toxic retinopathy with retinal haemorrhage, oedema, and papilloedema. We present a 32 year old woman with reduced visual acuity, retinal pigmentation, macular oedema, and abnormal ERG after taking mitotane. While primary hypoadrenalinemia in Addison’s disease has never been reported to cause any retinal problems secondary hypoadrenalinemia in adrenocortical dystrophy is associated with pigmentary retinopathy and other ocular findings. We postulate that the retinal problems secondary to mitotane treatment may act via a similar mechanism.

Case report

A 30 year old woman had a left nephrectomy, adrenalectomy, and chemotherapy in September 1997 following diagnosis of an adrenal carcinoma. In 1999, she was found to have secondary tumours in her lungs and liver. She was commenced on intra-arterial cisplatin and oral mitotane of up to 4.5 g daily for 6–8 months, ceasing in December 1999 because of weight loss, malaise, and soon after that, marked decrease of visual acuity in both eyes. The patient had no family history of any retinal disease. She had worn glasses for myopia for 9 years with best corrected visual acuity of 6/4 in each eye previously.

On 2 March 2000 her visual acuity was 6/12 in the right eye and 6/60 in the left eye. She also had facial pigmentation. Fundoscopy showed extensive pigmentary clumping in each eye and macular oedema in the left side (Fig 1). She was commenced on cortisone acetate and fludrocortisone to attempt to improve her vision.
One week later her visual acuity was 6/18 in the right and 6/24 in the left. Fundus examination showed mottled pigmentation at the macula and mid-periphery of both eyes but macular oedema in her left eye had resolved. Disc and vessels were normal. Full field electoretinography (ERG) was performed. Her rod response (scotopic, dim blue stimulus) b-wave was 50% of normal, the cone response (photopic, red stimulus) had b-wave 75% of normal amplitude and delayed implicit time (Fig 2). The amplitude of 30 Hz flicker responses was also reduced. There was no significant asymmetry between the two eyes. On 30 March 2000, her visual acuity improved to 6/12 in the right and 6/9 in the left. Her facial pigmentation had also faded.

Comment
Surgical resection is the treatment of choice for adrenocortical carcinoma. Mitotane (o,p'-DDD) is the only drug that causes regression of metastases and improves survival. Its biochemical action is unknown but data suggest that it modifies peripheral steroid metabolism and directly suppresses the adrenal cortex. The incidence of ocular side effects was 4% in a study of 132 patients. These effects include visual blurring, diplopia, lens opacities, optic neuritis, and a toxic retinopathy with features of papilloedema and retinal haemorrhage. In another study involving 19 patients, three patients had toxic retinopathy that included papilloedema, small retinal haemorrhages, and oedema and another patient had a subcapsular cataract. To our knowledge neither pigmentary retinopathy nor abnormal ERG findings have been reported previously. Previous studies also did not mention side effects reversibility except for a case of lenticular opacities that disappeared 5 days after discontinuance of mitotane. In our case, the patient’s visual acuity did improve significantly, with drying out of macular oedema after cessation of therapy and initiation of steroid replacement.

It is possible, however, that the ocular changes in this patient were not caused by mitotane but were secondary to cancer associated retinopathy (CAR). Its characteristic findings include attenuated retinal arterioles, with limited, if any, clinically apparent retinal pigmentary changes and cells in the vitreous humour. CAR was considered to be unlikely in this case because of the normal calibre of the retinal arterioles, the absence of vitreous cells, and the timing of the onset. Patients with CAR experience visual symptoms that often precede or are concurrent with the tumour diagnosis. Although primary hypoadrenalism in Addison’s disease has never been reported to cause retinal problems, secondary hypoadrenalism in adrenoleucodystrophy is associated with pigmentary retinopathy and other ocular findings. Adrenoleucodystrophy is a rare lipid storage disorders with increased serum level of long chain fatty acids (C24–C30). Ocular findings in adrenoleucodystrophy include visual loss secondary to visual tract demyelination and primary retinal ganglion cell degeneration, squint, cataracts, loss of corneal sensation, abnormal visual evoked potentials, and macular pigmentary changes.

The retinal pigmentary changes observed histologically were different from those in retinitis pigmentosa.

We postulate that the retinal side effects of mitotane could occur via a similar mechanism that affects the metabolism of long chain fatty acids. Mitotane causes a reduction in plasma 17-hydroxy corticosteroids level but an increase in the levels of 6-beta-hydroxyl cortisol, cholesterol, liver enzymes, corticosteroid binding globulin, and sex hormone binding globulin. At the time of writing, there was no published information on whether mitotane treatment affects serum long chain fatty acid levels. The availability of such data will be valuable for evaluating this postulation.

### References


### Coxackievirus B4 associated uveoretinitis in an adult

Coxackievirus is a kind of enterovirus, which may cause respiratory and gastrointestinal symptoms, erythema, meningocerephalitis, myocarditis, pericarditis, and myositis. Although Coxackievirus B4 is apparently a rare cause of chorioretinitis, Coxackievirus infection and concurrent posterior segment inflammation have been disclosed in a few cases. However, iridocyclitis, scattered haemorrhagic dots, and occlusive retinal vasculitis have not previously reported as being features of Coxackievirus B4 infectious ocular disease.

### Case report

A 34 year old man was referred to our hospital with a 2 week history of inferior visual field defect and visual loss in his right eye. His left eye was asymptomatic. A few days before the onset of visual manifestations, the patient noted a prodrome of viral infection, consisting of severe headache and high grade fever of 38.0–39.2°C, joint pain, and general malaise. There was no significant medical or family history. At the initial ophthalmic examination, his best corrected visual acuities were 10/20 in the right eye and 25/20 in the left eye, and intraocular pressure was normal in both eyes. Slit lamp examination showed moderate cellular infiltration in the anterior chamber in both eyes. Humphrey central visual field testing demonstrated relative scotoma inferior to fixation in the right eye. Goldmann peripheral visual field testing was within normal limits in both eyes. Ophthalmoscopic examination disclosed subretinal exudates with haemorrhage in the parafovea and the mid-periphery of bilateral eyes (Fig 1). Moreover, scattered...
haemorrhagic dots and blots were observed along retinal vessels. The optic discs appeared normal. Fluorescein angiography demonstrated occlusive retinal vasculitis characterised by blocked fluorescence and mild dye leakage from the retinal vessels corresponding to the lesions detected clinically, and irregular choroidal background fluorescence (Fig 2). Laboratory examination findings were normal except a white blood cell count of 10 500 x 10^3/L, GOT of 76 IU/L, GPT of 209 IU/L, gamma-GTP of 88 IU/l, and LDH of 771 IU/l. Serological tests, however, revealed a slight elevation of serum antibody to Coxsackievirus B4, at a titre of 1:8 (neutralisation test).

The patient's general condition improved gradually without specific medication. Cellular infiltration in the anterior chamber gradually disappeared. The retinal exudative lesions reduced in size, and haemorrhage tended to decrease. Eight months after the initial ophthalmic examination, bilateral exudative lesions had improved and were replaced by scars, and most of the retinal haemorrhagic lesions had disappeared. Visual acuity was restored to 25/20 in the right eye and 30/20 in the left eye. However, relative paracentral scotoma persisted in his right eye. Antibody titre to Coxsackievirus B4 had increased, demonstrating a 16-fold rise between the acute (1:8) and convalescent sera (1:128). Retrospectively, we diagnosed the ocular lesions in this patient as uveoretinitis induced by Coxsackievirus B4 infection.

**Comment**

Viruses are one of the most common causes of infections involving the posterior segment of the eye. Such infections may be congenital or acquired, and may affect primarily the retina or the choroid. Retinitis and choroiditis caused by viruses such as measles, influenza, Epstein-Barr virus, and Rift Valley fever virus typically occur on an acquired basis, subsequent to an acute viral systemic illness. Coxsackievirus-associated chorioretinitis is thought to elicit the same clinical course as in other viral chorioretinitis. Coxsackievirus infection and concurrent posterior segment inflammation have been reported in a few cases, in which clinical signs and antibody titres suggested that Coxsackievirus was the responsible agent.12 Among them, two cases of choriorretinitis associated with Coxsackievirus B4 infection were reported.13 One was a paediatric case, and the ophthalmoscopic manifestation was described as scattered white lesions in mid-periphery of the retina.7 The other was an adult case, with choriorretinitis similar to the multiple evanescent white dot syndrome.1 However, the clinical features observed in our patient consisted of iridocyclitis, scattered haemorrhagic dots, and occlusive retinal vasculitis were apparently different from the two earlier reports.

Coxsackievirus infection is apparently a rare cause of uveoretinitis but, nevertheless, should be considered in the appropriate clinical setting, since it is possible that overt clinical manifestations and complications may occur depending on the severity and the location of the uveoretinal lesions.

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**References**


**Evaluation of telemedicine for slit lamp examination of the eye following cataract surgery**

Ophthalmic surgery is well suited to the use of telemedicine in diagnosis and management,1 but diagnostic accuracy and reliability are critical if it is to be widely introduced.3 Moorfields Eye Hospital is located in central London and runs community outreach clinics. One clinic and operating list in Ealing is 17 miles from the central hospital. The surgical teams operate in Ealing and return to the central hospital. We therefore investigated the use of a telemedicine link for review of postoperative cataract surgical patients aiming to assess what can be seen clearly and reliably using telemedicine and to identify what the observer using this link may potentially miss.

**Case report**

This study had Moorfields Eye Hospital research and ethics committee approval. Patients consecutively admitted for cataract surgery were recruited to this study and full informed written consent was obtained. A telelink of a pair of Global Telemed mobile workstations connected by three ISDN lines, with a video transmission rate of 384 bits/s was used for examinations and high resolution examination being achieved by static images. Two surgeons conducted examinations, one using the slit lamp and the other using the telemedicine link. Slit lamp signs graded by the two observers comprised the presence and degree of the following—central corneal oedema, corneal oedema at the limbal section, Siedel’s sign, folds in Descemet’s membrane, anterior chamber depth, flare and cells, intraocular lens decentration, and lens stability. Both observers in person independently examined a group of postoperative cataract patients in order to determine agreement between practitioners.

Twelve consecutive postoperative cataract patients were recruited to the study with 10 others as a control group to assess interobserver agreement by in-person examination. The results of the study are given in Table 1.

**Comment**

Although interobserver variation means that telelink reliability cannot be precisely calculated in terms of sensitivity and specificity, these findings give an indication of the accuracy of slit lamp examination after cataract surgery using 384 K bandwidth. Examination by video telelink was relatively reliable in detecting oedema at the central cornea but did not consistently detect oedema at the corneal section, or anterior chamber flare and failed entirely to detect DM folds or anterior chamber cells. Patients enjoyed the telemedicine experience finding it reassuring to see as well as interact with their surgeon via the telelink.

Video compression algorithms used in streaming video are “lossy” and higher bandwidth increases video quality. The process begins with a good video source with low noise, since noise does not compress well. A poor quality video image is very difficult to stream successfully because of this difficulty in compression. The videoconferencing standard H-261 was implemented to provide for video compression to a given ISDN bandwidth. With increasing bandwidth there is correspondingly better quality video. A tele-video link for corneal assessment in Canada using six ISDN lines (768 K) was found to be acceptable, whereas three lines (384 K) were unacceptable (personal communication, Dr M Pop, Montreal, Canada).

The video display also affects the information observed. Broadcast video monitors are accurately colour calibrated, whereas digital compressed video to 384 K bandwidth has...
inherited retinal dystrophy and asymmetric axial length

The prenatal and postnatal development of the eye is determined by complex interactions between a number of genes, their products, and certain environmental factors. Since each eye is influenced by precisely the same processes as its fellow, mutations in regulatory genes usually lead to symmetric phenotypes. In this report, we describe two siblings of Asian ethnicity, born to unrelated parents with no family history of ocular disease, who have an unusual bilateral retinal dystrophy associated with very asymmetrical ocular growth.

Case report

Sib 1 was an 8 year old boy with epilepsy who had been born at term after an uneventful pregnancy. Pendular nystagmus had been noted soon after birth and his visual acuity in each eye was 3/60; refraction –17.00 DS right, plano left. Anterior segment examination was unremarkable with normal intraocular pressures. Examination of the right eye (Fig 1A and B) revealed generalised retinal pigment epithelial and choroidal atrophy, a macula “coloboma,” sheathed and occluded retinal vessels inferotemporally, and a shallow infero-temporal retinal detachment with subretinal fibrosis. The left eye appeared similar; however there was no macular abnormality. By ultrasound, the axial length and ocular volume were 26.70 mm and 9.9 ml right, 20.70 mm and 4.6 ml left respectively.

Sib 2 was a 6 year old girl born at term with no significant past medical history. Her corrected visual acuity was 3/60 in each eye; refraction –17.00 DS right, plano left. Anterior segment examination was normal. Funduscopy of the right eye revealed a “macula coloboma,” extensive retinal pigment epithelial and choroidal atrophy, intraretinal pigment migration and preretinal fibrosis. The appearances of the left eye were similar. No retinal detachment was noted. Axial lengths and ocular volumes were 26.1 mm and 9.4 ml right, 20.7 mm and 4.6 ml left respectively.

Electrophysiology was performed on both children based on the ISCEV standards for adults, but initially using surface electrodes on the lower eyelids. No definite ERG response could be recorded from the emmetropic left eye of either child (even with high intensity stimulation or photopic stimulation), a finding consistent with severe generalised dysfunction involving rod and cone photoreceptors. The electrical responses of both children’s myopic fellow eye showed a milder degree of dysfunction: there were reduced b:a ratios in both scotopic and photopic responses which, together with the abnormal 30 Hz flicker ERGs, suggest a mid-retinal locus affecting phototransductional cone and rod systems. The skin recordings obtained with surface electrodes were confirmed under anaesthesia using gold foil corneal electrodes.

It is reasonable to conclude that these two siblings represent original probands with a novel inherited, probably autosomal recessive, retinal dystrophy. The particularly interesting feature of this disorder is the asymmetry of the axial lengths and ocular volumes of the eyes, rare in genetically determined ocular diseases. Indeed, we are aware of only one other report that identifies ocular asymmetry in association with retinal degeneration. Laflat et al reported a single patient who had bilateral Stargardt’s disease and unilateral myopia.

Normally, axial elongation and ocular enlargement are carefully coordinated to equalise growth of fellow eyes in the quest for emmetropia. The differences observed in the two siblings described here reflect a decoupling of this developmental synchrony. It has been demonstrated that form deprivation in various species produces progressive axial growth and myopia of the postnatal eye. However, in both our sibs the ERG responses were much better preserved in the highly myopic right eye where, in addition, there was a low b:a ratio. Similar findings do not occur in myopia without retinal pathology.

The bilateral symmetrical retinal dystrophy noted in both our patients suggests a genetic basis for the disease but a single genetic defect cannot explain the additional asymmetry of eye size and electrophysiological measurements. Most probably, the phenotype results from at least two separate events, a germline mutation in a retinal or retinal pigment epithelial specific gene leading to degeneration, thereby creating a susceptible background on which a second event could occur. This event, leading to the asymmetry of ocular volume, may be an environmental factor or a mutation in a second gene important in the regulation of eye growth. If this were a somatic rather than germline mutation it would explain the asymmetry of the disease.

References


Inherited retinal dystrophy and asymmetric axial length

The prenatal and postnatal development of the eye is determined by complex interactions between a number of genes, their products, and certain environmental factors. Since each eye is influenced by precisely the same processes as its fellow, mutations in regulatory genes usually lead to symmetric phenotypes. In this report, we describe two siblings of Asian ethnicity, born to unrelated parents with no family history of ocular disease, who have an unusual bilateral retinal dystrophy associated with very asymmetrical ocular growth.

Case report

Sib 1 was an 8 year old boy with epilepsy who had been born at term after an uneventful pregnancy. Pendular nystagmus had been noted soon after birth and his visual acuity in each eye was 3/60; refraction –17.00 DS right, plano left. Anterior segment examination was unremarkable with normal intraocular pressures. Examination of the right eye (Fig 1A and B) revealed generalised retinal pigment epithelial and choroidal atrophy, a macula “coloboma,” sheathed and occluded retinal vessels inferotemporally, and a shallow infero-temporal retinal detachment with subretinal fibrosis.
Adenoma of ciliary pigment epithelium: a case series

Adenoma of ciliary pigment epithelium is a rare tumour. Many are diagnosed retrospectively either after excision or enucleation, as malignant melanoma is suspected. We report a series of four patients found to have adenoma of ciliary pigment epithelium and discuss the clinical features and unusual behaviour of these neoplasms.

Case reports

We reviewed the histopathological reports in the ophthalmic pathology archive dating from 1980 to date and identified four patients who had the histopathological diagnosis of adenoma of ciliary pigment epithelium. We crosschecked the details with the clinical oncology database. We reviewed their notes for features that would help us to identify this ciliary body tumour clinically. The salient features of these patients are given in Table 1.

Patient 1 was reported elsewhere in 1994. He had a dark brown multinodular mass in the inferotemporal anterior chamber angle of the left eye. His tumour was a relatively small but invasive lesion. Patient 2 was the only non-white patient with this condition in our series. Her tumour was an incidental finding when she presented to an ophthalmologist with allergic conjunctivitis. The tumour was small and dark brown. The tumour had invaded the anterior chamber angle and the root of the iris occupying one clock hour of the iris, also showing the localised cataract through the pupil. Patient 3 was initially treated for acute angle closure glaucoma in another hospital. Trabeculectomy was performed to achieve control of intraocular pressure. Postoperatively, he was found to have a lesion behind the crystalline lens. He underwent phacoemulsification with intraocular lens implantation to improve visualisation of the lesion. A black ciliary body mass was seen (Fig 1E). This prompted his referral to the oncology service in June 1999. Control of the intraocular pressure proved refractory even with additional medical treatment.

Table 1

<table>
<thead>
<tr>
<th>No</th>
<th>Age/sex/race</th>
<th>VA</th>
<th>Size (mm)</th>
<th>Clinical features</th>
<th>Surgery</th>
<th>Recurrence</th>
<th>Year of diagnosis</th>
<th>Complication</th>
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<tr>
<td>1</td>
<td>40/M/W</td>
<td>6/5</td>
<td>8x7x1</td>
<td>Angle invasion</td>
<td>Local resection</td>
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<td>1992</td>
<td>RD repair</td>
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<td>2</td>
<td>62/F/A</td>
<td>6/6</td>
<td>5x3x1</td>
<td>Angle invasion, cataract</td>
<td>Local resection</td>
<td>No</td>
<td>1996</td>
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<tr>
<td>3</td>
<td>65/F/BrC</td>
<td>6/18</td>
<td>8x4x4</td>
<td>Sentinal vessel, pigment dispersion</td>
<td>Local resection</td>
<td>No</td>
<td>1998</td>
<td>RD repair</td>
</tr>
<tr>
<td>4</td>
<td>54/M/W</td>
<td>1/60</td>
<td>5x4x4</td>
<td>Secondary glaucoma, pigment dispersion</td>
<td>Enucleation</td>
<td>No</td>
<td>1999</td>
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</table>

M = male, F = female, W = white, A = Asian, RD = retinal detachment, VA = visual acuity.
Pigment dispersion was seen in patients 3 and 4. This was mainly in the vitreous of patient 3 but in both the vitreous and the anterior segment of patient 4. There was heterochromia of the iris in patient 4. No angle invasion was seen in these two patients.

Other associated features were a localised cataract in patient 2, tracional retinal detachment and secondary glaucoma in patient 4. Patient 3 had an episceral sentinel vessel over the tumour. None of these patients had any history of ocular trauma or intraocular inflammation. Ultrasound biomicroscopy (Fig 1C) helped us to evaluate these tumours in more detail.

The first three patients underwent local resection of the tumour in the form of iridocyclectomy under hypotensive anaesthesia. The last patient had enucleation as he opted to have the eye removed because of the poor visual prognosis for that eye as a result of secondary glaucoma, extensive pigment dispersion, and tracional retinal detachment.

Histopathologically, these tumours showed characteristic features of adenoma. Mitotic activity was absent or low. Invasion of ciliary muscle and the iris root was seen in patients 1 and 2. Patient 3 had a cystic adenoma with cells forming gland-like structures around central cysts.

**Comment**

Our series highlights the paradoxical behaviour of adenomas of the ciliary pigment epithelium. Smaller lesions invaded the angle and larger lesions caused extensive pigment dispersion although non-invasively. Angle invasion resulted in these tumours being seen and resulted in the presentation of patients 1 and 2. Blurred vision due to pigment dispersion in the vitreous resulted in the presentation of patient 3. Angle closure glaucoma and pigment dispersion were the main features of patient 4. Shields et al. presented a series of eight patients with adenoma of ciliary pigment epithelium and described their clinical features. In their series they found an association with cataract, vitreous haemorrhage, and neovascular glaucoma.

Pigment dispersion was seen in two of our patients. The presence of pigment clumps and extensive pigment dispersion in the vitreous of patient 3 (Fig 1D) is unique and has not been reported before. Chang et al. in 1979 reported the presence of pigment in the retinal space adjacent to the tumour in a case of adenoma of ciliary pigment epithelium. Extensive pigment dispersion in vitreous had been reported in malignant melanoma of the choroid but to our knowledge not in adenoma of ciliary pigment epithelium.

Secondary glaucoma from intraocular tumours is well known. In their survey of intraocular tumours causing secondary glaucoma Shields et al. reported on 2704 eyes. Of the five adenomas of the ciliary body one was from ciliary pigment epithelium. None of these had secondary glaucoma. Of the ciliary body melanomas, 17% had secondary glaucoma. Angle closure was responsible for secondary glaucoma in 12% of the eyes with ciliary body melanoma. In their series in 1999 Shields et al. had one patient who had neovascular glaucoma secondary to adenoma of ciliary pigment epithelium. Patient 4 in our series presented with secondary angle closure glaucoma.

Malignant melanoma of ciliary body is known to invade the anterior chamber angle. Chang et al. reported angle invasion in adenoma of the pigment epithelium of ciliary body. Shields et al. reported a patient in whom invasion of the iris stroma by an adenoma of ciliary pigment epithelium was documented with progressive growth. They initially suspected this to be a tumour of the iris but on later evaluation showed the origin from the ciliary body. The presenting feature in patients 1 and 2 in our series was similar, although the ciliary body origin was recognised initially. In patient 2 the diagnosis of adenoma of ciliary body was strongly suspected preoperatively, as the tumour that had invaded the angle was very small. Invasion of the angle by ciliary body melanomas usually does not occur until they have attained a larger size. Invasion of the angle has also been described in melanocytoma of the ciliary body. These too tend to be relatively larger when they invade the angle, unlike the adenomas that we described. Iris melanocytomas undergo central necrosis and cause pigment dispersion and glaucoma. However, the necrotic centre is absent in adenomas.

One of our patients (patient 1) had a sentinel vessel. Sentinel vessels are typically thought to be associated with malignancy. However, this is not always the case. Presence of a sentinel vessel indicates ciliary body involvement. Fine needle aspiration biopsy may be considered for aiding diagnosis of malignancy. However, its role in the diagnosis of these lesions may be limited. Absence of malignant cells does not always rule out the presence of malignancy.

Our study highlights the paradoxical behaviour of adenoma of the pigment epithelium of ciliary body that has not been emphasised before. Adenoma of the pigment epithelium of the ciliary body should be kept in mind if there is extensive pigment dispersion by larger tumours and invasion of the anterior chamber angle by relatively small tumours.

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### References

dilatation and in the anaesthetic room preoperatively in the 2.5% group (Fig 1) showed a mean rise in systolic BP of 22 mm Hg ($p=0.003$) in the normotensive group. In the hypertensive group all patients recorded statistically significant rises in systolic and diastolic BP (respectively $p=0.010$, $p=0.009$). In the 10% group (Fig 2, 10 patients in the normotensive group showed a mean rise in systolic BP of 54.4 mm Hg ($p=0.001$) and 10.5 mm Hg in diastolic BP ($p=0.077$). In the hypertensive group 6 patients recorded a rise in systolic BP with a mean of 22.8 mm Hg ($p=0.015$) and a diastolic rise with a mean of 16.8 mm Hg ($p=0.033$) (Table 1) (Figs 3 and 4).

**Comment**

Our study showed that patients in both groups of phenylephrine doses experienced a rise in systolic blood pressure which was statistically significant; the mean of which is higher in the 10% group. The maximal systolic blood pressure rise is around 10–20 minutes after administration, which corresponds to the time of maximal plasma levels of phenylephrine as demonstrated by other authors, but the paired t-test failed to show any significance between the results of the groups of either concentration. Postoperative results showed some elevations in systolic and diastolic BP but weren’t statistically significant. Using Altman’s normogram the study was limited in its power. Although the aim of this study was not designed to investigate the effect of anxiety on systemic blood pressure, this element should be taken into consideration since it is known to elevate the systolic blood pressure. We have also used topical anaesthesia so that adrenaline 1:200 000, which is always included in local anaesthesia, is excluded which might cause elevation of the blood pressure although the concentration is too low for any significant systemic effect.

**Table 1**

<table>
<thead>
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<th></th>
<th>Mean</th>
<th>SD</th>
<th>Significance*</th>
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</thead>
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<td>Normotensive/sys</td>
<td>-12.44</td>
<td>19.08</td>
<td>0.86</td>
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<tr>
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<td>2.88</td>
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<td>0.61</td>
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<td>Hypertensive/sys</td>
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<td>Hypertensive/diast</td>
<td>-4.16</td>
<td>14.00</td>
<td>0.49</td>
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Two tailed t test, comparing the 2.5% group and 10% group both hypertensive and normotensive patients.

* $p=0.05$.

**References**


**Effect of preoperative detection of photoreceptor displacement on postoperative foveal findings in eyes with idiopathic macular hole**

In idiopathic macular hole formation, Gass' hypothesised that macular holes enlarge with displacement of the photoreceptors without tissue loss around the fovea. Jensen and Larsen' developed binocular kinematic perimetry that could evaluate local retinal photoreceptor displacement and also confirm centrifugal photoreceptor displacement away from the foveola in eyes with a macular hole. Using the binocular perimetry technique and scanning laser ophthalmoscope microperimetry, we confirmed that the patients with a unilateral macular hole with preoperative photoreceptor displacement had better postoperative visual function when compared to patients without preoperative photoreceptor displacement.' We also previously reported that the postoperative foveal findings were correlated with the postoperative visual function in eyes with macular hole.' The purpose of the present study was to investigate whether or not detecting photoreceptor displacement preoperatively affects the postoperative foveal findings in eyes with an idiopathic macular hole.

**Methods and results**

Fifteen patients (12 women, three men; mean age 62 (SD 5) years) with a unilateral idiopathic macular hole that resolved after vitreous surgery were examined. All had undergone surgery at our institution. Eleven eyes were classified as having a stage 3 hole, four stage 2, and one stage 4. The best corrected visual acuity (VA) levels in the unaffected eyes were 0.7 or better, and no patients had ocular diseases except for mild cataract.

To detect photoreceptor displacement in eyes with the disease preoperatively, we performed binocular kinetic perimetry.' The anatomical status of the repaired macular holes was assessed 3 months after the operation using optical coherence tomographic equipment (OCT 2000, Humphrey Instrument, division of Carl Zeiss, San Leandro, CA, USA). Good postoperative foveal findings were defined as the presence of a foveal depression, as previously reported by Imai and associates' (Figs 1 and 2).

Photoreceptor displacement was detected in 11 of the 15 (73%) patients preoperatively. Good postoperative foveal findings were observed in nine of the 11 (82%). Photoreceptor displacement was detected preoperatively in all nine patients. The prevalence of good postoperative foveal findings was significantly higher in patients in whom photoreceptor...
displacement (82%, 9/11) was detected preoperatively than in those in whom displacement was not detected (0%, 0/4) (p = 0.01, χ² test with Yates’s correction). The mean preoperative VA tended to be better and the mean VA measured 3 months postoperatively was significantly better in patients in whom photoreceptor displacement was detected preoperatively (0.15 and 0.79, respectively) than in those whom it was not detected (0.08 and 0.25, respectively) (p = 0.06 and p = 0.001, respectively, unpaired t test). Visual acuities were converted to the logarithm of the minimum angle of resolution (logMAR) for statistical analysis.

Comment

Jensen and Larsen reported that in two macular hole patients with the longest duration of symptoms, photoreceptor displacement was not detected. The reason why patients with a macular hole with longer duration of symptoms did not have photoreceptor displacement might be that there was more damage to the displaced photoreceptors. A longer disease duration probably induced functional damage to the displaced photoreceptors, and consequently no photoreceptors are present within the fovea. We also reported that the detection of photoreceptor displacement preoperatively should affect postsurgical visual function, and photoreceptor damage might occur in eyes in which photoreceptor displacement was not detected preoperatively, resulting in worse postoperative visual function.

Our results demonstrated that in patients with a macular hole in whom photoreceptor displacement was detected preoperatively, the postoperative foveal findings and visual acuities were better when compared to patients in whom displacement was not detected preoperatively. It was also reported that better anatomical foveal recovery in eyes after macular hole surgery resulted in better improvement of postoperative visual function. Although limitations of this study are based in its sample size, we think that a degree of preoperative photoreceptor damage influences not only postoperative visual function but also anatomical recovery.

The authors have no proprietary interest in any instruments used in this study.

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References


Nodular scleritis in a patient with sarcoidosis

Sarcoidosis is an immune mediated disease that may affect any organ. Scleral involvement has rarely been described with sarcoidosis. We report on a patient with unilateral nodular anterior scleritis who was found to suffer from sarcoidosis. Here, the clinical and histopathological features are described.

Case report

A 53 year old white woman had persistent moderate tenderness in her left eye that lasted for several weeks. Visual acuity was 20/20 in both eyes. The slit lamp appearance of the right eye was unremarkable. A scleral nodule in the inferior nasal quadrant was found in the left eye (Fig 1), and a moderate tenderness was determined in this area. The intraocular pressures were within normal ranges. On ophthalmoscopy, the posterior segment of both eyes was normal. The ultrasound images did not indicate posterior scleritis.

The medical history was remarkable for an episode of hepatitis A 20 years ago, and the patient suffered from exercise dependent dyspnoea, night sweats, and repeated headaches. An extensive examination for associated systemic diseases was performed, including rheumatological assessments for signs of RA, CRP, ANCA, ANA, and PPD tests: all were within normal ranges and consultant examinations by the internal medicine, rheumatology, neurology, and ENT departments were performed. The chest x ray examination revealed a bilateral hilar lymphadenopathy and basal interstitial pulmonary infiltration typical of sarcoidosis stage 2. In the body plethysmography, a mild restriction and a moderately reduced diffusion capacity was determined. The bronchoalveolar lavage revealed an increased proportion of CD4+ T cells and an increased CD4/CD8 ratio, consistent with active pulmonary sarcoidosis. The level of angiotensin converting enzyme was 51.3 U/I (normal, 8–52) in the high normal range. The magnetic resonance images revealed an increased enhancement and thickening of the inferior nasal part of the sclera of the left eye. A biopsy was taken from the scleral nodule under peribulbar anaesthesia. Histopathological studies on the paraffin embedded tissue that was performed by the pathologists revealed a perivascular lymphocytic cell infiltration, with non-casating granuloma-like cell accumulation (Fig 2). Some histiocytes and very few giant cells were found. The scleral tissue was oedematous and the vessels were dilated, but necrosis, vasculitis or malignant cell configurations were absent. Additionally, Ziehl-Neelson and Grocott’s silver methanamine stains excluded the possibility of causative infections.

Treatment with oral prednisone with an initial dosage of 1 mg/kg was performed. The corticosteroid dosage was tapered within 6 weeks, and was maintained at 20 mg. Under this regimen, the pain and the scleral nodule resolved immediately, and the ACE level fell to 24 U/I (normal).

Comment

Scleral involvement has been previously reported in a few patients with sarcoidosis. In a series of 537 patients with histologically proved sarcoidosis, the scleral plaques anterior to the equator have been found in four cases, and this was seen in the acute stage of disease and in association with erythema nodosum and bilateral lymphadenopathy. In another series by Jabs and Johns, within a group of 183 patients with chronic sarcoidosis, scleral involvement was detected in one patient. However, the clinical course and histopathological appearances have not been described previously. Posterior scleritis has been seen only rarely with sarcoidosis.

In our patient, the sparse systemic symptoms made the diagnosis difficult and, therefore, scleral biopsy was done in order to rule out infections or malignancies and to disclose the histopathological evidence for sarcoidosis. Although a classic non-caseating granuloma...
has not been found in our patient, a granuloma-like perivascular cell infiltration was seen, consisting primarily of lymphocytes, some histiocytes, and very few giant cells, which are typical for sarcoidosis. To the best of our knowledge, this is the first histopathological description of two thirds of eyes with sarcoid lesion in the sclera. The mainstay of treatment for sarcoidosis is corticosteroids, and the sclera was infiltrated in our patient.

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References

Panophthalmitis following contact diode laser cyclophotocoagulation in a patient with failed trabeculectomy and trabeculectomy for congenital glaucoma

Diode laser trans-scleral cyclophotocoagulation (TSCPC) is a well accepted method for the management of patients with refractory glaucoma. A lasting decrease in IOP has been described in patients with refractory glaucoma. A lasting decrease in IOP has been described in patients with refractory glaucoma. A lasting decrease in IOP has been described in patients with refractory glaucoma. A lasting decrease in IOP has been described in patients with refractory glaucoma.

His visual acuity was no light perception in the right eye and 20/60 in the left. There was right sided, peribulbar oedema, restricted motility, and purulent discharge with matting of the eyelashes. Rupture of the globe with prolapse of the iris was evident along the superior limbus. A severe, medically uncontrolled glaucoma followed TSCPC using G-probe delivery, severe, medically uncontrolled glaucoma followed TSCPC using G-probe delivery, severe, medically uncontrolled glaucoma followed TSCPC using G-probe delivery, severe, medically uncontrolled glaucoma followed TSCPC using G-probe delivery.

A diagnosis of panophthalmitis of the right eye was made. As no response to intravenous antibiotic treatment was observed over the next 48 hours, evisceration was undertaken with the patient’s informed consent. Staphylococcus epidermidis was grown on culture.

Comment
Its efficacy notwithstanding, diode laser cyclophotocoagulation has been associated with complications, although less frequent than with other cyclodestructive procedures. Con- junctival burns, corneal decompensation, neurotrophic corneal defects, uveitis, IOP spikes, phthisis, 0.5%, chronic hypotony (1%), macular puckering (0.5%), intraocular haemorrhage (0.5%), visual loss, and malignant glaucoma have all been reported.10 Possible correlation of outcome with perim- eteral pigmentation and the hearing of audible “pop,” especially in patients with dark brown irides, has been suggested.

Scleral thinning is a recognised risk factor for complications, but scleral perforation following this procedure has been reported earlier in one patient only. This patient had scleral thinning following previous cataract surgery. Possibly, the sharp probe edge had cut conjunctival vessels causing bleeding and contamination of the probe tip. Thin adherent debris was then carbonised allowing the laser tip temperature to rise to 300°C, sufficient to cause scleral perforation. This report led to redesigning of the probe tip to protect the vascular structures from its sharp edges.

Our patient was treated as a part of a standard protocol using the modified G probe, and bleeding did not occur. The perforation could have resulted from the 90% mechanical pressure, or the effect of the laser on the thinned out sclera, which the patient probably had as a result of buphthalmos and previous ocular surgery. There are no known “corrective factors” to utilise in this type of scleral thinning, and if treatment is absolutely necessary, a 40% lower energy should be used, and areas of thinning and possible wound dehis- cence, such as the bleb, should be avoided. We emphasise caution in undertaking contact diode laser trans-scleral cyclophotocoagulation in operated eyes with thin sclera.

Case report
An 18 year old male patient presented to our emergency service with a 4 day history of pain, redness, and peribulbar swelling in the right eye, accompanied by poor vision. One week earlier he had undergone 360 degree diode laser trans-scleral cyclophotocoagulation using contact G-probe in the right eye for controlled glaucoma. He had thrice undergone filtering surgery in both eyes for congenital glaucoma since 1985. During the last trabeculectomy performed in 1996, intra-operative mitomycin C (0.2 mg/100 ml applied for 3 minutes beneath the scleral flap) had also been used. But it failed in the right eye and the patient had developed a painful blind eye.

Bilaminar interepithelial bodies within fingerprint dystrophy-like changes in bilateral iridocorneal endothelial syndrome

In most cases the iridocorneal endothelial (ICE) syndrome affects women unilaterally and shows endothelial degeneration, thickening of Descemet’s membrane and subendothelial deposits including flakes and lamellae. The infantile iris atrophy, and the Cogan-Reese syn- drome. We present a unique case of an ICE syndrome, which we would subclassify as Chandler’s syndrome, with concomitant finger- print dystrophy and multiple interepitheli- al bilaminar bodies within the fingerprint- like striae.

Case report
Ten years earlier a now 45 year old patient presented with a bilateral ICE syndrome and fingerprint dystrophy. On slit lamp microscopy, the epithelial layer showed fingerprint lines, while the endothelial layer had a “hammered” appearance. Iris atrophy, corectopia, ectropion uveae and anterior synchiae were obvious (Fig 1A) and progressive in both eyes, nevertheless the intraocular pressure and the visual acuity were normal. Eight years later both corneas developed oedema (Fig 1B) and the left eye displayed cataract with reduction of visual acuity from 1.0 to 0.25 and 0.9 to 0.7 in the left and right eye, respectively. Com- bined perforating keratoplasty and extracap- sular cataract extraction were performed on the left eye in June 1998. None of the patient’s family members had a similar condition.

The histological examination of the paraffin embedded corneal specimen revealed fingerprint-like striae containing small basophilic deposits between the cells (Fig 2A). The van Kossa stain for calcium was negative and no hydroxyapatite crystals were seen. At the ultrastructural level the deposits were found to be round and bilaminar showing an electron dense core in the centre (Fig 2B). Some of the deposits coalesced to form oval...
the ICE syndrome, owing to obvious morphological changes of the iris despite non-specific endothelial changes. The striking features of Chandler's syndrome are corneal endothelial dystrophy and subsequent oedema. Corneal epithelial lesions were pronounced.

was degenerated, and intercellular interdigitation of the membrane was not thickened, the endothelial cells was normal without deposits in the basement membrane. The cytoplasm of the adjacent epithelial cells was normal without deposits in basement membrane structures. The cytoplasm of the adjacent epithelial cells was normal without deposits in relation to the cell membranes (uranyl acetate, lead citrate, bar = 250 nm). The endothelial cells possessed microvilli, their mitochondria were degenerated, and intercellular interdigitations were pronounced.

Comment
We consider our case as one of Chandler's syndrome, which is part of the spectrum of the ICE syndrome, owing to obvious morphological changes of the iris despite non-specific endothelial changes. The striking features of Chandler's syndrome are corneal endothelial dystrophy and subsequent oedema. Corneal epithelial lesions were pronounced. Second, an endothelial dystrophy—namely, a fingerprint dystrophy—is present in addition to the ICE associated changes. To our knowledge this association has not been described in the literature before.

Thirdly, the fingerprint striae contain bilaminar deposits with an electron dense core. Several types of deposits have been described in association with endothelial dystrophies. Patches of "unknown substance" were noticed in fingerprint dystrophy along basal laminae of the basal epithelial cells with no similarity to the bilaminar structures described here. Bröderick et al mentioned intercellular dense oblong bodies which were embedded in condensed matrix and were much smaller than the deposits in our case. In a cornea with a Meesmann dystrophy intraepithelial "peculiar electron dense structures" were shown, also with a totally different appearance compared to that seen in the fingerprint striae of the specimen we present here. In a patient with ocular tumoral calcinosis, nodules from the palpebral conjunctiva contained calcified "spherules," which show some similarity to our—nevertheless, uncalcified—deposits. Our deposits were located within the fingerprint striae, close to the epithelium. It seems to be obvious that the epithelial cells must have produced those bilaminar bodies, since no other cell type was present nearby, but there was no evidence of extrusion from the basal cells.

Our case shows that ICE syndrome can occur bilaterally in men, and can be associated with a fingerprint-like dystrophy. Further studies should be carried out to confirm our electron microscopical observation of bilaminar deposits within fingerprint-like striae and directed towards an analysis of their chemical composition of the extracellular material and origin.

Acknowledgements
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This case was presented as a poster at the meeting of the Association for Research and Vision in Ophthalmology in Fort Lauderdale in May 2001.

References

Fine retinal crystalline deposits observed by confocal scanning laser ophthalmoscopic examination using infrared light
Bietti's crystalline retinopathy is a tapetoretinal degeneration characterised by numerous tiny sparkling yellow-white spots mainly located in the deeper layer of the posterior pole retina. Since retinal crystalline deposits tend to become small and decrease in number over time, the fundus appearance becomes indistinguishable from other retinal dystrophies with time. The advent of confocal scanning laser ophthalmoscopy (SLO) with infrared light has enabled high sensitive examination of the deep layer of the retina. We studied the deep retinal abnormalities in a case with a typical Bietti's crystalline retinopathy and in another case with myopic choriotinal atrophy throughout the posterior pole. Consequently, we detected numerous fine crystalline deposits in both cases, which were not detected with other funduscopic examinations.

Case reports
Case 1
A 52 year old man was referred to our hospital by his ophthalmologist who found an abnormal fundus appearance. There was no consanguinity. Best corrected visual acuity was 20/24 in his right eye and 20/20 in his left eye. No abnormalities were found in the anterior segment and media. Fine crystalline deposits were not detected in the corneal stroma of either eye. Funduscopic examination revealed numerous fine reflective crystalline deposits throughout the posterior pole and mid-peripheral retina of both eyes. Most of these deposits were in the deep retina and subretina but some deposits were superficial. Goldmann perimetry showed relative scotomas in a zone within 30–40 degrees of the central field in both eyes. Fluorescein angiography showed indiscernible hyperfluorescent spots surrounded by hypofluorescent lesions in the early phase. Indocyanine green angiography also showed the atrophy of the choriocapillaris in the posterior pole. Crystalline deposits did not show any fluorescence in either fluorescein or indocyanine green angiography.

The retinal crystalline deposits were more clearly visible with the fundus examination using SLO (Rodenstock, Germany) using infrared light compared to the routine funduscopic examinations (Fig 1). Each crystal deposit appeared larger in size. In addition, numerous fine crystal deposits were evident, which were not observed by conventional funduscopic examinations including ophthalmoscopy and
and no fine crystal deposits were found at
limbs of either cornea. Both eyes were aphakic
and the posterior lens capsules were intact but
relatively opaque. Funduscopy examination
revealed the posterior staphyloma and the
extensive atrophy of the retinal pigment
epithelium and choroid. Staphyloma extended
from the posterior to mid-peripheral retina in
both eyes. Optic disc showed myopic appear-
ance. Retinal vessels and peripheral retina
showed normal appearance. Standard elec-
trorretinogram was non-recordable. Gold-
mann perimetry showed marked constriction
of all isoptres and loss of visibility of 1–4-e
isoptre for both eyes. During a 5 year follow-
up, visual acuity and retinal appearance were
stationary. Fluorescein angiography per-
formed at the age of 57 demonstrated hypofluorescence throughout the posterior
pole in the early phase and diffuse hyperflu-
orescence in the late phase. Indocyanine green
angiography disclosed the loss of background
clear-like fluorescence throughout the poste-
rior pole due to the choriocapillaris atrophy.
Fundus examination by the confocal SLO
with infrared light disclosed numerous tiny
crystalline deposits in the posterior pole (Fig
2), which were not evident with other fundu-
scopic examinations, including ophthalmos-
copy and biomicroscopy with a contact lens,
or with fluorescein and indocyanine green
angiographic studies. The deposits scattered
light when the source of light was moved to
cause them sparkle, similar to the fine
crystalline deposits observed in the fundus of
case 1.
Amino acid analysis and other examina-
tions did not show any abnormal value.

Comment
The diagnosis of Bietti’s crystalline retin-
opathy in case 1 is based on the retinal
crystalline and supported by the characte-
ristic island-like hypofluorescence observed by
fluorescein angiography. Fundus examina-
tion by SLO with the infrared light disclosed
numerous tiny crystalline deposits, which
were not observed by conventional fundus
examinations. Thus, in Bietti’s crystalline
retinopathy, there are more numerous ac-
cumulation of retinal crystalline deposits than
can be observed by funduscopy examina-
tions.
In case 2, numerous tiny retinal crystalline
deposits were detected by SLO with infrared
light, although no other examinations dis-
closed these crystalline deposits. We have not
observed such retinal crystalline deposits in
other cases with myopic chorioretinal atrophy,
suggesting the accumulation of crystalline
deposits is not a generalised feature of myopic
chorioretinal degeneration. Crystalline depos-
ts, prerequisite for the diagnosis of Bietti’s
crystalline retinopathy, are difficult to detect in
advanced cases. Since crystalline deposits
do not block or transmit fluorescence in
fluorescein or indocyanine green angi-
ography, diagnosis of Bietti’s crystalline retin-
opathy in advanced cases without fundus-
oscopically apparent crystalline deposits have
been established only when family history
was contributory. Because the presence of
degenerative myopia in advanced cases
suggests an autosomal recessive inheritance,
similar with some reported cases with Bietti’s
crystalline retinopathy with degenerative myopia has not been reported in
the literature. However, the presence of
components in retinal degeneration and the\n
Figure 1 Ocular fundus of case 1. Fundus
photograph [A], grey scale image of the
same fundus photograph [B] and SLO image
with infrared light [C] are shown. Note that
the crystalline deposits are most evident with
the infrared imaging.

Figure 2 Ocular fundus of case 2. Fundus
photograph [A], grey scale image of the same
fundus photograph [B] and SLO image with infrared light [C]. Magnified image of the boxed
area is shown in (D). Note that numerous crystalline deposits are observed with the SLO imaging.

Tonic pupils from giant cell arteritis
The tonic pupil is characterised by poor
reactivity to light, a slow tonic constriction
and redilation to a near target, and supersen-
sitivity to topical dilute pilocarpine.1 Most
instances do not have an identifiable cause;
however, a variety of conditions including
herpes zoster, orbital trauma including sur-
gery, and paraneoplastic syndromes may
result in tonic pupils. The mechanism(s)
which produce tonic pupils are not known,
although axonal loss within the ciliary gan-
nlion is generally believed to result in super-
sensitivity of the iris sphincter muscle. Giant
cell arteritis (GCA) has been reported as an
uncommon cause of tonic pupils.2 Further-
more, the cause of tonic pupils in GCA is
unknown and direct evidence for an ischae-
mic cause is not convincing.
We used orbital colour Doppler imaging to
study orbital and ocular blood flow in a
patient with GCA, unilateral visual loss, and
bilateral tonic pupils.

Case report
A 58 year old woman presented with visual
loss of the left eye for 2 days. She had had
pupils constricted slowly and tonicly to a pupil no longer reacted to direct light. Both unchanged 12 days later, except that her right eye and neuro-ophthalmic examination was topical 0.125% pilocarpine (Fig 2C).

Five earlier reports have documented tonic pupils in patients with GCA. Davis and coworkers described a 69 year old woman who developed polymyalgia and anisocoria. The involved pupil constricted to 2.5% methacholine. In another report of tonic pupils from GCA mild supersensitivity to 0.1% pilocarpine was noted; however, the patient had counting fingers vision in each eye from bilateral optic neuropathy, suggesting that the light near dissociation may have been caused by bilateral afferent dysfunction. The authors cited pathological studies which have suggested ischaemia of the extraocular muscles as a cause of ophthalmoplegia in some patients with GCA, and they implied that ischaemia was the cause of tonic pupils in their patient, although the site of damage was not specified. Currie and Lessell reported a 63 year old woman who had jaw claudication and loss of neck discomfort and intermittent jaw claudication over the previous 2 weeks. She had no past medical or ocular problems.

Visual acuity was 20/20 in the right eye and no light perception in the left eye. The right pupil reacted briskly to light, with a normal consensual response, the left was amaurotic. She identified all of the Ishihara pseudoisochromatic colour plates with the right eye, and automated perimetry of the right eye was normal. Extraocular motility was normal. Funduscopy of the right eye (Fig 1A) was normal, and the left optic disc was pale and swollen, with retinal cotton wool infarcts within the retina, chiefly within the left macula (Fig 1B). The superficial temporal arteries were supple, pulsatile, and non-tender. The Westergren erythrocyte sedimentation rate (ESR) was 144 mm in the first hour.

She was hospitalised and received intravenous methylprednisolone 250 mg every 6 hours. Temporal artery biopsy was positive for GCA, including the presence of numerous giant cells. After 12 doses of methylprednisolone she was discharged on 80 mg of oral prednisone per day.

Six days later her visual acuity remained 20/20, and with a +2.00 dioptre add over a plano lens she read J1 print at 13 inches with the right eye. Automated perimeter and funduscopy of the right eye remained normal. However, the right pupil now reacted sluggishly to light (Fig 2A) while the left remained amaurotic. In addition, there was light near dissociation in both eyes, with bilateral tonic constriction to a near target followed by slow redilation. Slit lamp examination on the right revealed an irregular pupil (Fig 2B) with sectoral iridoplegia. Both pupils constricted following the instillation of topical 0.125% pilocarpine (Fig 2C).

She remained asymptomatic in her right eye and neuro-ophthalmic examination was unchanged 12 days later, except that her right pupil no longer reacted to direct light. Both pupils constricted slowly and tonically to a near target. Orbital colour Doppler imaging revealed no blood flow in the left central retinal artery (CRA), and blood flow in the right CRA was attenuated at 4.7 cm/s (normal 10 cm/s). There was no flow detected in the short posterior ciliary arteries of the left eye, and attenuated flow in the short posterior ciliary arteries on the right. Although her ESR fell to 4 mm in the first hour and her jaw claudication resolved, the prednisone dose was maintained because of the attenuated blood flow within the right orbit. Her vision remained unchanged and her pupils remained non-reactive to light but with denervation supersensitivity, confirmed by constriction to 0.125% pilocarpine, 2 months after the onset of visual loss.

**Comment**

Two other authors have suggested that ischaemic damage may not be the sole cause of tonic pupils in patients with GCA. Bilateral tonic pupils with conjunctival injection were the only ocular manifestations in a 60 year old woman with biopsy proved GCA. Because there was no other clinical evidence of orbital or ocular ischaemia the authors postulated that damage to the ciliary ganglion may be mediated by an immunological mechanism. However, they did not quantify the orbital or ocular blood flow, and their patient shows that clinical signs of ischaemia, except for tonic pupils, may be absent despite markedly decreased orbital blood flow.

Wilhelm reported five patients with bilateral visual loss from anterior ischaemic optic neuropathy or central retinal artery occlusion and tonic pupils from GCA. Assessment of orbital blood flow was performed using ultrasonography in three of these patients. In two patients no flow was noted in the suprachoroidal arteries, while blood flow in the third was normal. The author noted that the ultrasonographic results supported ischaemia as a cause for the tonic pupils. In the patient with normal orbital blood flow, ischaemia below the resolution of ultrasonography was presumed to cause the tonic pupils.

The blood supply to the ciliary ganglion arises from several sources. Eliskova studied 18 human cadaveric orbits after injection of dye into the internal carotid artery. The ciliary ganglion was supplied with blood from one to four arteries, with the posterior lateral ciliary artery and the lateral muscular artery the most common sources, followed by the ophthalmic and central retinal artery. The vasculature of the ganglion itself is composed of a network of capillaries.

Orbital colour Doppler imaging is a non-invasive way to quantitate blood flow to the orbit and eye. Decreased blood flow in the ophthalmic artery, central retinal artery, and short posterior ciliary arteries may be detected on orbital colour Doppler imaging in patients with GCA. Markedly reduced blood flow was found with orbital colour Doppler imaging in both orbits in our patient, despite her visual loss being unilateral. We were unable to find a previous report of bilateral tonic pupils in a patient with unilateral visual loss from GCA. Although anterior segment ischaemia can cause loss of iris sphincter tone, we do not believe that the pupillary findings in our patient resulted from iris ischaemia. Iris ischaemia would result in poorly reactive pupils to both light and accommodation, without a tonic near reaction or denervation hypersensitivity. Therefore, the findings in our patient strongly suggest that the tonic pupils resulted from ischaemic damage to the ciliary ganglion or the postganglionic ciliary nerves which are responsible for pupillary constriction.

The authors do not have any proprietary interests in any of the contents of this manuscript.

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![Figure 1](http://bjo.bmj.com/)

![Figure 2](http://bjo.bmj.com/)
Frozen cucumber as a mount for vitreoretinal specimens

Vitreoretinal specimens are extremely difficult to process as a frozen specimen because of their small size and tendency to roll up. However, in order to perform immunohistochemical studies it is sometimes necessary to have frozen specimens as antigens may be destroyed if a fixative agent is used.

Dua et al. and Scott et al. suggested the use of frozen cucumber as a mount for conjunctival and corneal tissue. We modified this method for vitreoretinal specimens. We describe our technique and provide examples of its results.

Case report

Fresh cucumber (obtained from a greengrocer) was cut into small (1 cm) blocks; the part devoid of seeds was used. We found that with cucumbers older than 2 days the membranes did not adhere sufficiently well. These blocks were then stored at 4°C until required. Pig eyes were obtained and stored at 4°C until required. Subsequently, the eye was placed under the dissecting microscope and basic salt solution injected in through the vitreous cavity to enable easier dissection. The cornea was removed and a vitrectomy performed. The retinal specimens were stained with Indian ink in order to facilitate subsequent localisation.

Following vitrectomy, membranes were removed from humans (these included diabetic membranes, subretinal neovascular membranes, and epiretinal membranes); they were initially placed in Hartmann’s solution. Subsequently, they were placed on the cucumber under a dissecting microscope; it was possible to place the membrane flat without it rolling up because of the texture of the cucumber. These membranes were also stained with Indian ink before placing on the cucumber. The cucumber with the membrane on its “side” surface was placed in an aluminium foil cup and covered with a cryomatrix of Tissue-Tek OCT compound (Fig 1). The foil cup was then put in a plastic container and the contents flash frozen in liquid nitrogen.

The membranes were cut with a cryostat in 4–5 µm sections. We stained one slide from each specimen with haematoxylin and eosin and performed immunohistochemistry on the others (Figs 2 and 3). No specimens were lost while performing this technique of processing specimens.

We were able to maintain the orientation of the specimens and managed to obtain sufficiently satisfactory specimens to perform immunohistochemical studies. The use of Indian ink allowed us to locate the specimen easily when cutting sections. Unlike previous studies we found that the specimen attached to the cucumber without the use of albumin.

Comment

Swan and Davis first described the biopsy cucumber unit for processing cervical specimens. Frozen cucumber has been described as a mount for bladder and lung biopsy specimens. Ocular tissues that have been processed in this fashion are conjunctiva and cornea. Whittle et al. described a technique using cucumber as a mount for processing cadaveric human retina, which enabled indirect immunofluorescence studies.

Retinal specimens are difficult to process because of their size, tendency to roll up and, hence, difficult orientation. Nevertheless, it is necessary to process specimens in this way to perform certain immunohistochemistry techniques.

We suggest that cucumber is a suitable mount for vitreoretinal membranes that are required as frozen specimens for immunohistochemistry. It should be noted that most modern immunohistochemistry may be performed on fixed tissue.

References

Case reports

Case 1

A 20 year old white male, with no history of herpetic virus infection, presented with a 10 day history of progressive blurred right vision with floaters, significant pericentral discomfort, and pain on ocular motility. Acuity was 20/200 right eye, 20/20 left eye, with a right relative afferent pupillary defect (RAPD). There was swelling of the right optic nerve (Fig 1A) together with increasing non-reno-virous and ischaemia; seven days later (Fig 1C and D), several enlarging foci of retinitis, typical of ARN, developed in the retinal mid-periphery. PCR of a vitreous biopsy amplified HSV-1. There was no further reduction in visual acuity following intravitreal foscarnet and encephalic doses (10 mg/kg three times daily) of intravenous aciclovir for 10 days.

Case 2

A 47 year old healthy white female, with no history of prior herpetic virus infection, presented with a 3 day history of progressive blurred left vision. She also complained of significant pericentral discomfort exacerbated by eye movement. Acuity was 20/40 left eye, 20/15 right eye, with a left RAPD. There was swelling of the left optic nerve head together with a moderate non-granulomatous panuveitis. The right eye was unaffected. Fluorescein angiography showed marked optic disc hyperfluorescence and peripheral retinal ischaemia. Six days later, several enlarging foci of retinitis, typical of ARN, developed in the inferior retinal periphery. Polymerase chain reaction (PCR) of a vitreous biopsy amplified HSV-1. There was no further reduction in visual acuity following encephalic doses (10 mg/kg three times daily) of intravenous aciclovir for 10 days.

Case 3


References

Cutaneous angiosarcoma of the eyelids

Angiosarcoma of the eyelid is a rare potentially life-threatening tumour arising from the vascular endothelium. Through the presentation of a case, the clinicopathological features and management of this condition are described.

Case report

A 69-year-old white man presented with multifocal red-blue maculopapular lesions located in his right cheek and eyelids (Fig 1A). The patient had first noticed the lesions 9 months previously. An incisonal biopsy from the upper eyelid was performed. In subsequent staging investigations no metastases were found. The patient was otherwise in good health and was HIV negative. He underwent surgical resection of all clinically detectable lesions followed by adjuvant radiotherapy (tumour dose 45 Gy). After 34 months of follow up there was no evidence of local recurrence or distant metastases.

Histopathologically, the specimens consisted of skin and subcutaneous tissue. The lesions proved to be well differentiated angiosarcoma characterised by capillary vessels lined by plump endothelial cells with hyperchromatic nuclei. Using Ki-67, the proliferation rate was assessed as ≤5%–7% (Fig 1B).

Immunohistochemically, the tumour cells showed a positive reaction for vimentin, CD34 (human haemopoietic progenitor cell antigen) and FVIII-RA. They were consistently negative for S100 and HMB45 (results not shown).

Comment

Cutaneous angiosarcoma is a distinct subgroup of angiosarcomas most commonly seen in the skin and superficial soft tissues in patients over 55 years. The majority of these tumours arise in the head and neck area, particularly the scalp. They are aggressive and tend to recur locally and disseminate widely with a 5-year survival of approximately 12%–29%. To our knowledge, there are only seven well-documented cases involving the eyelid. Clinically, the lesions in our patient resembled those previously reported in view of the variant papillary projections into the cutaneous papillary projections into the cutaneous lamellae. In areas, malignant endothelial cells pile up producing typical papillary projections into the lumina (short arrow) (PAS >200).

Histologically, angiosarcomas display a spectrum from well to poorly differentiated varieties. The present case was consistent with a well differentiated angiosarcoma with characteristic interfacing channels lined by atypical endothelial cells and expression of immunohistochemical markers of vascular differentiation (CD34 and FVIII-RA). Often tumours display both vasomorphic and undifferentiated areas. The present case was noteworthy, in that the entire tumour appeared to be well differentiated. The proliferation rate was low to moderate compared to the reported rate for angiosarcoma (>10% in 72% of cases assessed by Ki-67). Miotic index has been observed as an independent histological prognostic marker for cutaneous angiосарcoma although other histological markers remain poorly defined. In the present case, low proliferation index and well differentiated histological features may have contributed to a better outcome.

The most important clinical prognostic factor is lesion size, with tumours <5 cm in diameter having a better prognosis. This underlies the importance of early diagnosis and appropriate management. The mainstay of treatment for cutaneous angiosarcoma includes surgery and/or radiotherapy although, due to the rarity of this condition, there are no standard guidelines. As illustrated by our case, the primary lesions were contained within the anterior lamellae of the lids and it was possible to surgically encompass the primary lesions avoiding exenteration and without compromising prognosis. In such cases, the surgical aim is to respect all clinically identifiable disease. In areas of doubt, microscopic control of surgical margins may have a role in guiding the extent of resection. Angiosarcomas usually respond to radiotherapy to some degree and most authors suggest that combined surgery and radiotherapy offers the best chance for long term control. In one series of 28 head and neck angiosarcomas, Meis et al have reported better survival after 32 months (median) with combined surgery and radiotherapy compared with surgery alone. In cases unsuited to surgery, radiotherapy alone may be considered, however usually only partial responses are achieved. The role of chemotherapy remains undefined.

In summary, cutaneous angiosarcoma should be included in the differential diagnosis of vascular or atypical eyelid lesions as early recognition is critical to optimise outcome. Classification of the mitotic index and degree of histopathological differentiation may be useful criteria in predicting the biological behaviour. Treatment needs to be individualised to the patient and in selected cases complete excision of clinically evident disease may be appropriate. Adjunctive radiotherapy is recommended due to the aggressive natural history of angiosarcoma.

Acknowledgements

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References

Permanent visual loss in a child with a rash

Endophthalmitis is an unusual but well described complication of bacteraemia. Here we report endophthalmitis arising in the course of a mild illness resembling chickenpox.

Case report

A previously healthy 6 year old white girl awoke in the early hours feeling hot and complaining of headache. She was given ibuprofen and later said she felt warm, and vomited. Next morning she complained of right eye and later said she felt warm, and vomited. Examination under aseptic conditions revealed a rolled up, red eye and a large hypopyon (Fig 1). Erythematous maculopapular lesions were present on her arms, legs, and face.

The patient was apyrexial and remained clinically well during her hospital admission. At discharge after further days of parenteral antibiotics (ceftriaxone) the rash had completely resolved. In the absence of a positive culture sensitivities could not be confirmed, although all the stated antibiotics except vancomycin are generally effective against meningococcus. At follow up the eye had become phthisical, with visual acuity reduced to perception of light only.

Comment

In this case meningococcal endophthalmitis, usually seen in association with clinically apparent septicaemia and meningitis, occurred in the setting of a mild illness thought to be chickenpox. Although the usual hallmark of meningococcal sepsis were absent, evidence for seeding of the eye in the course of a transient meningococcalae was provided by Gram negative intracellular diplococci in the aqueous humour and detection of meningococcal DNA in both the aqueous humour and cutaneous material. Reports of meningococcal endophthalmitis in clinically well, non-bacteraemic patients are rare, with just four published cases in English. The patients were 13 months,

References


Those excrescences on Descemet’s membrane

We read with interest the recent report, “Screening human donor corneas during the organ culture for the presence of guttata.”

Even the title made us question whether we were the only ones still using the original name for the condition cornea guttata. A quick perusal of Medline reveals a trend away from guttata, and towards guttata (Fig 1). This trend may be even more pronounced, as the literature tends to be conservative. The next generation of cornea specialists, or at least the small sample that goes through our training programme, tends to fall into three categories:

Those who say guttata, those who correct others for saying guttata, and those who try their best not to mention the term for fear they’re saying the wrong thing. Many of our faculty have now entered this last group.

When asked what is so bad about guttata, some insist that it is not the plural for gutta, the Latin word for drop. We’re not sure that anyone ever said it was. More erudite dissenters recognise that not only is guttata not a plural, it is not even a noun—it’s an adjective.

The closest English counterpart to guttata is probably guttate, drop-like, or speckled, so it’s incorrect to use guttata alone as a noun, or so the thinking goes. Some have chosen to use cornea guttata when referring to the condition, and guttae when referring to the excrescences themselves.
"Guttae were present" may well have grabbed the ears of an ancient Roman, since guttae is feminine singular, and Romans liked their subjects to agree with their verbs in number as much as the rest of us. However, it is not so clear that "Flavia's guttata is impressive" would be at all offensive, particularly if criticised. In fact, it may be a reason for the learned still do, at least when they refer to themselves as the learned. Those particularly offended by using an adjective substantively (as a noun) should be careful not to slip up with words like cornea. Both cornea and conjunctiva started out their ophthalmological careers as adjectives, modifying tunica (coat, feminine singular), which is why cornea and, in turn, guttata are feminine singular in the first place.

What then should we call those excrescences on Descemet’s membrane? Well, if you thought they are drops, then by all means call them drops, although you probably don’t need to invent yet another term from a dead language. Vogt called them “tropfige” (drop-like) at school of Public Health), Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, NT, "causation" within their study. The authors do not have definitive evidence to support their cause of sampling bias, although the severity or asymptomatic, and cases in which the presence of disease alters or entirely removes the exposure, are missed.

Water "hardness" remains unproved as a risk factor for acanthamoeba keratitis being no more than an observational quirk. While we have had good molecular based evidence for the association of acanthamoeba keratitis with home tap water," recent work using 18S rDNA typing and tracking has shown that there can be different subtypes of keratitis causing strains of Acanthamoeba spp (T4) between the tap water in the home and the cornea, making the epidemiology more complicated.

We believe that the suggested correlation by the authors of acanthamoeba keratitis with water hardness is spurious and demonstrates the danger of projecting “correlation” to “causation” within their study. The authors do not have definitive evidence to support their cause of sampling bias, although the severity or asymptomatic, and cases in which the presence of disease alters or entirely removes the exposure, are missed.

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Surgery for glaucoma

We would like to thank Drs Mermoud and Ravinet for their online comments on the editorial and also for their comments on O’Brart’s article (letter posted on www.bpjophthalmol.com 5 Aug 2002). We are delighted that there is agreement that subconjunctival drainage is an important component of non-penetrating filtering surgery. Blebs, albeit diffuse, are clearly visible in all cases of deep sclerectomy, and in many cases of viscosocanalostomy. However, although Ravinet and Mermoud and ourselves are certain that this is an important component for drainage in non-penetrating filtration surgery, it is important to clarify this point. Although we are certain of the importance, there is still controversy over the relative importance of subconjunctival drainage versus other routes such as trans-scleral or via Schlemm’s canal in the case of viscosocanalostomy.

On the second point, although Mermoud and Ravinet use antimetabolites before creation of the deep sclerectomy flap, we do not use antimetabolites in the deep scleral dissection. We believe these drugs should be withheld until after the macroperforation, clearly the team is laudable but incomplete. Ethical considerations of informed consent require full disclosure of all aspects of the proposed treatment. The following points might be considered for inclusion.

(1) Occlusion therapy has never been scientifically validated with a randomised, controlled study.

(2) The dose/response relationship has never been defined. Flynn et al stated that “Success was not related to the duration of occlusion therapy, type of occlusion used ...”. The variability of treatment protocols accentuates another dilemma “owing to our paucity of knowledge on the dose-effect relation—a situation one finds hard to imagine for any comparably established therapy outside ophthalmology. In other words we have no understanding of the dose-effect relation of occlusion in amblyopia therapy.”

(3) The application of “greater levels of occlusion being prescribed for more severe amblyopia” is compromised by the observation “that success was related to ... the depth of visual loss before treatment ...”

(4) The benefits of treatment are likely to deteriorate following cessation of patching.

(5) Visual acuity improves as children grow older and from school age to adulthood. This may well be exacerbated by inaccurate occlusion therapy.

(6) Both the occluded and the amblyopic eyes improve at the same rate during treatment.

In conclusion, we are pleased that the article and editorial have led to further healthy discussion. Mermoud and his colleagues are to be commended for the work they continue to do in non-penetrating filtration surgery. However, as we said in our editorial, current studies do reveal that none of the current operations for glaucoma are totally ideal yet, and further research, particularly on surgical methods and wound healing control, is needed so that optimal long-term pressure control can be achieved for all our patients with a minimum of complications.

References


Ocular and financial health

We read with interest the article by Khaneke A et al detailing the results of the 1996–7 Oman Eye Study: 1 They found a prevalence of blindness of 16.8% in those 60+ years of age, and comment that the population of this age group in Oman is predicted to double by the year 2020. This has serious implications for planning the provision of health care and specifically for eye care services.

This observation is true for most countries; the global population for the over 65 age group is projected to increase from approximately 400 million to 800 million people by the year 2020, contrasting with the under 5 years of age population, which is estimated to see a 6% growth in the same period.

“Vision 2020—the right to sight” was launched by the WHO and JABP in 1999, aiming for the elimination of avoidable blindness by the year 2020. In 1995 the estimate of global blindness (<3/60 better eye) was 44 million, and this is projected to rise to 76 million by 2020 if there is no change in current trends. 3 Vision 2020 prioritises five diseases for global attention—cataract, refractive errors, trachoma, onchocerciasis, and vitamin A deficiency. Action against diabetic retinopathy and glaucoma is also deemed important in countries where ocular infections have been controlled. Vision 2020 contends that the current increase in blindness, estimated at 1–2 million people per year, can be reversed if human and financial resources are targeted at these priority diseases in the countries with the highest prevalence and number of blind people. It is estimated that the result of a successful programme, achievable at a cost of $2 billion, will be 429 million blind person years avoided over the next 20 years, and a total saving in excess of $100 billion, by avoidance of lost productivity. 4 This would make the effective implementation Vision 2020 not only ethically important but also a cost effective strategy for poverty alleviation.

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References
2 www.census.gov/ipc/prod/wg98/wg98.pdf
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 secentl@secentl.org; web site: www.secentl.org).

16th Annual Meeting of German Ophthalmic Surgeons
The 16th Annual Meeting of German Ophthalmic Surgeons will be held 8–11 May 2003 in Nürnberg, Germany, Messezentrum. Organised by the Professional Association of German Ophthalmologists Ophthalmic Surgeon Group the conference will cover cataract surgery, refractive surgery, glaucoma surgery, vitrectoretinal surgery, corneal surgery, eye surgery in developing countries, and orbita, lacrimal and lid surgery. Further details: MCN Medizinische Congress organisation Nürnberg AG, Zerzabelshofstr 29, 90478 Nürnberg, Germany (tel: +49 911 3931621; fax: +49 911 3931620; email: doc@mcnag.info; web site: www.doc-nuernberg.de).

3rd British Oculoplastic Surgery Society Meeting
The 3rd British Oculoplastic Surgery Society Meeting will be held 18–19 May 2003 in Birmingham, UK. For further details please contact the Secretary of the British Oculoplastic Surgery Society Jane Olver (tel: +44 (0)121 424 5464; fax: +44 (0)121 424 4464; email: Martii@ihcaevnids.nlhs; web site: www.bopss.org).

13th Meeting of the EASD Eye Complication Study Group
The 13th Meeting of the EASD Eye Complica- tion Study Group will be held on the 23–25 May 2003, in Prague, Czech Republic. The scientifc programme includes keynote lectures from Professor John H Fuller (UK) on The epidemiology of diabetic retinopathy; Dr P Martin van Hagen (The Netherlands) on Growth factors and diabetic retinopathy; Professor Tereza Pelikanova (Czech Republic) on Pathophysiology of diabetic microvascular complications; Dr Tomas Sosna (Czech Republic) on Risk and protective factors of diabetic retinopathy.