Intravitreal injection of triamcinolone acetonide as treatment for chronic uveitis

Chronic intraocular inflammation such as chronic idiopathic uveitis can lead to cystoid macular oedema, papilloedema, and vitreous opacities temporarily or permanently reducing visual acuity. Chronic uveitis has usually been treated by topical or systemic application of steroids. Topical treatment, however, often has not been sufficiently effective to suppress intraocular inflammation and to reduce cystoid macular oedema. Systematic treatment with steroids inevitably leads to secondary side effects, and local or systemic suppression of the whole immune system and Cushing's syndrome. Taking into account that the eye comprises only 0.01% of the whole body volume, and considering that for achieving high concentrations of a drug at its site of action it is best to apply it directly into the region of required action, we describe the clinical outcome in a patient receiving an intravitreal injection of a crystalline corticosteroid.

Case report

A 17 year old woman suffering from chronic idiopathic uveitis in both eyes for 5 years had been treated topically, peribulbarly, and systematically with corticosteroids. As a steroid responder, she had developed secondary ocular hypertension. Steroid induced cataract in her right eye was operated on by phaco aspiration, transpupillary anterior vitrectomy, and posterior chamber lens implantation. To reduce the systemic side effects of steroid treatment, systemic cyclosporin A had been added to the treatment scheme since January 1998. In February 2000, she presented again with a severe uveitis with papilloedema and cystoid macular oedema. Despite intensive topical treatment with steroids given hourly, and systemic acetazolamide, visual acuity remained in the range 0.10–0.16. To avoid the side effects of systemic steroid treatment and to achieve high and longstanding concentrations of steroids in the eye, we injected 20 mg crystalline triamcinolone acetonide into the vitreous cavity of the right eye in July 2000 with topical anaesthesia.

Within the next 5 weeks, visual acuity increased to 0.5. Intraocular pressure increased to a maximum of 38 mm Hg, and was reduced to the normal range with topical antiglaucomatous medication. Four months after the injection, the steroid crystals were resorbed, visual acuity returned to the preoperative level of 0.1, and with topical steroids given, intraocular pressure decreased to values of less than 23 mm Hg without further antiglaucomatous medication.

Comment

In ophthalmology, corticosteroids applied topically or systemically are well known and have widely been used to suppress intraocular inflammation. Based on experimental studies performed by Macherer, Peyman and others, as well as on clinical observations, intravitreal injections of triamcinolone acetonide have increasingly been reported as treatment for intraocular neovascular, oedematous, or inflammatory diseases. These include diffuse diabetic macular oedema, proliferative diabetic retinopathy, neovascular glaucoma, exudative age related macular degeneration, and uveitis. In agreement with these previous studies, the results of the present report suggest that the intravitreal injection of triamcinolone acetonide may be an additional option in the treatment of chronic uveitis. Future studies may also consider different types of uveitic intraocular steroid injection are best for, and whether the use of intravitreally implanted slow release devices can decrease the recurrence rate of uveitis for a longer period than a single intravitreal injection dose.

Proprietary interest: none.

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Ophthalmodynamometric estimation of cerebrospinal fluid pressure in pseudotumour cerebri

Measurement of the cerebrospinal fluid pressure usually requires a lumbar puncture or craniotomy to get direct access to the cerebrospinal fluid space. These techniques, however, are invasive and so carry the risk of complications such as infections and damage to the neural structures. Furthermore, owing to the leakage of cerebrospinal fluid during the puncture, the cerebrospinal fluid pressure will be altered in the moment the measurement is performed. It would be desirable to have a non-invasive method allowing the estimation of the intracerebral pressure without requiring a direct access to the brain or spinal cord. We describe a patient in whom ophthalmodynamometry strongly suggested an increased intracerebral pressure which was confirmed by eventual direct measurement.

Case report

A 12 year old female patient presented with acute vomiting, massive headache, and bilateral abducens nerve palsy. Visual acuity was 20/20 in both eyes, and visual fields were unremarkable, except for an enlarged blind spot. Both optic discs showed a prominence of 5 mm (right eye) and 0.6 mm (left eye) as measured by confocal laser scanning tomography. Intraocular pressure measured 18 mm Hg. With topical anaesthesia, a Goldmann contact lens fitted with a pressure sensor mounted into its holding ring was put onto the cornea (Fig 1). Pressure was asserted onto the globe by slightly pressing the contact lens, and the pressure value at the time when the central retinal vein started pulsating was noted. The measurements of this new technique of ophthalmodynamometry were repeated nine times in both eyes.

The central retinal vein collapse pressure as the sum of the ophthalmodynamometric value plus the intraocular pressure, measured 103 relative units right eye and 98 relative units left eye. These values were significantly higher than normal values (6.1 (SD 8.4) relative units) determined previously in normal subjects (own data). Direct measurement of cerebrospinal fluid pressure by lumbar puncture performed about 5 hours later revealed a value of 107 cm water column (equivalent to 82.5 mm Hg). In combination with other clinical findings, the diagnosis of pseudotumour cerebri was made.

Comment

The central retinal vein is the only structure whose appearance depends on its inner pressure, and which runs through the cerebrospinal fluid space and which is accessible from outside the body without any invasive procedure being performed. After exit from the eye through the optic disc, the central retinal vein goes through the retrobulbar part of the optic nerve before it traverses the subarachnoidal and subdural spaces of the optic nerve and pia mater of the optic nerve meninges. The pressure in the central retinal vein is thus at least as high as the cerebrospinal fluid pressure. The central retinal vein collapse pressure may be measurable by ophthalmodynamometry since the vein will start to pulsate, if the sum

Figure 1 Photograph showing the Goldmann contact lens with a pressure sensor mounted into the holding ring of the contact lens and connected to a display.
of intraocular pressure plus an external pressure exerted onto the eye equals the diastolic pressure of the central retinal vein: \( p_{\text{ICP}} = p_{\text{IOP}} + p_{\text{EC}} \). The intraocular pressure can be determined by application tonometry, and the additional pressure exerted onto the globe can be measured by the ophthalmodynamometer. In the ophthalmodynamometers used in the 1960s and 1970s, determinations of the central retinal vein pressure were often difficult or almost impossible so that the central retinal vein pressure has usually not been measured.3 The new ophthalmodynamometer used in the present study (Fig 1) may overcome some of the problems associated with the old ophthalmodynamometers. In a previous study on the reproducibility of the new technique, the variation of the central retinal vein collapse pressure was 15.9% (SD 11.9%). The present study suggests that, in patients with markedly increased intracerebral pressure, the new, Goldmann lens associated, ophthalmodynamometer may provide information about the intracerebral pressure by estimating the central retinal vein collapse pressure. It may be helpful for the neuroophthalmological diagnosis of diseases associated with increased intracerebral pressure.

Proprietary interest: none.

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References

Treatment of atopic blepharitis by controlling eyelid skin water retention ability with ceramide gel application

Atopic blepharitis is one of the major ocular complications of atopic dermatitis (AD).1,2 It has been pointed out that atopic patients have dry skin accompanied by barrier disruption and water deficiency. Previously, we assessed the water retention ability of eyelid skin by measuring the water content and water evaporation rate from the eyelid in patients with atopic blepharitis. The water content positively correlated and water evaporation from the eyelid negatively correlated with the severity of blepharitis.3

Ceramide comprises about 30% of stratum corneum lipids, which have an important role in both the water retention and barrier function of the skin.4 Ceramide abnormalities in several skin disorders, such as AD, have been reported.5 Decreased levels of ceramides may be attributable to the insufficient water retention of the skin in AD. Appt Gel (Zenyaku Kogyo, Tokyo, Japan) is a product containing galactosyl ceramides extracted from horses as a major moisturising ingredient. In this study, we assessed the efficacy and safety of this gel product in patients with mild atopic blepharitis by measuring the water retention ability of the eyelid skin before and after treatment.

Methods and results

Sixteen lids of eight patients (five males and seven females, 7–35 years old, average age 16.0 (SEM 8.4) years) diagnosed as having AD by dermatologists, according to Hanifin and Rajka’s criteria,6 were examined. Because ceramide gel has no anti-inflammatory effect, cases with severe inflammation were excluded from this investigation. After informed consent was obtained, patients were instructed to place Appt Gel on their eyelids two to five times a day after washing their faces. Assessment of clinical findings using measurement of water retention ability were performed as previously described before and 4 weeks after the beginning of application. Statistical analysis was carried out by non-parametric tests (Wilcoxon test). A p value of 0.05 or less was considered statistically significant.

Water content of eyelid skin was significantly increased after treatment (30.6% (6.0%) before treatment, 41.2% (8.5%) after treatment; \( p<0.05 \)) (Fig 1). No slit lamp findings indicating toxicity were observed during the course of the study.

Comment

As the eyelid is a borderline lesion between dermatology and ophthalmology with influences on ocular homeostasis, dermatologists often hesitate in prescribing sufficient medication to the eyelids. The assessment and treatment of atopic blepharitis is therefore an important aspect of ophthalmological examination in atopic patients.

Ceramide gel treatment for 4 weeks significantly improved the water retention ability of eyelid skin of patients with mild atopic blepharitis. Among various moisturising products, the application of the ceramide gel is reasonable, because ceramide deficiency has been reported in the skin of atopic patients. Ceramide gel alleviates dryness without stickiness, and patients experience little discomfort. Comfort during application is thought to be one of the important factors for the compliance of patients. Some patients interrupt application of ointments, such as petrolatum (Vaseline), to the eyelids because of stickiness or because the shiny appearance around the eyes is cosmetically conspicuous. Although strong anti-inflammatory drugs are necessary in acute exacerbations of atopic blepharitis, moisturising of the skin using ceramide gel application represents a useful supplementary therapy during periods of relatively light inflammation.

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An unusual tumour of the lacrimal gland

Lacrimal gland swelling is usually due to an inflammatory or neoplastic process. We report an oncocytoma as the cause of lacrimal gland swelling, and review the literature. Oncocytoma of the lacrimal gland is extremely rare and has been described only three times before.

Case report

A 72 year old man experienced periodic swelling of his right eyelid over a period of 9 months. For 2 months he complained of vertical diplopia. He had a past medical history of chronic obstructive airways disease and a past ocular history of early cataract, asteroid hyalosis of the vitreous body, and primary open angle glaucoma.

On examination visual acuity of both eyes was 0.50. External examination showed a right hypopyon. There was a 3 mm of proptosis of the right eye. Eye movements in that eye were restricted in all directions. Slit lamp evaluation showed bilateral mild cata-

A 58 year old man reported by Biggs had a 6 month history of postproptosis of his eye and diplopia. On presenta-

Despite radical resection and postoperative radiation therapy, he died 6 months later from liver metastases. Dorello et al described a similar case of a 59 year old patient with an oncocytic carcinoma of the lacrimal gland with intraocular extension. The patient died approximately 2 years after development of his clinical symptoms (exophthalmos and diplopia), despite orbital exenteration and radiation therapy. A 81 year old woman reported by Biggs had a 6 month history of proptosis due to an oncocytoid adenocarcinoma of the lacrimal gland. No follow up information was given.

In summary, a 72 year old man was found to have an oncocytoma of the lacrimal gland. Oncocytoma has to be added to the differential diagnosis of lacrimal gland swelling.

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References


Morning glory disc anomaly: an atypical case

When optic disc elevation is encountered on funduscopic examination, the first concern is whether it represents true disc oedema. We present a patient who was urgently referred to us by a retinal specialist because of unilateral “disc swelling.” Further evaluation suggested that the patient had a congenital optic disc anomaly that was atypical in appearance, but most likely a variant of the morning glory disc anomaly (MGDA).

Case report

A 40 year old African-American woman with no visual complaints went to her local optometrist for a routine eye examination. At the optometrist’s office she was told that “in the back of her right eye she had a dark spot with a ring around it” and was referred to a retinal specialist. The retinal specialist found a mildly decreased visual acuity and a “swollen disc” in the right eye and the patient was referred for neuro-ophthalmic consultation. To her knowledge, she previously had never had a dilated funduscopic examination.

Best corrected visual acuity was 20/30 distance and J1 near in the right eye and 20/20 distance and J1+ near in the left eye. There was a 0.9 log unit right relative afferent pupillary defect. Her colour vision, external
examination, slit lamp biomicroscopy, intraocular pressures, and motility were all normal in both eyes. Funduscopic examination was normal in the left eye with an optic nerve cup to disc ratio of 0.4. The appearance of her right optic nerve (Fig 1) was that of an elevated ring around the centre of the disc, interrupted from about 7 o'clock to 9 o'clock by an area of pigmentation. The vasculature was mildly obscured as it crossed the elevation. There was no venous engorgement, haemorrhage, cotton wool spots, or exudate. Funduscopic examination gave the appearance of a parapapillary excavation of retina and retinal pigment epithelium surrounding the optic disc. An elevated annulus of apparent fibroglial tissue surrounds most of the disc. Fibroglial tissue is not unexpected, as the visible peripapillary pigmentation in MGDA dissipates over time. This decrease in peripapillary pigment over time is believed to be secondary to a metaplasia of hamartomatous RPE into fibroglial tissue and hyperplasia of the fibroglial tissue.

While MGDA is usually diagnosed by funduscopic examination alone, our case was atypical and not diagnosed immediately for several reasons: the patient had good visual acuity in the affected eye; she was African-American; there was no central fibrous tuft; and there was only a mild amount of peripapillary pigmentation. It is likely that our patient's visual acuity was spared because of relative sparing of the papillomacular bundle.

The term “morning glory syndrome” was coined for its ophthalmoscopic resemblance to the morning glory flower. In MGDA the optic nerve lies centrally within an excavation of the posterior globe. The size of the excavation varies from being relatively small, as in this particular case, to cases in which the excavation encompasses the macula, termed macular capture. In most cases there is a central fibrous tuft that obscures the central 12 o'clock. The macula and periphery were normal. Automated perimeter showed an enlarged blind spot and a relative superior altitudinal defect on the right and a full field on the left. Fluorescein angiography showed staining of optic nerve tissue but no leakage of fluorescein outside the disc margin, confirming the absence of true disc swelling (Fig 2). Magnetic resonance imaging of the brain and orbits with gadolinium showed no pathology. B-scan ultrasonography excluded optic disc drusen. Optical coherence tomography (OCT) of the optic nerve showed the elevated annulus of tissue seen on fundus examination and also showed a parapapillary excavation of the retinal pigment epithelium (RPE) adjacent to the optic nerve (Fig 3). This was thought to be most consistent with MGDA.

Another atypical feature of our patient is the small amount of peripapillary pigment seen in the affected eye. The only area of pigmentation is between 7 and 9 o'clock. The remaining clock hours have elevated fibrovascular tissue but no visible pigment. This finding is not unexpected, as the visible peripapillary pigment in MGDA dissipates over time. This decrease in peripapillary pigment over time is believed to be secondary to a metaplasia of hamartomatous RPE into fibroglial tissue and hyperplasia of the fibroglial tissue. Our patient was 40 years old at diagnosis of MGDA and it is possible that she had more peripapillary pigment when she was younger, as the glial hyperplasia tends to progressively elevate the disc over time. This is a condition that is not typically an inherited condition or part of a multisystem genetic disorder, although it has been reported as part of the renal-coloboma syndrome and trisomy 4q. This condition is not typically an inherited condition or part of a multisystem genetic disorder, although it has been reported as part of the renal-coloboma syndrome and trisomy 4q.

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Idiopathic anterior hyaloid vessels
Anterior hyaloid fibrovascular proliferation (AHFP), the growth of vessels across the anterior hyaloid face from an origin in anterior retina,1 was first described in phakic eyes following diabetic vitrectomy surgery,2 but also reported following cataract surgery in diabetics.3 Complications include cataracts, vitreous haemorrhage, tractional retinal detachment, ciliary body detachment, and phthisis bulbi.4 We present the first report of this entity occurring in a non-diabetic patient without previous ophthalmic surgery.

Case report
A 20 year old Asian man presented with acute right sided visual loss. There was no history of ocular trauma or family history of note. He was systemically well. Visual acuity was 6/12 right eye, 6/9 left. Vessels were visible on the right posterior lens capsule, associated with localised cataract and anterior vitreous opacity (Fig 1A). Clinical examination revealed no other ocular abnormality, but the temporal peripheries of the right retina was obscured. Anterior segment fluorescein angiography confirmed perfusion of the vessels (Fig 1B). Posterior segment fluorescein angiography

Figure 1 Right eye. (A) Abnormal vessels on temporal aspect of posterior capsule associated with localised cataract and anterior vitreous opacity. (B) Anterior segment fluorescein angiography confirming perfusion of vessels.

Figure 2 B-scan ultrasonography with Doppler showing a feeder vessel.

was of poor quality in the right eye and revealed no fundus or peripheral abnormality in the left eye. Fluorescein angiography of the patient’s only surviving parent was normal. Fasting blood glucose, Tizocara/Ttoxoplasma serology, haemoglobin electrophoresis, and skull/chest radiographs were normal. Doppler studies indicated a probable feeder vessel derived from anterior retina but no evidence of tumour or other pathology (Fig 2).

Progressive cataract reduced visual acuity to 6/24 within 2 weeks of presentation. Phacoemulsification surgery was performed, followed by closure of the abnormal vessels with krypton laser (568 nm). Nd:YAG capsulotomy was performed 6 weeks after surgery. Two weeks later, fibrinous anterior uveitis and vitritis developed which resolved with topical steroid medication. A year later, vessels persisted in the rolled capsular edge, and opacification of the anterior hyaloid necessitated Nd:YAG laser discission. Two and a half years after presentation, he is asymptomatic with 6/9 visual acuity in the right eye.

Comment
In most instances of retinal neovascularisation, an angiogenic stimulus, such as capillary non-perfusion or inflammation, can be identified.1 In this case, no evidence was found of capillary non-perfusion, or any, other than postoperative, inflammation. A number of specific diagnoses were considered. No temporal traction on vascular arcades or optic disc was seen in the affected eye, and peripheral retinal vasculature was normal, where seen in the affected eye, and in the fellow eye. There was no history of low birth weight or prematurity as in retinopathy of prematurity. There was no family history, and the retinal peripheries of the parent were normal, which makes dominant exudative vitreoretinopathy unlikely. No inflammation, other than post-surgical, was identified in anterior or posterior segment, and visible pars plana appeared normal. Tizocara and Ttoxoplasma serology was negative. There was no visible peripheral retinal vasculitis or systemic evidence of sarcoidosis or multiple sclerosis. No evidence of ocular trauma was found, and no foreign body was identified radiographi-

cally. The patient is not diabetic, screening for haemoglobinopathy was negative, and there were no ophthalmoscopic or angiographic signs of retinal vascular occlusion. No evidence of a hyperviscosity syndrome was found.

The patient’s age, sex, and race are typical of patients suffering from idiopathic peripheral vaso-occlusive retinopathy (Eales’ disease), but the absence of retinal vasculitis, vitreous and retinal haemorrhage, and the unilateral nature of disease, are less characteristic. No sign of tumour was apparent on ultrasonogra-

py, and no evidence of arteriovenous malfor-
mation was found in the eye or body. He had

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no features of incontinentia pigmenti or facial-cranialomal muscular dystrophy. The diagnosis therefore remains uncertain.

Treatment was mandated on the basis of visual loss. Vitreoretinal opinion preferred phacoemulsification surgery to pars plana vitrectomy because of the absence of definable vitreous or peripheral retinal disease. The vessels were closed with laser before capsulotomy to avoid haemorrhage, as has been described after capsulotomy in diabetic patients with AHFP.

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References

Iris pigment epithelial cyst induced by topical administration of latanoprost

Latanoprost is an ester prodrug analogue of prostaglandin F₂α that enhances uveoscleral outflow and reduces intraocular pressure. Several adverse side effects associated with topical administration of latanoprost have been described. Iris cyst can be primary or secondary; the secondary iris cysts are usually caused by trauma, intraocular surgery, inflammation, and prolonged use of strong miotic agents, etc. We report one female patient, with advanced chronic angle closure glaucoma, who developed an iris cyst in her left eye 9 months after topical administration of latanoprost in both her eyes.

Case report
A 67 year old female patient initially presented with advanced chronic angle closure glaucoma in 1994. Laser iridotomy was performed on her both eyes in April 1994. After that, both eyes were treated with 2% pilocarpine and B blocker to maintain her intraocular pressures in the low teens. Because she preferred to use monotherapy, latanoprost had been used once a day at bedtime since July 2000. The intraocular pressures were maintained between 12 and 15 mm Hg with latanoprost monotherapy. No abnormal responses except mild hyperaemia of the conjunctiva were noticed during follow up examinations. Unfortunately, in May 2001 (about 9+ months after latanoprost monotherapy), it was noticed that the iris of her left eye bulged forward between 7 o’clock and 8 o’clock. The lesion was gradually increasing its size, and in September 2001 an iris pigment epithelial cyst was found at the posterior iris surface through a mid-dilated pupil (Fig 1). Latanoprost was then discontinued and her antiglaucomatous medication was changed to dorzolamide and β blocker twice a day in both eyes. The iris cyst gradually decreased in size and completely disappeared from the pupil margin in February 2002 (Fig 2). During the follow up period of 4 months, there have been no visual complications or signs of recurring cyst.

Comment
Our report demonstrates another case of rare adverse side effects of latanoprost involving the iris. Although no ultrasonic biomicroscopy was used to follow up this case, the slit lamp biomicroscopy strongly suggested that the patient had a secondary pigment epithelial cyst arising from the posterior surface of the iris. The iris cyst developed in her left eye about 9+ months after topical administration of latanoprost in her both eyes, and it progressively decreased in size and completely disappeared 5 months after topical latanoprost was discontinued. The iris cyst in our case took more time to develop and a longer time to disappear than previously reported. We propose that if it took more time to develop an iris cyst after topical administration of latanoprost, it would need more time for the iris cyst to regress.

The topical latanoprost was administered to her both eyes, but only her left eye developed the iris cyst. We propose that both her eyes might have different sensitivity to the development of an iris cyst when exposed to topical latanoprost. (If her right eye was exposed to latanoprost for a longer time, an iris cyst might occur later.) Although cysts of uveal tissue might occur under uveitis, no definite symptoms and signs of uveitis were noticed in our patient during the follow up period. The most likely cause of this adverse side effect may be the increasing uveoscleral outflow on topical use of latanoprost; increasing uveoscleral outflow leads to an enhanced aqueous flow through the ciliary muscle and the intraepithelial space of the posterior iris. The iris cyst can occur at anytime during topical administration of latanoprost. Ophthalmologists should be aware of this possible rare side effect of topical administration of latanoprost.

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Paravertebral primitive neuroectodermal tumour presenting with Horner’s syndrome

We describe a peripheral primitive neuroectodermal tumour (PPNET) arising from the cervical paravertebral region of a 34 year old woman, who presented with Horner’s syndrome, and a cervical radiolucent tumour. PPNETs are rare malignant small round cell tumours. This appears to be the first documented case of localised PPNET with Horner’s syndrome at initial presentation.

Case report
A 34 year old woman presented with acute left scalpula pain, numbness of her left forearm, a left upper lid ptosis, and left hemifacial anhydrosis. Her symptoms disappeared spontaneously within a fortnight, but returned 2 months later with greater intensity. Examination then revealed wasting of the small muscles of her left hand with reduced power in the distribution of C8 and T1; there was loss of light touch and pinprick in the C8 dermatome. The presence of left 1 mm upper lid ptosis, miosis, hemifacial anhydrosis, and 1 mm lower lid (“upside down”) ptosis was highly suggestive of a preganglionic left Horner’s syndrome (Fig 1A). Magnetic resonance imaging (MRI) of the neck showed a large mass arising from the T1, T2 intervertebral foram extending to the root of the left side of the neck and the region of the apex of the left lung (Fig 1B).

A diagnostic biopsy was performed through a posterolateral approach, excising the extradural component of the tumour within the
nerve root canal. Histology revealed a malignant round cell tumour consistent with a peripheral primitive neuroectodermal tumour (PPNET, Fig 2). Immunohistochemical studies demonstrated positivity for focal vimentin, cytokeratins, synaptophysin, and MIC-2, but were negative for GFAP (gliial fibrillary acidic protein), S100, desmin, and the lymphoid markers LCA, CD3, and CD20. Staging investigations showed no evidence of metastatic disease and therefore the diagnosis of localised PPNET was made.

The patient received chemotherapy as primary treatment followed by radiotherapy and has been in remission since. A year later she was referred to our unit for correction of her left ptosis, and underwent left anterior levator resection with a satisfactory result. Four years following her initial diagnosis she remains free of recurrent disease.

**Comment**

Horner's syndrome is caused by an oculosympathetic deficit to the pupilodilator and supratrochlear nerve fibres, resulting in miosis and anhidrosis. Therefore the diagnosis of localised PPNET was made.

The patient received chemotheraphy as primary treatment followed by radiotherapy and has been in remission since. A year later she was referred to our unit for correction of her left ptosis, and underwent left anterior levator resection with a satisfactory result. Four years following her initial diagnosis she remains free of recurrent disease.

**Comment**

Horner's syndrome is caused by an oculosympathetic deficit to the pupilodilator and superior and inferior tarsal retractor muscles. It is manifest by upper lid ptosis, ipsilateral miosis, apparent enophthalmos due to lower lid (“upside down”) ptosis, and often facial anhidrosis. The presence of all these features in our patient, together with the left facial anhidrosis, was indicative of a preganglionic lesion, since the sympathetic facial sweat fibres branch distal to the superior cervical sympathetic ganglion and central neurological tract signs were absent. Preganglionic Horner's syndrome is frequently associated with neoplasms of the pulmonary apex, mediastinum, or neck, as illustrated by the location of the PPNET seen in our patient (Fig 1B).

PPNET is a rare malignant small round cell tumour that can affect any age group but is thought to peak in adolescence, with no sex predilection. The most common location is the thoracopulmonary region, followed by the head and neck. Extrasosseous Ewing's sarcoma is now considered a form of PPNET, through immunohistochemical, ultrastructural, and histogenetical similarities. Both tumours express elevated levels of glycoprotein p30–32, a product of the MIC-2 gene, in a unique and highly selected fashion, as well as specific translocations involving a gene on chromosome 22q12. Indeed, the tumour cells of our patient exhibited immunohistochemical reactivity for MIC-2, consistent with a PPNET. The association of Horner's syndrome with C8/11 cervical radiculopathy is classically seen in Pancoast's syndrome, which is due to superior sulcus tumours of the lung that involve the chest wall and spinal column. The presentation of PPNET with Pancoast's syndrome has been reported previously, but in this case the tumour had massively infiltrated the left thorax with widespread metastases, resulting in death at 17 weeks following initial diagnosis. In contrast, our patient presented with Horner's syndrome at a relatively early stage of the disease, since the PPNET was localised with no evidence of metastatic spread. The malignant nature of this tumour highlights the importance of comprehensively investigating any patient with Horner's syndrome, especially when associated with cervical radiculopathy.

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**Association of keratoconus and Avellino corneal dystrophy**

Keratoconus is an idiopathic, progressive, non-inflammatory ectasia of the axial cornea. It is associated with other systemic disorders or ocular disease have been reported, but its specific origin remains unknown. Recently, Munier and associates detected that four types of autosomal dominant corneal dystrophy result from mutation in the human transforming growth factor β induced gene (Btg3). The product of which has shown to be the protein keratoepithelin (R55SW for granular corneal dystrophy, R535Q mutation for Reis-Bückler's corneal dystrophy, R1246H mutation for laryngeal corneal dystrophy type I, and R124H mutation for Avellino corneal dystrophy). Molecular genetic analysis of various corneal dystrophies which had previously presented an insuperable challenge to clinical diagnosis, now clearly demonstrates the distinct phenotypes. We report a rare case of bilateral keratoconus in association with Avellino corneal dystrophy diagnosed by molecular genetic analysis.

**Case report**

A 35 year old man had complained blurred vision in both eyes for several years. His general health was good and there was no history of atopic disease, connective tissue disease, or ocular trauma. His familial history was unknown. His best corrected visual acuity was RE 20/50 and LE 20/100. Slit lamp examination revealed bilateral non-inflammatory corneal thinning with protrusion of the central thinning areas. Fleischer ring was found in both corneas. Central corneal thickness was 428 µm on the right and 421 µm on the left measured by ultrasonic pachymetry. There was also clinical evidence of granular corneal dystrophy in both eyes. Discrete grey-white opacities and star-shaped stippled opacities

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**References**


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**Figure 1** [A] Left Horner’s syndrome with left upper lid ptosis, miosis, and “upside down” lower lid ptosis. [B] Magnetic resonance imaging of neck (coronal section), showing a mass arising from between the first and second thoracic intervertebral foramen, extending into the root of the left side of the neck and the left pulmonary apical region (arrow).
were seen in anterior stroma (Fig 1, top). Computed corneal topography showed inferior steeping consistent with the diagnosis of keratoconus (Fig 1, bottom). With rigid gas permeable contact lenses his visual acuity corrected to 20/20 right and 20/25 left. The remainder of the ocular examination was unremarkable.

After obtaining informed consent, we collected venous blood from the patient and extracted genomic DNA. Using appropriate primers, we amplified exons 4 and 12 of the \( \text{igH3} \) gene by polymerase chain reaction (PCR) and directly sequenced the products. We detected a heterozygous G\( \rightarrow \)A transition in codon 124 that results in a substitution from arginine to histidine in this patient (Fig 2). These genetic findings were consistent with Avellino corneal dystrophy.

**Comment**

To our knowledge, this is the first molecular genetic report of a bilateral association of keratoconus with Avellino corneal dystrophy. There is only one case report in the literature of a patient with keratoconus associated with Avellino corneal dystrophy. Sassani and associates reported the bilateral association of keratoconus and Avellino corneal dystrophy, which was diagnosed histopathologically. On the other hand, there are five reports with keratoconus associated with granular corneal dystrophy.** However, those cases were diagnosed clinically, not histopathologically or genetically.** A clinical diagnosis of the different types of corneal stromal dystrophy is difficult, especially for granular corneal dystrophy and Avellino corneal dystrophy. Some cases previously reported as granular corneal dystrophy might be actually cases of Avellino corneal dystrophy.

The involvement of genetic factors has been reported in keratoconus, but its hereditary pattern was not identified. A gene for at least one form of hereditary keratoconus has been mapped to human chromosome 21.** In our case, it is unclear whether a genetic factor had a role in the simultaneous development of keratoconus and Avellino dystrophy. There may be some linkage between the genes responsible for these two abnormalities. In our case, molecular genetic analysis clearly demonstrated the presence of distinct phenotype, which had not previously been presented clinically.

The authors have no proprietary interest in any aspects of this work.

**References**


**Presence of vitronectin in neovascularised cornea of patient with gelatinous drop-like dystrophy**

Gelatinous drop-like corneal dystrophy (GDL) is a rare autosomal recessive disorder that is most often seen in Japan. This bilateral dystrophy usually presents in the first decade of life and is associated with a decrease of visual acuity. Typically, a muttonberry-like opacity is present with proliferative subepithelial mounds that grow with age. Corneal neovascularisation (NV) also accompanies advanced cases. Corneal transplantation is the major therapeutic option for GDL, but because NV can significantly increase the risk of graft rejection, a better understanding of the mechanism(s) for the corneal NV would be valuable.

**Case report**

A 39 year old Japanese man with GDL was studied. His right eye had band-shaped corneal opacities in the interpalpebral area with a number of gelatinous prominences, and vascular invasions from the superior limbus into the clear cornea (Fig 1A). Because the visual acuity of the right eye had decreased to 20/800, penetrating keratoplasty was performed, and the diagnosis of GDL was confirmed by characteristic histopathological findings of amyloid deposits beneath the corneal epithelium and mutation of the M151 gene.
It was recently reported that vitronectin, a multifunctional extracellular matrix adhesion molecule, is often a component of the abnormal extracellular deposits in various age-related human diseases such as age-related macular degeneration and amyloidosis. This suggested that similar pathways may be involved in the pathologies of other age-related diseases. Because the disease state of GDLD deteriorates with age, we hypothesised that similar vitronectin related pathways may also be associated with GDLD, and examined whether vitronectin was expressed in the GDLD cornea.

An antibody directed against vitronectin (Santa Cruz Biotechnology, Santa Cruz, CA, USA) exhibited intense reactivity with the infiltrating leukocytes and corneal epithelium around the deposits. Substantial levels of vitronectin protein were also present in the deposits (Fig 1B, C).

Because the production of new extracellular matrix proteins has a crucial role in supporting cell proliferation that are necessary for new blood vessel growth, and because vitronectin receptors, such as integrin αβ3 and αβ5, are involved in angiogenesis, we further hypothesised that the accumulated vitronectin in the GDLD cornea may also have a role in the accompanying corneal NV.

We therefore examined the angiogenic potential of vitronectin using an in vivo corneal assay. Five µl hydron pellets (Interferon Sciences, New Brunswick, NJ, USA) containing 1 µg of mouse vitronectin (Invitrogen, Carlsbad, CA, USA) were implanted into the cornea of anaesthetised male Sprague-Dawley (S-D) rats and, after 7 days, the animals were killed, and the corneal vessels were photographed. Vitronectin elicited a strong angiogenic response, but administration of phosphate buffered saline (PBS) alone did not (Fig 2).

**Comment**

These results provide the first evidence for the expression of vitronectin in the cornea with GDLD, and for the in vivo induction of angiogenesis by vitronectin. The results indicate that vitronectin may have a role in corneal NV in patients with GDLD. Therefore, further studies exploring mechanisms of corneal NV mediated by vitronectin-integrin system, and how mutation of M1S1 leads to accumulation of vitronectin with more samples, may eventually offer a novel insight in understanding the angiote of corneal NV associated with GDLD.

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**Factor XII deficiency and recurrent sixth nerve palsy**

Factor XII deficiency is associated with thrombosis. Severe deficiency increases an individual’s prothrombotic tendency but with marked reduction in levels there is less certainty. We present a case of recurrent sixth cranial nerve palsy due to severe factor XII deficiency. To our knowledge, this is the first reported case of a recurrent cranial nerve palsy associated with factor XII deficiency.

**Case report**

A 58 year old white male presented with an acquired constant inconstant horizontal diplopia. He had had a previous episode of horizontal diplopia of 3 weeks’ duration 6 months previously with spontaneous resolution and a further similar episode 18 years before that had been otherwise well. He was not hypertensive or diabetic, of normal weight and a non-smoker, and without any cardiovascular disease. There was no family or personal history of venous or arterial thrombosis.

An examination by the浦作氏 found he to have bilateral sixth nerve palsies without any associated headache or papilloedema. Detailed magnetic resonance imaging with contrast and lumbar puncture opening pressure and investigation was normal.

Owing to the recurrent nature of the nerve palsy and the initial young age of presentation, a further prothrombotic examination was undertaken. Laboratory investigations showed a normal full blood count, plasma viscosity, liver function tests, glucose, homocysteine, prothrombin time, and fibrinogen assay. There was a significantly prolonged activated partial thromboplastin time of 74.7 seconds (normal range 24–32), which was still abnormal on repeat testing (90 seconds). Further laboratory studies demonstrated severe factor XII deficiency which was consistent on repeated testing (<1% of normal levels) but otherwise normal levels of protein S, protein C, antithrombin antibodies, factor VIII, von Willebrand factor, and the factor V Leiden mutation or prothrombin G20210A allele were not found.

**References**


He was fitted with Fresnel prisms to relieve his diplopia and was followed up 3 weeks later. By that time his diplopia had completely resolved and he had full abduction in both eyes.

Comment
With this recurrent and resolving pattern of cranial nerve palsy in a patient with no other risk factors for arteriosclerosis and a normal magnetic resonance image and lumbar puncture, the most likely predisposing factor in this case is his prothrombotic state associated with severe factor XII deficiency.

Severe factor XII deficiency is a genetic determinant for thrombosis. It is not associated with any other clinical manifestations and prolonged activated thromboplastin time is a consistent finding in any level of factor XII deficiency. The only previously reported ophthalmic complications of factor XII deficiency are two cases of central retinal vein occlusion in patients without any vasculopathic risk factors. Assay for factor XII is not routinely done on thrombophilia screening protocols. It has been suggested that the frequency of factor XII deficiency (1.5–3%) is high enough to warrant the inclusion of factor XII assays in routine thrombophilia screening. Detailed thrombophilia screening of healthy populations may produce an identifiable abnormality in 10% but clearly 10% of the population are not clinically affected in their lifetime. Therefore the need for additional management should be assessed according to the presence of concurrent risk factors in an algorithmic fashion. Since his cranial nerve palsy resolved quickly and there was no family history of vascular thrombosis he was treated empirically with aspirin, although there is no evidence to support its benefit in this condition.

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Unilateral proptosis: the role of medical history
The most common cause of bilateral and unilateral exophthalmos among adults is Graves’ disease. Unilateral exophthalmos, although frequently seen in connection with thyroid diseases, has a much larger differential diagnosis than bilateral exophthalmus. With this recurrent and resolving pattern of diplopia and was followed up 3 weeks later. By that time his diplopia had completely resolved and he had full abduction in both eyes.

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A 30 year old homosexual HIV+ man was referred to the uveitis department complaining of blurred vision in the left eye. He was taking zidovudine, lamivudine, ritonavir, and saquinavir. His last CD4+ count was 128 cells ×10^6/l and viral load 1 300 000.

His visual acuities were 6/6 in the right eye and counting fingers in the left. There was no inflammation in the anterior chambers or in the vitreous. Ophthalmoscopy revealed a yellowish choroidal lesion surrounded by fluid and haemorrhages in the macula of the left eye (Fig 1). Fluorescein angiography showed an angiomatos-like lesion corresponding to those seen clinically. Blood tests were ordered including VDRL, toxoplasmosis serology, Lyme disease serology, ELISA for toxocariasis and were all negative. Computed tomography (CT) scan and serum studies were unremarkable. Blood sample was sent to CDC Atlanta for Bartonella serology. Since clinical diagnosis was cat scratch disease and most patients show good recovery without treatment we decided not to treat before results of blood tests. We kept examining the patient every week with ophthalmoscopy and fluorescein angiography (Fig 2A, B). The lesion progressively increased in size but he did not show visual acuity deterioration.

A month after presentation the lesion had increased and four small lesions appeared in the right eye. His visual acuity dropped to hand movements. Although we did not have the results of Bartonella serology, we decided to give him ciprofloxacin. Bartonella henselae serology was positive for IgG, 1:256, and IgM negative.

Fifteen days after treatment was started the lesions in the right eye disappeared and the macular lesion in the left eye resolved completely.

**Comment**

There was a well established association between neuroretinitis and cat scratch disease although many different clinical presentations have been described. Ormerod et al reported two patients with small areas of retinitis and arterial occlusions. Pollock and Kristinnson described one patient with cat scratch disease and helioid unifocal choroiditis. Hong et al first described this syndrome when they reported six young patients with a solitary round yellow choroidal lesion associated with subretinal fluid. There was no association with inflammatory or infectious diseases. Fish et al described a case of peripapillary angiomatosis associated with neuroretinitis. Our patient presented with clinical features of helioid unifocal choroiditis but after angiogram we could see an angiomatos-like lesion.

The treatment of ocular cat scratch disease remains controversial. Pollock and Kristinnson reported a case that improved in visual acuity from 6/12 to 6/6 occurred after 3 weeks without treatment. One of the cases described by Ormerod showed some benefit after treatment although his recovery was very slow. The second patient showed improvement without treatment. Warren et al reported an HIV+ patient with cat scratch disease whose lesion enlarged without treatment. Once the diagnosis of Bartonella was confirmed by polymerase chain reaction of the retina sample, the patient was started on systemic antibiotics with good results. Considering that spontaneous recovery could occur we decided not to treat until our patient showed deterioration in the left eye and involvement in the fellow eye.

Ophthalmologists should be aware of this unusual presentation of cat scratch disease with helioid unifocal choroiditis and angiomatos-like lesions. Although larger series and control studies are needed, HIV+ patients with intraocular manifestations of cat scratch disease may benefit from systemic treatment with antibiotics.
various complications that have been experienced with its use. In this report, we describe an unusual complication associated with limited macular translocation in a patient with long standing choroidal neovascularisation.

Case report
A 35 year old woman was referred to our department because of a gradual decrease in visual acuity in her right eye. At the first visit, her best corrected visual acuity was right eye, 20/40, with a refractive error of −17.5 dioptres in the spherical equivalent. Clinical and angiographic examinations showed a juxtapfoveal choroidal neovascularisation. During the subsequent follow up period, choroidal neovascularisation and surrounding retinal pigment epithelial atrophy gradually expanded and involved the subfoveal region (Fig 1A). We gave the patient detailed information on the available therapeutic options, including macular translocation, but she chose conservative follow up rather than surgical intervention. Three years after her first visit, the visual acuity in her right eye worsened to 20/100. At that time, the patient decided to have surgical treatment. With her consent, we performed limited macular translocation. After the surgery, the visual acuity in her right eye improved to 20/40, with a refractive error of 5–8 dioptres.

Figure 1  [A] A preoperative fluorescein angiogram shows myopic choroidal neovascularisation with marked atrophy of the surrounding retinal pigment epithelium. An area of relatively healthy retinal pigment epithelium is shown inferonasal to the fovea. [B] A fluorescein angiogram taken 3 months after the surgery demonstrates a sharply delineated blockage of choroidal fluorescence in the translocated macular area (white arrow). The original macular area shows hypofluorescence with larger choroidal vessels well visualised (black arrow). This finding may indicate that the retinal pigment epithelium of the original macular region has been torn away and subsequent atrophy of the underlying choriocapillaris has occurred.

Comment
In many eyes with choroidal neovascularisation, the macula can easily be separated from the subjacent fibrovascular tissue. In some eyes with long standing choroidal neovascularisation, however, the outer portion of neurosensory retina may adhere firmly to the subjacent tissue. In such cases, an inner portion of fibrovascular tissue may be torn off and translocated with overlying neurosensory retina during macular translocation. The underlying healthy retinal pigment epithelium covered with the translocated abnormal tissue may not be able to fulfil its physiological roles on the overlying neurosensory retina, and good functional recovery of the translocated macula is unlikely to be achieved. As documented here, simultaneous translocation of the underlying abnormal retinal pigment epithelium associated with long standing choroidal neovascularisation can occur during limited macular translocation and result in an unsatisfactory visual outcome. When patients are deciding whether to consent to surgical intervention with limited macular translocation in such cases, they should be informed of the benefits and risks of the treatment, with due consideration of this complication.

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Figure 2  [A] A horizontal optical coherence tomography section taken through the translocated macula displays highly reflective double layers underneath the fovea that probably correspond to the native retinal pigment epithelium and simultaneously translocated retinal pigment epithelium. (B) A postoperative indocyanine green angiogram shows well visualised larger choroidal vessels in the original macular area probably caused by the absence of the retinal pigment epithelium-choriocapillaris complex (black arrow). The relative hypofluorescence in the translocated macular area may represent blockage of choroidal fluorescence by the translocated retinal pigment epithelium (white arrow).

Fungal and bacterial chronic endophthalmitis following cataract surgery
Endophthalmitis, although rare, is one of the most vision threatening complication of cataract surgery. The majority of these infections...
are bacterial in the Western world. The occurrence of fungal endophthalmitis after cataract surgery is rare as well as polymicrobial infections.1

We report a case of chronic postoperative endophthalmitis caused by bacterial and fungal pathogenic agents.

Case report
A 73 year old woman was referred for pain and redness in the left eye. Her past history was remarkable for an extracapsular cataract extraction in the left eye with a posterior chamber intraocular lens implant that had been performed in Turkey in 1998. The patient had recurrent episodes of decreased vision and ocular pain in the postoperative course and was treated with peribulbar injections of corticosteroids over 2 years. On presentation, visual acuity was hand movements in the left eye. Slit lamp examination of the left eye showed a white corneal infiltrates involving the superior and nasal quadrant (Fig 1). There was a moderate anterior chamber reaction and a 1 mm hypopyon. The implant was in good position and no residual cortical material was seen. The vitreous showed 2+ cells. Intraocular pressure was normal. Examination of the right eye was normal except for a mild nuclear cataract. The diagnosis of chronic infectious endophthalmitis was suspected.

The aqueous cultures were sterile for bacteria and fungi. Intravitreal injections of vancomycin and amikacin were performed. Postoperatively, the patient was given intravenous ciprofloxacin, piperillin, and topical fortified tetracillin, gentamicin, and vancomycin drops. The eye showed no improvement during the next 3 weeks and the patient subsequently underwent removal of the implant and capsular bag which grew Candida parapsilosis, Corynebacterium striatum, and Staphylococcus epidermidis.

Intravitreal injection of amphotericin B (5 µg, weekly), topical amphotericin B (7 mg/ml eye drops every 2 hours), and oral fluconazole (400 mg a day) were then added to the patient’s regimen. A decrease in intraocular inflammation and corneal infiltrates was noted after 1 month of treatment that was stopped by the patient. Six months later, visual acuity of the left eye was no light perception and biomicroscopic examination showed panophthalmitis. The patient refused medical treatment and enucleation was performed.


Figure 1 Anterior segment photograph of the left eye showing an hypopyon and a crystalline keratopathy.

Figure 2 Higher magnification showing crystalline keratopathy.

Comment
Species of candida, other than Candida albicans, are normal flora of cutaneous and mucocutaneous surfaces and are only rarely incriminated as agents of clinical disease in immunocompetent hosts. Candida parapsilosis is in fact one of the most common fungi cultured from the normal human external eye.1 However, it can cause infectious crystalline keratitis or, more typically, suppurative stromal keratitis. Candida parapsilosis has also been reported as a cause of small epidemics of postoperative endophthalmitis.1 In the present case, Candida parapsilosis could be considered as a true pathogenic organism because of its localisation, in the capsular bag, and also because antifungal therapy was effective.

On the other hand, Staphylococcus and Corynebacterium species are frequently identified in postoperative endophthalmitis.1 This case is, to our knowledge, the first documented report of bacterial and fungal endophthalmitis following cataract surgery. The use of broad spectrum antibiotics, the administration of steroids, and the increased number of patients with local or systemic immunosuppression could explain the development of such infections which are frequent in post-traumatic endophthalmitis but extremely rare after cataract surgery.1 However, cross contamination by hospital personnel may also account for increase in yeast infections in certain environments. A recent survey of hospital personnel revealed that 70% of nurses and non-nursing hospital personnel carried yeasts on their hands, 70% of nurses and non-nursing hospital personnel carried yeasts on their hands, particularly subungal spaces, with Candida parapsilosis being those most frequently recovered.5 This case demonstrates the atypical presentation and the poor prognosis of polymicrobial endophthalmitis. Identification of all the organisms is essential before the onset of therapy, emphasising the need for complete microbiological evaluation of any postoperative endophthalmitis.

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Surgical performance for specialties undertaking temporal artery biopsies: who should perform them?

We read with interest the paper by Galloway and colleagues which suggests that ophthalmologists are best suited to perform temporal artery biopsies.1 We recently completed a retrospective study of all the temporal artery biopsies performed at four teaching hospitals (Hammersmith Hospital, Charing Cross Hospital, St Mary’s Hospital and The Western Eye Hospital) in north London between January 1998 and January 2002. Ninety one patients underwent 92 biopsies. Of these, 15 were positive for temporal arteritis implying a 16% positive biopsy rate which is compatible with Galloway et al’s results. Ophthalmologists performed 54 biopsies (59%) while general and vascular surgeons (GVS) performed 38 biopsies (31%). Both groups had similar positive biopsy rates—ophthalmologists 10/54 (19%) and GVS 5/28 (13%). In an analysis of the ability to perform biopsies, the ophthalmologists had two failed biopsies (one specimen—no artery identified and one specimen—crushed artery) while the GVS group had one failed biopsy (no artery identified). The average artery length was 13.0 mm (range 5–21 mm) for the ophthalmologists and 14.0 mm (range 7–22 mm) for the GVS group.

We disagree with Galloway and colleagues’ assertions that ophthalmologists are best suited to performing temporal artery biopsies as our study found that both groups of...
surgeons obtained similar lengths of artery and had similar positive biopsy rates. We note that in their study, while the vascular surgeons only performed two of the 41 biopsies, the average length of specimen obtained was 22.5 mm, thus suggesting that the vascular surgeons may well be the best equipped to perform these biopsies. In Charing Cross Hospital, the vascular surgeons routinely use a Doppler ultrasound probe to help detect and demarcate the superficial temporal artery before biopsy and this practice has been advocated by other studies to help improve the yield of the biopsy. In our study, all the biopsies performed by ophthalmologists were done under local anaesthetic and seven cases had to be performed outside normal working hours as emergencies because of lack of theatre time. The fact that ophthalmologists performed 59% of biopsies may be due to the fact that all four hospitals had large neurological and rheumatological units attached to them. We feel that both ophthalmologists and general and vascular surgeons are equally capable of performing temporal artery biopsies and that guidelines should be designed locally to decide who should perform the biopsies. More importantly, the follow up of these patients should be clearly stated and be ideally under the care of the rheumatologists.

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References

Major complications of endoscopic sinus surgery: a comment

We were pleased to read the informative article by Rene et al. The authors refer to “Onodi cells” as “aerated posterior ethmoid air cells, along the optic canal.” Could it be that they meant to refer to the cells as “Onodi cells”? Onodi described a number of variations of posterior ethmoid anatomy.1 Endoscopic sinus surgeons refer to the pattern of extramural pneumatization of the ethmoid lateral or superolateral to the sphenoid, where the posterior ethmoid is indented by the optic canal, as an Onodi cell. More recently it has been suggested that this cell is better characterised as a “sphenethmoidal cell.”2

The main significance of the Onodi or sphenethmoid cell is that while sinus surgeons expect to find the optic nerve in the sphenoid sinus, they are not routinely looking for it in the posterior ethmoid. Consequently, the chance of iatrogenic injury is likely to be higher in patients with Onodi cells and even higher in patients with extensively pneumatized Onodi cells. The exact incidence of Onodi cells is unclear. Endoscopic dissection studies suggest an incidence as high as 39% or 42%. CT imaging studies suggest a lower incidence of 7%. Driben et al.3 feel that the likelihood of a sphenethmoid cell increases with increasing obliquity of the anterior sphenoid wall. Racial factors may also have a role as Onodi cells appear to be more common in Asian patients.4

Every effort should be made to identify these cells preoperatively and to be aware of intraoperative clues such as anterior sphenoid wall alignment, which may point to the presence of an Onodi cell. Attention to these important details will increase the likelihood of uncomplicated endoscopic sinus surgery at the sphenethmoid junction.

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References

Trabeculectomy augmented with antimetabolites with a viscosanalostomy technique for the management of open angle glaucoma

In a small randomised, prospective study, O’Brart and co-workers5 attempted to prove that trabeculectomy (n = 25) provided better intraocular pressure (IOP) control than viscosanalostomy (n = 25). However, we have serious concerns about the methodology of this study that need to be addressed. Viscosalostomy is characterised by the injection of viscoelastic into Schlemm’s canal.6 However, this was not performed in half of the patients randomised to this group. This would be inappropriate as the use of an antimetabolite in only half the patients in the trabeculectomy group, and then comparing this entire group to the viscosanalostomy group. Although the authors do state that the subgroup of patients with intracanalicular viscoelastic injection was inferior to trabeculectomy, this cohort consisted only of 12 patients and it is not known how many had completed follow up after 18 months. Surely, these small numbers are insufficient to draw such conclusions.

We are also concerned that the use of intraoperative antimetabolites in the trabeculectomy group added a major confounding variable in this study. This is particularly perplexing as the authors’ viscosanalostomy technique primarily relied on subconjunctival filtration, as evidenced by their scleral flap design and looser suturing technique in which only three 10/0 nylon sutures were used. Furthermore, their excellent early success rates (80% at 6 months), the presence of filtering blebs in their successful viscosanalostomy procedures, the lack of one in their failures, and the need for postoperative bleb needling and 5-fluorouracil injections all suggest that the use of mitomycin-C and 5-fluorouracil improved the success rate of glaucoma filtering surgery by reducing episcleral fibrosis, and probably explains the difference in success rates in this study. We wonder what the results would have been had the use of intraoperative antimetabolites been used in all groups, or if none was used at all.

In contrast with the authors’ technique, we employ Stegemann’s approach to viscosanalostomy in using a parabolic superficial flap secured tightly with five sutures in a relatively watertight fashion.7 Although postoperative blebs may develop, most patients achieve IOP lowering through multiple alternate pathways including uveoscleral, through Schlemm’s canal, and subconjunctival.8 Certainly in higher risk cases, we might consider the use of antimetabolites in non-penetrating surgery, and have found it to be safer when used with trabeculectomy.

It should be mentioned that quite often we do rely on subconjunctival filtration in non-penetrating procedures (that, is, deep sclerectomy) but advocate the use of a collagen wick9 or hyaluronic acid implant with an intraoperative antimetabolite for higher risk cases to obtain optimal IOP control.

Fibrosis and loss of permeability of the trabeculo-Decemet’s window (TDW) is a well described cause of postoperative elevation in IOP after non-penetrating glaucoma surgery. Postoperative Nd:YAG gonipuncture of the TDW in these cases is a relatively easy adjunctive procedure and may be needed in up to 41% of non-penetrating procedures.10 It has been reported to successfully lower postoperative IOP in over 80% of cases.6 Yet, we are dismayed that the authors decided not to attempt laser gonipuncture in those viscosanalostomy cases with postoperative IOP elevations because “such interventions clearly convert a ‘non-penetrating’ technique into a penetrating, full thickness procedure.” We vehemently disagree with this line of reasoning as we feel gonipuncture is an extremely useful adjunctive procedure and converting to a penetrating (not “full thickness”) procedure in the safety of the controlled postoperative period is completely reasonable. This is akin to suturing a lysis in trabeculectomy. Several authors feel that performing suture lysis constitutes conversion of a guarded trabeculectomy into a full thickness undigrated trabeculectomy and thus cannot be fairly compared. Although gonipuncture was performed in only three eyes at 18 months, we wonder what results would have been obtained if this was done in all cases with uncontrolled IOPs at any point in the postoperative period.

Although we are critical of this study, we applaud O’Brart and colleagues for attempting to investigate this evolving area of glaucoma surgery. Although they may have shown that trabeculectomy with the use of an antimetabolite is superior to a modified form
of viscocanalostomy dependent on subconjunctival filtration without the use of an antimetabolite, this study unfortunately does not fairly compare the efficacy of Stegmann’s viscocanalostomy technique versus trabeculectomy. Non-penetrating glaucoma surgery remains an improved safety profile and surely as future well designed controlled studies become available, the efficacy of these procedures compared to trabeculectomy will become clearer.

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References

Argon laser and trichiasis

We were interested to read the approach taken by Sahni and Clark1 to facilitate the effective argon laser treatment of trichiasis. They have ably reviewed the complications of trichiasis, the different forms of management of trichiasis, the advantages of argon laser treatment in the management of trichiasis, the technique of argon laser trichiasis therapy, and the limitations of laser lash therapy.

We take issue with the authors in two areas. Firstly, the almost certain consequence of using a duration of laser treatment of 0.1 second is that if the laser “takes,” the lash will disappear within the space of a few laser shots, effecting preclusion the destruction of that particular lash follicle. We have particularly made it a point that when teaching trainees the technique of laser lash, we ensure that the energy burst lasts long enough to commence visible lash destruction as well as destruction of the subcutaneous lachrymal, as the burn is directed towards the lash follicle. Thus we always use a duration of several seconds, or even continuous energy, and aim to achieve instantaneous destruction of the lash follicle above the lid level after the first shot, or certainly within three shots. Thus, 1–3 second duration bursts may be required, depending on the individual lash. Just a few more shots will effectively and completely destroy the subcutaneous lash and its follicle.

Secondly, the article by Bartley and Lowry quoted by the authors, describes using a “drop of ink from a fountain pen” to facilitate laser lysis. Presumably in the interests of sterility, Sahni and Clark have used the ink from a “blue skin marker pen” to allow improved absorption of argon laser energy. While use of a fresh marker pen for each patient may be relatively efficient, it could not be regarded as cost effective. By contrast, in a procedure described by us2 we found that transferring a tiny drop of the patient’s own blood, whether still liquid or already coagulated, to the lash base on the lid margin is a simple, rapid, cheap, safe, and highly effective method of getting the laser reaction started when the lashes are pale. We have found that the required amount of blood is invariably present on the patient’s own lid skin at the site of local anaesthetic infiltration. We usually transfer it by picking it up with a sterile drawing up needle. This is achieved remarkably easily on the laser slit lamp, which allows adequate magnification for the accurate siting of the transferred blood.

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References

Management of age related macular degeneration: still room for improvement

The aim of Mitchell et al’s study1 in collaboration with the Macular Disease Society (MDS) was to assess the perceived quality of health care of people with macular disease in the United Kingdom. The study was performed in 1999 and took the form of a retrospective audit by postal survey of 2000 members of the MDS. I performed a similar but smaller study earlier.2 This study took the form of a retrospective audit by postal survey of 200 members of the MDS. The aim was to assess the clinical management of patients with age related macular degeneration (ARMD) in the United Kingdom from the patients’ perspective. The final response rate was 79% (similar to 77% of Mitchell et al) and patients had attended over 105 ophthalmic units throughout the United Kingdom. Mitchell et al omitted a question from their discussion but comparison of the two studies raises some noteworthy points.

The prevention2 and treatment3 of ARMD remains far from ideal both for the majority of patients and their ophthalmologists. Members of the MDS are likely to be a highly motivated study group and it is not clear how their views will reflect the views of patients as a whole. We outlined our study in an email to the MDS mailing list. It is therefore conceivable that a proportion of the original study group also formed part of the second, larger study. What is clear enough is that the fact that patients want information on ARMD and the fact that there are readily available sources (for example, free information booklets on ARMD from the Royal College of Ophthalmologists or Royal National Institute for the Blind), ophthalmic units throughout the United Kingdom are still failing routinely to provide such information to their patients.

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References

Standardised clinical photography in ophthalmic plastic surgery

We read with keen interest the path breaking article by Calista et al regarding the successful treatment of an eyelid squamous cell carcinoma with intrascleral cidofovir. The dramatic response of this highly malignant lesion to such a non-invasive form of therapy is remarkable and certainly worth further clinical evaluation. However, we would like to raise a few crucial issues pertaining to the documentation of this case.

The authors have presented an extreme close up of the affected eye in Figure 1 which highlights the pretreatment appearance of the lesion quite adequately. However, the post-treatment photograph (Fig 2) is almost half the magnification of Figure 1. Therefore, although there is an indubitable reduction in the size of the tumour, the two clinical photographs are not strictly comparable since a decrease in magnification results in visual clues that lead to the lesion being perceived as smaller in size.

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Secondly, the pretreatment view (Fig 1) has been taken in primary gaze and reveals a right lower lid retraction as well as the semblance of a mild lid notch. In contrast, the post-treatment view (Fig 2) has been photographed on the left and slight downgaze and does not reveal either of the above findings. Now, it is difficult to discern whether there is an actual disappearance of these pretreatment findings or it is due to the inherent lack of comparability of these pictures because of their being in totally different positions of gaze, which is compounded by the magnification factor mentioned earlier. It would be of immense benefit to the readers if the authors could clearly point out exactly which of the discrepancies, which have arisen due to dissimilar photographs.

It is imperative that extremely high standards of clinical photography be maintained in plastic surgery/oncology and photographs be taken with similar parameters to ensure valid pretreatment and post-treatment comparisons that accurately reflect the results. The standards and recommendations for clinical photography have been widely documented and should be universally practised. These include using the same camera lens, settings, lighting, film, magnification, and patient position to ensure reproducibility and comparability. Even small variations in the pictures may cause drastic changes in the clinical and research value of photography and, unless stringent criteria are met, the photographs may lose their relevance and overall impact.

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References

Entonox as an analgesic agent

We read with great interest the paper on Entonox as an analgesic agent by Cook et al. We congratulate the authors for their work. However, we would like to clarify certain aspects of the study.

We recently performed a randomised, placebo controlled, double blind study with 100 patients on the analgesic effect of Entonox for pain relief during local anaesthetic injections in minor eyelid procedures (accepted for publication). We administered Entonox for 30 seconds in our study and found no statistically significant difference in the pain scores between the placebo and the study groups. No side effects were reported but in our pilot study, where the patients were given Entonox for 60 seconds, all the Entonox group patients reported light headedness and required prolonged observation before being discharged.

We attributed our results of lack of statistically significant pain relief with Entonox to less than 50 seconds’ administration of Entonox. Waud et al.1 have shown that optimal administration of Entonox should cover 50 seconds, based on theoretical calculations for effective analgesia. Based on the above experience, we would like to know if the authors administered Entonox throughout the laser treatment and, if so, did it interfere with the laser delivery? Since the inhalation process is likely to be associated with head movements? If the Entonox was given for a shorter duration, we need to know the duration of administration since that would be of practical benefit to the readers. The authors have concluded that Entonox is useful in young patients, those undergoing re-treatments, and in patients who have previously not been able to tolerate the full treatment. We would like to know how the authors came to this conclusion, as there is no mention of the type of patients selected for their study.

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References

Mechanism of ophthalmic artery occlusion following pars plana vitrectomy

Saito et al. present a patient with Terson’s syndrome and dense vitreous haemorrhage who underwent pars plana vitrectomy and was noted postoperatively to have developed an ophthalmic artery occlusion. They propose that the ophthalmic artery was occluded by the spontaneous release of an embolus from an atheromatous plaque in the internal carotid artery. This seems unlikely in a 59 year old man without a previous history of symptomatic atherosclerotic disease. Although the authors identified plaques in the patient’s carotid artery by ultrasound, these can be seen in 11% of asymptomatic males under age 40 and may therefore be an incidental finding in this case.1

An alternate explanation for the patient’s ophthalmic artery findings is trauma from the retrobulbar injection. Intravascular injection into the ophthalmic artery has been reported as a complication of retrobulbar anesthesia.1 It is possible that either an intravascular injection or simply needle tip trauma resulted in thrombus formation with obstruction of flow in the ophthalmic artery. It should also be noted that although acute ophthalmic artery occlusion is the presumed diagnosis, the same findings could result from simultaneous obstructions of the retinal and choroidal circulations, also a consequence of errant retrobulbar injection. The possibility that the patient’s chorioretinal disturbance could have been iatrogenic highlights the importance of a thorough preoperative discussion with patients about the risks and benefits of different methods of delivering anaesthesia for ophthalmic surgery.

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References

Dynamics of corneal endothelial cell death in organ culture

We read with interest the remarks of Crowston et al. on our article.1 We showed that the TUNEL technique revealed a far higher percentage of endothelial cells (ECs) irreversibly engaged in a cell death process than that obtained by trypan blue staining. The two techniques were performed sequentially: after observation of trypan blue staining, corneas were immediately fixed in formaldehyde for TUNEL. Crowston et al suggest that the trypan blue itself and/or the time spent outside the organ culture medium before fixing in formaldehyde caused an artefactual increase in the percentage of TUNEL positive ECs. Two arguments counter this remark.

(1) The trypan blue staining procedure is identical to that used, during endothelial examination(s) of grafts, in all European cornea banks that use organ culture during endothelial examination(s) of grafts. Neither the low concentration of trypan blue (1 mg/ml nor the short exposure time (about 1 minute) nor the short incubation in the presence of 0.9% NaCl has ever been incriminates in the over-mortality of ECs in routine practice. Moreover, the innocuity of injections of trypan blue into the anterior chamber, a common feature during cataract surgery, has been well demonstrated.2

(2) The time spent outside the organ culture medium before fixing in formaldehyde, a period required for vital staining and microscopic examination of the endothelium, lasts only a few minutes. The cornea remains under the microscope for about 1 minute only, the time needed for image acquisition. Such rapidity is possible by using a prototype automatic analyser of the endothelium, which we developed and have recently published.3 This is very probably insufficient time for DNA fragmentation to occur at the level we observed. Moreover, the fixing of the endothelial layer in 10% formaldehyde is immediate, and prevents any continuation of fragmentation phenomena. On balance, it is highly unlikely that the succession of markings is responsible for the discrepancy between the positivity percentages of the two techniques. In conclusion, we chose to also perform the two techniques simultaneously on paired corneas or on the halves of one cornea because we wanted to superimpose the two stains on the same cornea and thus obtain a double cell staining.

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Downloaded from http://bjo.bmj.com/ on June 23, 2017 - Published by group.bmj.com
Role of optometry in Vision 2000

The latest issue of Community Eye Health (No 43) discusses the mobilisation of optometry to deal with uncorrected refractive error, which is now a major cause of functional blindness.

NOTICES

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 Surgery Group the conference will cover cataract surgery, refractive surgery, glaucoma surgery, vitreoretinal surgery, corneal surgery, eye surgery in developing countries, and orbita, lacrimal and lid surgery. Further details: MGN Medizinische Congress organisation Nürnberg AG, Zerrablöffhöfstr 29, 90478 Nürnberg, Germany (tel: +49 911 3931621; fax: +49 911 3931620; email: doc@mcnag.info; web site: www.doc-muenrberg.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 Ollier (tel: +44 (0)121 424 5464; fax: +44 (0)121 424 4464; email: MartiDi@heartsol.wmds.nhs.uk; web site: www.bopss.org).

13th Meeting of the EASD Eye Complication Study Group

The 13th Meeting of the EASD Eye Complication Study Group will be held on the 23–25 May 2003, in Prague, Czech Republic. The scientific 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 Terzic Pelikanova (Czech Republic) on Pathophysiology of diabetic microvascular complications; Dr Tomas Sosna (Czech Republic) on Risk and protective factors of diabetic retinopathy.

Three travel grants of £1000 each, sponsored by GlaxoSmithKline for young scientists (under 35 years at the time of the meeting). Applications should be made with the submission of abstracts. The deadline for abstracts is 14 February 2003.

Further details: Ortopedie Centrum, s.r.o., Strekovské nabrezi 51, 400 03 Usti nad Labem, Czech Republic (tel: +420 47 521 6588; fax: +420 47 533 40 77; email: oortcenu-lu@volny.cz; web site: www.ortopedie-centrum.cz).

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

The detachment course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding Annual Meeting of Iranian Society of Ophthalmology will be held on 29–30 November 2003 and 1–4 December 2003 respectively, at the Razi Conference Center, Hemmat Byw, Tehran, Iran. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Breuningerbau, 72076 Tuebingen, Germany (tel: +49 70 71 293209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Mashehchki, Dr Siham Moradian, Dept of Ophthalmology, Labbanfinejad Medical Center, Pasdaran Ave, Boostan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbafi@hotmail.com).
Trabeculectomy augmented with antimetabolites with a viscocanalostomy technique for the management of open angle glaucoma

I I K Ahmed and C Kranemann

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