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. Systemic treatment with steroids inevitably leads to secondary side effects such as systemic suppression of the whole immune system and Cushings syndrome. Taking into account that the eye comprises only 0.01% of the whole body volume, and considering that for achieving high concentrations of steroids, a drug at its site of action 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 cortisone.

Case report
A 17 year old woman suffering from chronic idiopathic uveitis in both eyes for 5 years had been treated topically, peribulbarly, and systemically with corticosteroids. As a steroid responder, she had developed secondary ocular hypertension. Steroid induced cataract in her right eye was operated on by phacoaspiration, 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. Intracocular pressure increased to a maximum of 38 mm Hg, and was reduced to the normal range with topical steroids. Topical treatment, however, often has not been sufficiently effective to suppress intraocular inflammation and to reduce cystoid macular oedema. Systemic treatment with steroids inevitably leads to secondary side effects such as systemic suppression of the whole immune system and Cushings syndrome. Taking into account that the eye comprises only 0.01% of the whole body volume, and considering that for achieving high concentrations of steroids, a drug at its site of action 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 cortisone.

References

Ophthalmodynamometric estimation of cerebrospinal fluid pressure in pseudotumour cerebi

Measurement of the cerebrospinal fluid pressure usually requires a lumbar puncture or cranionomy 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 therefore 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 0.5 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 began 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 measurements of cerebrospinal fluid pressure by lumbar puncture performed about 5 hours later revealed a value of 107 cm water column (equivalent to 82.3 mm Hg). In combination with other clinical findings, the diagnosis of pseudotumour cerebi 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 exiting the eye through the optic disc, the central retinal vein goes through the retrobulbar part of the optic nerve before it traverses the subarachnoid 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
of intraocular pressure plus an external pressure exerted onto the eye equals the diastolic pressure of the central retinal vein. 

The intraocular pressure can be determined by applanation 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. 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.

J B Jonas, B Harder
Department of Ophthalmology, Faculty of Clinical Medicine Mannheim, University of Heidelberg, 68167 Mannheim, Germany

Correspondence to: Dr J Jonas, Universitäts-Augenklinik, Theodor-Kutzer-Ufer 1-3, 68167 Mannheim, Germany; Jost.jonas@augen.uni-heidelberg.de

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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). 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.

Ceramide comprises about 30% of stratum corneum lipids, which play an important role in both the water retention and barrier function of the skin. Ceramide abnormalities in several skin disorders, such as AD, have been reported. Decreased levels of ceramides may be attributable to the insufficient water retention of the skin in AD. Apytt 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 prescription.

Methods and results

Sixteen lids of eight patients (five males and three 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, 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 Apytt 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<0.025) (Fig 1). Water evaporation values were significantly decreased after treatment (4.5 (1.4) ×10⁻³ g/cm²/s before treatment, 3.5 (0.9) ×10⁻³ g/cm²/s after treatment; p<0.05) (Fig 2). No slit lamp findings indicating toxicity were observed during the course of the study.

Comment

As the eyelid is a border line 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 eyelid 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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N Asano-Kato, K Fukagawa, Y Takano, T Kawakita, K Tsubota, H Fujishima
Department of Ophthalmology, Tokyo Dental College, Japan

S Takahashi
Department of Dermatology

Correspondence to: Naoko Asano-Kato, MD, Department of Ophthalmology, Tokyo Dental College, Chiba, Japan, S Takahashi, Department of Dermatology, Tokyo Dental College, Chiba, Japan, naokato@bc.itc.or.jp

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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 hypotropia. There was a 3 mm of proptosis of the right eye. Eye movements in that eye were restricted in all directions. Slit lamp examination showed bilateral mild cataract. Intraocular pressures were normal (12 and 10 mm Hg respectively). Funduscopic examination yielded cup-disc ratios of 0.8. Computed tomography (CT) of orbit (Fig 1) showed an extraconally solid space occupying mass in the lacrimal fossa. The tumour displaced the right eye inferomedially. The tumour was thought to arise from the lacrimal gland. A fine needle aspiration cytology demonstrated proliferation of oncocytic cells. The tumour was removed in toto via a lid crease incision.

Histological macroscopic description yielded a spherically shaped, partially encapsulated tissue (measuring 2.8 × 3.0 × 1.9 cm). After preparation it had a homogeneous brownish tuberiform aspect. Microscopic examination (Fig 2) showed fibrous tissue with focally pre-existing remnants of lacrimal gland tissue. There was a sharply demarcated tumour composed of tubular structures lined by large cells. These transformed epithelial cells consisted of eosinophilic, granular cyttoplasm with round to oval shaped relative small nuclei and nucleoli. No evidence of angioinvasive growth was seen. A diagnosis of a radically removed oncocytoma was made.

Postoperative visual acuity of the right eye was reduced because of poor tear secretion confirmed on Schirmer testing and slit lamp examination showed filamentary keratopathy. Lubricating eye drops were prescribed and collagen punctal plugs were inserted in the superior and inferior puncta. Diplopia and the motility disturbance diminished. Follow up to date is 1½ years and no regrowth has been assessed.

Comment

Oncocytomas (synonyms: oxyphilic adenomas or oncocytic adenomas) are benign, epithelial tumours arising in the ductal cell lining of apocrine glandular structures. In an oncocytoma the oncocyes can form nests, cords, and tubules. Oncocytes can be found among the epithelial cells of various normal organs. Oncocytomas occur frequently and have been described in salivary, thyroid, parathyroid, buccal mucosa, breast, kidney, pharynx, and larynx. If they occur near the eye, they are usually located in caruncle, lacrimal sac, accessory lacrimal glands of the conjunctiva and rarely in the lacrimal gland. For unknown reasons oncocytic lesions begin to appear in early adulthood and proliferate with age.

Most lacrimal gland masses represent inflammatory disease, either responding to antibiotics or anti-inflammatory medication. Various malignant tumours of the lacrimal gland have been described varying from lymphoproliferative disorders to epithelial neoplasms. Approximately 50% of epithelial tumours are benign mixed tumours (pleomorphic adenomas) and about 50% are carcinomas. In rare cases a lacrimal gland mass is found to be an oncocytoma, as described in our case and in three other cases in the literature, or as an oncocytic carcinoma.

Beskid and Zarzcka described a 39 year old woman with an oncocytoma of the lacrimal gland. Riedel et al reported a 1.5 year old African girl with an oncocytoma of the lacrimal gland. This is the only case in the literature that reported on an oncocytoma in childhood. Riedel et al also reported a 76 year old woman with an oncocytoma of her lacrimal gland, that presented with a 2 month period of swelling of her lacrimal fossa without proptosis. After excision there was no regrowth during a follow up period of 3.5 years. Proliferations of oncocytic cells can also show malignant features and become malignant oncocytomas. A 58 year old man reported by Riedel had a malignant oncocytoma (synonym: oxyphilic adenocarcinoma) of the lacrimal gland. He had a 3 month history of proptosis of his eye and diplopia. On presentation the tumour had intracranial extension. 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 intracranial 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 oncocytic 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.

L J C Hartman, M P Maurit
Department of Ophthalmology, University Medical Center, Heidelberglaan 100, Utrecht, Netherlands

M R Conninga-van Dijk
Department of Pathology

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 20/20 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 peripapillary excavation of retina and retinal pigment epithelium surrounding the optic nerve seen on fundus examination and also tissue surrounding the optic nerve. Automated perimetry 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 peripapillary excavation of the retinal pigment epithelium (RPE) adjacent to the optic nerve (Fig 3). This was thought to be most consistent with MGDA.

Comment

Morning glory disc anomaly is a congenital anomaly of the optic disc that is typically unilateral for review see Brodsky’s. The majority of patients have a visual acuity between 20/200 and counting fingers in the affected eye, although cases with 20/20 vision and no light perception have been reported. It is more common in females than males and is less common in African-Americans than white people. 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-coloobra syndrome and trisomy 4q.

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 part of the disc and a variable amount of peripapillary pigment.

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 (Fig 1). Indeed, the peripapillary annulus of tissue surrounding the optic nerve spared a small area temporally from about 7 to 9 o’clock. In this region there was some pigment disturbance, but little if any apparent fibrosis, compared to the rest of the optic nerve. While a previous study of eight patients’ suggested there was “no correlation between optic disc configuration and visual acuity” there was no patient in that study with a documented visual acuity better than 20/100.

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 thin connective 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. There is controversy regarding the aetiology of MGDA. Some believe it is a form of optic disc coloboma. This theory is supported by evidence that MGDA is seen along a continuum of other optic disc anomalies including coloboma in the renal coloboma syndrome. Based on the findings of a scleral defect, vascular anomalies, central glial tuft, and adipose and smooth muscle tissue in histopathological specimens, it has been hypothesised that MGDA may be a primary mesenchymal disorder or an abnormality in the relative growth between the mesoderm and neuroectoderm. Another theory proposes that an abnormal enlargement of the distal optic stalk during eye development allows the inner layer of the optic cup to enter, causing an excavation at the entry site. One problem in determining the aetiology has been the lack of clinical confirmation (primarily a lack of fundus photography) in previous histopathological reports.

In this report we present OCT data that confirm these pathological findings in MGDA. Common to all of the histopathological reports is a layer of RPE that lines the peripapillary excavation. This histological feature is confirmed in the present case with OCT, which shows RPE extending posteriorly within the peripapillary scleral excavation as it approaches the optic nerve (Fig 3). We are currently evaluating other patients with MGDA using OCT and comparing these findings with the OCT appearances of other optic nerve anomalies, including optic disc coloboma.

MGDA is sometimes associated with a basal encephalocele or up to a third of patients with MGDA will develop a retinal detachment. Hence, the first step in the management of MGDA is recognising these associated conditions. Our patient did not have the characteristic facial features (flattened nasal bridge or cleft lip) nor did she have any neurological, endocrine, or respiratory symptoms to suggest she had a basal encephalocele and an magnetic resonance imaging confirmed its absence. Funduscopic examination showed no evidence of retinal detachment, and she will be followed carefully for this potential complication. Although this is an atypical case, with no central fibrous tuft and little peripapillary pigment, this patient demonstrates the peripapillary excavation characteristic of MGDA. To our knowledge this is the first report of OCT of an eye with MGDA and confirms previous histopathological reports of MGDA showing RPE lining the central peripapillary excavation. Ongoing studies are using OCT to quantify the changes that occur with MGDA over time and to compare the features of MGDA with those of other optic nerve anomalies, including optic disc coloboma.
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J N Newman
Departments of Ophthalmology, Neurology, and Neurosurgical Surgery

Correspondence to: Nancy J Newman, MD, Neuro-ophtalmology Unit, Emory Eye Center, 13658 Clifton Road, NE, Atlanta, GA 30322, USA; ophtnjn@emory.edu

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References


Idiopathic anterior hyaloid vessels

Anterior hyaloid fibrovascular proliferation (AHFP), the growth of vessels across the anterior hyaloid face from an origin in anterior retinala, was first described in phakic eyes after diabetic vitrectomy surgery; but also reported following cataract surgery in diabetics. Complications include cataracts, vitreous haemorrhage, tractional retinal detachment, ciliary body detachment, and phthisis bulbi. 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 opacities (Fig 1A). Clinical examination revealed no other ocular abnormality, but the temporal periphery of the right retina was obscured. Anterior segment fluorescein angiography confirmed perfusion of the vessels (Fig 1B). Posterior segment fluorescein angiography 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, Tzunara/Toxoplasma 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, fibrous 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. 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. Tzunara and Toxoplasma 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 radiographically. 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 hypercoagulable 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 ultrasonography, and no evidence of arteriovenous malformation was found in the eye or body. He had...
Iris pigment epithelial cyst induced by topical administration of latanoprost

Latanoprost is an ester produg analogue of prostaglandin F_2\alpha 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 both her eyes in April 1994. After then, 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 January 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 B 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 both her 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 after 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 intrapapillary 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.
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 (glial 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
Horners syndrome is caused by an oculosympathetic deficit to the pupiloliddor and superior and inferior tarsal retractor muscles.1 It is manifest by upper lid ptosis, ipsilateral miosis, apparent enophthalmos due to lower lid ("upside down") ptosis, and often facial anhydrosis.2 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 anhydrosis, was indicative of a preganglionic deficit to the pupilodilator and superior tarsal retractor muscles.3 Horner's syndrome is caused by an oculosympathetic deficit, 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.

Association of keratoconus and Avellino corneal dystrophy

Keratoconus is an idiopathic, progressive, non-inflammatory ectasia of the axial cornea. Its association 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 (R535W for granular corneal dystrophy, R535G mutation for Reis-Bückler’s corneal dystrophy, R124C mutation for Avellino corneal dystrophy type 1, and R124H mutation for Avellino corneal dystrophy).1 Molecular genetic analysis of various corneal dystrophies which had previously presented an insuperable challenge to clinical diagnosis, now clearly demonstrates the distinct phenotypes.2 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 42 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 spicular opacities...
were seen in anterior stroma (Fig 1, top). Computed corneal topography showed inferior steepening 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 βH3 gene by polymerase chain reaction (PCR) and directly sequenced the products. We detected a heterozygous G→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. There is only one case report in the literature of keratoconus and Avellino corneal dystrophy.

Some aspects of this work 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 presented clinically.

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

S Igarashi
Department of Ophthalmology, Asahikawa Medical College, Asahikawa, Japan

Y Makita
Department of Public Health, Asahikawa Medical College, Asahikawa, Japan

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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 aetiologies 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 by immunohistochemistry.

An antibody directed against vitronectin (Santa Cruz Biotechnology, Santa Cruz, CA, USA) exhibited intense reactivity with the infiltrating leucocytes and basal epithelial cell layer. Diffuse staining for vitronectin is also found in the deposits. Anti-vitronectin also appears to stain the superficial layer of corneal epithelial cells, although we cannot totally rule out the possibility that this might represent an edge artefact (haematoxylin counterstain, original magnification ×200).

Factor XII deficiency is associated with thrombosis.3,4 Severe deficiency increases an individual's prothrombotic tendency but with marked reduction in levels there is less certainty.1 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 incomitant 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 also been otherwise well. He was not hypertensive or diabetic, of normal weight but had been otherwise well. He was not sure what caused these episodes but felt confident that he had had the episodes for over the years. He was an active sportsman and had been involved in various sports but had not experienced any injury to the head.

On examination he was found to have hypermetropic astigmatism OD and myopic astigmatism OS. He was in good general health and not taking any medication. On ophthalmological examination his visual acuity was normal. A routine fundus examination was normal but after dilatation his near vision was found to have a left inferior band-shaped opacities occupying the inferior cornea. There is also neovascular infiltration from the superior limbus of the cornea, some of the vessels are indicated by arrows. (B) Light microscopic photograph of a corneal button section surgically excised from the 39 year old man. Histological section of this cornea shows eosinophilic amorphous material in the subepithelial region, and the overlying epithelium was degenerated. Note the prominent inflammatory infiltrate (haematoxylin and eosin, original magnification ×200). (C) Immunostaining of GDLD cornea with vitronectin showing immunopositivity in the infiltrating leucocytes and basal epithelial cell layer. Diffuse staining for vitronectin is also found in the deposits. Anti-vitronectin also appears to stain the superficial layer of corneal epithelial cells, although we cannot totally rule out the possibility that this might represent an edge artefact (haematoxylin counterstain, original magnification ×200).

Factor XII deficiency and recurrent sixth nerve palsy
Factor XII deficiency is associated with thrombosis.3,4 Severe deficiency increases an individual’s prothrombotic tendency but with marked reduction in levels there is less certainty.1 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.
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.

A Kipioti, O C Backhouse, P M Jacobs, M R Howard
Department of Ophthalmology, York District Hospital NHS Trust, York, UK

Correspondence to: A Kipioti, Eye Department, Clarendon Wing, Leeds General Infirmary, LS2 9NS, UK, tknapiloti@doctors.org.uk

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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 exophthalmos. With unilateral presentation one should think of orbital pseudotumour, orbital cellulitis, cavernous sinus thrombosis, or intraorbital neoplasms.1

Graves’ ophthalmopathy (GO) usually is associated with Graves’ hyperthyroidism (GH) although the temporal relation to thyroid disease is not consistent. It has been estimated that 77% of GO patients are hyperthyroid, 21% euthyroid, and 2% hypothyroid.2,3 In this case the interval between GH and GO is 3.3 years in men and 3.6 years in women. Two thirds of patients present with orbital symptoms within 18 months of diagnosis of thyroid disease. The following cases of Graves’ ophthalmopathy are described because of their unusual presentation: a long interval between thyroid disease and the development of predominantly unilateral Graves’ ophthalmopathy. Both cases were seen at the department of ophthalmology of the University Hospital Groningen, Netherlands.

Case 1
A female patient born in 1922 became hyperthyroid with minimal eye signs with possibly some lid retraction in 1948. She was treated by thyroidectomy and became clinically euthyroid. In 1988 an abnormal function antigen titre was tested because of complaints of dizziness. Thyroid hormones were within normal ranges and thyroxoperoxidase (TPO) antibody and antiparietal cell antibodies (APCA) were negative. In January 1983 she presented with complaints of sharp pains in both eyes, a feeling of pressure, and an exophthalmos of her right eye. On ocular examination there were no abnormalities except for exophthalmos of the right eye. Hertel exophthalmometer values were 24 mm right eye and 19 mm left eye. In December 1983 signs and symptoms worsened. Examination at that time showed an obvious retraction of the upper eyelid of the right eye. The exophthalmos was stable. There was diplopia caused by a right hypotropia in primary position of 1.5 degrees with restriction of elevation. A computed tomography (CT) scan showed enlargement of all external eye muscles of the right eye without involvement of the tendons. During this exacerbation laboratory testing showed thyroid hormone levels within normal ranges and again TPO and colloid antibodies tests were negative. The diplopia resolved without treatment.

Case 2
A male patient born in 1944 was diagnosed with hyperthyroidism in 1979. Thyroid hormones were abnormal. FT4 was decreased, TSH was increased, antibodies against colloid were positive, and APCA and TPO antibodies were negative. Because of this primary hypothyroidism levothyroxine therapy was started and he gradually became euthyroid. His ophthalmic history mentioned a disorder of the central retinal pigment epithelium and glaucoma which was treated with timolol.

Comment
Unilateral proptosis as a result of Graves’ disease cannot be rejected as a diagnosis, even 20 or 30 years after the onset of thyroid disease. Only one retrospective study among 557 patients mentioned intervals up to 25 years without giving exact numbers and underlying thyroid disease.7 For hypothyroidism and GO one study suggests intervals exceeding 15 years.3

Thyroid hormone testing should be performed to rule out abnormalities in thyroid hormone levels although thyroid status does not seem important as the active phase of ophthalmopathy may occur during hyperthyroidism, hypothyroidism, and euthyroidism.1 Thyroid antibody testing may be supportive for the diagnosis. A CT scan can be essential in further analysis showing enlargement of extraocular muscles with sparing of the tendons.2 It is also known that a CT scan can demonstrate contralateral eye muscle involvement in 50–90% of patients with clinically unilateral eye involvement.2

Our two patients illustrate that the medical history is important in evaluating proptosis. One should always think of Graves’ disease as a possible cause of unilateral exophthalmos even though a patient may have had thyroid disease more than 20 years earlier.

In August 2000 he presented with unilateral proptosis and progressive loss of vision. Ocular examination showed chemosis and oedema of the eyelids in both eyes. Visual acuity was 20/60 right eye and 20/30 left eye. Papillary reflexes were normal and symmetrical. Clinically and biochemically he presented as euthyroid. A CT scan showed bilateral enlargement of the eye muscles, more marked on the right side. He was treated with 60 mg prednisone daily.

One month later visual acuity of the right eye dropped further and he was referred to our hospital. Ocular examination showed marked soft tissue signs, worse in the right eye, bilateral chemosis, and unilateral exophthalmos of 26 mm in the right eye. Visual acuity was 20/400. Another CT scan was performed which showed enlargement of all recti muscles of right eye and in lesser extent of the left eye with signs of compression of the right optic nerve. An orbital decompression through a Caldwell–Luc approach was performed on the right side. Postoperatively, visual acuity of the right eye improved to 20/30 and proptosis in the right eye was reduced by 8 mm. A strabismus convergens of the right eye was the main side effect.

In April 2001 a bilateral medial recession and a recession of the inferior rectus of the right eye were performed to treat the strabismus convergens. Binocular single vision was achieved in the primary position with some diplopia at extremes of gaze.
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/l and viral load 1,300,000.

Unusual presentation of cat scratch disease in HIV+ patient

Intraocular cat scratch disease may present with different clinical features including neuroretinitis, retinitis, retinal infiltrates, arterial and vein occlusions. Most of the cases show spontaneous recovery without therapy. There are only few reports of intraocular cat scratch disease in HIV+ patients. We report an unusual case of cat scratch disease presenting as helioid unilateral choroiditis in an HIV+ patient that showed good response to systemic therapy.

Case report

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/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 angiomatous lesion corresponding to those seen clinically. Blood tests were ordered including VDRL, toxoplasmosis serology, and treponemal antibodies which were all non-contributory. Two weeks later, the patient was referred 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 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, 1:1256, 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 is a well established association between neuroretinitis and cat scratch disease although many different clinical presentations have been described. Ormerod et al. described two patients with small areas of retinitis and arterial occlusions. Pollock and Kristinsson described one patient with cat scratch disease and helioid unilateral choroiditis. Hong et al. first described this syndrome when they reported six young patients with a solitary round yellow chorioretinal lesion associated with subretinal fluid. There was no association with inflammatory or infectious diseases. Fish et al. reported a case of peripapillary angiomatosis associated with retinal pigment epithelium. Our patient presented with clinical features of helioid unilateral choroiditis but after angiogram we could see an angiomatous-like lesion.

The treatment of ocular cat scratch disease remains controversial. Pollock and Kristinsson reported a case that improve-ment 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 unilateral choroiditis and angiomatous-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.

References


Simultaneous translocation of the macula and underlying retinal pigment epithelium during macular translocation surgery in a patient with long standing myopic neovascular maculopathy

Limited macular translocation has been reported to be a promising treatment for some patients with choroidal neovascularisation. Although this technique has the advantage of being less invasive, there is documentation of...
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 correct 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 juxtafoveal 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 consultation and surgery. With her consent, the patient decided to have surgical treatment. With her consent, the patient's visual acuity has not improved. During a follow-up period of 15 months, it has remained at the same level as her preoperative vision.

Postoperatively, fluorescein angiography showed an extrafoveal neovascular membrane with a foveal shift of 0.7 disc diameter (Fig 1B). Sharply demarcated hypofluorescence in the macular area was also demonstrated. Biomicroscopic examination revealed a slightly hyperpigmented lesion underneath the translocated macula, which corresponded to the area of hypofluorescence. A horizontal optical coherence tomography (Humphrey Systems, San Leandro, CA, USA) section taken through the translocated macula displayed highly reflective double layers (Fig 2A). Two reflective bands observed on an optical coherence tomography scan were highly reflective double layers underneath the fovea (Fig 2B). Despite sufficient foveal displacement, the patient's visual acuity has not improved. During a follow-up period of 15 months, it has remained at the same level as her preoperative vision.

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 the neurosensory retina may adhere firmly to the subjacent tissue. In such cases, an inner portion of the 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 role 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 considering 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.

M Ichibe, K Imaizumi, M Ohji, Y Oya, T Yoshizawa, H Abe
Department of Ophthalmology, Niigata University School of Medicine, Niigata, Japan

Reference

Fungal and bacterial chronic endophthalmitis following cataract surgery
Endophthalmitis, although rare, is one of the most vision-threatening complications 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 periocular 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 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 albicans, 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 a light perception and biomicroscopic examination showed panophthalmitis. The patient refused medical treatment and enucleation was performed.


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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 group 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.11 In our study, all the biopsies performed by ophthalmologists were done as extra cases in theatre 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 neuro- logical 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.

K Fong, V Ferguson
The Western Eye Hospital, Marylebone Road, London NW1 2YY

Correspondence to: Dr Kenneth Fong; fongcck@yahoo.co.uk

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Major complications of endoscopic sinus surgery: a comment
We were pleased to read the informative article by Rene et al. The authors report the high incidence of postoperative complications.1 Endoscopic sinus surgery may result in the formation of ethmoid and 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 “sphenoidethmoidal” cell.2

The main significance of the Onodi or sphenoidethmoidal 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 extensive pneumat- ised Onodi cells.3 The exact incidence of Onodi cells is unclear. Endoscopic dissection studies suggest an incidence as high as 59% or 42%. CT imaging studies suggest a lower incidence of 7%. Driben et al. feel that the likelihood of a sphenoidethmoid 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

We are also concerned that the use of intra-operative antimitabolites 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 excellent early success rate of viscosanalostomy (95% 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 injection. All use of mitomycin-C and 5-fluorouracil improves the success rate of glaucoma filtering surgery by reducing episcleral fibrosis,3 and probably explains the difference in success rates in this study. We wonder what the results would have been had the use of intra-operative antimitabolites been used in all groups, or if none was used at all.

In contrast with the authors’ technique, we employ Stegmann’s approach to viscosanalostomy in using a parabolic superficial flap secured tightly with five sutures in a watertight fashion. Although early blebs may develop, most patients achieve IOP lowering through multiple alternate pathways including uveoscleral, through Schlemm’s canal, and subconjunctival.5 Certainly in higher risk cases, we employ antimitabolites for antimitabolites in non-penetrating surgery, and have found it to be safer than 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 wick or hyaluronic acid implant with an intraoperative antimitabolite in higher risk cases to obtain optimal IOP control.

Fibrosis and loss of permeability of the trabeculo-descemet’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 adjunc- tive procedure and may be needed in up to 41% of non-penetrating procedures.6 It has been reported to successfully lower postoperative IOP in over 80% of cases.7 Yet, we are dis- mayed that the authors decided not to attempt laser gonipuncture in those viscosanalostomy cases with postoperative IOP eleva- tions because “such interventions clearly con- vert 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 early cautery lysis in trabeculotomy cases. Although some authors feel that performing suture lysis constitutes conversion of a guarded trabeculec- tomy into a full thickness unguarded trab- eculectomy and thus cannot be fairly compared to a TDW.8 Although myotomy 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 antimitabolite is superior to a modified form.
of viscocanalostomy dependent on subcon- 
junctival filtration without the use of an 
antimetabolites. A randomised, prospective study comparing 
trabeculectomy will certainly have 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.

I K Ahmed, C Kranemann
University of Toronto, Toronto, Ontario, Canada

Correspondence to: I K Ahmed, 43 Bywood Drive, 
Toronto, Ontario M9A 1M1; ike.ahmed@utoronto.ca

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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 techni- 
que 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 
argon laser treatment of trichiasis, and the 
aim to achieve destruction of the subcutaneous 
lash and its follicle.

Secondly, the article by Bartley and Lowry 
quoted by the authors, describing using a “drop 
of ink from a fountain pen” to facilitate lash 
laser. 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 us in 1994,2 we found that trans- 
ferring 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 
aesthetic 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 magnifi- 
cation for the accurate sitting of the trans- 
fused blood.

G A Wilcsek, I C Francis
The Ocular Plastics Unit, Prince of Wales Hospital, 
and the University of New South Wales, Randwick, 
Sydney, Australia

Correspondence to: Ian C Francis; 
ili@student.unsw.edu.au

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Management of age related 
macular degeneration: still room for improvement

The aim of Mitchell et al’s study1 in collabora- 
tion with the Macular Disease Society (MDS) 
was to assess the perceived quality of health 
care of people with macular disease in the 
United Kingdom. This 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 in 
collaboration with the MDS 7 years earlier.2 This also took the form of a retrospec- 
tive 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 (ARM) in the United 
Kingdom from the patients’ perspective. The 
final response rate was 79% (similar to 77% of 
Mitchell et al1 and patients had attended over 
100 ophthalmic units throughout the United 
Kingdom. Mitchell et al omitted this early 
study from their discussion but comparison of 
the two studies raises some noteworthy 
points.

The prevention1 and treatment2 of ARM 
remains far from ideal both for the majority of 
patients and their ophthalmologists. Members 
of the MDS are likely to be a highly moti- 
vated study group and it is not clear how their 
views will reflect the views of patients as a 
whole with ARM. However, both studies 
highlighted the fact that lack of information 
and advice on ARM is a significant cause of 
dissatisfaction for these patients. Mitchell et al1 
asked if the diagnostic consultation with the 
eye specialist was satisfactory: 59% responded 
“yes” and 41% “no.” The two most common 
reasons given for a negative answer were the 
attitude of the specialist and lack of informa- 
tion and advice (43.5% and 43.4% respecti- 
vely of those answering this question). The 
earlier study specifically asked whether pa- 
tients were given written information on 
ARM, were informed about support groups 
or informed about relevant radio programmes 
by the eye department.3 Only 6% received 
written information, 14% were given details 
of support groups, and 3% details of relevant 
programmes. By definition 100% of the study 
group would have liked to have been in- 
formed of the existence of the Macular Disease Society by the eye unit. However, nine 
different sources were cited for discovering 
the MDS; over 80% from radio and magazines 
with only 12% from the ophthalmic unit. 
Unfortunately, despite that fact that the two 
studies are separated by 7 years, any temporal 
comparison has to be undertaken with 
cautions. The protocol of both surveys included 
questionnaires being sent to members of the 
MDS chosen at random from the MDS 
mailing list. It is therefore conceivable that a 
proportion of the original study group also 
formed part of the second, but is not stated. 
What is clear though, is that despite the fact 
that patients want information on ARM and 
the fact that there are readily available sources 
(for example, free information booklets on ARM 
from the Royal College of Ophthalmolo- 
mists or Royal National Institute for the 
Blind), ophthalmic units throughout the 
United Kingdom are still failing routinely to 
provide such information to their patients.

L J Howe
Moorfields Eye Hospital, City Road, London 
EC1V 2PD, UK; lucy.robson@btpenworld.com

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Standardised clinical 
photography in ophthalmic 
plastic surgery

We read with keen interest the path breaking 
article by Califia et al regarding the successful 
treatment of an eyelid squamous cell carcino-
toma with intrallesional cidofovir.4 The dra- 
matic response of this highly malignant lesion to 
such a non-invasive form of therapy is 
remarkable and certainly worth further clini- 
cal evaluation. However, ARMD 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 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 indisputable reduction in 
the size of the tumour, the two clinical photo- 
graphs are not strictly comparable since a 
decrease in magnification results in visual 
clues that lead to the lesion being perceived as 
smaller in size.
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 pain relief. Based on our personal 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 importance 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.

**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 39 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 were not identified by the authors in their study. This is very probably insufficient time for DNA fragmentation to occur at the level we observed. Moreover, the innocence of injections of trypan blue into the anterior chamber, a common feature during cataract surgery, has been well demonstrated.\(^2\)

(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 (0.9% NaCl) nor the short exposure time (about 1 minute) nor the short incubation in the presence of 0.9% NaCl has ever been incriminated 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.\(^3\)

(2) The time spent outside the organ culture medium before fixation is quite short, which has caused an artefactual increase in the percentage of TUNEL positive ECs. Two arguments counter this remark.

**Dynamics of corneal endothelial cell death in organ culture**

We read with interest the remarks of Crowston et al.\(^4\) on our article.\(^5\) They showed that the TUNEL technique revealed a far higher percentage of endothelial cell (EC) death 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 fixation 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 (0.9% NaCl) nor the short exposure time (about 1 minute) nor the short incubation in the presence of 0.9% NaCl has ever been incriminated 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.\(^3\)

(2) The time spent outside the organ culture medium before fixation 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 recently published.\(^7\) 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 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.
The second remark by Crowston et al is particularly interesting. We too were surprised by the high percentage of TUNEL positive ECs (mean 12.7%, SD 16.4). This may imply that all the cells died within 8 days, which was evidently not the case. We believe this apparent contradiction can be explained by the following theory. The TUNEL staining is positive during a relatively long window (24–48 hours). The TUNEL index, measured at a given moment, provides a global view of all the cells with fragmented DNA. However, the DNA fragmentation may be at different stages, and the cells very likely spread according to a Gaussian distribution. Therefore the cells, which are TUNEL positive at a given moment, will not all die instantaneously and simultaneously. Only the cells furthest to the right on the curve will die in the very short term, and it is probably these that are liable to be correctly counted if it were possible to perform TUNEL on two consecutive days, but the percentage of positive cells revealed would probably be very similar, but a large majority of the positive cells recorded on the second day would have already been counted on day one. It is, however, undeniable that the cells that are TUNEL positive at a given moment will all die eventually. In other words, we believe that, at the end of storage, corneas contain a number of ECs engaged in an irreversible cell death process far more extensive than the highly unreliable trypan blue staining technique suggests.

G Thuret, C Chiquet, L Campos, P Gain
Cell Death and Neoplasia Laboratory EA 3063, University Hospital, St Etienne, France
Correspondence to: Gilles Thuret; gilles.thuret@univ-st-etienne.fr

References

NOTICES

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.

For further information please contact: Journal of Community Eye Health, International Centre for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0)20 7608 6910; fax: +44 (0)20 7250 3207; email: eyeresource@ucl.ac.uk; web site: www.jceh.co.uk). Annual subscription (4 issues) UK£25/US$40. Free to workers in developing countries.

International Centre for Eye Health

The International Centre for Eye Health has published a new edition of the Standard List of Medicines, Equipment, Instruments and Optical Supplies (2001) for eye care services in developing countries. It is compiled by the Task Force of the International Agency for the Prevention of Blindness. Further details: Sue Stevens, International Centre for Eye Health, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0)20 7608 6910; email: eyeresource@ucl.ac.uk).

Second Sight

Second Sight, a UK based charity whose aims are to eliminate the backlog of cataract blind in India by the year 2020 and to establish strong links between Indian and British ophthalmologists, is regularly sending volunteer surgeons to India. Details can be found at the charity web site (www.secondsight.org.uk) or by contacting Dr Lucy Mathen (lucymathen@yahoo.co.uk).

Specific Eye ConditionS (SPECS)

Specific Eye ConditionS (SPECS) is a not for profit organisation which acts as an umbrella organisation for support groups of any conditions or syndrome with an integral eye disorder. SPECS represents over fifty different organisations related to eye disorders ranging from conditions that are relatively common to very rare syndromes. We also include groups who offer support of a more general nature to visually impaired and blind people. Support groups meet regularly in the Boardroom at Moorfields Eye Hospital to offer support to each other, share experiences and explore new ways of working together. The web site www.eyesconditions.org.uk acts as a portal giving direct access to support groups own sites. The SPECS web page is a valuable resource for professionals and may also be of interest to people with a visual impairment or who are blind. For further details about SPECS contact: Kay Parkinson, SPECS Development Officer (tel: +44 (0)1803 524238; email: k@eyesconditions.org.uk; web site: www.eyesconditions.org.uk).

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: 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-muennerberg.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 Oliver, tel: +44 (0)121 424 3646; fax: +44 (0)121 424 4464; email: Martid@hotmail.com.

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 before the submission of abstracts. The deadline for abstracts is 14 February 2003.

Further details: Ortopedice Centrum, s.r.o., Strekovské nabrezi 51, 400 03 Uhersk Hradec, Czech Republic (tel: +420 47 521 6588; fax: +420 47 533 40 77; email: ortcentrum-ul@volny.cz; web site: www.ortopedice-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 Razie Conference Center, Hammat Byow, Tehran, Iran. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Breuningerbau, 72076 Tuebingen, Germany (tel: +49 7071 293209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Masheychi, Dr Siamak Moradian, Dept of Ophthalmology, Labbanfinejad Medical Center, Pasdaran Ave, Boosan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labafi@hotmail.com).

PostScript

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