Intravitreal injection of triamcinolone acetonide as treatment for chronic uveitis

Chronic intraocular inflammation such as chronic 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 due to systemic suppression of the whole immune system and Cushing's syn-drome. 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 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 1999. 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.

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 Machemer, 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 address the relative efficacy of different types of uveitis and determine whether injection of intravitreal 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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References

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 therefore be desirable to have a non-invasive method allowing the estimation of the intracerebral pressure without requir- ing 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 2/20 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 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 82.3 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 exiting 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. 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. \(^1\) 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. \(^3\) 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 intraocular pressure, the new, Goldmann lens associated, ophthalmodynamometer may provide information about the intraocular pressure by estimating the central retinal vein collapse pressure. These measurements may be helpful for the neuro-ophthalmological diagnosis of diseases associated with increased intracerebral pressure.

Proprietary interest: none.

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). \(^1\) 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 of eyelid skin of patients with mild atopic blepharitis. Among various moisturizing 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, moisturizing of the skin using ceramide gel application represents a useful supplementary therapy during periods of relatively light inflammation.

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References
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). Fundoscopic examination showed 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 oncocyic cells. The tumour was removed in toto via a transcutaneous approach.

For unknown reasons oncocytic lesions begin in the conjunctiva and rarely in the lacrimal gland. 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,1,4,5 or as an oncocytic carcinoma.6

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,1,4,5 or as an oncocytic carcinoma.6

Beskid and Zaracka described a 39 year old woman with an oncocytoma of the lacrimal gland. Riedel et al.7 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.7 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 oncocyic cells can also show malignant features and become malignant oncocytomas. A 58 year old man reported by Riedel7 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 radiotherapy therapy, he died 6 months later from liver metastases.

References


Morning glory disc anomaly: an atypical case

When optic disc elevation is encountered on funduscopy 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 funduscopy 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...
anomaly of the optic disc that is typically uni-

Morning glory disc anomaly is a congenital

If found is not unexpected, as the visible peripap-

Figure 1 Photograph of the patient’s right optic disc. An elevated annulus of apparent fibroglial tissue surrounds most of the disc although it appears to spare part of the papillomacular bundle. An excavation of retina and retinal pigment epithelium surrounding the optic disc can be appreciated from about 12 o’clock to 7 o’clock.

eexamination, slit lamp biomicroscopy, in-

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 cen-

tral fibrous tuft that obscures the central area of the disc and a variable amount of peripap-

Comment

Morning glory disc anomaly is a congenital anomaly of the optic disc that is typically uni-
lateral (for review see Brodsky). 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 re-

Figure 2 Fluorescein angiography of the patient’s right eye. Both early (left; 32.6 seconds after injection) and late (right; 6 minutes and 14 seconds after injection) images show no evidence of fluorescein leakage outside the disc margin.

Another atypical feature of our patient is the small amount of peripapillary pigment seen in the affected eye. The only area of pig-

Figure 3 Optical coherence tomography (OCT) of the patient’s right optic nerve. Representative OCT shows retinal pigment epithelium lining an excavation surrounding the optic nerve, characteristic of morning glory syndrome.

eating 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 elevated ring from about 12 o’clock to 7 o’clock. The macula and periphery were normal. 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 reti-

nal pigment epithelium (RPE) adjacent to the optic nerve (Fig 3). This was thought to be most consistent with MGDA.

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 con-
tinuum 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 his-

topathological specimens, it has been hypoth-

suggested that MGDA may be a primary mesen-

chymal disorder or an abnormality in the relative growth between the mesoderm and ectoderm. **Another theory is that MGDA is an abnormal enlargement of the dural optic stalk during eye development allows the inner layer of the optic cup to enter, causing an excursion at the entry site.** One problem in determining the aetiology has been the lack of clinical confirmation (primarily a lack of fun-

dus photography) in previous histopathologi-

tal reports. **In this report we present OCT data that confirm these pathological findings in MGDA.** Common to all of the histopatho-

logical reports is a layer of RPE that lines the peripapillary excavation. This histological fea-

ture 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 find-

ings with the OCT appearances of other optic nerve anomalies, including optic disc colo-

boma.

MGDA is sometimes associated with a basal encephalocele and 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 (fla-

ttened nasal bridge or cleft lip) nor did she have any neurological, endocrine, or respira-

tory 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 care-

fully 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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Idiopathic anterior hyaloid vessels

Anterior hyaloid fibrovascular proliferation (AHFP), the growth of vessels across the anterior hyaloid face from an origin in anterior retina, was first described in phakic eyes after diabetic vitreoretinopathy; 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, 6/5 left. Vessels were visible on the right posterior lens capsule, associated with localised cataract and anterior vitreous opacification (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, Toxocara/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 arcade 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. Toxocara 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 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 ultrasonography, and no evidence of arteriovenous malformation was found in the eye or body. He had...
no features of incontinentia pigmenti or facio-cutaneous 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 definite 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 AHFPP.

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Iris pigment epithelial cyst induced by topical administration of latanoprost

Latanoprost is an ester prodrug analogue of prostaglandin F α that enhances uveoscleral outflow and reduces intraocular pressure.1 Several adverse side effects associated with topical administration of latanoprost have been described.2 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.3 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 a β 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 hyperacrnia 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 a 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.2 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 after uveitis,2 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 (PNPET) arising from the cervical paravertebral region of a 34 year old woman, who presented with Horner’s syndrome and a cervical radiolucent mass. PNPETs are rare malignant small round cell tumours. This appears to be the first documented case of localised PNPET with Horner’s syndrome at initial presentation.

Case report

A 34 year old woman presented with acute left scapula 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 foramen 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 extraspinal component of the tumour within the

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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

Horner’s syndrome is caused by an oculosym pathetic deficit to the pupillodilator 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.1,2 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.1 The most common location is the thoracopulmonary region, followed by the head and neck. Extraosseous 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/T1 cervical radiculopathy is classically seen in Pancoast’s syndrome, which is due to superior sulcus tumours of the lung that involve the thoracic apex, resulting in death at 17 weeks following initial diagnosis.1,8 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.

Acknowledgement

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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, Muñier 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, R535Q mutation for Reis-Bückler’s corneal dystrophy, R1246 mutation for keratoconus, and R124H mutation for Avellino corneal dystrophy type 1, and R124H mutation for Avellino corneal dystrophy type 2).17 Molecular genetic analysis of various corneal dystrophies which had previously presented an insuperable challenge to clinical diagnosis, now clearly demonstrates the distinct phenotypes.17 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 20/30 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

References

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 beta3 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 in codon 124 from arginine to histidine in this patient (Fig 1).

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.

Comment

To our knowledge, this is the first molecular genetic report of a bilateral association of keratoconus with Avellino corneal dystrophy.

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 mulberry-like opacity is present with protuberant 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.
vitronectin in the GDLD cornea may also have
we further hypothesised that the accumulated
macular degeneration and amyloidosis. This
molecule, is often a component of the abnor-
multifunctional extracellular matrix adhesion
sary for new blood vessel growth,
supporting cell proliferations that are neces-
sary matrix proteins has a crucial role in
deposits (Fig 1B, C).

It was recently reported that vitronectin, a
multifunctional extracellular matrix adhesion
molecule, is often a component of the abnor-
mal 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.6 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 leukocytes and corneal epithelium
around the deposits. Substantial levels of vit-
ronectin protein were also present in the
deposits (Fig 1B, C).

Because the production of new extracellu-
lar matrix proteins has a crucial role in
supporting cell proliferations that are neces-
sary for new blood vessel growth,4 and
because vitronectin receptors, such as integrin
αvβ3 and αvβ5, are involved in angiogenesis,6 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 (Inter-
feron Sciences, New Brunswick, NJ, USA)
containing 1 µg of mouse vitronectin (Invitro-
gen, 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 administra-
tion 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 angi-
genesis 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 MISO leads to accumulation
of vitronectin with more samples, may even-
tually offer a novel insight in understanding
the aetiology of corneal NV associated with
GDLD.

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Involvement of interleukin-8, vascular
endothelial growth factor, and basic fibroblast
growth factor in tumor necrosis factor
alpha-dependent angiogenesis. Mol Cell Biol

Factor XII deficiency and
recurrent sixth nerve palsy

Factor XII deficiency is associated with
thrombosis.7,8 Severe deficiency increases an
individual’s prothrombotic tendency but with
moderate reduction in levels there is less
certainty.9 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 male presented with an
acquired constant incontinent horizontal di-
diplopia. He had had a previous episode of hori-
zontal diplopia of 3 weeks’ duration 6 months
previously with spontaneous resolution and a
further similar episode 18 years before that
but had been otherwise well. He was not
hypertensive or diabetic, of normal weight
and a non-smoker, and without any cardio-
vascular disease. There was no family or
personal history of venous or arterial throm-
bosis. On examination he was found to have
bilateral sixth nerve palsies without any asso-
ciated 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 presenta-
tion, a further prothrombotic examination
was undertaken. Laboratory investigations
showed a normal full blood count, plasma vis-
cosity, liver function tests, glucose, homo-
cysteine, 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 se-
vere factor XII deficiency which was consist-
ent on repeated testing (<1% of normal
levels) but otherwise normal levels of protein
S, protein C, antiphospholipid antibodies, fac-
tor VIII, von Willebrand factor, and the factor
V Leiden mutation or prothrombin G20210A
alleles were not found.

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tor VIII, von Willebrand factor, and the factor
V Leiden mutation or prothrombin G20210A
alleles were not found.
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.1 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.2 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.3 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.4 Since his cranial nerve palsy resolved quickly and there was no family history of vascular thrombosis he was treated empirically with warfarin 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 exophthalmus among adults is Graves’ disease. Unilateral exophthalmus, although frequently seen in connection with thyroid diseases, has a much larger differential diagnosis than bilateral exophthalmus. 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 In the untreated 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.3 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 thyroideotomy and became clinically euthyroid. In 1988 she noticed blurry vision and function and diagnosis of thyroid disease were tested because of complaints of dazziness. Thyroid hormones were within normal ranges and thyreroxiprotein (TPO) colloid and antinuclear antibodies (APCA) were negative. In January 1983 she presented with complaints of sharp pains in both eyes, a feeling of pressure, and an exophthalmus of her right eye. On ocular examination there were no abnormalities except for exophthalmus 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 again showed an obvious retraction of the upper eyelid of the right eye. The exophthalmus was stable. There was diplopia caused by a right hypotropia in primary position of 1.5 degrees with restriction of elevation. A computed tomograph (CT) scan showed enlargement of all external eye muscles of the right eye without involvement of the tendons. During this exacerbaration laboratory testing showed thyroid hormone levels within normal ranges again TPO and colloid antibodies tests were negative. The diplopia resolved without treatment.

Case 2
A male patient born in 1944 was diagnosed with hypothyroidism 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 oculary history mentioned a disorder of the thyroid, 21% euthyroid, and 2% hypothyroid. In the untreated

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. Pupillary reflexes were normal and symmetric. 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 exophthalmus of 26 mm in the right eye. Visual acuity was reduced and 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.

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 of patients mentioned intervals up to 25 years without giving exact numbers and underlying thyroid disease.4 For hypothyroidism and GO one study suggests intervals exceeding 15 years.5 Thyroid hormone testing should be performed to rule out abnormalities in thyroid hormone levels although thyroid status does not seem important as the inactive phase of ophthalmopathy can occur during hyperthyroidism, hypothyroidism, and euthyroidism.6 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.7 It is also known that a CT scan can demonstrate contralateral eye muscle involvement in 50–90% of patients with clinically unilateral eye involvement.8

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.

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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 whose lesion enlarged without treatment. Although we did not have the 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 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.

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 arteriolar occlusions. Pollock and Kristinsson 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 reported a case of peripapillary angiomatosis associated with neuroretinitis. Our patient presented with clinical features of helioid unifocal choroiditis but angiogram we could see an angiomatic-like lesion.

The treatment of ocular cat scratch disease remains controversial. Pollock and Kristinsson reported a case that improve 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 achieved myopic neovascular maculopathy.

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

Figure 1 Fundus photograph of yellowish choroidal lesion surrounded by fluid and haemorrhages.

Figure 2 (A) Fundus photograph at second visit, yellowish lesion increased. (B) Anteriovenous phase fluorescein angiogram showing an angiomatous-like lesion.
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, limited macular translocation was performed on her right eye, as described previously. 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 image may have corresponded to the native retinal pigment epithelium and abnormal retinal pigment epithelium translocated with the macula. These findings may indicate that the abnormal subfoveal retinal pigment epithelium, which adhered tightly to the overlying neurosensory retina, probably because of the long history of neovascular maculopathy, was translocated with the macula during surgery. Indocyanine green angiography findings supported this speculation (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 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 fulfill 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.

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. 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 were 2+ cells. Intracanal pressure was normal. Examination of the right eye was normal except for a vitreous showed 2+ cells. The vitreous showed 2+ cells. However, it can cause infectious crystalline keratopathy or, more typically, suppurative granulopathy.

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. However, it can cause infectious crystalline keratopathy or, more typically, suppurative granulopathy.

In the present case, Candida parapsilosis could be considered as a true pathogenic organism because of its localization, 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. 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. 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, particularly subungal spaces, with Candida parapsilosis being those most frequently recovered.

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. 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 performed 38 biopsies (31%). Both groups had similar positive biopsy rates—a difference of 15% (10/54) compared with 14/38 (37% for ophthalmologists, 14/40 (35%) for the GVS group. 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 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 trained 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 in 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 neurological and rhematological 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 rhematologists.

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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 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. 2 More recently it has been suggested that this cell is better characterised as a “sphenohethmoidal” cell. 3 The main significance of the Onodi or sphenohethmoidal 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 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 neurological and rhematological 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 rhematologists.

We are also concerned that the use of intraoperative antmitabolites in the trabeculectomy group added a major confounding variable in this study. This is particularly perplexing as the authors’ viscoanalostomy technique primarily relied on subconjunctival filtration, as evidenced by their scleral flap design and lesser suturing technique in which only three 10/0 nylon sutures were used. Furthermore, their excellent early success rate of viscoanalostomy (95% at 6 months), the presence of filtering blebs in their successful viscoanalostomy procedures, the lack of one in their failures, and the need for postoperative bleb needling and 5-fluorouracil injections all argue that use of mitomycin-C and 5-fluorouracil improves 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 antmitabolites been used in all groups, or if none was used at all.

In contrast with the authors’ technique, we employ Stegmann’s approach to viscoanalostomy in using a parabolic superficial flap secured tightly with five sutures in a relatively watertight fashion. 4 Although our success rates 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 feel it is reasonable to use antmitabolites in non-penetrating surgery, and we have found it to be safer than when used with trabeculectomy.

We would also recommend that quite often we do rely on subconjunctival filtration in nonpenetrating procedures (that is, deep sclerectomy) but advocate the use of a collagen wick 6 or hyaluronic acid implant with an interoperative antmitabolite in 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 goniodiopuncture of the TDW in these cases is a relatively easy adjunctive procedure and may be needed in up to 41% of non-penetrating procedures. 7 It has been reported to successfully fully lower IOP in over 80% of cases. 8 Yet, we are dismayed that the authors decided not to attempt laser goniodiopuncture in those viscanostomy 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 goniodiopuncture 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 performing suture lysis in trabeculo-Decemet’s window. The authors feel that performing suture lysis constitutes conversion of a guarded trabeculectomy into a full thickness unguarded trabeculectomy and thus cannot be fairly compared. Although suture lysis 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 antmitabolite is superior to a modified form.
of viscocanalostomy dependent on subconjunc-
tival filtration without the use of an anti-
metabolite. This study unfortunately does not 

fairly compare the efficacy of Sahni’s viscocanalostomy technique versus trab-
ceucalotomy. Non-penetrating glaucoma sur-
gery has 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

1 O’Brart DPS, Rowlands E, Islam N, et al. A 

randomised, prospective study comparing 

trabeculectomy augmented with 

anti-metabolites with a viscocanalostomy 

technique for the management of open angle 

glaucoma uncontrolled by medical therapy. Br 


2 Sahni and Clark have used the ink from a 

“blue skin marker 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, 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 

anaesthetic infiltration. We usually transfer it 

by picking it up with a sterile drawing up nee-

dle. This is achieved remarkably easily on 

the laser slit lamp, which allows adequate magni-

fication for the accurate sitting of the trans-

ferred blood.

A G Wilsec, I C Francis

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Sydney, Australia

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Management of age related 

macular degeneration: still room for improvement

The aim of Mitchell et al’s study 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. A patient baseline evalu-

ation and audit by postal survey of 200 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, baseline survey. 

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 Ophthalm-

ologists 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


Perceived quality of health care in macular 
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Macular Disease Society. Br J Ophthalmol 


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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 carcino-

ma with intralidodiol. 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 docu-
mntation of this case.

The authors have presented an extreme close in of the affected eye (Fig 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 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.

PostScript
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 were small and did not show either of the above findings. The authors administered Entonox throughout the procedure and we noted that although acute ophthalmic artery thrombus formation with obstruction of flow can be seen in 11% of asymptomatic males aged 40–59 years, also a potential consequence of errant injections during the administration of anaesthesia for ophthalmic surgery.

References


Entonox as an analgesic agent

We read with interest the remarks of Crowston et al on our article. 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 (0.4%) 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.

(2) The time spent outside the organ culture medium before fixing in formaldehyde, a common feature during cataract surgery, has been well demonstrated.

We would like to clarify these points of discrepancy, which have arisen due to dissimilar photographic techniques.

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 photographs 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


Dynamics of corneal endothelial cell death in organ culture

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References

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 the high percentage of TUNEL positive ECs is 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 really blue. If it were possible to perform TUNEL on two consecutive days, 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 probably be very similar, but a large majority of the percentage of positive cells revealed would be truly blue. If it were possible to perform TUNEL on two consecutive days, 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 probably be very similar, but a large majority of the percentage of positive cells revealed would be truly blue. If it were possible to perform TUNEL on two consecutive days, 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.

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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 7230 3207; email: eyeresource@ucl.ac.uk; web site: www.jech.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 condition or syndrome with an integral eye disorder. SPECS represents over fifty different conditions 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.eyeconditions.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@eyeconditions.org.uk; web site: www.eyeconditions.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 orbit, lacrimal and lid surgery. Further details: MGN Medizinische Congress organisation Nürnberg AG, Zerzabelshofstr 29, 90478 Nürnberg, Germany (tel: +49 911 3931621; fax: +49 911 3931620; email: doc@mcngaz.info; web site: www.doc-nuernberg.de).

3rd British Oculoplastic Surgery Society Meeting
The 3rd British Oculoplastic Surgery Society Meeting will be held 18–19 May 2003 in Birmingham, UK. For further details please contact the Secretary of the British Oculoplastic Surgery Society. Jane Oliphant (tel: +44 (0)121 424 3564; fax: +44 (0)121 424 4464; email: MartiDI@heartso1.wmids.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: Ortopedické Centrum, s.r.o., Střešovské nábřeží 51, 400 01 Ústí nad Labem, Czech Republic (tel: +420 47 521 6588; fax: +420 47 533 40 77; email: ortcentrum-ul@volnv.cz; web site: www.ortopedické-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 Razavi Conference Center, Hemmat Byr, 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 Mashehchi, Dr Siamak Moradian, Dept of Ophthalmology, Labbanfiranejad Medical Center, Fardaran Ave, Boosan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbafi@hotmail.com).

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