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LETTERS TO THE EDITOR

Indocyanine green angiography in choroidal tuberculomas

EDITOR,—An 85 year old white woman presented with progressive asthenia, fever, coughing, and dyspnoea. Chest roentgenogram showed interstitial pulmonary infiltrates and right pleural effusion. Cultures of the bronchoalveolar lavage fluid subsequently confirmed the presence of *Mycobacterium tuberculosis*.

On admission, best corrected visual acuity was 20/400 in a right amblyopic eye and 20/50 in the left eye. Biomicroscopic examination revealed no sign of anterior or posterior inflammation. Multiple choroidal lesions (Fig 1) were present in both eyes. The choroidal lesions were deep, white-yellowish, with indistinct borders. Fluorescence angiography (FA) revealed early nodular hypofluorescence, and
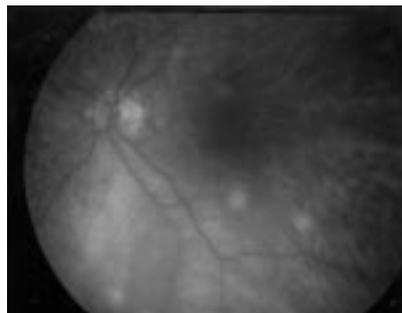

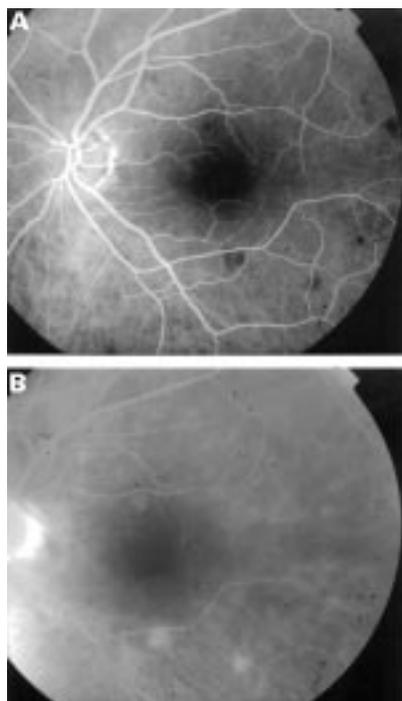*Figure 1   Multiple choroidal granulomas in the left posterior pole.*

Figure 2   (A) Early prolonged blockage and (B) late moderate hyperfluorescence of the choroidal lesions on fluorescein angiography.
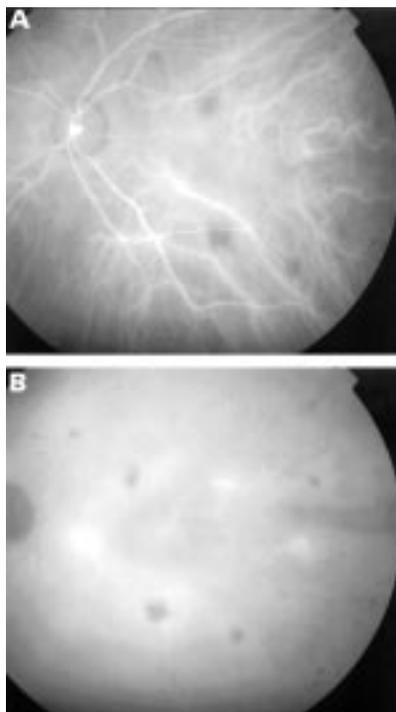

Figure 3   ICG angiograms reveal early (A) and late (B) phase blockage by the choroidal granulomas.

late moderate hyperfluorescence (Fig 2). Indocyanine green (ICG) angiography revealed prolonged hypofluorescence and in the late stage images, moderate delineation of the lesions by a peripheral hyperfluorescent ring (Fig 3).

COMMENT

Ocular tuberculosis may occur by haematogenic spread from a pulmonary focus. Choroidal tuberculomas are rare ophthalmic findings even in miliary tuberculosis.[1] Previous reports indicate that these lesions have prolonged hypofluorescence in FA, and late mild hyperfluorescence.[2] [3]

Only one description of ICG angiography in a case with presumed ocular tuberculosis has been reported previously in the literature.[4] We found similar angiographic characteristics in our case, which represents, to our knowledge, the first ICG angiography description of multiple choroidal tuberculomas in microbiologically confirmed miliary tuberculosis. Hypofluorescence in ICG images may be due to a masking effect of the choroidal vessels by the overlying granulomas.

Ophthalmic examination may be contributive when disseminated tuberculosis is suspected. In this case ICG angiography, which was performed to assess the choroidal involvement, showed prolonged hypofluorescence.

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Diagnosis of an atypical case of ocular toxoplasmosis using the demonstration of intraocular antibody production and the polymerase chain reaction

EDITOR,—Ocular toxoplasmosis is the most frequent infectious cause of chorioretinal inflammation in immunocompetent individuals.[1] Diagnosis is usually made by observing the typical fundus lesion, by detecting the presence of anti-*Toxoplasma* antibodies in the serum, and by excluding other causes of necrotising fundus lesions.[2] In unusual cases, invasive procedures may be required to aid diagnosis.[3]

CASE REPORT

A 17 year old white male presented complaining of floaters and reduced visual acuity in the left eye. Visual acuity was 6/9 in the left eye, 6/6 in the right. Examination revealed moderate anterior chamber activity, marked vitritis, and an active retinochoroiditis adjacent to an area of old chorioretinal scarring inferonasal to the optic disc. A diagnosis of ocular toxoplasmosis was suspected, and topical and oral steroids, and oral clindamycin were commenced. Peripheral blood anti-*Toxoplasma* IgG antibodies, measured using the dye test, were positive (16 IU/ml). Despite treatment, the ocular inflammatory signs increased and 5 weeks following initial presentation he developed a confluent area of retinal necrosis in the peripheral retina leading to a superotemporal retinal detachment. This was distinct from the original area of inflammation. The presence of severe vitreous inflammation and peripheral retinal necrosis suggested a unilateral acute retinal necrosis syndrome.[4] Three port trans pars plana vitrectomy with perfluorocarbon liquid and fluid/silicone exchange was performed. At vitrectomy, vitreous humour was taken for anti-*Toxoplasma* and antiviral antibody levels and a retinal biopsy was also obtained. Postoperatively, he was commenced on sulphadiazine, pyrimethamine, and folinic acid and continued on oral steroid medication. Levels of IgG, IgA, and IgM were measured in serum and vitreous aspirate at the same time. The Goldmann–Witmer coefficient using IgG was greater than 59, using IgA greater than 45, and using IgM greater than 65. This is evidence of intraocular antibody production. Samples were negative for antiviral antibodies. Intraocular *Toxoplasma* DNA was demonstrated by a polymerase chain reaction (PCR) assay using primers for the P30 gene. PCR testing for viral DNA was negative. Insufficient material was obtained to attempt to isolate the parasite using tissue culture or animal inoculation. Retinal biopsy demonstrated a mixed inflammatory response without a specific infective agent. The patient subsequently responded to treatment and the intraocular inflammatory signs subsided.

COMMENT

Ocular toxoplasmosis is a common cause of retinochoroiditis, and can usually be diagnosed clinically. Rarely is it possible to obtain vitreous and retinal biopsies to aid diagnosis, but in doubtful cases, it may be appropriate to perform anterior or posterior chamber aspirate to confirm the diagnosis. The assessment of *Toxoplasma* antibodies in serum is of limited use, unless rising titres can be demonstrated, since the incidence of *Toxoplasma* infection in the general population is high. The demonstration of antibody production within the eye is particularly valuable in the diagnosis of difficult cases. The finding of higher anti-*Toxoplasma* antibody levels in the aqueous humour than in the serum (the Goldmann–Witmer coefficient) indicates intraocular antibody production.[5] A further investigation which is extremely useful is the demonstration of parasite DNA within ocular fluid by PCR.[6] With PCR a sequence of DNA is amplified from minuscule amounts of DNA making it amenable to direct analysis.[7][8] De Boer *et al* used a combination of the demonstration of intraocular antibody production and PCR analysis in the diagnosis of a variety of infectious uveitis cases.[9] In this case we initially made a diagnosis of ocular toxoplasmosis, but the disease progressed clinically and did not respond to treatment. The patient was compliant with prescribed medication, and had no evidence of immunocompromise. Retinal detachment is unusual in ocular toxoplasmosis,[10] but is typical of acute retinal necrosis syndrome, suggesting an alternative diagnosis in this case. We were, however, able to confirm the diagnosis of toxoplasmosis by evidence of intraocular antibody production and by positive PCR amplification.

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Protein C and protein S deficiency associated with retinal, optic nerve, and cerebral ischaemia

EDITOR,—Deficiencies in the vitamin K dependent factors protein C and protein S can lead to arterial or venous thrombosis. Branch and central retinal arterial and venous occlusions[1-4] have been associated with deficiencies in these plasma proteins, as have amaurosis fugax[5] and stroke.[6] We report, to the best of our knowledge, the first case of ischaemic optic neuropathy associated with combined protein C and protein S deficiency.

CASE REPORT

A 47 year old woman with non-insulin dependent diabetes mellitus with documented absence of previous retinopathy presented with blurring of vision and bright flashing lights in her right eye for 2 weeks, associated with vague periocular discomfort and left sided facial and leg numbness. Best corrected visual acuity was 20/30 right eye and 20/25 left eye. The anterior segment examination was unremarkable and the intraocular pressures were 15 mm Hg right eye and 14 mm Hg left eye. A large cotton wool spot was present inferotemporal to the right optic disc (Fig 1). The overlying vitreous was clear. The retinal venules appeared moderately tortuous but undilated. Fluorescein angiography revealed normal arterial filling but markedly delayed arteriovenous filling and late disc hyperfluorescence. When she returned 2 weeks later, this cotton wool spot was smaller, but other cotton wool spots superior to the disc had appeared (Fig 2). The patient underwent carotid Doppler and cerebral angiography studies which revealed near complete occlusion of the right internal carotid artery. Coumadin therapy was instituted and extensive diagnostic evaluation was pursued. She returned 2 weeks later and all the cotton wool spots were resolving.

Three days later she was admitted to the hospital with syncope and left hemiparesis due to an infarct in the territory of the right middle cerebral artery. She also suffered sudden,

Figure 1 Initial large cotton wool spot inferotemporal to right optic disc.
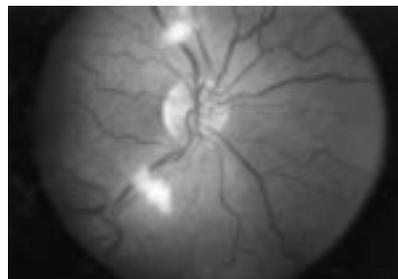

Figure 2 Initial cotton wool spot along the inferotemporal vessel resolving 2 weeks later with appearance of new cotton wool spots superiorly.

Figure 3 Six weeks after initial presentation. Note pale disc with narrowing of the retinal arterioles and an overall reduction in venous calibre and tortuosity.

painless loss of vision to the level of hand movements in the right eye. Fundus examination 6 weeks later revealed a pale optic disc with both generalised and focal narrowing of the retinal arterioles, and an overall reduction in venous calibre and tortuosity (Fig 3). Three months later, at which time the visual acuity remained at hand movements, electroretinography (ERG) was performed to distinguish retinal vascular pathology from optic nerve embarrassment. The right eye exhibited modest reductions in scotopic b-wave amplitudes in response to dim white flash (33%) and to bright white flash (20%) compared with the left eye. Cone b-wave implicit time on 30 Hz flicker testing was only slightly longer in the right eye compared with the left eye (30.5 ms versus 29.5 ms). Oscillatory potential amplitudes were normal in both eyes. These results were interpreted as showing insufficient evidence for ischaemic retinal damage as an explanation for her profound loss of vision. The patient was diagnosed with ischaemic optic neuropathy in the right eye based on clinical findings and the ERG results. Laboratory testing revealed that protein C antigen was 47% and protein S antigen 46% of normal levels. Activated protein C and antithrombin levels were normal, and no lupus anticoagulant activity was detected.

COMMENT

This patient, with combined protein C and protein S deficiency, suffered ipsilateral retinal, optic nerve, and cerebral ischaemia within a period of 6 weeks. The rapid changes in the appearance of cotton wool spots over a period of several days, which is not consistent with their natural course in diabetic retinopathy,[7] combined with neurological symptoms prompted us to search for systemic causes of ischaemia, including evaluation for hypercoagulable states. We suggest that new cotton wool spots in a patient free of other signs of vascular retinopathy such as microaneurysms or retinal haemorrhages should raise the spectre of a systemic basis for the ischaemia. As the ERG was not compatible with occlusion of the ophthalmic or central retinal arteries, demonstrating only mild retinal ischaemia, we ascribed the sudden visual loss in the face of diffuse disc pallor to optic nerve ischaemia, perhaps from occlusion of multiple ciliary vessels. Ischaemic optic neuropathy has, to our knowledge, not previously been associated with protein C or protein S deficiency, and expands the spectrum of ophthalmic manifestations of the hypercoagulable state.

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Macular hole following YAG capsulotomy

EDITOR,—Since the initial identification of macular holes as pathological entities in the middle of the 19th century,[1] there has been an evolution in the understanding of their aetiology. Tangential macular traction by perifoveal vitreous cortex is now accepted as the causative factor in the development of idiopathic macular holes.[2 3]

With the widespread use of extracapsular cataract extraction procedures, posterior capsule opacification is a frequent complication. YAG laser capsulotomy, although a noninvasive procedure, has been associated with a number of complications, including retinal detachment, cystoid macular oedema, and raised intraocular pressure (IOP).[4 5] A much rarer complication of YAG capsulotomy herein reported is the formation of a macular hole after YAG capsulotomy.[4]

CASE REPORT
A 71 year old woman underwent an uncomplicated extracapsular cataract extraction with posterior chamber lens implantation in her left eye. Her ocular history was significant for chronic open angle glaucoma. In the immediate postoperative period, there was an acute rise in IOP to 40 mm Hg that responded to Diamox (acetazolamide) orally. Three months postoperatively, best corrected visual acuity was 20/20 in both eyes with IOPs of 17 mm Hg in the right eye and 13 mm Hg in the left.

Two years later, the best corrected visual acuity was found to have decreased to 20/80 in the left eye attributable to significant posterior capsule opacification. Posterior capsulotomy was performed with a Nd:YAG laser (4.1 mJ/pulse, total energy 109.5 mJ). Postoperatively, there was no increase in IOP and no vitreous prolapse into the anterior chamber. Two weeks after the Nd:YAG laser capsulotomy, the patient noted a decrease in visual acuity, along with a black spot in her central vision. On examination, a stage 3 macular hole was seen with best corrected visual acuity 20/400 left eye. Retinal consultation confirmed the diagnosis and the patient underwent a pars plana vitrectomy, with C_3F_8 gas instillation and facedown positioning.

Evaluation of the patient 4 weeks after surgery revealed an improvement of visual acuity in the left eye to the level of 20/25. Visual acuity 6 months after surgery remained at the level of 20/25 with the macular hole closed.

COMMENT
The most common complication of extracapsular methods is a late opacification of the posterior capsule. Surgically opening the posterior capsule has been shown in several studies to increase the incidence of both cystoid macular oedema and retinal detachment.[4 5] With the advent of the Nd:YAG laser, the ease of posterior capsulotomy has been greatly simplified. Retinal complications following YAG laser capsulotomy are well documented.[4 5] Winslow and Taylor[4] reported one retinal flap, two macular holes, six cases of cystoid macular oedema, and 10 retinal detachments following YAG laser capsulotomy. In this series, macular hole formation occurred 1 and 3 months after capsulotomy while in our case it occurred within 2 weeks.

Over the years, several mechanisms have been proposed to explain the increased incidence of retinal complications following posterior capsulotomy including increased vitreous liquefaction, changes in vitreous composition, acoustic transients, and direct retinal damage. Osterlin[6] reported a greater decline in the hyaluronic acid content in vitreous samples from monkey eyes having undergone intracapsular cataract extraction as opposed to extracapsular cataract extraction. He postulated that in the eyes that had undergone intracapsular cataract extraction, hyaluronic acid in the vitreous had diffused anteriorly, resulting in the vitreous instability and subsequent retinal complications. Thus, the intact capsule acts as a diffusion barrier for hyaluronic acid. This concept of a diffusion barrier was again employed by Miyake[7] to theorise a role for the posterior capsule in the development of cystoid macular oedema due to iris synthesised prostaglandins.

Significant liquefaction of the vitreous, postulated to be the result of acoustic transients accompanying the laser irradiation, has been documented in monkey and rabbit eyes following Nd:YAG laser irradiation of the posterior capsule.[8] Other more direct injuries to the retina with the formation of macular holes have been reported in industrial accidents involving the Nd:YAG laser.[9]

In a case report by Blacharski and Newsome,[10] bilateral macular holes were reported following Nd:YAG laser posterior capsulotomies. In the first eye, a macular hole formed 21 days after capsulotomy in the absence of vitreous prolapse or an elevated IOP post laser. In the second eye, careful biomicroscopy was performed before Nd:YAG capsulotomy and despite the absence of complication, a macular hole formed 10 days after treatment. These authors believed it unlikely that the shock wave generated by the pulse directly caused the macular hole as relatively low energies were used on both occasions (18 mJ and 29 mJ).

In our case, we propose that the macular hole was formed secondary to the perifoveal vitreous contraction initiated by the YAG capsulotomy. The possible mechanisms of Nd:YAG laser initiation of vitreous contraction could include the well documented acoustic transients generated by a YAG laser pulse, as well as vitreous instability secondary to the vitreous liquefaction demonstrated in

both human and monkey eyes following YAG posterior capsulotomy.[4]

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Magnetic resonance imaging of colobomatous optic hypoplasia

EDITOR,—Retinochoroidal coloboma is a common ocular malformation that can occur as an isolated finding in an otherwise healthy individual or as part of a complex malformation syndrome.[1] Histologically, it is a well demarcated, excavated, infrapapillary area of absent retina, pigment epithelium, Bruch's membrane, and choriocapillaris, with variable attenuation of the choroid.[2] Some retinochoroidal colobomas incorporate the optic disc and cause the inferior aspect of the optic disc to appear retruded or absent within the excavation.[3] The purpose of this study was to determine whether such malformations are associated with hypoplasia of the intracranial optic nerve.

Five patients with unilateral retinochoroidal coloboma involving the optic disc underwent magnetic resonance imaging (MRI) of the head to rule out associated intracranial malformations. Patients consisted of two males and three females with ages at the time of MRI ranging from 2 weeks to 4 years. All patients had large unilateral retinochoroidal colobomas that incorporated the optic disc (Fig 1).

MRI consisted of sagittal T1 weighted images, axial T2 weighted images, and coronal T1 weighted thin section images (with 3 mm slice thickness and 0.3 mm gaps) through the chiasm, intracranial optic nerves, and orbits. T1 weighted coronal MR images of the
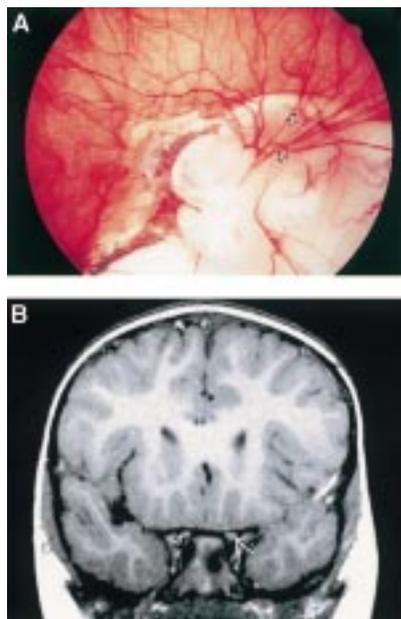
Figure 1 (A) Retinochoroidal coloboma incorporating the segmentally hypoplastic right optic disc (open arrows). The major retinal vessels delimit the lower margin of the optic disc. (B) T1 weighted coronal MR image (case 1) demonstrating hypoplasia of the right optic nerve (small arrow). The area of the right optic nerve is approximately half the size of the normal left optic nerve (large arrow).

intracranial optic nerves were examined to compare the size of the intracranial optic nerve corresponding to the colobomatous eye with that corresponding to the normal eye. Cases 1–3 had no associated systemic or intracranial malformations. Case 4 had Goldenhar syndrome with hemifacial microsomia, cerebral hemispheric asymmetry without disorganisation, and colobomatous microphthalmos on the involved side. Case 5 had Aicardi syndrome with agenesis of the corpus callosum and bilateral grey matter heterotopia.

In all patients, coronal MRI showed a smaller intracranial optic nerve on the side corresponding to the retinochoroidal coloboma (Fig 1). The degree of intracranial optic nerve hypoplasia varied according to the ophthalmoscopic configuration of the optic disc. When the inferior aspect of the optic disc was present but retruded posteriorly within the colobomatous defect (case 1), the corresponding intracranial optic nerve was only slightly diminished in size relative to the normal optic nerve. When only the inferior aspect of the optic disc was absent within the colobomatous defect (cases 2–5), a moderate reduction in intracranial optic nerve size was seen.

In 1988, Novakovic *et al* demonstrated that focal retinal lesions can produce segmental hypoplasia of corresponding sectors of optic disc.[4] In 1990, Brodsky *et al* showed that T1 weighted MRI can be used to confirm the clinical diagnosis of optic nerve hypoplasia by showing a reduction in size of the intracranial optic nerve(s).[5] In the present study, MRI showed that colobomatous retinochoroidal malformations involving the optic disc are consistently associated with hypoplasia of the ipsilateral intracranial optic nerve, corresponding to the inferior segmental hypoplasia observed ophthalmoscopically. The nosological overlap between colobomatous derangement of the optic nerve and segmental hypoplasia, which has gone largely unrecog-

nised, reflects the timing of colobomatous dysembryogenesis early in gestation and implicates a primary developmental failure of inferior retinal ganglion cells.[4] MRI of other segmental optic disc malformations (for example, congenital tilted disc syndrome, unilateral high myopia) may disclose similar reductions in intracranial optic nerve size.

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Orbital haemangiopericytoma simulating an intraocular mass

EDITOR,—Most patients with orbital tumours present with proptosis.[1][2] It is uncommon for an orbital mass to cause symptoms and signs simulating intraocular disease. We report the case of a patient with an orbital tumour that was initially suspected to be an intraocular tumour.

CASE REPORT
A 71 year old woman noted a photopsia, diplopia, and peripheral scotoma in her left eye. She was evaluated and underwent laser treatment for suspected retinal hole at the margin of a presumed retinal detachment. After non-resolution of the "detachment", a second ophthalmologist raised the possibility that the fundus lesion was a choroidal melanoma. The patient was then referred to the oncology service for further management. Ocular examination revealed corrected visual acuity of 6/7.5 in both eyes. Proptosis of 3 mm with minimal limitation of supraduction and infraduction was noted. Fundus examination showed an elevated choroidal mass with normal appearing retinal and choroidal vessels overlying the mass. The mass did not shift with eye position. Fluorescein angiography demonstrated retinal and choroidal isofluores-

Figure 1 Late venous phase of fluorescein angiography showing retinal and choroidal isofluorescence.

Figure 2 Coronal T1 weighted magnetic resonance image demonstrating intraconal orbital mass compressing the globe.

cence in the area of the mass (Fig 1). B-scan ultrasonography showed an echolucent mass compressing the sclera, measuring 16×16×12 mm. Based on these findings, an orbital tumour producing globe compression was suspected.

Magnetic resonance imaging was performed to more clearly delineate the soft tissue mass. A well circumscribed intraconal mass was found adjacent to the sclera inferomedially, producing globe compression and inferior rectus displacement (Fig 2). On T1 weighted images, the lesion was isointense and on T2 weighted images, hyperintense with respect to muscles. Marked enhancement of the lesion with gadolinium was found. Our differential diagnosis included orbital cavernous haemangioma, neurofibroma, schwannoma, fibrous histiocytoma, and haemangiopericytoma.

The patient underwent transconjunctival excisional biopsy. The pink encapsulated mass was composed of spindle cells with moderate mitotic activity. Staghorn vascular channels were evident, and in several areas the tumour cells invaded the pseudocapsule.

The histopathological diagnosis was benign haemangiopericytoma. The patient has been followed for 1 year without further problems.

COMMENT
Haemangiopericytoma is a rare vascular tumour derived from an abnormal proliferation of pericytes. It rarely occurs in the orbit, accounting for only 1% of all orbital biopsies.[1–5] Orbital haemangiopericytoma occurs as a painless, unifocal tumour often in the muscle cone.[1–5] The majority of cases are recognised between the ages of 20–70 years.[1–5] In most cases there is progressive proptosis. However, in our case mild proptosis but marked compression of the globe was seen. Orbital haemangiopericytoma poses a risk for recurrence and metastasis, especially when the tumour invades beyond the pseudocapsule.[3–5]

Orbital haemangiopericytoma generally is a slow growing tumour that has an ocular and systemic prognosis.[1–5] There is a risk for recurrence and metastasis when the pseudocapsule is breached.[4] In one series, a 30% recurrence rate was noted with recurrences generally occurring 1 month to 7 years after surgery.[3] Our patient may be at risk of developing orbital recurrence in the future because there was invasion of the pseudocapsule.

Orbital tumours should be included in the differential diagnosis of a solid intraocular mass. Those orbital tumours that arise in the muscle cone adjacent to the sclera may produce these confusing clinical features.

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Delayed suprachoroidal haemorrhage following trabeculectomy bleb needling

EDITOR,—Transconjunctival needling of trabeculectomy blebs is a relatively safe, simple outpatient procedure that can successfully re-establish aqueous flow in failed trabeculectomies.[1 2] We report a severe delayed suprachoroidal haemorrhage occurring secondary to this procedure in an aphakic patient receiving aspirin therapy.

CASE REPORT
Fifteen years previously a 75 year old myopic man underwent bilateral intracapsular cataract extractions. He developed secondary open angle glaucoma but was intolerant of topical β blockers because of bradycardia. He was managed on pilocarpine drops 4% four times daily but control of intraocular pressure (IOP) was inadequate with deterioration in visual fields. Twelve years after the cataract extractions he underwent bilateral trabeculectomies with postoperative 5-fluorouracil. Three years later the left visual acuity was 6/18 with an IOP of 22 mm Hg despite pilocarpine. In view of progressive cupping of the left optic disc in association with this pressure, the patient was offered needling of the left filtering bleb.

Figure 1   B scan ultrasound examination of the patient's left eye 24 hours after trabeculectomy bleb needling and 17 hours after he noticed sudden pain and loss of vision in that eye. Findings are consistent with vitreous haemorrhage and haemorrhagic choroidal detachments.

Figure 2   B scan ultrasound examination of the same eye 4 months later showing a fixed funnel retinal detachment.

The needling was performed at the slit lamp with immediate development of a shallow bleb. The anterior chamber was well maintained with an IOP of 4 mm Hg. Subconjunctivally, 5 mg of 5-fluorouracil was administered and the patient was discharged with topical steroids and antibiotics. When he bent over 7 hours later he experienced sudden pain in his left eye with immediate reduction of vision. He presented for examination the following day when the visual acuity was noted to be reduced to hand movements with a left relative afferent pupillary defect (RAPD). There was a large subconjunctival haemorrhage, a total hyphaema, and IOP of 7 mm Hg. There was no fundal view but B scan ultrasound showed vitreous haemorrhage and haemorrhagic choroidal detachments (Fig 1). Further direct questioning revealed that the patient was taking 75 mg of aspirin "for his heart" on his family doctor's advice.

The patient was managed conservatively with serial ultrasound examinations. Despite initial subjective improvement in vision, the sight remained reduced at hand movements with a persistent RAPD and a soft eye. B scan ultrasound 4 months after needling showed an open funnel retinal detachment (Fig 2) which, in view of the poor visual prognosis, was not felt to be amenable to vitreoretinal surgery.

COMMENT
Delayed suprachoroidal haemorrhage is a well recognised but fortunately rare complication of all forms of intraocular surgery, especially filtering procedures. Pathological study of eyes enucleated within hours of the haemorrhage occurring have suggested the cause to be rupture of necrotic posterior ciliary arteries.[3] A number of risk factors for delayed suprachoroidal haemorrhage have been reported including aphakia, high myopia, a large peroperative reduction in IOP, postoperative hypotony, and systemic vascular disease.[3 4] The patient reported here was myopic, aphakic, had ischaemic heart disease and additionally was on aspirin.

Two cases of haemorrhagic choroidal detachments have been reported[1 2] after bleb needling with adjunctive mitomycin C. Precise details of the individual cases were not supplied, however, so it is not clear if these patients had predisposing risk factors or the result of their final visual outcome. A large choroidal effusion occurring after bleb needling has been reported in a pseudophakic patient,[5] the effusion resolving after surgical reformation of the anterior chamber. Our patient was managed conservatively owing to early subjective improvement in his visual acuity. It is possible, however, that the outcome may have been improved with surgical drainage of the suprachoroidal haemorrhage at an early stage, as has been advocated by some authors.[4] The contribution that aspirin played in the development or exacerbation of the haemorrhage is unknown but has not been previously reported as a risk factor. This report emphasises that, while needling of trabeculectomy blebs is usually a safe procedure, severe complications may arise and these need to be taken into consideration, especially when managing high risk patients.

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Spontaneous intracorneal haemorrhage

EDITOR,—Spontaneous intracorneal haemorrhage leading to corneal discoloration is an uncommon occurrence. The few such reported cases of spontaneous intracorneal haemorrhage have been due to contact lens related deep stromal neovascularisation, erosion of a vessel due to corneal ulceration, and rupture of reopened ghost vessels in a patient with interstitial keratitis and systemic hypertension. Corneal blood staining clinically represents a reddish-brown, or greenish-yellow discoloration of the cornea resulting from blunt trauma and subsequent hyphaema with raised intraocular pressures or less commonly by intrastromal haemorrhage in the presence of corneal vascularisation.[1] The term "corneal blood staining" has been used to refer only to the latter in this case report—a case of spontaneous intracorneal haemorrhage related to acne rosacea associated corneal vascularisation.

CASE REPORT
A 72 year old man was seen at the eye casualty unit with a 3 week history of reduced vision and ocular discomfort in his left eye. There was no history of trauma.

His ocular history included chronic posterior blepharitis and peripheral corneal ulcers (upper cornea) in the left eye related to acne rosacea. This patient had also had an uneventful cataract surgery in the same eye some 3 years previously and had not been seen in the eye department since. Relevant medical history includes treatment for paroxysmal atrial fibrillation with sotalol. The patient had been taking

Figure 1   Left eye on presentation.

Figure 2 The same eye 7 months later showing shrinkage of the area of staining and underlying stromal vessels.

warfarin until 2 months before the original presentation. The clinical findings were left eye visual acuity −6/60 (Sn) improving to 6/12p with a pinhole; circumciliary congestion and an area of reddish-brown discoloration (6.0–6.5 mm) inferotemporally on the cornea, clinically resembling corneal blood staining. The intraocular pressure was within normal limits and no other ocular abnormality was detected. The other eye had a visual acuity of 6/6p with pinhole and appeared to be normal. On review, 2 months after initial presentation, he was noted to have shrinkage of the area of discoloration revealing underlying prominent superficial and deep stromal corneal vessels adjacent to the area of discoloration, and some lipid deposition close to the deeply vascularised limbus. On further follow up 5 months later, the patient had retained the same visual acuity of 6/60 (Sn) improving to 6/12p with pinhole. Though the ciliary congestion persisted, the patient was not in any discomfort. The area of discoloration had a greenish-yellow tinge now and measured 5.7–4.2 mm.

COMMENT

Deep intracorneal haemorrhage is most often seen after intraocular surgery, after direct, blunt ocular trauma, and in a vascularised cornea. The contribution of systemic factors such as diabetes or hypertension is unclear.[2] Acne rosacea is known to cause peripheral vascularisation especially involving the infero-nasal and inferotemporal quadrant. These vessels are known to progress in the absence of acute symptoms.[3] In our patient, the corneal blood staining was a result of direct bleeding into the corneal stroma from the deep stromal vessels. The deep stromal vascularisation appears to have developed insidiously as in similar cases reported subsequent to contact lens wear.[2 3] Corneal blood staining either from persisting hyphaema or deep intra-corneal haemorrhage represents deposition of haemoglobin and its breakdown products within the cornea.[4] A histopathological analysis of blood stained corneas, most of which were associated with raised intraocular pressures, indicated a gradient of haemoglobin degradation from the posterior to the anterior corneal stroma, extracellular haemoglobin particles being concentrated more posteriorly while haemosiderin laden keratocytes predominated anteriorly.[1] Animal model experiments in rabbits utilising total persistent hyphaema with sustained increased intraocular pressures have also revealed similar results.[5] Endothelial degeneration accompanies corneal blood staining and keratocytes appear to be actively involved in haemoglobin degradation.[5] Porphyrin induced photosensitivity producing cytotoxic oxygen species within the blood stained cornea have also been considered as contributing to endothelial and keratocyte degeneration.[6] Clearing of blood staining is thought to be a result of the phagocytic action of the keratocytes and from a diffusion of haemoglobin into the conjunctival circulation and the anterior chamber.[7] The pattern of peripheral, posterior, and anterior stromal clearing observed seems consistent with diffusion of haemoglobin breakdown products out of the cornea as the primary mechanism of clearing.[4] In the absence of a hyphaema, therapeutic efforts are directed towards prevention of corneal blood staining—for example, treating the corneal ulcer vigorously, correction of entropion or treatment of systemic hypertension.[2 7] The presence of deep stromal vascularisation secondary to any cause must be watched carefully and managed as a potentially vision threatening complication especially in contact lens wearers. Once intracorneal bleeding has occurred, Giessler *et al* advise waiting for a spontaneous clearing, although it may take 2 or 3 years or more.[7] In the absence of severe associated pathology, corneal blood staining has been noted to clear without permanent corneal opacity changes.[4] Penetrating keratoplasty may be considered.

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Indocyanine green angiography in choroidal tuberculomas

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