Indocyanine green angiography in choroidal tuberculomas

EDITOR,—An 85 year old white woman presented with progressive asthenia, fever, coughing, and dyspnoea. Chest roentgenograms 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 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).

**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.

**COMMENT**

Ocular tuberculosis may occur by haematogenic spread from a pulmonary focus. Choroidal tuberculomas are rare ophthalmic findings even in miliary tuberculosis. Previous reports indicate that these lesions have prolonged hypofluorescence in FA, and late mild hyperfluorescence. Only one description of ICG angiography in a case with presumed ocular tuberculosis has been reported previously in the literature. 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.

**DAN MILEA**

**CHRISTINE FARDEAU**

**LIVIA LUMBRICO**

Department of Ophthalmology, Hôpital de la Pitié-Salpêtrière, Paris, France

**THOMAS SIMILOWSKI**

Department of Respiratory and Intensive Care Medicine, Hôpital de la Pitié-Salpêtrière, Paris, France

**PHUC LE HOANG**

Department of Ophthalmology, Hôpital de la Pitié-Salpêtrière, Paris, France

Correspondence to: Phuc Le Hoang, MD, Service d’Ophthalmologie, Hôpital de la Pitié-Salpêtrière, 47-83 Boulevard de l’Hôpital, 75651 Paris Cedex 13, France.
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, since the incidence of toxoplasmosis, as have amaurosis fugax and stroke. 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 periorcular 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 vessels 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, 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. Labora
tory testing revealed that protein C antigen was 47% and protein S antigen 34% of normal levels. Activated protein C and anti-thrombin levels were normal, and no lupus anticoagulant activity was detected.

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.
Macular hole following YAG capsulotomy

EDITORS—Since the initial identification of macular holes as pathological entities in the middle of the 19th century, 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. The widespread use of extracapsular cataract extraction procedures, posterior capsule opacification is a frequent complication. YAG laser capsulotomy has been associated with a non-invasive procedure, has been associated with a number of complications, including retinal detachment, cystoid macular oedema, and raised intracranial pressure (IOP). A much rarer complication of YAG capsulotomy herein reported is the formation of a macular hole after YAG capsulotomy.

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 20/20 in both eyes with IOPs of 17 mm Hg in the right eye and 13 mm Hg in the left. Significant liquefaction of the vitreous, postoperative IOPs of 17 mm Hg in the right eye and 13 mm Hg in the left.

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. With the advent of YAG laser capsulotomy, the case of posterior capsulotomy has been greatly simplified. Retinal complications following YAG laser capsulotomy are well documented. Winslow and Taylor 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 reported a greater decline in the hyaluronic acid content in vitreous samples of 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, rendering 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 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, postoperative IOPs of 17 mm Hg in the right eye and 13 mm Hg in the left.

In a case report by Blacharski and Newsome, 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 biomicroscopic examination before Nd:YAG capsulotomy and despite the absence of compication, a macular hole formed 10 days after treatment. These authors believed unlikely that the shock wave generated by the procedure initiated the macular hole as relatively low energies were used on both occasions (18 mm and 29 mm).

In our case, we propose that the macular hole was formed secondary to the perifoveal vitreous cortex as a result of vitreous contractile forces generated 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.5

The authors have no proprietary interest in any of the products mentioned in this article.

RIPAN CHAUDHARY
Department of Ophthalmology, University of Alberta, Edmonton, Canada

TOM SHEIDOW
JOHN R GONDER
MOHAMMAD N MERCHEA
Department of Ophthalmology, University of Western Ontario, London, Canada

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Magnetic resonance imaging of colobomatous optic hypoplasia

EDITORS—Retinocochorial 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. Histologically, it is a well demarcated, excavated, intrapapillary area of absence of retinal, pigment epithelium, Bruch’s membrane, and choriocapillaris, with variable attenuation of the choroid. Some retinochoroidal colobomas incorporate the optic disc and cause the inferior aspect of the optic disc to appear retracted or absent within the excavation. The purpose of this study was to determine whether such malformations are associated with hypoplasia of the intracranial optic nerve.

To our patients with unilateral retinocochorial 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 presentation of MRI ranging from 2 weeks to 4 years. All patients had large unilateral retinocochorial 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
focal retinal lesions can produce segmental optic nerve hypoplasia, which has been largely unrecognised, reflects the timing of colobomatous dysmorphogenesis early in gestation and implicates a primary developmental failure of inferior retinal ganglion cells. 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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MICHAEL C BRODSKY
University of Arkansas for Medical Sciences, Little Rock, Arkansas

Correspondence to: Arkansas Children’s Hospital, 800 Marshall, Little Rock, AR 72202, USA

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Orbital haemangiopericytoma simulating an intraocular mass

EDITOR,—Most patients with orbital tumours present with proptosis. 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 correct 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 isofluorescence 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 intraocular mass was found adjacent to the sclera intermedially, producing globe compression and inferior rectus displacement (Fig 2). On T1 weighted images, the lesion was isointense and on T2 weighted images, hyperintense in respect with 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. Orbital haemangiopericytoma occurs as a painless, unifocal tumour often in the muscle cone. The majority of cases are recognised between the ages of 20–70 years. 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.

Orbital haemangiopericytoma generally is a slow growing tumour that has an ocular and systemic prognosis. There is a risk for recurrence and metastasis when the pseudocapsule is breached. In one series, a 30% recurrence rate was noted with recurrences generally occurring 1 month to 7 years after surgery. 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.

Dr Ralph C Eagle Jr performed the interpretation of the histopathology.

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CAROL L SHIELDS

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

Figure 2 Coronal T1 weighted magnetic resonance image demonstrating intraocular orbital mass compressing the globe.
Delayed suprachoroidal haemorrhage following trabeculectomy bleb needling

EDITOR,—Transconjunctival needling of trabeculectomy blebs is a relatively safe, simple outpatient procedure that can successfully treat suprachoroidal haemorrhage in certain cases. We report a 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 trabeculectomy blebs 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.

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 4 months after needling showed an open funnel retinal detachment (Fig 2) which, in view of the poor visual prognosis, was felt to be amenable to vitreoretinal surgery.

COMMENT

Delayed suprachoroidal haemorrhage is well recognised but unfortunately 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. A number of risk factors for delayed suprachoroidal haemorrhage have been reported including aphakia, high myopia, a large peripapillary reduction in IOP, postoperative hypotony, and systemic vascular disease. The patient reported here was myopic, aphatic, had ischaemic heart disease and additionally was on aspirin.

Two cases of haemorrhagic choroidal detachments have been reported after bleb needling with adjunctive mitomycin C. Precise details of these cases have not been published, 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, 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. 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.

LUCY J HOWE
PHILIP BLOOM
The Western Eye Hospital, Marylebone Road, London NW1 3BY

Correspondence to: Lucy Howe.
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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 hypoxemia with raised intraocular pressures or less commonly by intrastromal haemorrhage in the presence of corneal vascularisation. 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...
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 intra-
ocular 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.4 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.5 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
tear wear.6 Cornal 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 analy-
is of blood stained corneas, most of which
were associated with raised intraocular pres-
ures, 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 pre-
dominated anteriorly.7 Animal model experi-
ments in rabbits utilising total persistent
hyphaema with sustained increased intraocu-
lar pressures have also revealed similar
results.5 Endothelial degeneration accompa-
nies corneal blood staining and keratocytes
appear to be actively involved in haemoglobin
degradation.8 Porphyrin induced photosensi-
tivity producing cytotoxic oxygen species
within the blood stained cornea have also been
considered as contributing to endothelial and
keratocyte degeneration.9 Clearing of blood
staining is thought to be a result of the phago-
cytic action of the keratocytes and from a dif-
fusion of haemoglobin into the conjunctival
circulation and the anterior chamber.10 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.1 In the absence of a

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

hiyphaema, 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.1,7 The
presence of deep stromal vascularisation
secondary to any cause must be watched care-
fully and managed as a potentially vision
threatening complication especially in contact
tes 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.1 In the absence of severe
associated pathology, corneal blood staining
has been noted to clear without permanent
corneal opacity changes.1 Penetrating kerato-
plasty may be considered.

V SUDHA
Department of Ophthalmology, Princess Margaret
Hospital, Swindon, Wiltshire

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Orbital haemangiopericytoma simulating an intraocular mass

WALTENIO V DINIZ, CAROL L SHIELDS, JERRY A SHIELDS, KAAN GUNDUZ and ALAN BRACKUP

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