Ultrasound biomicroscopic images of the anterior chamber angle of a patient with posterior polymorphous dystrophy

EDITOR,—Posterior polymorphous dystrophy (PPD) is a hereditary corneal dystrophy that is typically asymptomatic and non-progressive. It rarely results in severe visual dysfunction due to corneal decompensation and/or glaucoma. We examined the anterior chamber angle of a PPD patient with corneal oedema and broad iridocorneal adhesion by using ultrasound biomicroscopy (UBM). The examination indicated a unique iridocorneal adhesion that could not be seen in gonioscopy.

CASE REPORT
A 39 year old woman was examined for progressive loss of vision (30/200) in her left eye and increased foreign body sensation. Slit lamp examination of the left eye revealed diffuse corneal oedema and bullous keratopathy (Fig 1). Broad based iridocorneal adhesion extending anteriorly to the Schwalbe’s line was found by gonioscopy from 3 o’clock to 8 o’clock and 9 o’clock to 1 o’clock. Intraocular pressure was 14 mm Hg in the right eye and 15 mm Hg in the left eye. A small space (arrow) between the trabecular meshwork and the iris is shown.

COMMENT

Patients with PPD usually demonstrate normal vision, but endothelial decompensation and/or glaucoma can develop, resulting in visual loss. Intraocular pressure (IOP) elevation in PPD patients occurs in approximately 15% of PPD patients. It seems that extent of synechial closure is not fully correlated with IOP.


HIV retinopathy at seroconversion

EDITOR,—HIV retinopathy is a benign abnormality of uncertain aetiology which was first described in AIDS patients in 1982. The retinal findings comprise cotton wool spots similar in appearance to those found in diabetes mellitus and immune complex disorders. While it may be associated with asymptomatic HIV infection, the retinopathy is generally regarded as a feature seen in patients with laboratory evidence of significant immune deficiency. We report a patient in whom HIV retinopathy was noted during an acute seroconversion illness—a finding which has not been previously described.

CASE REPORT
A 44 year old heterosexual white woman presented to the regional infection unit with a 5 day history of myalgia, arthralgia, fever, anorexia, and watery diarrhoea. On examination a diffuse macular rash was noted, there was generalised lymphadenopathy. Cotton wool spots were noted on ophthalmoscopy of the right retina (Fig 1). Her diarrhoea persisted despite use of enteropaths and she was found to have an inflammatory infiltrate in jejunal biopsy. Despite normal appearances on barium enema and multiple normal colonic biopsies she was thought likely to have mild inflammatory bowel disease and was treated with corticosteroids and mesalazine. Following discharge on this regimen her diarrhoea settled but she continued to lose weight. When readmitted 2 months later the cotton wool spots were again noted on examination. No other disease process liable to cause these was identified; her blood pressure was never higher than 140 mm Hg systolic/80 mm Hg diastolic, erythrocyte sedimentation rate was only 30 mm in the first hour, and autoantibody screen was negative—and in light of her ongoing weight loss, the patient was tested for HIV and found to be antibody positive. Retrospective analysis of stored serum from her first admission showed the presence of HIV antigen with undetectable antibody, indicating that she was undergoing a seroconversion illness at that time. Her absolute CD4+ lymphocyte count on the second admission was 220 cells ×103. Her later clinical course following the diagnosis of HIV infection showed a rapid progression of the disease with a marked decline in CD4+ cell count and development of AIDS within 6 months of diagnosis (AIDS was defined by AIDS.gov4 but had not been formally diagnosed). Her response to antiviral therapy was poor, leading to death 21 months after presentation.

COMMENT
HIV retinopathy is a benign feature of HIV disease which is principally recognised in patients with symptomatic disease or significantly reduced CD4+ cell counts.5 The aetiology is poorly understood and has variously been suggested to be due to circulating immune complexes or to direct infection of the retina by the human immunodeficiency virus.6

In the patient described cotton wool spots were ultimately attributed to her HIV disease and had been present since her presentation during a seroconversion illness. This finding has not been previously reported. Although she had a significantly depleted CD4+ cell count when it was first measured (several months after seroconversion) no measurement of her lymphocyte subsets was made at the onset of her illness and the degree of immunodeficiency associated with the acute seroconversion is therefore unknown. It is recognised that patients can progress rapidly from seroconversion to profound immunodeficiency and AIDS7 and that seroconversion itself can be associated with a marked fall in CD4+ cell count. The latter may have been relevant in our patient as lymphopatapathy noted has reflected the severity of her seroconversion and the concomitant CD4+ lymphocyte depletion. Whether this finding of retinopathy during seroconversion is of any clinical value in aiding diagnosis or as a predictor of the subsequent clinical course is not clear, although it is interesting to note that progression to AIDS was rapid in this case. In the experience of the authors the finding proved helpful in stimulating consideration of the diagnosis in a patient with no obvious risk factors for HIV infection.

J FLINLAYSON
R B LAING
A CADWGAN
F GREEN
Infection Unit, Aberdeen Royal Infirmary, Aberdeen, Scotland
Accepted for publication 21 April 1998

ance and adaptation can take place. Chronic nutritional deficiency states, liver disease, and bacterial overgrowth are common complications.\(^2\) Bacterial overgrowth can exacerbate the malabsorption and this was probably the case with our patient. The manifestations of hypovitaminosis A appear after prolonged depletion. This can often mean a significant time lag between the causative event and presentation.\(^3\)

As vitamin A plays a key role in the elaboration of visual pigment, night blindness is often the earliest sign of vitamin A deficiency. Bitot spots and xerosis of the ocular surface occur after more prolonged deficiency. Keratomalacia is a liquefactive necrosis of the cornea leading to scarring or perforation. The latter may be precipitated by intercurrent infection\(^4\) creating demands that cannot be met by the depleted stores of vitamin A. The importance of a complete medical history is demonstrated by this case. While the early ocular manifestations of hypovitaminosis A are readily reversible,\(^4, 5\) the late changes cause permanent corneal damage and visual loss. In addition, there is an increased childhood morbidity and mortality\(^6\) associated with vitamin A deficiency which can be reduced by restoring vitamin A levels to normal.\(^7\)

Night blindness had been present for at least 3 years and the initially mild lid and conjunctival changes were misinterpreted as being secondary to allergy, possibly because the patient’s skin condition (itself the result of vitamin A depletion, hyperkeratosis) was thought to reflect atopy. Although the clinical presentation was entirely consistent with vitamin A deficiency, the diagnosis was not initially considered because it is so infrequently encountered in developed countries, resulting in an unnecessary delay in instituting the correct treatment.

A history of previous bowel resection, in the presence of intestinal surface abnormalities, should raise the possibility of inadequate absorption and storage of essential vitamins. Patients with short bowel syndrome need regular review and screening for possible deficiencies.\(^8, 9\)

**Table 1** Investigation results

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Pretreatment</th>
<th>Post-treatment (2 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>11.0 g/dl</td>
<td>12.6 g/dl</td>
</tr>
<tr>
<td>Vitamin A (25.8–48.7 µg/dl)</td>
<td>&lt;20 µg/dl</td>
<td>216 µg/dl</td>
</tr>
<tr>
<td>Vitamin E (11.5–35 mmol/l)</td>
<td>Undetectable</td>
<td>0.3 mmol/l</td>
</tr>
<tr>
<td>25 Hydroxy vitamin D (40–195 mmol/l)</td>
<td>7 mmol/l</td>
<td>2.46 mmol/l</td>
</tr>
<tr>
<td>Calcium (2.12–2.65 mmol)</td>
<td>1.81 mmol/l</td>
<td>2.46 mmol/l</td>
</tr>
<tr>
<td>Albumin (3–50 g/l)</td>
<td>30 g/l</td>
<td>48 g/l</td>
</tr>
<tr>
<td>Prothrombin time (10–14 seconds)</td>
<td>32 seconds</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Visual impairment due to bilateral corneal endothelial failure following simultaneous bilateral cataract surgery

**COMMENT**

Previous studies of patients undergoing simultaneous, bilateral modern cataract surgery have reported no bilateral, vision threatening postoperative complications.\(^5\) Even so the possibility of rendering the patient temporarilly or permanently blind cannot be completely ruled out. In a recent consultation section on simultaneous bilateral cataract surgery\(^7\) the main cause of concern among surgeons was the possibility of bilateral endophthalmitis. There was, however, no mention of bilateral secondary endothelial failure resulting in poor vision. Secondary endothelial failure accounts for approximately 25% of patients requiring cataract grafts\(^8\) and they have a higher rate of graft failure and rejection.\(^9\) The visual prognosis is also poorer in this group of patients and it can take up to a year to reach an optimum level.\(^10\) Therefore, patients requiring corneal grafts for bilateral secondary endothelial failure following simultaneous bilateral cataract surgery can potentially be rendered visually handicapped for a long time.

We are not aware of the reasons for our patient having simultaneous bilateral cataract surgery. Unfortunately, despite an apparently normal corneal examination she still developed bilateral secondary endothelial failure resulting in severe visual impairment for a long time.

This case therefore demonstrates that the possibility of bilateral visual loss due to secondary endothelial failure is another strong argument against routine simultaneous bilateral cataract surgery. We suggest that patients who are being offered this surgery should be made aware of the risks and consequences of secondary endothelial failure. Preoperatively, a meticulous examination of their corneal endothelium should be undertaken. If significant corneal endothelial pathology is noted, than only unilateral cataract surgery should be performed. The second eye should have the cataract surgery only after the first eye has been successfully rehabilitated.

**Figure 1** Shows right failed corneal graft with vascularity in one quadrant and a clear corneal graft in the left eye.


**CASE REPORT**

A 76 year old white woman was referred to our cornea clinic with complaint of poor vision. She had undergone an uncomplicated bilateral phacoemulsification with posterior chamber intraocular lens implant in March 1995 at another hospital. A few months before her surgery the visual acuity had been noted to be 6/24 in either eye. She had bilateral cataracts and the corneas were reported as normal. Following the surgery her vision gradually deteriorated in both eyes over 6 months to 0/60 in both eyes. With posterior chamber intraocular lens implant in the left eye. She was referred to our cornea clinic with complaint of poor vision.

On presentation to us in September 1997 her vision was 1/60 right eye and 6/60 left. Examination revealed a right failed corneal graft with vascularity in one quadrant and a clear corneal graft in the left eye (Fig 1). Intraocular lens implants were in situ. The fundus appeared grossly normal in the right eye. Early retinal pigment epithelial changes were noted at the left macula. She was offered a repeat right corneal graft with a guarded prognosis but she decided against it.

**COMMENT**

Previous studies of patients undergoing simultaneous, bilateral modern cataract surgery have reported no bilateral, vision threatening postoperative complications.\(^5\) Even so the possibility of rendering the patient temporarily or permanently blind cannot be completely ruled out. In a recent consultation section on simultaneous bilateral cataract surgery\(^7\) the main cause of concern among surgeons was the possibility of bilateral endophthalmitis. There was, however, no mention of bilateral secondary endothelial failure resulting in poor vision. Secondary endothelial failure accounts for approximately 25% of patients requiring cataract grafts\(^8\) and they have a higher rate of graft failure and rejection.\(^9\) The visual prognosis is also poorer in this group of patients and it can take up to a year to reach an optimum level.\(^10\) Therefore, patients requiring corneal grafts for bilateral secondary endothelial failure following simultaneous bilateral cataract surgery can potentially be rendered visually handicapped for a long time.

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**AJAI K TYAGI**

**PETER J MCDONNELL**

**Birmingham and Midland Eye Centre, Dudley Road, Birmingham B18 7QG**

Correspondence to: Aji K Tyagi.

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Symptomatic acute raised IOP following haemodialysis in a patient with end stage renal failure

EDITOR,—We report a case of a 45 year old man with chronic renal failure presenting with symptomatic bilateral acute raised intraocular pressure (IOP) following haemodialysis. The pressures were successfully reduced with a topical β blocker and following the commencement of regular topical treatment his symptoms were controlled with no further record of raised IOPs.

CASE REPORT

A 45 year old white man was referred to the eye casualty department by the renal physi-cian, complaining of bilateral blurred vision and a dull frontal headache following haemo-dialysis. The blurred vision resolved spontane-ously within 2 hours of onset but the headache persisted. The headaches had been recurrent following every haemodialyses which he had undergone and could last up to 10 hours. The blurred vision was a less consistent feature, only occurring occasionally. He had end stage renal failure due to glomerulonephritis and had been commenced on haemodialysis in a patient who had previously healthy eyes. Carbonic anhydrase inhibitor is relatively contraindicated in this condition as it can precipitate severe metabolic acidosis. Regular topical β blocker can be used to control this condition.

YEE FONG CHOONG
M MJ MENAGE
Eye Department, Leeds General Infirmary, Leeds LS2 SNS

Correspondence to: Dr Choong.
Accepted for publication 7 May 1998

COMMENT

Symptomatic raised IOP following haemodialysis is rarely diagnosed. Asymptomatic raised IOP following haemodialysis has been reported in the medical literature. Several studies have shown that raised IOP follows haemodialysis in a significant number of patients while others have failed to show this relation.1 4 The prevalence of this phenomenon among patients undergoing haemodialysis is not known and the pathophysiology involved is not certain. The elevation of IOP may be due to a decrease in outflow facility and an osmotic influx of water into the eye because of hyperosmolality of intraocular fluids following dialysis.5 In all the studies, the raised IOP was of questionable clinical significance. All except one patient who had a history of narrow angle glaucoma were asymptomatic. To our knowledge this is the first case reported of symptomatic acutely raised intraocular pressure following haemodialysis in a patient who had previously healthy eyes. Carbonic anhydrase inhibitor is relatively contraindicated in this condition as it can precipitate severe metabolic acidosis. Regular topical β blocker can be used to control this condition.


Episcleral melanoma without conjunctival or uveal involvement

EDITOR,—Melanocytic lesions of the episclera include Axenfeld nerve loop, episcleral melanocytosis, ochronosis, conjunctival nae-vus, cellular blue naevus, melanocytoma, conjunctival melanoma with deep extension, extraocular extension of uveal melanoma, or metastatic melanoma.1 2 The occurrence of an episcleral melanoma without conjunctival, uveal, or skin involvement is extremely rare. We report an unusual case of malignant melanoma occurring as an isolated tumour on the episcleral surface.

CASE REPORT

A 36 year old healthy man developed a pigmented epibulbar lesion in the left eye over a 1 year period. There was no history of ocular trauma, cutaneous melanoma, or dys-plastic naevus syndrome. Ocular examination revealed visual acuities of 6/6 in both eyes. In the left eye, there was an episcleral pigmented mass located 2 mm from the limbus at the 10 o’clock position, measuring 4.0 × 3.5 mm in base (Fig 1). The conjunctiva was freely mobile over the lesion. Anterior segment examination was otherwise normal with no sign of conjunctival naevus, primary acquired melanosis, or malignant melanoma. There was no evidence of an intraocular melanoma by funduscopy. On transillumination, blockage of light transmission by the epibulbar lesion was noted. The differential diagnosis included an
episceral cellular blue naevus, melanocytoma, melanoma, or foreign body.

The epibulbar lesion was removed via “no touch” partial lamellar scleroconejunctivectomy approach with wide margins and supplemental cryotherapy to the surrounding conjunctiva. After surgery, there was no transillumination light blockage. Pathological examination disclosed a deep subconjunctival lesion comprised of epithelioid melanocytes with prominent nuclei, consistent with malignant melanoma (Fig 2). Mitoses were not observed. There was no evidence of primary acquired melanosis. Similarly, an emissarial scleral canal at the base of the mass was tumour free. Subsequent systemic evaluation revealed no sign of primary melanoma elsewhere. The patient has been followed for 1 year with no evidence of recurrence or metastasis.

COMMENT
Episcleral melanoma may be impossible to clinically differentiate from cellular blue naevus or melanocytoma. A careful conjunctival and funduscopic examination is necessary to rule out extracellular extension from an uveal melanoma or contiguous spread from a conjunctival melanoma. In order to thoroughly rule out metastatic melanoma, a systemic evaluation should be performed. Melanoma originates mostly in the skin (86%), followed by the eye (11%), vulva (1%), soft tissues (<1%), rectum (<1%), and vagina (<1%). In our patient, there was no evidence of primary melanoma elsewhere, although we realise that metastatic melanoma can occur in the body without detection of a primary site. In a series of 10 metastatic tumours to the conjunctiva, there were two cases of cutaneous metastatic melanoma both of which also had uveal metastasis. However, our patient had no evidence of an intraocular tumour.

The origin of the episcleral melanoma remains obscure, although most probably it arises from melanocytes deep in the subconjunctival tissues. Malignant melanoma should be considered in the differential diagnosis of an episcleral pigmented lesion.

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KAAN GUNDÜZ
JERRY A SHIELDS
CAROL L SHIELDS
Oncology Service, Wills Eye Hospital, Thomas Jefferson University, Philadelphia, PA, USA

RALPH C EAGLE JR
Pathology Department, Wills Eye Hospital, Thomas Jefferson University, Philadelphia, PA, USA

Correspondence to: Jerry A Shields, MD, Director, Oncology Service, Wills Eye Hospital, 900 Walnut Street, Philadelphia, PA, 19107, USA
Accepted for publication 15 April 1998


Congenital lenticular pigmentation

Editor,—Pigment deposits on the anterior lens capsule may be seen in a variety of conditions. In this case report, we describe unusual bilateral pigmentation of the anterior lens surface. The 21 year old woman with myopia was referred for evaluation of pigmentary dispersion syndrome. There was no previous history of ocular inflammation, trauma, or use of topical or systemic medications. There were no visual complaints.

On examination, her best corrected visual acuity was 20/15 right eye and 20/20 left eye with –1.25 D sph both eyes and J1+ for near. Slit lamp examination revealed clear corneas bilaterally with no pigment deposits over the posterior corneal surfaces. The anterior chambers were deep and quiet. No irides transillumination defects were noted. Intraocular pressure was 14 mm Hg before and after pupillary dilatation. On pupillary dilatation, the anterior surface of the lens in the right eye demonstrated clumps of pigmented cells. The cells were paraxial in location, closely packed with a few isolated cells in the pupillary axis (Fig 1). The cells were rounded, fusiform orstellate in shape (Fig 2). In the left eye, similar stellate pigmented cells were seen in the pupillary axis. The zonal attachments in the right eye were prominent. However, no pigment deposits were noted in the peripheral capsule, zonules, or on the posterior lens capsule. On gonioscopy, the angles were wide open with ciliary body visible 360° both eyes (E 40 q, Spach classification). No pigment deposits were noted in the angle structures. Fundus examination revealed a clear vitreous, cup to disc ratios of 0.5 right eye and 0.6 left eye, an unremarkable posterior pole, and periphery.

COMMENT This case represents an unusual asymmetric pigmentation of the anterior capsule of the lens. The closely packed fusiform pigmented cells resembled iris pigment epithelial cells or pigmented ciliary epithelial cells in culture. In the absence of signs of intraocular inflammation, and other causes of primary and secondary of pigmentary dispersion, it is likely that the pigmented cells were implanted on the lens surface in utero from the developing iris pigment epithelium. It is possible that there was migration of some of these implanted cells into the visual axis but at present appear non-progressive and do not impair the patient’s vision. The paraxial location of congenital lenticular pigmentation is unusual. Previously described cases of congenital pigmentation showed a radial distribution of the pigmentation in the mid and peripheral portions of the anterior lens capsule. These radial pigmented lines were pathologically confirmed to be melanin pigment granules incorporated within the lens zonules. Pigmentation of the anterior lens surface may be seen in many conditions including anterior segment inflammation with posterior synchiae, pigmentary dispersion syndrome, siderosis, the aging eye, pseudoxfolliation syndrome, antipsychotic medication usage, and remnants of the tunica vasculosa lentis. These must be differentiated from congenital lenticular pigmentation as seen in this case.

This work is supported by an unrestricted grant to Washington University from Research to Prevent Blindness, and EY 02687 Core Grant for Vision Research.

DEEPAK P EDWARD Department of Ophthalmology, University of Illinois Eye Center, Chicago, USA

MARTIN B WAX Department of Ophthalmology and Visual Sciences, Washington University School of Medicine, St Louis, USA

Correspondence to: Martin B Wax, MD, Department of Ophthalmology, Washington University School of Medicine, PO Box 8096, 660 S Euclid Avenue, St Louis, MO 63110, USA. Accepted for publication 7 May 1998

Fluorescein and indocyanine green angiography in arteritic anterior ischaemic optic neuropathy

Editor,—Anterior ischaemic optic neuropathy (AION) is the most common cause of visual loss in giant cell arthritis (GCA). However, other presentations have been described including posterior ischaemic optic neuropathy, choroidal ischaemia, retinal artery occlusion, branch retinal artery occlusion, cilioretinal artery occlusion, and occipital cortex infarction. We present the first indocya-

nine green angiography (ICGA) findings in a case of GCA with simultaneous optic nerve and choroidal ischaemia.

CASE REPORT A 65 year old woman was admitted because of bilateral blindness. She had had complete, painless visual loss in her right eye 72 hours before admission followed 48 hours later by visual loss in the left. One week earlier she had noted jaw claudication and neck pain. Ophthalmic examination revealed no light perception (NLP) with disc oedema in both eyes. Wester-gren erythrocyte sedimentation rate (ESR) was 76 mm in the first hour, and magnetic resonance imaging was normal. She was given 50 mg/day oral prednisone, with no improvement. One week later she was referred to our institution for a neuro-ophthalmological evaluation. Visual acuities were still NLP with mid-dilated, unreactive pupils; funduscopic examination showed pale disc oedema in both eyes. ESR was 30 mm in the first hour. Fluorescein angiography (FA) (Fig 1) showed marked delay in optic nerve and choroidal filling; mild optic nerve leakage, and peripheral hyperfluorescent spots with RPE mottling were seen in the late angiographic phases in the left eye. ICGA (Fig 2) confirmed the severe ischaemia of the optic nerve and on the temporal side and highlighted staining of several peripheral choroidal vessels. Temporal artery biopsy was positive for GCA. Predni-sone was increased to 100 mg/day but there was no recovery of visual function at follow up.

COMMENT The association of choroidal ischaemia and AION is particularly suggestive of GCA as indicated by Hayreh. Mack et al and Siatkowski et al performed FA in GCA and found significant delay of choroidal filling in comparison with either normal subjects or patients with non-arteritic AION. We report a case of optic nerve and simultaneous choroi-
dal ischaemia in GCA. Choroidal hyperper-

fusion was more severe on the temporal side suggesting a distinct involvement of the lateral posterior ciliary arteries (PCAs). FA also highlighted areas of RPE atrophy and pigmented migration in the peripheral retina. Similar abnormalities of the outer retina in GCA were attributed to arteritic involvement of the PCA supply to the choroid. Siatkowski et al performed FA in GCA and found significant delay of choroidal filling in comparison with either normal subjects or patients with non-arteritic AION. We report a case of optic nerve and simultaneous choroidal ischaemia in GCA. Choroidal hyperperfusion was more severe on the temporal side suggesting a distinct involvement of the lateral posterior ciliary arteries (PCAs). FA also highlighted areas of RPE atrophy and pigmented migration in the peripheral retina. Similar abnormalities of the outer retina in GCA were attributed to arteritic involvement of the PCA supply to the choroid.
This is the first ICGA study of choroidal circulation in GCA. ICGA clearly demonstrated the choroidal ischaemia but also showed staining of some peripheral vessels, probably related to an inflammatory infiltration of their wall not visible with ophthalmoscopy and FA. Even though no conclusions can be drawn from a single case, ICGA may a valuable diagnostic tool for differentiating arteritic from non-arteritic AION and even an interesting way to monitor the disease.

FEDERICO SADUN
ALFREDO PECE
ROSAR m BRANCATO
Department of Ophthalmology and Visual Science,
Scientific Institute H San Raffaele,
Milano-Roma, Italy

Correspondence to: Professer Rosario Brancato,
Institute H San Raffaele, Department of Ophthalmology, V le Olgettina 60, 20132 Milan, Italy.
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Intraocular and extraocular bleeding after intracameral injection of tissue plasminogen activator

EDITOR,—During the early postoperative period after glaucoma filtration surgery the seldrostomy can be blocked by haemorrhage or fibrin clot. In these cases tissue plasminogen activator (tPA) can be injected into the anterior chamber after paracentesis or subconjunctivally. It works rapidly so that within 3 hours the effect is usually apparent. This report describes a patient who had massive ocular bleeding after intraocular injection of tPA.

CASE REPORT
A 76 year old white man with uncontrolled advanced primary open angle glaucoma in the left eye underwent trabeculectomy with mitomycin C. Past ocular history was relevant for trabeculectomy with 5-fluorouracil, 8 years earlier, and a combined mitomycin C trabeculectomy, phacoemulsification, and intraocular lens implantation 2 years before. Medical history regarding bleeding or coagulation disorders was negative, although tests to exclude abnormalities in the coagulation system were not done. The patient did not take coagulation inhibitors before or after surgery.

The surgery was uneventful. One day after surgery the intraocular pressure (IOP) was 10 mm Hg and there was a large superotemporal filtering bleb. One week later the IOP was 30 mm Hg, with a very vascularised low bleb and a deep anterior chamber. Laser suture lysis (two sutures) and digital ocular compression did not lower the IOP. An intracameral injection of 15 µg of tPA was done. The following day the patient had a large (40%) hyphaema and a dense subconjunctival haemorrhage extending to the eyelids and the orbital rim (Figs 1 and 2). Mild vitreous haemorrhage was also present. Vision was hand movements and IOP was 5 mm Hg. Ocular trauma had...
not occurred. The blood resorbed over 3 weeks, and the function of the bleb remained satisfactory.

COMMENT
Recombinant tPA is a serine protease with clot specific fibrinolytic activity. tPA has been used successfully to lyse blood, fibrinous clots, and/or membranes after pars plana vitrectomy, cataract surgery, and glaucoma surgery. A dose of up to 25 µg of tPA is used for ophthalmic procedures. Hyphaema is the most frequent complication of intracameral tPA injection after glaucoma surgery (up to 36% of cases). Lundy et al suggested that a dose of 6–12.5 µg may be equally effective and reduces the risk of hyphaemases.

In this patient the bleeding source was probably intraocular, which extended to the subconjunctival space through the fistula (functioning after the tPA injection), and to the preseptal periorcular tissues because of the large volume of the haemorrhage. It is not know whether previous surgeries and/or ocular scarring might have contributed to the intensity of the bleeding. The use of mitomycin C and laser suture lysis were probably not related to this complication. The singular aspect of this case was the severity of the bleeding, and its extraocular extension.

AUGUSTO AZUARA-BLANCO
Department of Ophthalmology, Queen's Medical Centre, University of Nottingham, Nottingham

RICHARD P WILSON
Glaucoma Service, Wills Eye Hospital, Jefferson Medical College, Philadelphia, USA

Correspondence to: Augusto Azuara-Blanco, MD, Department of Ophthalmology, B-Floor, South Block, Queen's Medical Centre, Nottingham, NG7 2UH.
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