LETTERS TO THE EDITOR

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. [1] 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 12 mm Hg in the left eye. Pupillary distortion and glassy membrane were found but no iridial hole was noticed in the left eye (Fig 1). The right eye was almost normal except for three very tiny vesicular lesions on the posterior cornea. Specular microscopy of the right central cornea revealed diffuse endothelial changes with pleomorphism and polymegathism. The number of endothelial cells in the right eye was 500/mm^2 but in the left eye, the measurement was not possible. Her daughter's corneas demonstrated thickening of Descemet's membrane, band-like figures on posterior corneal surface and endothelial cell loss (1500/mm^2).

By UBM (Zeiss-Humphrey, San Leandro, CA, USA), the anterior iris was broadly adhered to the posterior corneal surface but the trabecular meshwork was not fully covered (Fig 2). The iridial surface adhered to the protruded Schwalbe's line, and the glassy membrane pulled the anterior iris to the cornea but the position of posterior iris was rather normal. UBM could not distinguish the glassy membrane from the iris.

COMMENT

Patients with PPD usually demonstrate normal vision, but endothelial decompensation and/or glaucoma can develop, resulting in visual loss. Intraocular pressure (IOP) elevation occurs in approximately 15% of PPD patients.[1] It seems that extent of synechial closure is not fully correlated with IOP.[1] In this case, three quarters of the anterior chamber angle especially in the superior and inferior quadrants was closed by gonioscopy. However, the aqueous outflow pathway was preserved because of tunnels of open trabecular meshwork below the synechiae as shown in Figure 2. The UBM findings explain why her IOP was not elevated in spite of the broad iridocorneal adhesion. Furthermore, the anterior layer of the iris were elevated to the cornea but those of the posterior layer was not. This finding suggests that only the anterior layer might be atrophic because of the maldevelopment of the iris and/or iris ischaemia.[1 2]

Glaucoma in PPD is believed to result from the aqueous outflow obstruction caused by synechial closure of the trabecular meshwork by metaplastic corneal endothelial cells and membranes.[1 3-6] However, our findings do not support this hypothesis. IOP elevation in PPD may be explained not only by synechial closure but also by abnormalities of neural crest cell differentiation or the basement membrane.[1 3-6] Recording of UBM images on more cases of PPD is needed to clarify mechanism of IOP elevation in this disease.

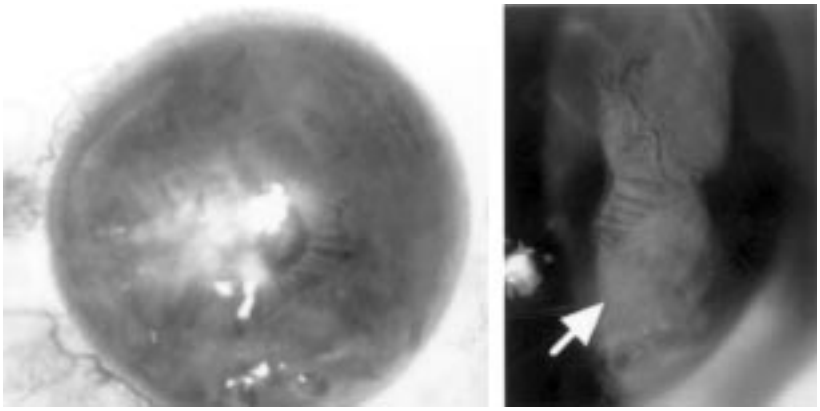

Figure 1 Anterior segment of the left eye. The glassy membrane (arrow) covered the normally appearing iris surface. The membrane broadly attached to the posterior corneal surface.

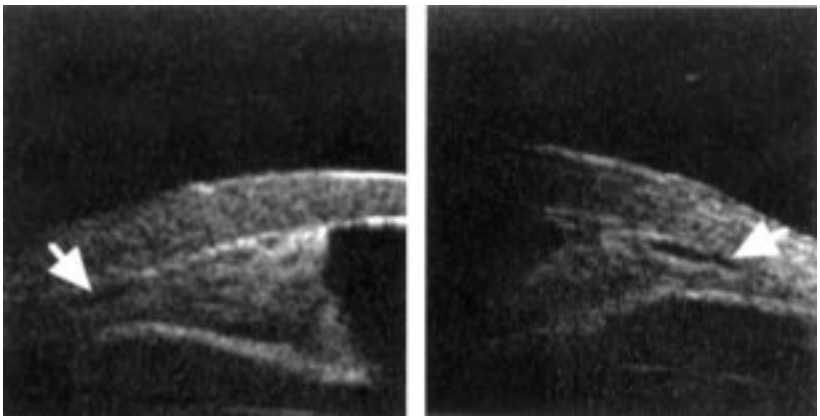

Figure 2 Ultrasound biomicroscopy cross sectional view through the anterior chamber angle in the left eye. A small space (arrow) between the trabecular meshwork and the iris is shown.

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HIV retinopathy at seroconversion

EDITOR,—HIV retinopathy is a benign abnormality of uncertain aetiology which was first described in AIDS patients in 1982.[1] The retinal findings comprise cotton wool spots similar in appearance to those found in diabetes mellitus and immune complex disorders.[2] 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.[3] 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 and cotton wool spots were noted on ophthalmoscopy of the right retina (Fig 1). Her diarrhoea persisted despite the absence of enteropaths and she was found to have an inflammatory infiltrate on rectal 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 $\times 10^6$/l.

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 wasting being the AIDS defining illness). 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.[4] The aetiology is poorly understood and has variously been suggested to be due to circulating immune complexes[1] or to direct infection of the retina by the human immunodeficiency virus.[5]

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 AIDS[6–8] and that seroconversion itself can be associated with a marked fall in CD4+ cell count. The latter may have been relevant in our patient whose retinopathy may have 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.

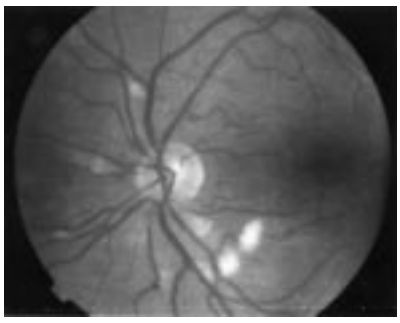

Figure 1 Cotton wool spots seen on ophthalmoscopy of the right retina.

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Xerophthalmia and short bowel syndrome

EDITOR,—Vitamin A deficiency due to malnutrition is the leading cause of childhood blindness worldwide[1] but is rarely seen in developed countries. Although case reports of xerophthalmia in developed countries have appeared in the general medical[2 3] and ophthalmic literature,[4 5] the relative rarity of the condition can lead to delay in diagnosis with potentially serious consequences. We present a patient with short bowel syndrome whose grossly depleted vitamin A stores only came to light with the onset of severe ocular surface disease.

CASE REPORT

The patient, now 8 years old, was born after a normal pregnancy by breech delivery at 36 weeks. At age 1 day he developed abdominal distension with bile stained vomiting. At laparotomy multiple small bowel atresia were excised and a jejunostomy fashioned. This was closed 3 weeks later and replaced by a jejunojejunal anastomosis. The latter required two revisions (at 10 days and 2 months after the initial anastomosis) with excision of more bowel and division of adhesions. For the first few months he was fed only intravenously, this being very gradually replaced by oral feeding over the subsequent 4 months. Vitamin and mineral supplements were included in his diet up to age 2 years. He has always been of small stature and low weight (below 3rd centile for both factors) and prone to recurrent infections and had frequent (up to six a day) motions which tended to float. Epiphyseal maturation has been delayed (bone age at 7.9 years of age was equivalent to that of a 3 year old child). He had dry flaky skin which was thought to be due to eczema. After his family moved he was lost to hospital follow up and general practitioner review had only been intermittent.

His mother had noted red eyes and swollen lids over the previous 2½ years. Difficulties seeing in the dark were also apparent. His grandfather recalled taking him to the cinema some 3 years earlier and noting that the child appeared not to see in the dark. A tentative diagnosis of allergic conjunctivitis had been made at a casualty department and topical steroid drops had brought about temporary improvement in the redness of his eyes. However, in the 3 months before presentation to us, the redness had recurred with increasing discomfort, sensitivity to bright light and decreased vision causing distress and severe disruption of education. Treatment with topical lubricant, antibiotics, and steroids was ineffective. He was referred to us for a second opinion regarding his chronic ocular surface problems.

At examination he had great difficulty in opening his eyes because of discomfort and photophobia. Visual acuity was reduced to hand movements in both eyes. The skin of the eyelids was red and thickened and the eyelashes were excessively long. Both eyes were injected but most striking was the marked xerosis of the conjunctiva and absence of corneal lustre. Both corneas showed confluent punctate staining with fluorescein but no epithelial defects or new vessels. Schirmer's test was normal but the ocular surface on both sides was totally unwettable. A diagnosis of vitamin A deficiency was made on the basis of his ocular signs. The child was then initially treated by his local paediatric team and referred on to Great Ormond Street for further care.

Biochemistry showed multiple deficiencies, in particular of the fat soluble vitamins and calcium (Table 1). His prothrombin time was prolonged. Endoscopy revealed chronic oesophagitis and dilated residual small bowel loops and a hydrogen breath test was suggestive of bacterial overgrowth. Treatment based on the World Health Organisation guidelines,[1] with 100 000 IU of vitamin A intramuscularly, oral vitamin K, and subsequently oral Ketovite and Forceval (vitamin and mineral supplements) and other supplements resulted in improvement in ocular symptoms and signs within 5 days. Two weeks after starting treatment, his visual acuity had recovered to 6/9 right eye, 6/6 left eye and examination was normal apart from a faint subepithelial corneal opacity just below the visual axis in the right eye. Systemic antibiotic therapy for the bacterial overgrowth was also instituted.

COMMENT

In developed countries vitamin A deficiency is seen in patients with severe liver disease leading to storage and metabolic abnormalities. Bowel disease or surgical shortening[3 6] can cause insufficient absorption resulting in deficiencies as in our patient. The amount and site of bowel resected determines how much toler-

Table 1 Investigation results

	Pretreatment	*P ost-treatment (2 weeks)*
Haemoglobin	11.0 g/dl	12.6 g/dl
Vitamin A (25.8–48.7 µg/dl)	<20 µg/dl	216 µg/dl
Vitamin E (11.5–35 mmol/l)	Undetectable	0.3 mmol/l
25 Hydroxy vitamin D (40–195 mmol/l)	7 mmol/l	—
Calcium (2.12–2.65 mmol)	1.81 mmol/l	2.46 mmol/l
Albumin (3–50 g/l)	30 g/l	48 g/l
Prothrombin time (10–14 seconds)	32 seconds	Normal

ance and adaptation can take place. Chronic nutritional deficiency states, liver disease, and bacterial overgrowth are common complications.[6] 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.[5 7]

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[1] 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,[2 4 7] the late changes cause permanent corneal damage and visual loss. In addition, there is an increased childhood morbidity and mortality[8] associated with vitamin A deficiency which can be reduced by restoring vitamin A levels to normal.[9]

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 ocular 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.[2–4 6 7]

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

EDITOR,—Although present day cataract surgery has a high success rate, simultaneous bilateral cataract surgery is not routinely performed.[1] The main cause for concern in patients undergoing simultaneous bilateral surgery is the possibility of visual impairment due to serious complications affecting both eyes. The potential problem that is most frequently highlighted in the literature is the risk of bilateral endophthalmitis.[1 2] We report a case of bilateral poor vision following simultaneous bilateral phacoemulsification and intraocular lens implant due to secondary corneal endothelial failure. To our knowledge this has not been previously reported.

CASE REPORT

A 76 year old white woman was referred to our cornea clinic with complaint of poor vision. She had undergone an uncomplicated simultaneous 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 3/60 right and hand movements left. This was due to bilateral diffuse corneal oedema secondary to endothelial failure. She underwent a left penetrating keratoplasty in September 1995 and subsequently the same operation was performed in her right eye in August 1996. Unfortunately the right corneal graft failed in the postoperative period.

On presentation to us in September 1997 her vision was 1/60 right eye and 6/36 left. Examination revealed a right failed corneal graft with vascularisation 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.[1 3] 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[2] 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 corneal grafts[4] and they have a higher rate of graft failure and rejection.[5 6] The visual prognosis is also poorer in this group of patients and it can take up to a year to reach an optimum level.[7 8] 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.

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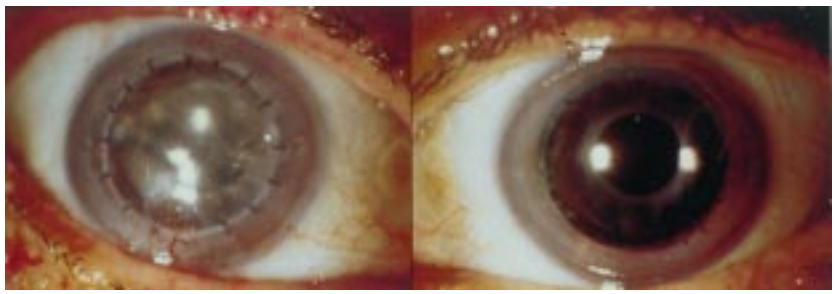

Figure 1 Shows right failed corneal graft with vascularisation in one quadrant and a clear corneal graft in the left eye.

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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 physicians, complaining of bilateral blurred vision and a dull frontal headache following haemodialysis. The blurred vision resolved spontaneously 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 three times a week. There was no relevant past ocular or family history of note. On examination unaided visual acuities were 6/5 in both eyes. The eyes were quiet and the corneas were clear. There was no relative afferent pupillary defect. The anterior chamber depths were estimated at 2.6 mm in both eyes using the Smith method.[1] The right IOP was 42 mm Hg and the left was 36 mm Hg. The angles were open at grade 3–4 using the Shaffer grading system with no peripheral anterior synechiae in both eyes.[2] There was no evidence of pigment dispersion. There was no significant elevation of IOPs following pupil dilatation. Both discs were healthy with good neuroretinal rims. We treated the raised ocular pressures with levobunolol 0.5% eye drops alone. Systemic carbonic anhydrase inhibitor was relatively contraindicated in renal failure. Within 1 hour the headache had resolved and the IOP decreased to 24 mm Hg in the right and 18 mm Hg in the left. Subsequently we arranged to measure his IOPs before and after haemodialysis to establish a causal relation. Before haemodialysis the IOPs were 18 mm Hg in the right and 16 mm Hg in the left. Following haemodialysis, the IOPs were 32 mm Hg and 28 mm Hg in the right and left eye, respectively. Consequently he was commenced on levobunolol 0.5% twice daily and since then his symptoms have improved markedly with minimal headaches and no visual disturbances.

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.[3–6] 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.[4] 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.[5] Regular topical β blocker can be used to control this condition.

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Episcleral melanoma without conjunctival or uveal involvement

EDITOR,—Melanocytic lesions of the episclera include Axenfeld nerve loop, episcleral melanocytosis, ochronosis, conjunctival naevus, cellular blue naevus, melanocytoma, conjunctival melanoma with deep extension, extraocular extension of uveal melanoma, or metastatic melanoma.[1–5] 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 dysplastic 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

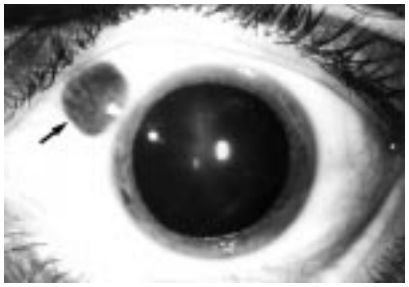

Figure 1 Anterior segment photograph of the left eye showing the epibulbar pigmented lesion (arrow).

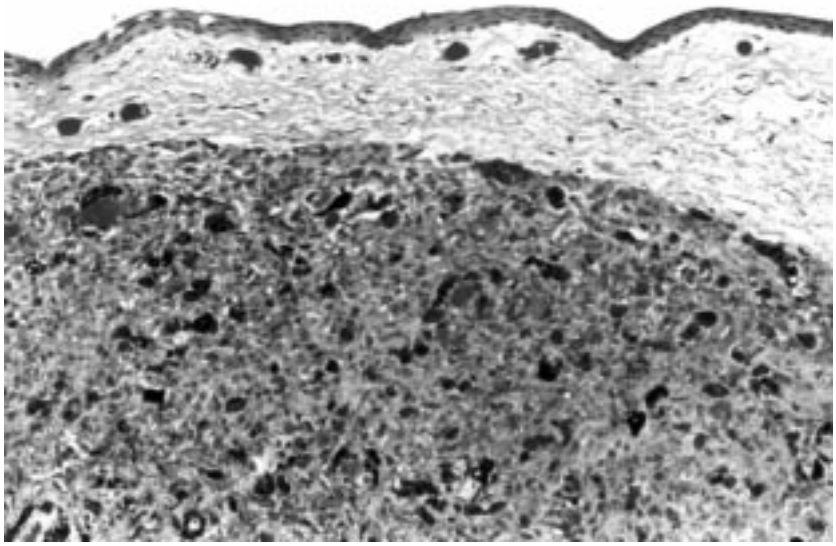

Figure 2 Photomicrograph demonstrating the deep subconjunctival lesion containing epithelioid melanocytes with prominent nucleoli, consistent with malignant melanoma (haematoxylin and eosin, original magnification, ×150)

episcleral cellular blue naevus, melanocytoma, melanoma, or foreign body.

The epibulbar lesion was removed via "no touch" partial lamellar scleroconjunctivectomy 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 nucleoli, 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 extraocular 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%).[6] 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.[5] 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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Intraocular metastasis of endodermal sinus tumour

Editor,—Ocular metastases were previously thought to be rare.[1] However, after having been extensively studied in patients with cancers, the incidence of ocular metastatic tumours increased.[1–5] Haematological malignancy—including leukaemia, lymphoma, and multiple myeloma—is the most common primary cancer whereas mammary and pulmonary carcinomas are regarded as the most frequent intraocular metastatic carcinomas.[1–5] We report an intraocular metastatic endodermal sinus (yolk sac) tumour which, to our knowledge, has not yet been described.

CASE REPORT

A 45 year old man presented with blurred vision in the left eye. Funduscopic study suggested a posterior choroidal tumour causing retinal detachment. Additional physical examination disclosed a 10 cm mass in the anterior wall of the chest.

Computed tomography of the thorax exhibited a mass, 13 × 11 × 8 cm, in the anterior mediastinum. The lesion with solid and cystic components extended into the right lung and anterior wall of the chest. Left enucleation was subsequently performed.

Grossly, the left eyeball revealed a grey white mass, 2 × 1.5 × 1.5 cm, with cystic degeneration in the posterior choroid. It protruded into the vitreous chamber (Fig 1). Microscopically, the ocular lesion was composed of glandular structures of various sizes lined by low columnar cells having large, oval, and basophilic nuclei with coarse granules of chromatin and inconspicuous nucleoli. Numerous eosinophilic hyaline globules were noted (Fig 2, upper). Schiller–Duval bodies, however, were not observed.

Immunohistochemical stainings revealed strong cytoplasmic positivity to α fetoprotein (Fig 2, lower), α-1-antitrypsin, epithelial membrane antigen, and cytokeratin. Stainings for S-100 protein, HMB-45, GFAP, NF, HCG, and CD30 were negative. The diagnosis was endodermal sinus tumour (EST). Subsequently, biopsy of the chest wall lesion consisting of a few pieces of grey tissue, 0.5–1 cm in greatest dimension, was obtained. Sections revealed round vacuolated tumour cells forming nests and glands. Immunohistochemical study showed the same result as previously described in the ocular lesion. Furthermore, a high serum α fetoprotein level of 6850 IU/ml was detected. The final diagnosis was primary anterior mediastinal EST showing prominent glandular differentiation with metastasis to the left eyeball.

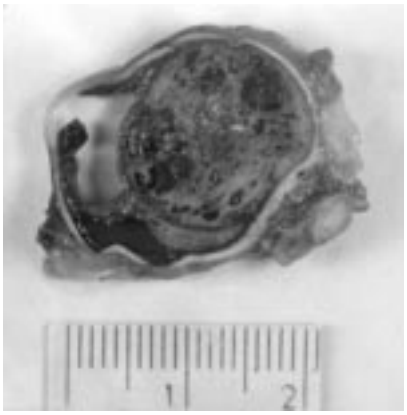

Figure 1 Gross specimen of the left eye showing grey mass with cystic degeneration in posterior choroid producing retinal detachment.

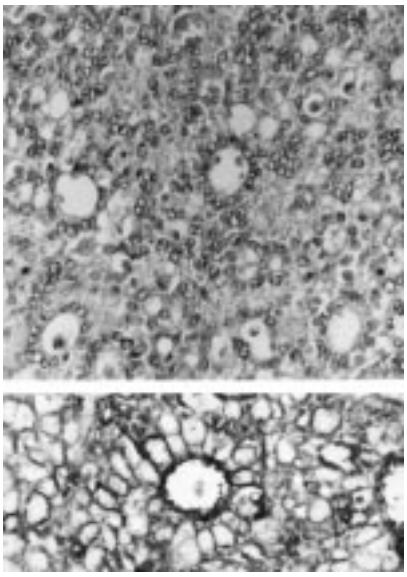

Figure 2 Metastatic intraocular endodermal sinus tumour. Upper, depicting tumour cells with glandular arrangement and abundant hyaline globules. Lower, tumour cells showing positivity to a fetoprotein.

COMMENT

EST has several histological features such as reticular, polyvesicular-vitelline, hepatoid, endometrioid-like, intestinal, and mixed variants.[6] Although Schiller–Duval body is the diagnostic hallmark of EST, it is not found in all cases.[7 8] In our example, the diagnosis of EST was based on the morphology of the tumour cells, numerous hyaline globules, and identification of α fetoprotein in neoplastic cells as well as in serum. This pattern of EST with prominent glandular differentiation is similar to that originally described in the ovary by Cohen *et al.*[7] A small sized ocular tumour compared with the mediastinal one as well as absence of any previous report on primary ocular EST makes us believe that the mediastinal EST is the primary cancer that metastasises to the left eyeball.

Malignant melanoma should be in differential diagnosis because it has diverse histopathological patterns and is common intraocular neoplasm in the West. However, hyaline globules are not the feature of malignant melanoma and there is no immunohistochemical finding to support. Moreover, the uveal melanoma is extremely rare in Thailand.[9]

In summary, we present an intraocular metastatic EST in which we suggest to spread from the anterior mediastinal EST. Although metastatic cancer to the eyes is currently believed to be the most common ocular malignancy,[5] EST has not yet been recorded to metastasise intraocularly.

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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 in a young woman. Recognition of this benign condition should diminish the clinician's concern that other potentially progressive disorders such as pigmentary glaucoma are likely.

CASE REPORT

A 21 year old white 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 iris transillumination defects were noted. Intraocular pressures were 15 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 or stellate in shape (Fig 2). In the left eye, similar stellate pigmented cells were seen in the pupillary axis. The zonular 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, Spaeth 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.[1]

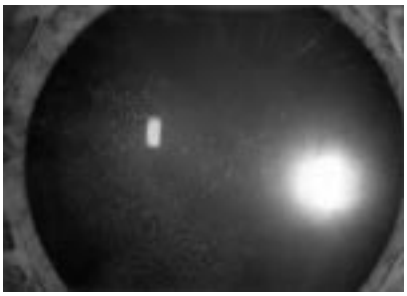

Figure 1 Slit lamp photograph showing the paraxial closely packed and the isolated central pigmented cells. Note the prominent zonular attachments superiorly.

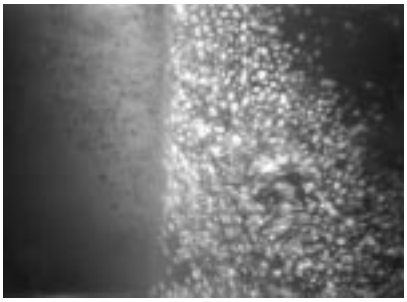

Figure 2 Slit lamp photograph at a higher magnification. Note the fusiform, stellate, and rounded pigmented cells on the anterior lens surface.

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.[2 3] These radial pigmented lines were pathologically confirmed to be melanin pigment granules incorporated within the lens zonules.[2] Pigmentation of the anterior lens surface may be seen in many conditions including anterior segment inflammation with posterior synechiae, pigmentary dispersion syndrome, siderosis, the aging eye,[2] pseudoexfoliation syndrome,[2] antipsychotic medication usage,[4] and remnants of the tunica vasculosa lentis.[3] These must be differentiated from congenital lenticular pigmentation as seen in this case.

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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 arteritis (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.[1] We present the first indocyanine 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 claudicatio and neck pain. Ophthalmic examination revealed no light perception (NLP) with disc oedema in both eyes. Westergren erythrocyte sedimentation rate (ESR) was 76 mm in the first hour; brain 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 choroid especially on the temporal side and highlighted staining of several peripheral choroidal vessels. Temporal artery biopsy was positive for GCA. Prednisone 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.[2] Mack *et al*[3] and Siatkowski *et al*[4] 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 hypoperfusion 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 pigmentary 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.[5]

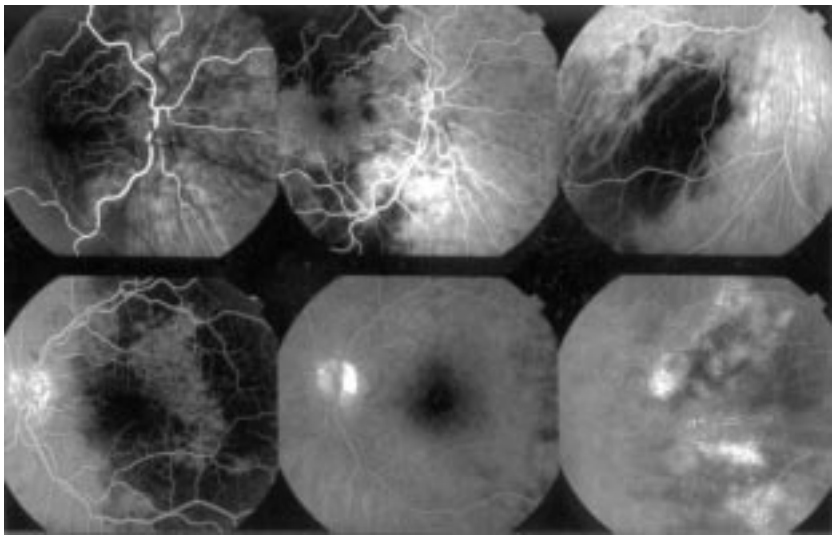

Figure 1 Fluorescein angiography showed marked delay in optic nerve perfusion (up to 20 seconds) and choroidal filling (up to 10 minutes) in both eyes as well as peripheral hyperfluorescent spots with RPE mottling in the late angiographic phases in the left eye (bottom, right).

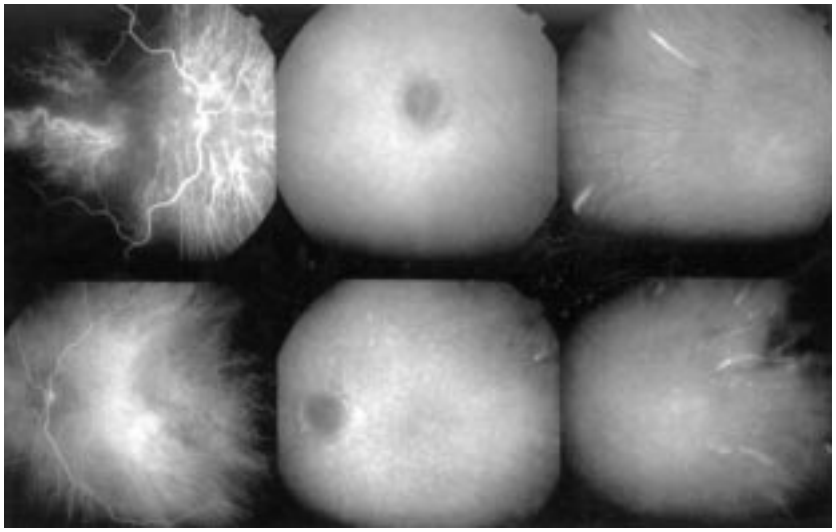

Figure 2 Indocyanine green angiography highlighted severe ischaemia of the choroid especially on the temporal side with staining of several peripheral choroidal vessels (top and bottom, right).

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.

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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 sclerostomy can be blocked by haemorrhage or fibrin clot.[1–4] 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.

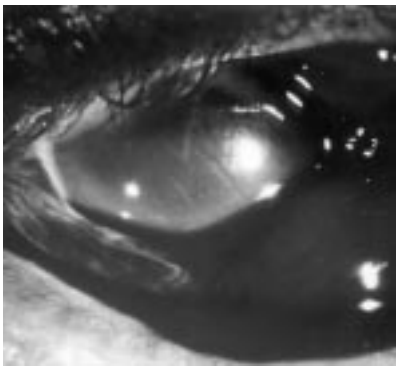

Figure 1 External photograph. Hyphaema and dense subconjunctival haemorrhage.

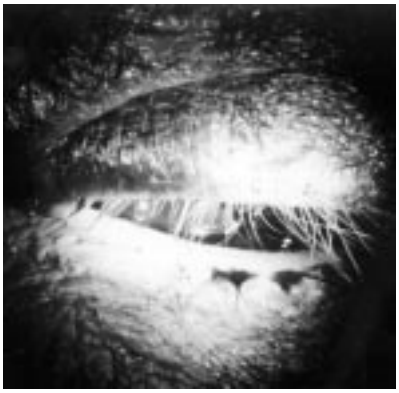

Figure 2 External photograph. Subcutaneous haemorrhage in the eyelids, extending through the orbital rim (not seen in the photograph).

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). A 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).[4] Lundy *et al* suggested that a dose of 6–12.5 µg may be equally effective and reduces the risk of hyphaemas.[4]

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 periocular 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.

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