LETTERS TO THE EDITOR

Involutional type of entropion in a child with cutis laxa

EDITOR,—The diffuse elastic tissue disease called cutis laxa (CL) is a serious, even lethal systemic illness, involving not only the skin but connective tissues throughout the body.[1] The skin hangs in loose folds, producing the appearance of premature ageing. Internal manifestations such as emphysema, ectasia of the aorta, and multiple hernias are usually present.

We report a child with cutis laxa, who presented with an unusual ophthalmic manifestation of the disease.

CASE REPORT

Our patient, who is now a 4 year old boy and the third child to a normal first degree cousin couple, was noted to have redundant skin and a hoarse cry at the age of 3 months. Skin biopsy was consistent with cutis laxa (elastin stain showed focal thickening of the elastic fibres with tapered ends). His 7 month old sister was also diagnosed as having cutis laxa at 3 months of age. Her ophthalmic examination revealed no abnormalities. Otherwise, the family history was negative for such skin problems.

Recently, he presented to our clinic with a 2 month history of a red right eye. Examination revealed an entropion of the right lower lid (Fig 1A). The lid position corrected temporarily upon manual eversion only to re-invert shortly after. The lids were hyperextensible but inelastic. Manual eversion of the lower lid resulted in significant fat prolapse into the fornix (Fig 1B). Slit lamp examination revealed moderate inferior corneal staining and injection of the medial and inferior bulbar conjunctiva. Fundus examination was normal. There was significant skin laxity over the eyelids, cheeks, neck, and trunk.

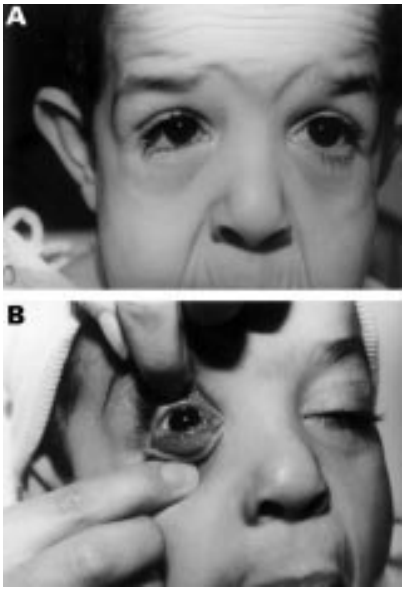

Figure 1 (A) Entropion of the right lower lid. (B) A shallow lower fornix with significant fat herniation upon manual inferior lid traction.

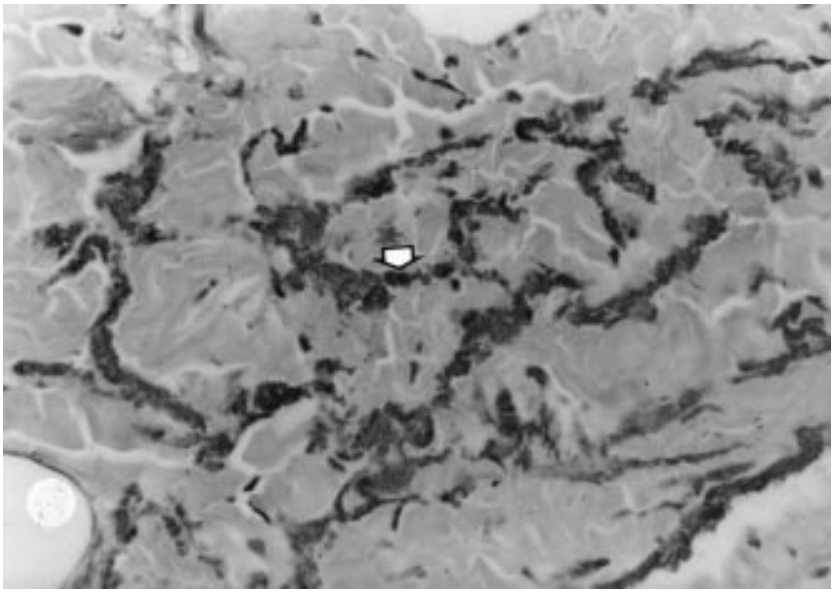

Figure 2 Eyelid tissue stained for elastic fibres showing marked granular degeneration of the elastic fibres. Aldehyde-fuscin, ×400.

Surgical correction was carried out using a lateral tarsal strip in addition to two full thickness lid sutures. A small piece of resected eyelid tissue was sent for pathological examination.

Staining for elastic tissue revealed marked granular degeneration of the elastic fibres (Fig 2). There was also a decreased number of elastic fibres, especially in the superficial dermis.

Postoperatively, the lower lid position was normal at 12 months' follow up.

COMMENT

Cutis laxa was first described by Alibert in 1832.[2] The rare syndrome of cutis laxa is a heterogeneous group of disorders characterised by inappropriate laxity of the skin which appears loosely folded to form a cuticular layer larger than the body it envelopes.[1] This leads to the production of a typical grotesque facies and the appearance of premature ageing.[3] The skin is hyperextensible but inelastic. These skin changes are frequently associated with systemic abnormalities, particularly of the lungs and heart.[1] There may be ophthalmic manifestations.

Cutis laxa may be inherited in an autosomal dominant or recessive manner.[4] The clinical features and prognosis differ considerably in the two forms.[3] In the autosomal dominant variety, complications are mild, and the patients have a normal life span. Conversely, in the autosomal recessive type, there is a high incidence of illness and death in childhood from pulmonary and cardiac involvement. Furthermore, autosomal recessive forms of CL can be divided into two types: CL with emphysema and CL with retarded development.[5] The first disorder usually leads to death within the first years of life from cardiopulmonary complications. The second disorder is not associated with pulmonary disease, but there are many systemic defects, among which gross delay in motor development is the most important.

Extensive analysis of the skin and other organs of patients with CL has demonstrated defective elastic fibres throughout the body.[6] This defect consists of a reduction in the amount and size of the elastic fibres and granular degeneration and fragmentation of the fibres with disruption of their normal arrangements; hence the term "generalised elastolysis".

Goltz and coworkers suggested an imbalance between the circulating pancreatic elastase and its inhibitor (pancreatic elastase inhibiting substance, EIS), with a diminution of the latter in patients with CL.[6] Recently, frame shift mutations in exon 30 of the elastin gene were identified in three affected individuals.[7]

The reported ophthalmic manifestations of CL include ectropion, blepharochalasis, epicanthic folds, hypertelorism, bilateral macular colobomas, fine retinal pigmentary changes,[5] and bilateral orbital fat prolapse.[8] The case reported here presents a new manifestation of the disease—namely, lower lid entropion, with all the criteria of the involutional type. Several anatomical abnormalities have been identified as causative factors in involutional entropion, including (a) horizontal lid laxity, (b) dehiscence or attenuation of lower lid retractors, (c) overriding of the preseptal over the pretarsal orbicularis muscle, and (d) enophthalmos, the role of which has been recently proved to be insignificant.[9]

Both lid lamellae and the canthal tendons contain elastic fibres.[10] Histopathologically, the eyelid specimen showed significant granular degeneration in addition to a decreased number of the elastic fibres, thus accounting for the horizontal lid laxity present in our patient. In addition, the lower lid retractors also contain elastic fibres.[10] The shallowness of the lower fornix and the fat herniation into it upon manual inferior lid traction confirm the laxity of the lid retractors and the orbital septum secondary to the disorder. These factors, horizontal lid and retractor laxity, allowed for the overriding of preseptal over pretarsal orbicularis and the inward rotation of the lid margin in a fashion similar to that which occurs with involutional entropion in elderly people.

In summary, cutis laxa is a systemic disease that relates to the presence of abnormal elastic fibres throughout the body. Involutional entropion in a 4 year old child is an unusual finding that was associated with marked laxity of the eyelid tissues present in cutis laxa.

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1 Ostlere LS, Pope FM, Holden CA. Cutis laxa complicating Ehlers–Danlos syndrome type II. *Clin Exp Dermatol* 1996;**21**:135–7.
2 Beighton P. The dominant and recessive forms of cutis laxa. *J Med Genet* 1972;**9**:216– 21.
3 Cashman ME. Cutis laxa. *Proc Roy Soc Med* 1957; **50**:719.
4 Pope FM. Cutis laxa. In: Beighton P, ed. *Mckusick's heritable disorders of connective tissues*. 5th ed. Baltimore: Mosby-Year Book, 1993:253.
5 Agha A, Sakati NO, Higginbottom MC, *et al.* Two forms of cutis laxa in the newborn period. *Acta Paediatr Scand* 1978;**67**:775–80.
6 Goltz RW, Hult A, Goldfarb M, *et al.* Cutis laxa. A manifestation of generalized elastolysis. *Arch Derm* 1965;**92**:373–87.
7 Zhang MC, He L, Giro M, *et al.* Cutis laxa arising from frameshift mutations in exon 30 of the elastin gene (ELN). *J Biol Chem* 1999;**274**:981–6.
8 Greaney MJ, Richards AB. Bilateral orbital fat prolapse in cutis laxa. *Br J Ophthalmol* 1998;**82**: 713–14.
9 Collin JRO, Rathbun JE. Involutional entropion. *Arch Ophthalmol* 1978;**96**:1058–64.
10 Bron AJ, Tripathi RC, Tripathi BJ. In: *Wolff's anatomy of the eye and orbit*. 8th ed. London: Chapman and Hall Medical, 1997:35, 148.

Unilateral arcus lipoides corneae with contralateral Sturge–Weber syndrome

EDITOR,—Arcus lipoides corneae usually occurs bilaterally and symmetrically.[1] Pronounced unilateral arcus lipoides corneae occurs in atrophic eyes, less marked with relative ocular hypotension[2–4] or contralateral carotid artery stenosis.[5–7] We report on a patient with unilateral arcus lipoides in the normal eye sparing the other one with Sturge–Weber syndrome.

CASE REPORT

A 33 year old patient showed a left sided secondary juvenile open angle glaucoma due to Sturge–Weber syndrome. The glaucoma diagnosed at the age of 10 months with a maximum intraocular pressure (IOP) of 40 mm Hg had been treated twice by diathermy of the ciliary body, and once by trabeculectomy during the first 6 years of life. Since then, the IOP has ranged between 16 and 20 mm Hg in the left eye, constantly being 4–6 mm Hg lower in the right eye.

A facial haemangioma involving the first and second branch of the facial nerve had been treated with radiotherapy after birth (Fig 1). There was no history of neurological deficits or epilepsy. Apart from intermittent hypercholesterolaemia up to 260 mg/dl, the young man's medical history was unremarkable.

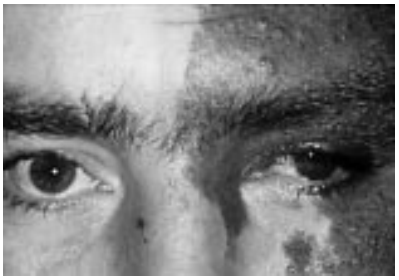

Figure 1 Sturge–Weber's syndrome with juvenile glaucoma in the left eye, a facial haemangioma involving the first and second branch of the facial nerve after radiotherapy.

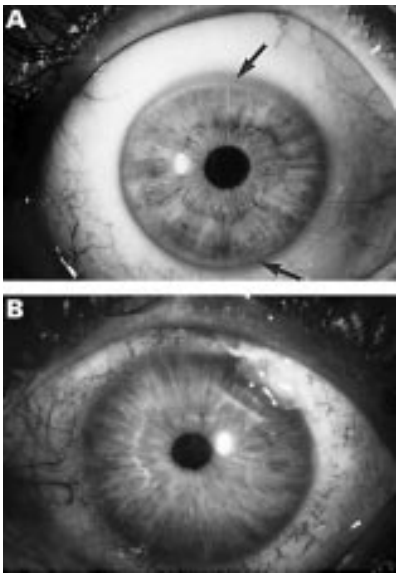

Figure 2 (A) Narrow arcus lipoides (arrow) most pronounced superiorly and inferiorly right eye. (B) Increased episcleral vascularisation due to the Sturge–Weber syndrome and a filtering bleb since age of 6 years. No arcus lipoides left eye.

Visual acuity was 20/15 right eye and 20/30 left eye, intraocular pressure 16 mm Hg right eye and 20 mm Hg left eye. The right eye disclosed a definite yellow-whitish arcus lipoides extending through the entire corneal thickness, separated from the limbus by a clear zone (Fig 2A). The arcus was more pronounced superiorly and inferiorly than in the medial and lateral quadrant. Apart from the arcus lipoides, the right eye did not reveal any abnormalities.

No arcus lipoides could be detected in the left eye (Fig 2B). The eye showed increased episcleral vascularisation. The optic disc presented with moderate glaucomatous disc change (optic disc size 3.0 mm, cup/disc ratio 0.78).

COMMENT

Arcus lipoides corneae is known to be usually associated with hyperlipoproteinaemia, especially type IIa and IIb, but may occur without predisposing factors.[1] Histologically, it mainly consists of extracellular deposits of cholesterol, phospholipids, and triglycerides in stroma, Bowman and Descemet membrane.[8] Generally, it is developed bilaterally and symmetrically.[1] Single cases of unilateral arcus lipoides corneae were reported in eyes with absolute or relative ocular hypotension,[2–4] or in patients with contralateral carotid artery stenosis.[5–7]

Our patient presented with a narrow, but clearly recognisable arcus lipoides which occasionally can occur in younger people, even in the absence of marked hyperlipoproteinaemia.[1]

In earlier reports,[8] it was suggested that vascular congestion and increased vascular permeability of limbal blood vessels might possibly be involved in the development of unilateral arcus lipoides. Analogously to cases of absolute ocular hypotension, one could assume that in our patient, the relative hypotension in the right eye with consecutively relatively higher blood supply may have facilitated the lipid deposition in the cornea, whereas the development of arcus was prevented by the higher IOP in the left eye.

In our opinion, this case implies that unilateral arcus lipoides corneae can occur in morphologically normal eyes with solely relative ocular hypotension.

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1 Barchiesi BJ, Eckel RH, Ellis PP. The cornea and disorder of lipid metabolism. *Surv Ophthalmol* 1991;**36**:1–22.
2 Naumann GOH, Küchle M. Unilateral arcus lipoides corneae with traumatic cyclodialysis in two patients. *Arch Ophthalmol* 1989;**107**:1121–2.
3 Naumann GOH, Küchle M. Unilateral corneal arcus lipoides [letter]. *Lancet* 1993;**342**:1185.
4 Capoferri C, Pissarello R, Paganoni G. Unilateral corneal arcus and ocular hypotony. *Can J Ophthalmol* 1994;**29**:155–6.
5 Smith JL, Susac JO. Unilateral arcus senilis: sign of occlusive disease of the carotid artery. *JAMA* 1973;**226**:676.
6 Bagla SK, Golden RL. Unilateral arcus corneae senilis and carotid occlusive disease. *JAMA* 1975;**233**:450.
7 Sugimura G. Unilateral corneal arcus. *Arch Ophthalmol* 1990;**108**:780–1.
8 Cogan DG, Kuwabara T. Arcus senilis and its pathology and histochemistry. *Arch Ophthalmol* 1959;**61**:553–60.

Sickle form macular whitening in a child after viewing a solar eclipse

EDITOR,—Individual cases of retinopathies after unprotected exposure to sunlight are frequently reported in patients with psychiatric disorders,[1] after religious practices,[2 3] or related to drug abuse.[4] Even though knowledge of the harmful effects of sun gazing dates back to ancient times[5] there are still many reports on epidemics of patients with solar retinopathy after viewing solar eclipses.[6] This case report demonstrates that, despite the availability of cheap and safe protection,[7] cases of eclipse retinopathy are still observed and so strong preventive efforts should be made for future eclipses, especially for groups at risk.

CASE REPORT

An 11 year old girl was evaluated after complaining of bilateral central scotomata after observing the subtotal solar eclipse of 11 August 1999 in south eastern Switzerland without protective eyewear. Visual acuity 5 days after the event was 20/25 on both eyes. Amsler grid testing showed bilateral small central scotomata. Anterior segments were unremarkable. Biomicroscopy showed sickle form oedematous areas parafoveal in the deep retinal layers in both eyes, corresponding to the shape of the subtotal solar eclipse (Fig 1).

After 3 months the visual acuity was 25/20, the patient denied any residual scotoma in the Amsler grid testing. The retinal oedema had resolved, but there were some subtle pigmentary irregularities to be observed (Fig 2).

COMMENT

Areas of retinal oedema outlining a sickle—that is, the "brand" of the sun, have been described after the solar eclipses of 17 June

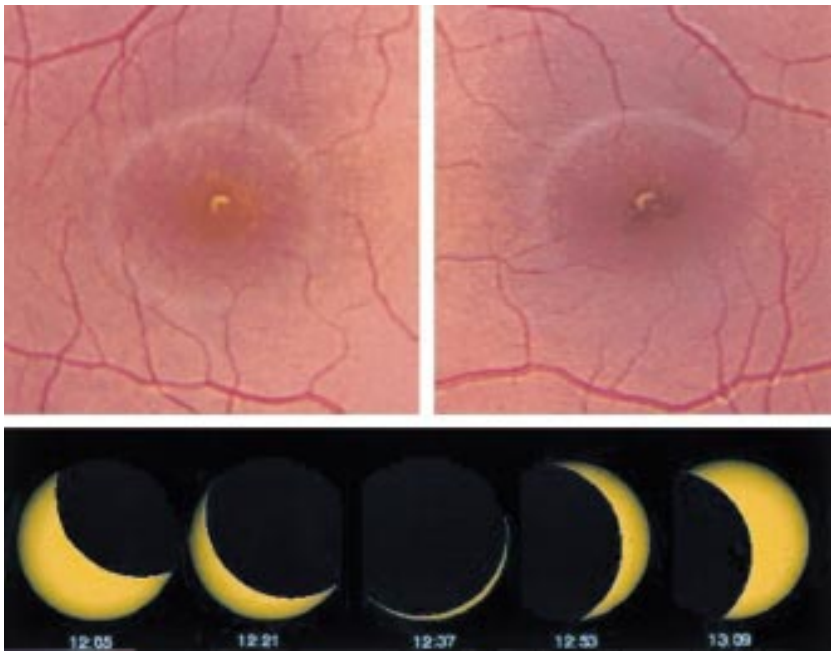

Figure 1 Sickle form oedematous areas parafoveally in the deep retinal layers in both eyes. They were similar in shape to the development of the subtotal solar eclipse calculated for the point of observation of our patient.

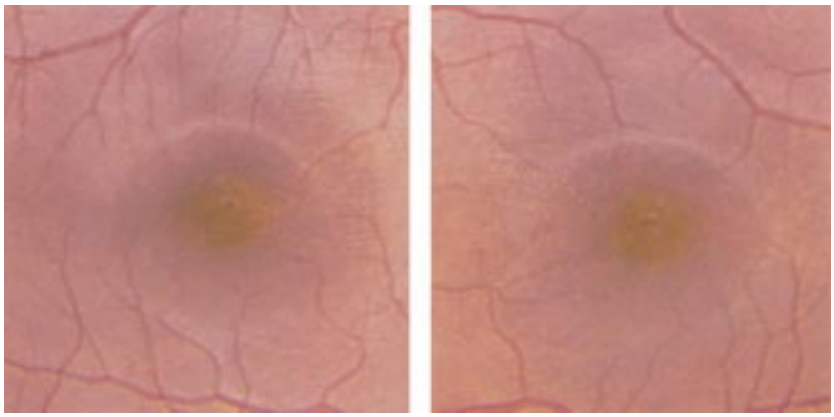

Figure 2 Subtle pigmentary irregularities in both maculas 3 months after eclipse retinopathy.

1890[8] and 29 April 1976.[9] If the patient does not gaze at the sun for brief periods but observes it constantly for a longer period of time during a defined phase of the eclipse the subsequent retinal lesion can be sharply defined. From the shape of the "brand" one can determine the time phase of exposure, which in the present case was the maximum of the subtotal solar eclipse (Fig 1).

Approaching the 1999 solar eclipse there was extensive information in the media about appropriate eye protection and special glasses for eclipse viewing were widely available. In addition, there was cloudy weather over most parts of central Europe. Only a few mild cases of eclipse retinopathy have therefore been reported in the eastern part of Switzerland.

Our patient observed the eclipse from the mountains of south eastern Switzerland, where the sky was clear. Although the family was in possession of appropriate protective glasses, the 11 year old girl did not use them.

Although prevention strategies have proved to be very effective[10] children seem to be the most at-risk group in the population for retinal damage from solar eclipses. We therefore recommend that special efforts be made to ensure eclipse viewing safety in children. The next total solar eclipse will take place over southern Africa and Madagascar on 21 June 2001.

We thank Mr Hugo Niederberger, ophthalmic photographer, for providing the pictures.

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1 Anaclerio AM, Wicker HS. Self-induced solar retinopathy by patients in a psychiatric hospital. *Am J Ophthalmol* 1970;**69**:731–6.
2 Hope-Ross M, Travers S, Mooney D. Solar retinopathy following religious rituals. *Br J Ophthalmol* 1988;**72**:931–4.
3 Cangelosi GC, Newsome DA. Solar retinopathy in persons on religious pilgrimage. *Am J Ophthalmol* 1988;**105**:95–7.
4 Fuller DG. Severe solar maculopathy associated with the use of lysergic acid diethylamide (LSD). *Am J Ophthalmol* 1976;**81**:413–6.
5 Duke-Elder S, MacFaul PA. Injuries; Part 2 Non-mechanical injuries. In: S*ystem of ophthalmology.* Duke-Elder, S, ed. London: Henry Kimpton, 1972:885–93.
6 Istock TH. Solar retinopathy: a review of the literature and case report. *J Am Optom Assoc* 1985;**56**:374–82.
7 Chou BR. Retinal protection from solar photic injury. *Am J Optom Physiol Opt* 1981;**58**:270–80.
8 Haab O, cited in: Zur Sonnenfinsternis vom 17. April. *Neue Zürcher Zeitung* April 13 1912;**133**:1.
9 Pau H. Abbildung einer partiellen Sonnenfinsternis in der Makula bei Sonnenbeobachtung. *Klin Monatsbl Augenheilkd* 1977;**171**:295–8.
10 Juan-Lopez M, Pena-Corona MP. Estrategia para prevenir daños a la salud ocasionados por la observación del eclipse solar en México. *Salud Publica Mex* 1993;**35**:494–9.

Serum antibodies to HSC71 in Vogt–Koyanagi–Harada disease

EDITOR,—Heat shock proteins (HSPs) are highly conserved immunogenic intracellular molecules, and are induced by heat, inflammatory mediators, and physiological stress.[1] The presence of antibodies to HSPs or heat shock cognate protein (HSC) has been reported in several autoimmune diseases.[1] However, despite the prevalence of these antibodies in these autoimmune diseases, their significance is not fully understood.

In this report, we investigated antibodies to HSC71 specific antibody levels in sera from patients with Vogt–Koyanagi–Harada disease (VKH), a systemic disorder that affects various organs that contain melanocytes and is believed to be an autoimmune disease.[2]

CASE REPORT

Serum samples were obtained from eight patients with VKH, and from nine unaffected volunteers. All samples were obtained from VKH patients during the acute phase of the illness with severe uveitis and were taken before steroid administration. Mean ages were 43 and 40 for VKH and controls, respectively. Informed consent was obtained from all of the patients and volunteers. All sera were stored at −70°C until use.

Cloning and expression of the recombinant HSC71 (rHSC71) were performed as previously described.[3] Anti-HSC71 antibody levels of sera were assayed by enzyme linked immunosorbent assay. In brief, flat bottomed microtitre plates were coated with 10 µg/ml rHSC71 in phosphate buffered saline (PBS). After incubation overnight, non-specific binding sites were blocked with PBS containing 0.05% Tween 20, 1 mM EDTA, 0.25% BSA, and 0.05% NaN_3. Wells were incubated with serum diluted 1:200 for 2 hours at room temperature. The plates were then washed and incubated with alkaline phosphatase conjugated anti-human polyvalent immunoglobulins (Sigma BioSciences, St Louis, MO, USA) diluted 1:40 000 for 2 hours at room temperature. For the colour reaction, the washed wells were incubated with *p*-nitrophenyl phosphate substrate solution and analysed by measuring the optical density (OD) at 405 nm. Results were analysed using the two tailed Student's *t* test. A p value of 0.05 or less was considered significant. Levels of anti-HSC71 antibody were significantly raised in patients with VKH ($p<0.00005$) compared with healthy volunteers (Fig 1).

Molecular analysis of specificity of anti-HSC71 antibody of VKH patient serum was done by western blot analysis as described previously.[3] The antiserum from VKH patients

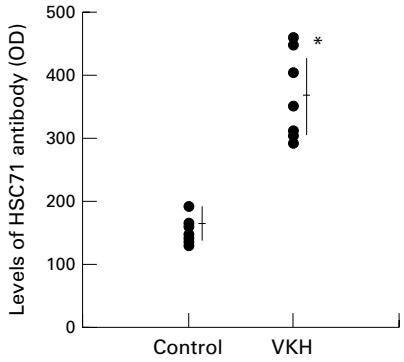

*Figure 1 Antibody to HSC71 in patients with VKH (VKH; n=8) and in normal healthy controls (n=9). Horizontal bars represent mean values of data. *p<0.00005 compared with the control.*

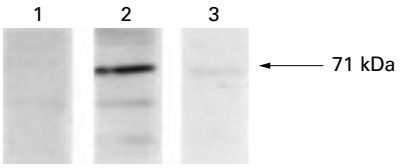

Figure 2 Representative data of western blot analysis of anti-rHSC71 antisera in patients with VKH and toxoplasmic retinochoroiditis. Lane 1, a healthy control; lane 2, VKH patient; lane 3, toxoplasmic retinochoroiditis patient.

reacted strongly with rHSC71, while the sera from a toxoplasmic retinochoroiditis patient and a healthy volunteer did not (Fig 2).

COMMENT

In postnatal ocular maturation, the level of HSC70 mRNA expression was still high in the retina, which suggests some important role of HSC70 protein in structural or functional maintenance of this tissue.[4] HSP70 proteins bound to the immunodominant epitope of interphotoreceptor retinoid binding protein indicates that they may play a part in the processing and presentation of antigens by antigen presenting cell.[5]

There are homologies between HSPs and unrelated self proteins as well as antibody cross reactivities.[1] It has been proposed that HSP reactive T cells or antibodies serve as amplifiers of ongoing autoimmune destruction. Stressed cells at the sites of inflammation produce elevated HSP levels and thus become targets of HSP reactive T cells. Currently, it appears more likely that self HSP reactive T cells are physiological constituents of the immune system, and that their activities are regulated by numerous factors including cross reactive peptides which are constantly taken up by food and occasionally introduced through infection. Increased HSC71 antibody levels in patients with VKH indicate that HSC71 may play an important role in pathogenesis of VKH. Alternatively, as anti-HSC71 antibodies may merely be an epiphenomenon reflecting tissue damage, it would be necessary to know serum antibody levels in disease controls—that is, in patients who have similar inflammation which is not of an autoimmune nature (infective retinitis/choroiditis). According to our data of serum from a patient with toxoplasmic retinochoroiditis, this possibility was shown to be unlikely although a definitive conclusion will be reached by analysing a larger number of cases and various types of similar inflammatory diseases. Further investigation is needed to elucidate how this autoimmune response is related to the pathogenesis and pathophysiology of VKH.

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2 Norose K, Yano A. Melanoma specific Th1 cytotoxic T lymphocyte lines in Vogt-Koyanagi-Harada disease. *Br J Ophthalmol* 1996;**80**:1002–8.
3 Yang T-H, Aosai F, Norose K, *et al*. Heat shock cognate protein 71-associated peptides function as an epitope for Toxoplasma gondii-specific CD4+ CTL. *Microbiol Immunol* 1997;**41**:553–61.
4 Tanaka Y, Kobayashi K, Kita M, *et al*. Messenger RNA expression of heat shock proteins (HSPs) during ocular development. *Curr Eye Res* 1995; **14**:1125–33.
5 Rengarajan K, de Smet MD, Chader GJ, *et al*. Identification of heat shock proteins binding to an immunodominant uveitopathogenic peptide of IRBP. *Curr Eye Res* 1994;**13**:289–96.

Subhyaloid haemorrhage from proliferative diabetic retinopathy after Nd:YAG laser iridotomy

EDITOR,—Various vascular and haematological disorders can cause a subhyaloid premacular haemorrhage leading to decrease in vision.[1 2] So far though, a premacular haemorrhage has not been recognised as a complication of peripheral laser iridotomy (PI). The following is a case report of such an occurrence with consideration of possible mechanisms.

CASE REPORT

A 58 year old diabetic woman complained of sudden reduction in vision immediately after a prophylactic Nd:YAG laser peripheral iridotomy to her left eye. Fundus examination revealed a dense premacular haemorrhage. The iridotomy was performed through an Abraham lens using a Coherent 7970 laser. Ten shots were needed with energy of 3–5.5 mJ.

The patient had proliferative diabetic retinopathy for which she underwent extensive peripheral retinal photocoagulation with residual neovascularisation at the disc (NVD). Several weeks before her current event, she presented with a spontaneous premacular haemorrhage following a Valsalva manoeuvre. She refused any surgical or laser intervention and the haemorrhage had completely resolved at the time of the PI.

COMMENT

The Nd:YAG laser creates a PI by photodisruption of tissues. Three mechanisms are involved in this process: (1) very high irradiances strip electrons from atoms creating a gaseous state of electrons and ions called "plasma"; (2) the plasma expands rapidly outwards creating shock and acoustic waves that mechanically disrupt adjacent tissue; and (3) latent stress in the tissues causes additional disruption when the laser makes an incision.[3] The peak pressure in the shock wave associated with plasma formation may exceed 1000 atm.[4]

Several investigators have previously reported retinal and choroidal damage following argon or Nd:YAG laser peripheral iridotomy.[5] This included peripheral retinal and foveal photocoagulation, choroidal and retinal detachment, and cystoid macular oedema and unexplained visual loss. Most of the complications were attributed to radiant laser energy rather then mechanical stress. In our patient, this is a remote possibility in view of the good laser focus on the targeted iris tissue through the Abraham lens. A likely explanation is a rebleeding of fragile NVD secondary to the posterior propagation of shock waves. This mechanism has been contemplated to explain retinal complications after posterior capsulotomy[6] as well as after excimer laser photorefractive keratotomy.[7 8] Experimentally, a pressure wave of 9–16 bar (130–230 psi) has been measured by Vogel *et al* 18 mm from the focal point of a Q switched Nd:YAG laser using a pulse energy of 5 mJ.[9] Changes in intraocular pressure during the procedure or while removing the contact lens is another possible explanation although unlikely; the patient, already experienced with laser procedures, was very cooperative. The iridotomy was technically easy and performed with particular care since this was her only seeing eye.

The Nd:YAG laser is used in the management of premacular haemorrhages.[10] In our patient, performing the PI with the same laser was the cause of such a one. Regardless of the mechanism(s) involved, the procedure should be performed with special care particularly in predisposed patients such as those with proliferative retinopathy and/or with a recent bleed. Using the least amount of energy is an obvious precaution. Pretreatment of the chosen iris site with the argon laser in a photocoagulative mode to stretch or thin the iris bed might also be helpful in lowering the Nd:YAG laser energy required.

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2 Gass JDM. *Stereoscopic atlas of macular disease: diagnosis and treatment.* 2nd ed. St Louis: CV Mosby, 1987:558–64.
3 Mainster MA, Sliney DH, Belcher CD III, *et al*. Laser photodisruptors; damage mechanisms, instrument design and safety. *Ophthalmology* 1983;**90**:973–91.
4 Lauterborn W. High-speed photography of laser-induced breakdown in liquids. *Appl Phys Lett* 1972;**21**:27–9.
5 Ritch R, Liebman JM. Laser iridotomy and peripheral iridoplasty. In: Rich R, Shields MB, Krupin T, eds. *The glaucomas.* 2nd ed. St Louis: Mosby, 1996:1549–73.
6 Winslow RL, Taylor BC. Retinal complications following YAG laser capsulotomy. *Ophthalmology* 1985;**92**:785–9.
7 Janknecht P, Soriano JM, Hansen LL. Cystoid macular oedema after excimer laser photorefractive keratectomy (letter). *Br J Ophthalmol* 1993;**77**:681–2.
8 Loewenstein A, Lipshitz I, Varssano D, *et al*. Macular hemorrhage after excimer laser photorefractive keratectomy. *J Cataract Refractive Surg* 1997;**23**:808–10.
9 Vogel A, Hentshel W, Holzfuss J, *et al*. Cavitation bubble dynamics and acoustic transient generation in ocular surgery with pulsed neodymium: YAG lasers. *Ophthalmology* 1986;**93**:1259–69.
10 Ulbig MW, Mangouritas G, Rothbacher HH, *et al*. Long-term results after drainage of premacular subhyaloid hemorrhage into the vitreous with a pulsed Nd:YAG laser. *Arch Ophthalmol* 1998;**116**:1465–9.

Bilateral endogenous bacterial endophthalmitis associated with pyogenic hepatic abscess

EDITOR,—Endogenous or metastatic bacterial endophthalmitis is rare, with a prevalence of approximately 2–8% of all cases of endophthalmitis.[1] Endogenous bacterial endophthalmitis is associated with chronic diseases such as diabetes mellitus and renal failure, invasive medical procedures, and non-ocular surgery, injecting drug abuse, or prolonged placements of central venous lines.[1] Gram positive bacteria are the most common causative organisms of endogenous bacterial endophthalmitis.[1]

A small number of cases of endogenous bacterial endophthalmitis due to *Klebsiella pneumoniae*, a Gram negative organism, have previously been reported, with the majority of the cases originating in Taiwan.[2–7] *K pneumoniae* endophthalmitis is associated with diabetes mellitus and hepatic abscesses, can be bilateral, and is also associated with a poor visual outcome.[2–7] We report the case of a Taiwanese seaman who developed bilateral endogenous bacterial endophthalmitis after presenting with a pyogenic hepatic abscess.

CASE REPORT

A 40 year old male surgical inpatient was reviewed after he complained of a 3 day history of bilateral painful red eyes and reduced visual acuity. The patient, a previously healthy Taiwanese seaman airlifted from a ship 1 week earlier, had a right hepatic lobe abscess measuring 3.6 cm × 7.5 cm. This had been treated by open drainage of the abscess followed by peritoneal lavage and intravenous gentamicin and tazocin (5 mg/kg three times daily and 4.5 g three times daily, respectively). Both pus samples and blood cultures grew *K pneumoniae* sensitive to these antibiotics and the patient's general condition had improved by the time the ophthalmic review was requested.

Initial best corrected visual acuity (BCVA) was 3/60 in both eyes, while examination revealed bilateral periorbital erythema and oedema with marked conjunctival chemosis and injection. This was accompanied by bilateral severe anterior uveitis (cells 4 plus, flare 4 plus) and bilateral posterior synechiae formation. Funduscopy demonstrated bilateral severe vitritis (plus 4 cells) with temporally located, white choroidal infiltrates associated with exudation corresponding to subretinal abscesses (Fig 1A). Bilateral vitreous aspirates were performed with an intravitreal injection of gentamicin 200 µg and a subconjunctival injection of cefuroxime 200 mg and gentamicin 80 mg. A second dose of these antibiotics was subconjunctivally administered 24 hours later. No organisms were isolated from the vitreous samples. The previous intravenous antibiotic regimen was continued to which intravenous ciprofloxacin 400 mg twice daily and hydrocortisone 100 mg three times daily were added. The anterior uveitis was treated with topical gentamicin, ceftazidime, and dexamethasone hourly plus atropine 1% twice daily.

After 16 days the BCVA increased to 6/36 and 6/24 in the right and left eyes. The periorbital erythema and oedema improved, while slit lamp examination demonstrated reduced anterior chamber activity (2 plus cells and 1 plus flare bilaterally). There were no residual posterior synechiae. Fundal examination demonstrated reduction of the vitritis (2 plus cells) and the areas of subretinal abscess had decreased in size leaving a surrounding area of RPE atrophy (Fig 1B). Medications included oral ciprofloxacin 750 mg twice daily, oral prednisolone 60 mg daily, topical gentamicin and ceftazidime six times a day, dexamethasone 2 hourly, and atropine 1% twice daily.

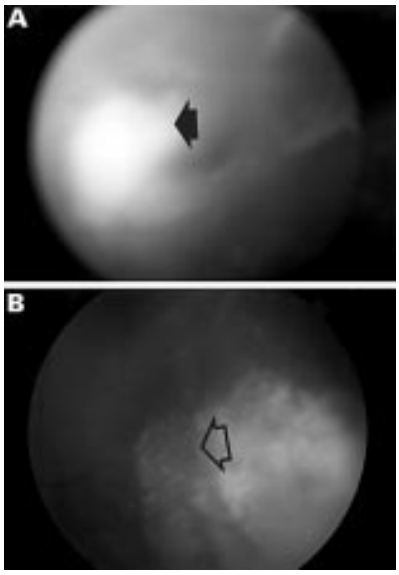

Figure 1 Fundus photograph of the left eye demonstrating (A) subretinal abscess (solid arrow) with associated severe vitritis. (B) Resolution of the subretinal abscess resulted in a surrounding area of retinal pigment epithelium atrophy (open arrow). There has also been a reduction in the severity of the vitritis.

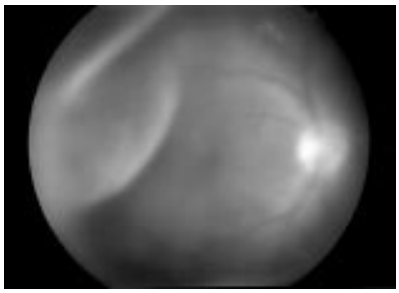

Figure 2 Fundus photograph of the right eye demonstrating a retinal detachment involving the macula.

Just before transfer home to Taiwan, the patient developed a right retinal detachment involving the macula (Fig 2). Two vitrectomy procedures involving retinopexy, encirclement, and ultimately silicone oil were subsequently performed in Taiwan. The patient's left retina also detached and required a trans pars plana vitrectomy with retinopexy, which resulted in successful reattachment of the retina. At 4 month follow up the BCVA is PL and 6/12 in the right and left eyes respectively, the right retina is redetached but the left retina remains flat.

COMMENT

Although rare, most cases of endogenous bacterial endophthalmitis are caused by Gram positive bacteraemias in patients with existing illness or injecting drug abuse.[1] A number of cases caused by the Gram negative organism *K pneumoniae* have been reported, mainly from Taiwan.[2–7] *K pneumoniae* is the leading cause of pyogenic liver abscess in Taiwan and patients with *K pneumoniae* endophthalmitis secondary to hepatic abscess are more likely to have diabetes mellitus.[2 6–8] However, like this patient, not all patients have diabetes and the role of unrecognised host or environmental factors leading to this unique association between pyogenic liver abscess and endophthalmitis is unclear.[2 4 6 7]

Typically the clinical symptoms of *K pneumoniae* endophthalmitis occur 2–3 days after drainage of a pyogenic liver abscess but before bacterial culture results and antibiotic sensitivities are available.[2 4 7] Systemic antibiotics are more valuable in endogenous rather than traumatic or postoperative endophthalmitis, probably due to breakdown of the blood-ocular barrier at the site of ocular seeding.[2] The choice of antibiotic reflects bacterial sensitivity results and while an increasing prevalence of *K pneumoniae* resistant to a large number of antibiotics, including gentamicin, has been reported most of the cases of *K pneumoniae* infection previously isolated in Taiwan are resistant only to ampicillin and sulbenicillin.[2 7]

The benefit of intravitreal antibiotics in endogenous endophthalmitis is unproved but potential benefits outweighed the risks in our patient who had bilateral disease.[1–3 6 7] Successful drainage of a subretinal abscess has been documented, but up to three quarters of retinal detachments in all types of endophthalmitis due to virulent organisms remain detached despite surgery.[5 9] Visual prognosis is poor in *K pneumoniae* endophthalmitis with 90% of reported eyes having visual outcomes of counting fingers or worse.[2 4 6] In view of this, a high of index of suspicion with prompt diagnosis and aggressive treatment is important, particularly in the 25% of patients who have bilateral disease.[1–7 9]

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1 Okada AA, Johnson RP, Liles WC, *et al.* Endogenous bacterial endophthalmitis. Report of a ten-year retrospective study. *Ophthalmology* 1994;**101**:832–8.
2 Margo CE, Mames RN, Guy JR. Endogenous Klebsiella endophthalmitis. Report of two cases and review of the literature. *Ophthalmology* 1994;**101**:1298–301.
3 Irvine WD, Flynn HW, Miller D, *et al.* Endophthalmitis caused by gram-negative organisms. *Arch Ophthalmol* 1992;**110**:1450–4.
4 Lee CC, Chen CY, Chen FH, *et al.* Septic metastatic endophthalmitis from Klebsiella pneumoniae liver abscess: CT and MR imaging characteristics—report of three cases. *Radiology* 1998;**207**:411–6.
5 Yarng SS, Hsieh CL, Chen TL. Vitrectomy for endogenous Klebsiella pneumoniae endophthalmitis with massive subretinal abscess. *Ophthalmic Surg Lasers* 1997;**28**:147–50.
6 Chee SP, Ang CL. Endogenous Klebsiella endophthalmitis—a case series. *Ann Acad Med Singapore* 1995;**24**:473–8.
7 Cheng DL, Liu YC, Yen MY, *et al.* Septic metastatic lesions of pyogenic liver abscess. *Arch Intern Med* 1991;**151**:1557–9.
8 Cheng DL, Liu YC, Yen MY, *et al.* Causal bacteria of pyogenic liver abscess. *J Formosan Med Assoc* 1989;**88**:1008–11.
9 Foster RE, Rubsamen PE, Joondeph BC, *et al.* Concurrent endophthalmitis and retinal detachment. *Ophthalmology* 1994;**101**:490–8.

Sustained remission of CMV retinitis in HIV-2 disease

EDITOR,—The only established risk factor for the development and progression of cytomegalovirus retinitis (CMVR) in individuals with HIV disease is a low CD4+ T lymphocyte count. The initiation of highly active antiretroviral treatment (HAART) has brought a significant change in the clinical outcome of CMV retinitis[1] with studies reporting longer times to CMV relapse. These findings correlate with increased CD4+ T lymphocyte counts and reduced HIV viral loads.

We report the case of an HIV-2 positive patient who showed no reactivation of CMV retinitis for 25 months despite a low CD4+ T lymphocyte count and a lack of response to HAART treatment. The role of HIV-2 seropositivity in our case is assessed and other relevant factors discussed.

CASE REPORT

A 47 year old black African male was diagnosed with AIDS (HIV-2 positive) in September 1996. The AIDS defining illness was enteric non-Hodgkin's lymphoma. He was given saquinavir, stavudine (d4T), and zalcitabine (DDC). Owing to his persistently low CD4+ T lymphocyte count (30 cells $\times 10^6$/l) he was referred for ophthalmic evaluation in August 1997. He was diagnosed with peripheral zone III inactive CMV retinitis in his left eye (Fig 1) and was started on maintenance treatment with oral ganciclovir (3 g per day).

Three weeks later this area of retinitis reactivated. He was then given intravenous ganciclovir 5 mg/kg twice daily for 4 weeks and then restarted on oral ganciclovir. His CD4+ T lymphocytes remained low with a peak reaching 70 cells $\times 10^6$/l in January 1998. In April 1998 antiretroviral treatment was discontinued because he showed no response to therapy.

The patient underwent frequent ophthalmic reviews during which inactive CMV retinitis was recorded on funduscopic and photographic assessment. No new lesions have been recorded in either eye. His current CD4+ T lymphocyte count is 7 cells $\times$ 10^6/l and his visual acuity is 6/5 in each eye.

COMMENT

In a group of patients not responding to HAART therapy Mitchell *et al*[2] showed a median time to CMVR progression of 18 days (95% CI: 8,91). Walker and Popescu[3] showed a median time to CMVR reactivation of 122 days (95% CI: 93-186) in patients receiving oral ganciclovir as maintenance treatment.

This remission period far exceeds reactivation rates in patients with low CD4+ T lymphocyte counts, further suggesting that the immune response to CMV is not solely related to the CD4+ T lymphocyte count[4].

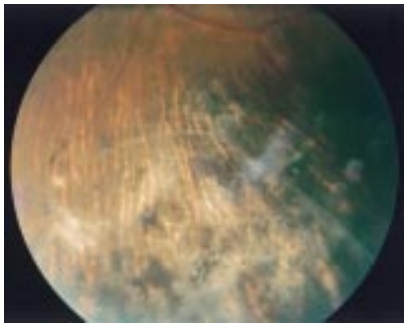

Figure 1 Peripheral zone III inactive CMV retinitis in the left eye.

We believe that HIV-2 seropositivity in this case is a relevant factor in modifying the natural history of CMVR. HIV-2 is biologically similar to HIV-1 but it has a reduced virulence.[5] The only study to our knowledge comparing ocular lesions between HIV-1 and HIV-2 infected individuals is by Monteiro-Grillo *et al*.[6] They reported less severe complications in patients infected by HIV-2.

Other factors relating to the immune response to CMV have also been suggested in the literature. Schrier *et al*[7] demonstrated that HIV infected individuals with HLA phenotypes A2B44, B51, and DR7 have low T cell immune responses to CMV and are predisposed to CMV retinitis as immunodeficiency progresses.

More studies with HIV-2 infected individuals are required to monitor the clinical course of CMVR in this group of patients and clarify whether HIV-2 virulence is an important factor modifying the immune response to CMV.

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1 Lee V, Subak-Sharpe I, Shah S M. Changing trends in cytomegalovirus retinitis with triple therapy. *Eye* 1999;**13**:59–64.
2 Mitchell SM, Membrey WL, Youle MS, *et al.* Cytomegalovirus retinitis after the initiation of highly active antiretroviral therapy: a 2 year prospective study. *Br J Ophthalmol* 1999;**83**:652–5.
3 Walker M, Popescu M. Oral ganciclovir for maintenance therapy of cytomegalovirus retinitis: experience from the European compassionate use programme. *Int Conf AIDS* 1998;**12**: 316 (abstract no 22270).
4 Nolan W, Mitchell S. Do all patients with CMV retinitis require life long anti-CMV therapy? *Br J Ophthalmol* 1998;**82**:844–5.
5 Marlink R, Kanki P, Thior I, *et al.* Reduced rate of disease development after HIV-2 infection as compared to HIV-1. *Science* 1994;**265**:1587–90.
6 Monteiro-Grillo M, Sousa AP, Galvao J, *et al.* Clinical correlations in HIV-1 and HIV-2 infected patients. *Eur J Ophthalmol* 1993;**3**:13–20.
7 Schrier RD, Freeman WR, Wiley CA, *et al.* CMV-specific immune responses and HLA phenotypes of AIDS patients who develop CMV retinitis. HNRC Group. HIV Neurobehavioral Research Center. *Adv Neuroimmunol* 1994;**4**:327–36.

Complete regression of retinal neovascularisation after therapy with interferon alfa in Behçet's disease

EDITOR,—One of the main problems in ocular Behçet's disease (BD) is severe posterior uveitis with retinal vessel occlusion and secondary ischaemic changes leading to retinal neovascularisation and to bad prognosis despite immunosuppression.

Previously we have shown the efficacy of interferon alfa (IFNα)-2a in posterior uveitis and especially in retinal vasculitis with reopening of occluded vessels.[1] Now we demonstrate the antiangiogenic effect of IFNα-2a in one BD patient with retinal neovascularisations. The IFNα-2a treatment has resulted in complete remission of the retinal neovascularisations without laser coagulation of nonperfusion areas, which would have been the standard therapy.

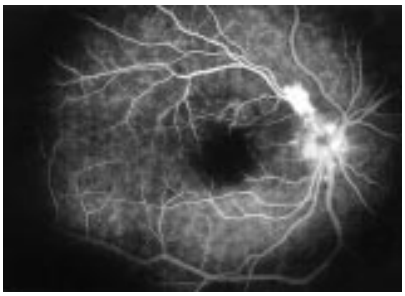

Figure 1 Fluorescein angiography of right eye before starting IFNα-2a therapy. Optic disc neovascularisation is seen among the superior artery branch. Visual acuity 20/100.

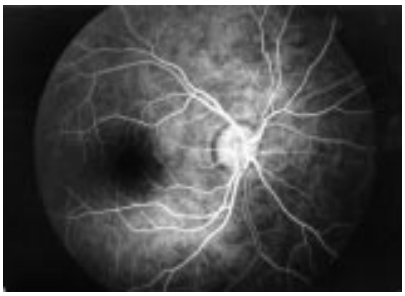

Figure 2 Fluorescein angiography of right eye 7 weeks after beginning IFNα-2a treatment. Optic disc neovascularisation had completely disappeared. Visual acuity 20/25.

CASE REPORT

A 27 year old man with recurrent oral aphtosis, pustular skin lesions, epididymitis, arthritis (elbow, sacroiliac joint), and retinal vasculitis of the left eye was diagnosed in October 1998 as having Behçet's disease (BD), according to the criteria of the international study group.[1] At this time therapy with cyclosporin A (3 mg/kg body weight) was initiated. Until June 1999 he had had no relapses. One month later the visual acuity in the right eye decreased to 20/100, but was stable in the left eye (20/600). Biomicroscopically there was no inflammation of the anterior chamber. Funduscopy of both eyes revealed vitreous infiltration and a macular oedema with gliotic changes and central sanguination. In addition, optic disc neovascularisation in the right eye (Fig 1) and optic disc oedema in the left eye were present. Peripheral vessel leakage as a sign of active vasculitis was shown by fluorescein angiography. At the same time oral aphtosis and pustular skin lesions recurred. Owing to active retinal vasculitis with marked decrease of visual acuity we stopped the cyclosporin A therapy and on the next day started IFNα-2a therapy with 6 million units/day subcutaneously. Six days later the visual acuity rose to 20/40 in the right eye and 20/300 in the left eye. Funduscopy revealed a decrease of vitreous infiltration, macular oedema, and retinal haemorrhages in both eyes; the optic disc oedema in the left eye and the optic disc neovascularisation in the right eye regressed. Seventeen days after initiating IFNα-2a treatment the ophthalmological examination disclosed a stable visual acuity in the right eye and an increased visual acuity of 20/200 in the left eye because of irreversible macular defects. The retinal vasculitis had improved in both eyes; additionally, optic disc neovascularisation in the right eye had completely disappeared. Another 5 weeks later there was further improvement with a visual acuity of 20/25 in the right eye (Fig 2) and 20/100 in the left eye.

When tapering down the interferon therapy to 3 million units IFNα-2a per day the patient had one relapse in the end of October 1999. Since then he has had no recurrences and a stable visual acuity of 20/25 in the right eye and 20/100 in the left eye since November 1999 with a dosage of 3 million units/6 million units interferon alfa every other day.

COMMENT

We have already shown the efficacy of IFNα-2a in posterior uveitis and especially in retinal vasculitis with reopening of occluded vessels,[1] but this case underlines the antiangiogenic effects of this cytokine in retinal neovascularisation.

It is known that IFNα-2a, a genetically engineered copy of one of the naturally occurring human interferons, has antiviral, immunoregulatory, and antineoproliferative properties and has been used clinically for many years in the treatment of viral hepatitis, solid tumours,[2] and haematological malignancies. More recently it has been used successfully to treat haemangiomas of infancy and childhood, where an antiangiogenic effect has been demonstrated.[3]

Up to now, this antiangiogenic effect was examined in ophthalmology for treatment of choroidal neovascularisation in age related macular degeneration[4] and for treatment of diabetic proliferative retinopathy,[5] but it failed to show an effect in controlled clinical trials.

This case report hints at the possibility of treatment by IFNα-2a and, eventually, prevention of retinal neovascularisations due to ocular inflammation.

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1 Koetter I, Eckstein AK, Stuebiger N, *et al.* Treatment of ocular symptoms of Behçet's disease with interferon α2a: a pilot study. *Br J Ophthalmol* 1998;**82**:488–94.
2 Kaban LB, Mulliken JB, Ezekowitz RA, *et al.* Antiangiogenic therapy of a recurrent giant cell tumor of the mandible with interferon alfa-2a. *Pediatrics* 1999;**103**:1145–9.
3 Ezekowitz AB, Mulliken JB, Folkman J. Interferon alfa-2a therapy for lifethreatening hemangiomas of infancy. *N Engl J Med* 1992;**326**: 1456–63.
4 Fung WE. Interferon alfa-2a for the treatment of age related macular degeneration. *Am J Ophthalmol* 1991;**112**:349–50.
5 Skowsky WR, Siddiqui T, Hodgetts D, *et al.* A pilot study of chronic recombinant interferon-alfa 2a for diabetic proliferative retinopathy: metabolic effects and ophthalmologic effects. *J Diab Compl* 1996;**10**:94–9.

An unusual cause of asthenopia: "pseudo-accommodative insufficiency" associated with a high AC:A ratio

EDITOR,—Asthenopia is characterised by ocular fatigue, frontal headache, and blurred vision, particularly during periods of sustained close work.[1] It can be caused by accommodative insufficiency, a condition in which the effort required to maintain accommodation for near objects produces troublesome symptoms. This may be associated with a low accommodative convergence: accommodation (AC:A) ratio, which the patient has to overcome using positive fusional reserves. When fusion is insufficient symptoms of asthenopia can occur. In contrast, a high AC:A ratio would not normally be associated with asthenopia, but rather with overconvergence, potentially resulting in a convergence excess type of esotropia.[2] We report two cases in which "pseudo-accommodative insufficiency" was identified as the cause of asthenopia and paradoxically associated with a high AC:A ratio.

CASE REPORTS

Case 1

A 12 year old male patient was referred complaining of difficulty with reading. His visual acuities were 6/6, N4.5 in the right eye, and 6/5, N4.5 in the left eye. Cycloplegic refraction showed no significant refractive error. A cover test revealed a 2 prism dioptre exophoria both for distance and near, and he had a full range of ocular motility. Convergence as measured using the RAF rule was well maintained to 6 cm, but accommodative amplitude for an N5 target was only 9 dioptres. It was noted at this stage that when accommodation failed a right esotropia developed transiently. The negative fusional vergence was 4 prism dioptres base-in for near, and 6 prism dioptres base-in for distance. Positive fusional vergence was 40 prism dioptres base-out for both near and distance. The AC:A ratio, measured using the distance gradient method, was 11:1. He was treated with exercises to build up negative fusional vergence.

Case 2

A 20 year old man was referred complaining of blurred vision for near work, associated with frontal headaches. His visual acuities were 6/5, N5 in each eye. Cycloplegic refraction showed no significant refractive error. A cover test revealed a 2 prism dioptre exophoria for near and a 4 prism dioptre exophoria for distance. He had a full range of ocular motility. Convergence as measured using the RAF rule was well maintained to 6 cm, but accommodative amplitude for an N5 target was only 8 dioptres. Again it was noted that when accommodation failed a right esotropia developed transiently. The negative fusional vergence was 12 prism dioptres base-in for near, and 4 prism dioptres base-in for distance. Positive fusional vergence was 20 prism dioptres base-out for near and 14 prism dioptres base-out for distance. The AC:A ratio, measured using the distance gradient method, was 12:1. He was treated with exercises to build up negative fusional vergence.

COMMENT

We believe that we have identified a cause of asthenopia, which paradoxically, is associated with a high AC:A ratio. Although both patients had signs and symptoms, which initially were suggestive of accommodative insufficiency, the sudden transient esotropia that was observed while testing accommodation, together with the high AC:A ratio, indicates that this diagnosis was incorrect. We speculate that these patients choose to relax their accommodation in order to maintain binocular single vision, but at the expense of clarity of vision for near work. If they continued to accommodate their high AC:A ratio resulted in a greater angle of esophoria, and when their negative fusional vergence reserves were no longer sufficient to compensate then a manifest deviation developed. We have coined the term "pseudo-accommodative insufficiency" to describe this phenomenon.

When assessing patients with asthenopic symptoms it is important to distinguish between true accommodative insufficiency and "pseudo-accommodative insufficiency". Treatment for the former using convex lenses would be inappropriate for the latter, as it does not address the underlying cause of the problem. Instead, management should be aimed at augmenting negative fusional reserves and negative relative vergence.

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1 Mehta A. In: Rosenbaum AL, Santiago AP, ed. *Clinical strabismus management. Principles and surgical techniques.* Philadelphia: Saunders, 1999:3.
2 Von Noorden GK. *Binocular vision and ocular motility. Theory and management of strabismus*. 5th ed. St Louis: Mosby, 1996:306.