Figure 3 (a) Periorbital skin biopsy (low magnification) showing diffuse infiltration of the dermis by cells with folded nuclei and pale cytoplasm. (b) Periorbital skin biopsy (high magnification) showing occasional cells staining positive with chloroacetate esterase, in keeping with a myeloid leukaemic infiltrate.

changes in blood viscosity and clotting status.⁷ Cutaneous lesions have also been well described.^{6,8,9} Involvement of the periorbital skin however is rare.^{6,10,11} Treatment of cutaneous lesions with radiation is effective in producing symptomatic relief.¹¹ This modality of treatment is of particular value in providing symptomatic relief in terminally ill patients while avoiding eyelid surgery.

This case illustrates the different ways in which acute myeloid leukaemia can manifest itself in the eye. It also demonstrates the efficiency of radiotherapy as a palliative treatment for subcutaneous eyelid leukaemic infiltration.

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Sir,

Solar retinopathy after the 1999 solar eclipse in East Sussex

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Introduction

We studied 15 patients who suffered solar retinopathy as a result of viewing the subtotal (98%) solar eclipse in East Sussex on 11th August 1999.

Methods

Patients who viewed the eclipse were defined as having solar retinopathy if they were symptomatic with visible retinal signs consistent with a solar burn. At 3 months achromatic contrast sensitivity (ACS) and

chromatic contrast thresholds (CCT) were tested. They were reviewed at 8–12 months if still symptomatic.

ACS and CCT were measured using a cathode-ray-tube based gratings system.¹ Our system tests the central four degrees of vision. ACS was measured at six spatial frequencies: 0.33, 0.66, 2.2, 3.4, 10.0, and 17.0 cycles per degree. The system presents vertical sinusoidal gratings of light and dark bars of varying luminance. CDT used low spatial frequency sinusoidal gratings with standardized equiluminance along either the red/green or tritan confusion axes.

Results

No symptom at presentation predicted long-term visual loss (Table 1). Most patients experienced a central, dark, positive scotoma.

One patient (case 9) used approved protective sunglasses bought from a supermarket. These were

double aluminised, neutral density 5 optical quality polyester lenses and claimed transmittance of 0.001% of visible light and no transmittance of ultraviolet or infrared light.

One patient (case 14) admitted to taking 'speed' (presumably an amphetamine-based drug) and viewed the eclipse for 30 min unprotected with resulting severe burns (Figure 1).

Only two eyes of two patients (cases 5 and 14) had worse than 6/6 vision at final follow-up and one of these was lost to follow-up after 3 months. However, in many patients subtle visual disturbances persisted. In none of the patients did visual acuity deteriorate after presentation.

Most patients initially had a deep white spot centrally or slightly eccentrically in the foveal region which persisted at 3 months in some of the worst affected (Figure 1) but eventually resolved. Pseudoholes remained in some cases (Table 1).

Significantly abnormal ACS and CCT (worse than two standard deviations from the means of age-matched controls) were only detected in two patients. In one eye of each of these patients there were moderate deficits at low and medium spatial frequencies but severe deficits at high spatial frequencies. CCT were also significantly worse than 2 SD from the mean CCT of aged-matched controls for both red-green and tritan contrast thresholds in these eyes. The other eye of patient 14 also had significantly affected red-green and tritan colour vision and milder but significantly affected CCS at low and high spatial frequencies.

Figure 1 Colour fundus photographs of the right and left eyes of a 17-year-old female patient taken 3 months after the eclipse. These show classical slightly eccentric foveal deep retinal hypopigmentation. She said she had watched the eclipse for 30 min under the influence of 'speed'. Visual acuity was 6/9 on the right and 6/6 left at this time (Case 14). On review at 1 year these signs had almost disappeared except for a small approximately 200- μ m pseudohole at the right fovea.

Discussion

Eclipse retinopathy is the commonest cause of solar retinopathy.² Victims of solar retinopathy may complain of blurred vision, a central black spot and metamorphopsia. After 6 months the visual acuity is usually in the range of 6/5 to 6/12 but frequently with a small central subjective scotoma. Visual acuity does not always recover and has reportedly remained as low as 3/60 with permanent retinal damage in the form of retinal holes and pseudoholes.³

After a week the initial deep yellow exudate shrinks and may be surrounded by a red halo followed by retinal pigment epithelium hypopigmentation with surrounding pigment clumping at 6 weeks. The appearance of a lamellar macular hole at or adjacent to the foveal reflex may develop.⁴

Ultrastructurally, photoreceptors suffer fragmentation and vesiculation of the rod and cone outer segment lamellae as well as pyknosis of nuclei especially of

Table 1 Clinical case summaries

Case	Sex	Age	Protection	Eye	Visual acuity				Signs at last review
					1st week	3/12	8/12	Persistence of symptoms	
1	m	22	nil	both	6/5 6/5	6/5 6/5	---	none at 8/12	pale spots 3/12
2	m	28	nil	left	6/6	6/5	6/5	black +ve scotoma 8/12	normal fundus 3/12
3	m	21	covered R eye	left	6/5	6/5	---	none at 3/12	pseudohole at 3/12
4	f	28	nil	right	6/36	6/6	6/6	small -ve scotoma 8/12	pseudohole 8/12
5	m	36	nil	right	6/36	6/24	*	white +ve scotoma 3/12	white patch 3/12
6	m	14	nil	both	6/5 6/6	6/5 6/5	---	none at 8/12	normal fundus at 3/12
7	m	37	covered R eye	left	6/5	6/5	6/5	crescentic white patch	white patch 8/12
8	m	27	covered R eye	left	6/12	*	*	*	white spot at presentation
9	f	36	approved**	right	6/5 6/5	6/5 6/5	6/5 6/5	black spot at 8.12	normal fundus at 8/12
10	m	33	nil	both	6/9 6/12	-	6/5 6/5	-ve scotomas L+R 11/12	bilateral pseudoholes 11/12
11	f	40	nil	both	6/4 6/4	6/4 6/4	---	no symptoms 3/12 onwards	normal fundus 3/12
12	m	20	nil	both	6/6 6/9	6/6 6/6	*	black spot both eyes 3/12	pale patch R+L 3/12
13	f	9	nil	left	6/9	6/9	6/6	black spot left eye	left pseudohole 8/12
14	f	17	nil	both	6/24 6/24	6/9 6/6	6/12 6/6	blurred patches both eyes	pseudohole R, normal L
15	m	29	sunglasses	left	6/18	6/6 6/6	---	no symptoms 8/12	left pseudohole 3/12

-, Missed appointment.

---, No symptoms at 8 months therefore not reviewed.

*, Lost to follow-up.

**, Wearing approved protection bought from a supermarket.

parafoveal rods. Relative survival of foveal cones may account for the good prognosis for visual acuity.⁵

Two large studies of solar retinopathy were found. Rai *et al*⁶ describe 3-year follow-up of 319 solar retinopathy patients, 126 of whom had observed a solar eclipse. They found a similar good prognosis for visual acuity and only a small proportion with longstanding metamorphopsia (11 eyes of 319 patients). Abnormal tritan colour contrast sensitivity was found in five patients investigated.

Another study⁷ was of 58 solar eclipse-related solar retinopathy patients with a mean of 4.2 years follow-up. This found a rapid initial improvement in visual acuity over the first month with no further improvement after 18 months.

Our study concurs that patients can make an excellent recovery in the first 3 months following a solar retinal injury. 6/6 visual acuity was attained in cases 4, 14 and 15 despite initial visual acuities of 6/36, 6/24 and 6/18 respectively. Persistence of foveal hypopigmentation at 3 months in cases 5 and 14 may have been predictive of worse long-term visual acuity as may have been significantly abnormal achromatic contrast sensitivity (ACS) and chromatic contrast thresholds (CCT).

Conclusion

Visual acuity can improve considerably in the majority of eclipse-related solar burns. However, the persistence

of visual symptoms in those with mild burns would suggest that even brief glimpses of a solar eclipse should be avoided.

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Sir,

Visual field loss and Alzheimer's disease

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Alzheimer's disease (AD) is a degenerative neurological condition of unknown aetiology affecting approximately 10% of the UK population aged 65 or over.¹ Clinical features include disturbances of memory, language, praxis and conceptual abilities. We describe a case of Alzheimer's disease presenting, unusually, with visual field loss.

Case report

A 52-year-old female teacher presented to her optometrist with an 18-month history of difficulty in writing. Optometric examination showed visual acuities of 6/5 in each eye. No abnormality of the anterior segments or fundi was detected and intraocular pressures were 20 mmHg bilaterally. Automated perimetry revealed a left inferior field defect in both eyes. These findings prompted referral to the glaucoma clinic, where a more detailed history was elicited. She first noticed problems with poor spelling and spidery handwriting. More recently she had become aware of an impaired ability to iron, orientate her dress, interpret the time on a clock and to read the schoolchildrens' handwriting. Occasionally her mind would go blank in mid-conversation and she had problems remembering the plot of a film.

She denied headache or other neurological symptoms. Her general health was good and she was not on medication.

The findings recorded by the optometrist were confirmed. In addition, Goldmann intraocular pressures were 16 mmHg in each eye and the optic

disc appearances were normal (cup:disc ratio 0.4). A slightly incongruous left inferior homonymous quadrantanopia was clearly defined by Goldmann and Humphrey full threshold visual field testing (see Figure 1). Apkarian flash visual evoked responses were normal from the left occipital lobe and reduced and delayed from the right. An electroencephalogram showed non-specific changes reflecting generalised cerebral dysfunction, suggesting the possibility of Jacob–Creutzfeld disease to be unlikely. On the basis of the long history, the nature of the presenting symptoms and the striking MRI findings a diagnosis of Alzheimer's disease was made after consultation with a neurologist.

Comment

Disturbances of the visual system in Alzheimer's disease (AD) are well documented, and importantly, can predate other manifestations of dementia.² Unfortunately, because of characteristically vague symptomatology and normal examination findings at presentation, the diagnosis may be overlooked.

Patients with AD usually seek an ophthalmic consultation because of disturbances of pattern processing and recognition. The commonest example of this is difficulty with reading which may present as skipping of words or lines on a page, 'dancing print' and blurred vision, and can progress to alexia in advanced disease.^{2,3} Other well-documented difficulties include an inability to recognize faces (prosopagnosia), to pick out individual objects in a group, or disorders of visuospatial processing,⁴ eg inability to grasp objects within the field of view (optic ataxia), neglect of objects to one side of the object of regard (simultanagnosia), dress disorientation (optic apraxia), and becoming lost in familiar surroundings.

Conventional examination findings are often unremarkable in patients with AD.⁴ Central visual acuity generally remains normal, at least early in the disease. Colour vision disturbances are not uncommon but may not always be apparent on formal testing. Optic disc pallor has been reported, but is not a characteristic finding. Subtle ocular motility disturbances have been demonstrated, but patients are usually asymptomatic.

Visual fields, though sometimes difficult to assess in AD patients, are usually full in the early stages.⁴ Abnormal perimetry measurements have been identified by Trick *et al* during apparently reliable automated field testing.⁵ However, the reported defects were mainly generalised loss of sensitivity or arcuate scotomata, and none had a clearly defined homonymous quadrantanopia. Our patient was young,