LETTERS TO THE EDITOR

Cystoid macular oedema after excimer laser photorefractive keratectomy

EDITOR,—A 54-year-old healthy woman underwent excimer laser photorefractive keratectomy (E-PRK) of her right eye. Pre-operative visual acuity was 0.4 right eye (-8.0 sphere $-1.75 \times 20^\circ$), 0.63 left eye (-4.75 sphere $-2.75 \times 165^\circ$). Both retinæ had mild myopic degenerations (which may have been a reason for the visual acuity not being optimal). One and 4 month(s) later the best corrected visual acuity was right eye 0.3. The retina was unchanged.

Six months later the patient was suffering from metamorphopsia and loss of visual acuity (0.05 right eye with $+4.0$ sphere $-2.75 \times 0^\circ$) for months. The left eye was unchanged.

Both retinæ showed focal areas of hypopigmentation and thinning of the pigment epithelium. In addition there was a parafoveal ring of gliosis in the right eye and late frame angiography revealed the typical petal pattern of cystoid macular oedema (CMO, Fig 1).

Three arguments make us think that our patient's CMO was caused by E-PRK: (1) Although the retina was not entirely void of myopic changes there was neither a retinal oedema nor a Fuchs's spot nor did the patient realise any metamorphopsias before PRK. The reduction in visual acuity occurred about 6 months after PRK and was not caused by a subretinal neovascularisation but by CMO as shown by angiography. And as there were no eye diseases that would have explained CMO and no events (trauma, inflammation) other than E-PRK, this therapy may be suspected as aetiology of our patient's CMO. (2) As there are late onset CMOs after YAG laser treatment^{1,2} the fact that CMO was not present 4 months after E-PRK does not refute our opinion. (3) Probably shock waves (besides other mechanisms) contribute to create a CMO after YAG laser therapy.^{3,4} YAG lasers cause pressure peaks of several kilobars which decrease to about 10 bar after a few millimetres.

Excimer lasers produce shock waves of 100 bar whose intensity does not decrease⁴ within at least 3 mm. Thus the pressure in the anterior vitreous after E-PRK is identical to the one

Figure 1 Angiogram of the right eye about 1 year after excimer photorefractive keratectomy. Late phase with petal pattern of hyperfluorescence typical of cystoid macular oedema.

after YAG capsulotomy and may perhaps elicit a CMO.

Of course, one case report cannot prove a link between E-PRK and CMO but should alert ophthalmologists to look at the retina of patients who have undergone E-PRK.

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Thymoxamine in the treatment of traumatic mydriasis

EDITOR,—Traumatic mydriasis after blunt trauma may persist, often affecting young adults, causing glare, blurred vision, and poor cosmesis. Treatment with the muscarinic agent, pilocarpine drops 1% four times daily, is available if recovery fails but it causes brow ache, miosis, and decreased accommodative amplitude.

The dilator pupillae muscle has α receptors¹ and an α agonist such as phenylephrine causes pupillary dilatation. Thymoxamine drops 0.5%, a competitive α antagonist, causes pupillary constriction. Thymoxamine affects accommodation less than pilocarpine 1%, and needs only be administered twice daily. These advantages prompted this study.

Six patients with an average age of 45 years (range 28 to 60 years). All had traumatic mydriasis for more than 1 month and were given thymoxamine 0.5% drops immediately. No patient had pupil sphincter rupture on slit-lamp biomicroscopy. Each eye was examined 1 hour later. Patients were given the thymoxamine 0.5% drops to take twice a day and examined 1 day later. The following were measured and repeated by a single unmasked observer:

(1) The pupil diameter in millimetres, using the standardised (700 lux) lighting conditions of a single Goldmann field analyser fitted with an eyepiece graticule in a dark room.

(2) The best corrected visual acuity both near (Jaeger type) and far (Snellen chart) with and without a pinhole.

(3) The monocular accommodative amplitude in dioptres was measured using an RAF rule standard technique (fine print target advanced to the patient until onset of blurring). The average of three readings was taken.

(4) Rating of subjective problems on a visual scale: 0=none and 10=maximum, for (a) degree of blurred vision, (b) discomfort on reading, (c) glare and light sensitivity, (d) cosmesis.

Three patients experienced transient discomfort and hyperaemia. Two cases (cases 1 and 2) had widely dilated, completely unreactive pupils and thymoxamine had no effect.

In case 3 the vision improved from 6/9 to 6/6, pupil diameter decreased from 6 mm to 4 mm with better cosmesis and less subjective blurring and light sensitivity.

In case 4 the pupil diameter reduced from 5 mm to 3 mm, with subjective improvement in cosmesis and light sensitivity but no visual improvement.

In case 5 the pupil diameter decreased from 4 mm to 3 mm, vision improved from 6/6 to 6/5 and J2 to J1. Light sensitivity decreased.

In case 6 the pupil diameter fell from 5 mm to 3 mm with no improvement in vision or symptoms except slight reduction in light sensitivity.

The two cases (cases 3 and 5) of visual acuity improvement found there was a definite improvement in their vision.

The range of accommodation increased slightly by 1 to 2 dioptres in all cases in which the pupil reacted to the thymoxamine drops (four cases in total); this may be due to the increased depth of focus produced by a smaller pupil. In the two cases in which the pupil did not react to the thymoxamine drops the accommodation range decreased slightly by 1 and 2 dioptres after the thymoxamine 0.5% drops. Sympathetic tone produces a small inhibition of accommodation in the order of 0.85 dioptres,² therefore blocking sympathetic tone with thymoxamine would also reduce the accommodation range. This reduction of accommodation may also be due to the transient myopia that can occur after blunt injury.³

It appears that thymoxamine has a variable but potentially beneficial effect on traumatic mydriasis with fewer side effects than pilocarpine. The effect of thymoxamine and other sympathetic α blockers in the management of traumatic mydriasis deserves further investigation.

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Fluorescein flushing of the forearm

EDITOR,—Fundus fluorescein angiography is widely used to study the retinal microcirculation. The incidence of complications of the procedure is low, serious complications being even rarer. A reported minor complication of the procedure is accidental arterialisation of the fluorescein dye.¹

A case of inadvertent arterialisation is reported here. A healthy diabetic man of 39 underwent fundus fluorescein angiography. A bolus of 5 ml of 20% fluorescein was injected through a size 21 butterfly cannula, inserted in the antecubital vein as described elsewhere.² During the procedure, the patient's arm was rested on the handlebar of the fundus camera with the forearm extended. At the end of the procedure, the patient was found to have unusual yellow discoloration of the forearm (Figs 1 and 2). The patient did not volunteer any symptoms but on direct questioning, complained of momentary tingling in the fingers towards the later part of the procedure. The skin discoloration faded slowly over 30 minutes while the patient was observed.