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. Preoperative visual acuity was 0.4 right eye (−8.0 sphere−1.75×20°), 0.63 left eye (−4.75 sphere−2.75×165°). 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 + 4.0 sphere−2.75×0°) for months. The left eye was unchanged.

Both retinæ showed focal areas of hypopigmentation and thinning of the pigmented epithelium. In addition there was a paravascular 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 Fuch’s spot nor did the patient realise any metamorphopias 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† 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.‡

YAG lasers cause pressure peaks of several kilobars which decrease to about 10 bar after a few milliseconds. Excimer lasers produce shock waves of 100 bar whose intensity does not decrease within at least 3 mm. Thus the pressure in the anterior vitreous after E-PRK is identical to the one 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 need treatment 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 receptors and an α agonist such as phentolamine 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 slitlamp biomicroscopy. Each eye was examined 1 hour later. Patients were given the thymoxamine 0.5% drops to take 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 eyelid apron 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 dioptries 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 lens 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 dioptries 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 dioptries after the thymoxamine 0.5% drops. Sympathomimetic agents produce a small inhibition of accommodation in the order of 0.85 dioptres, 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.†

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.†

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.†

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 discolorations in the skin of the hand (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.

Figure 1 Angiogram of the right eye about 1 year after excimer laser photorefractive keratectomy. L-posterior pattern of hyperfluorescence typical of cystoid macular oedema.

10=maximum, 0=none

Figure 1 Angiogram of the right eye about 1 year after excimer laser photorefractive keratectomy. L-posterior pattern of hyperfluorescence typical of cystoid macular oedema.
The causes of skin discoloration following fundus fluorescein angiography include extravasation of the dye which is localised and painful. Fundus camera handlebar can cause a tourniquet effect after injection of the dye in the vein on the dorsum of the hand, leading to pooling of the dye in the forearm. In our case, a tourniquet was applied to the arm but was released before the injection of the dye. The upper limit of the skin discoloration was well below the site of the tourniquet (Fig 3). Accidental arterial injection of the dye must have caused the discoloration in our case. A direct arterial injection of the dye is a possibility but in our case the cannula was sited in the superficial vein under direct visual supervision. The injection needle has to penetrate down to the deeper level to enter a tougher artery. The markedly prominent superficial veins making easy insertion of cannula and late onset of patient symptoms suggest an arterial puncture towards the later part of the injection. As the fundus camera handlebar is not very supportive for keeping the forearm extended, a minimal slip of the resting arm can cause flexion of the forearm. Because only bicipital aponeurosis separates the antecubital (median basilic) vein from the brachial artery, a needle from the vein can puncture into the artery on flexion of the forearm. Rubinstein suggested the use of a specially designed armrest with the fundus camera, which can help avoid this. Careful selection of a vein and careful insertion of the venous cannula are necessary to avoid inadvertent arterialisation until such a welcome accessibility is available.

I am grateful to Mr N W D Walshaw for his permission to publish this case and the medical illustration department for its help with the photographs.

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BOOK REVIEW


External ocular diseases make heavy demands on available resources for both primary and secondary ophthalmic care. There have been few recent major texts on this subject which is beginning to change rapidly with the development of new concepts of ocular surface disease and a better understanding of inflammatory processes underlying many of the conditions. A new textbook might have been expected to consolidate this new knowledge. Dr Ostler's book has been eagerly awaited.

The main strengths of this textbook are the high quality of the illustrations together with the coverage of a wide range of uncommon disorders. Its weakness is the use of the traditional approach. Individual disorders have been thoroughly dealt with in the text. This has been at the expense of providing concepts for the classification, diagnosis, and management of groups of diseases. This has resulted in a forbidding volume of text which is frequently repetitious. Summary tables have been used infrequently. The approach is didactic which may be appropriate for non-specialists in this field but does not permit the reader to evaluate the author's proposals critically. The lack of a comprehensive review of the literature will reduce the value of this book for the specialist reader.

The author has commented in the preface, that the book has been divided into two major sections. The first deals with the external eye and on an anatomical basis to facilitate diagnosis. This has been done in outline form for the busy clinician. This section is intended to be partly independent of the second section of book in which disease entities are dealt with in more depth for reference purposes. This has resulted in frequent repetition in both the first and second sections. The disadvantage of dealing with diagnosis on an anatomical basis is that some disorders, with manifestations affecting the lids, conjunctiva, and cornea are therefore dealt with in four chapters (for example, staphylococcal blepharitis and herpetic eye disease).

The first section of eight chapters and 365 pages consists of six chapters dealing with diseases of the different anatomical constituents of the external eye and adnexa, sandwiched between an introductory chapter on general principles of management, and a chapter entitled ocular surface disorders but which deals with this concept in a limited way.

The remaining seven chapters include six which are devoted to infectious disorders. These are segregated by organism with chapters on bacterial infections, chlamydial disease, rickettsial infections, viral infections, fungal ocular infections, and parasitic infections. A final chapter deals with diseases of non-infectious aetiology. One of the unusual features of the second part of the book is that the management of the systemic aspects of these diseases is discussed in some detail in a way which would be more appropriate in a general medical textbook. For example, the management of pneumonia and of the gastrointestinal complications of ulcerative colitis and Crohn's disease are outlined at greater length than the management of atopic keratoconjunctivitis or alkali burns of the anterior segment.

The division into two sections is less than successful because of the almost complete lack of cross referencing between the first and second sections. Establishing whether there is further information in the second section about a condition described in the first can only be established by resorting to the index. Some conditions in the first section are dealt with in more detail because they are not included in the second section in the text.

Diseases of the external eye have been difficult to classify, the pathogenesis is often obscure or ill understood, and clear management strategies are rarely available. This book will be useful in the library as a further source of material on external diseases. It will assist clinicians in the differential diagnosis of disorders of the external eye. Because of its bulk, layout, and approach it is unlikely to find a place on the shelf of the trainee or general ophthalmologist, or in the specialist's library. However, the deficiencies are more a reflection on the difficulty of handling this topic rather than on the author whose expertise as a clinician is frequently apparent.

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