Bilateral simultaneous spontaneous acute angle closure glaucoma in a herpes zoster patient

Sir,—Acute angle closure glaucoma is very common and may occur in predisposed eyes, frequently following precipitating conditions. However, having associated with unilateral acute angle closure, and the occurrence of simultaneous spontaneous attack is extremely rare. We have managed a case of simultaneous spontaneous bilateral acute angle closure in a patient with T-2 dermato- tome herpes zoster.

A 61-year-old woman was referred to our clinic because of severe headache, vomiting, and decreased visual acuity for two days prior to admission. Five days earlier a T-2 dermato- tome herpes zoster (Figs 1 and 2) was diagnosed and a course of oral acyclovir (Zovirax), 200 mg five times a day, was begun. On examination her visual acuity was counting finger OD and hand motion OS. The intra-ocular pressure on application tonometry was 48 mmHg OD and 58 mmHg OS. Both eyes had ciliary congestion, oedematous cornea, shallow anterior chamber, and mid dilated pupils. The lens showed a +2 nuclear sclerosis cataract. The retina was attached and the optic nerve heads showed some haemorrhage.

The patient was treated with manitol, acetazolamide 500 mg intravenously, pilocarpine 2%, and dexamethasone, and within 90 minutes the intraocular pressure was 10 mmHg OU. During the following two days laser iridotomies were performed in both eyes, and today in both eyes the best corrected vision is 20/30. Intraocular pressure is 14 mmHg OD and 13 mmHg OS; the fields of vision are normal, and laser iridotomies are patent.

Acute angle closure glaucoma is frequently precipitated by mydriasis caused by para- sympathetic tone, and subsequent mydriasis might initiate a roseus resulting in angle closure in predisposed individuals.

A. ALHALEL
A. HIRSH
S. MELAMED
M. BLUMENTHAL
Goldschlager Eye Institute, Sheba Medical Center, Tel Aviv University, Ramat Gan, Israel


The association of Fuchs’s corneal endothelial dystrophy with angle closure glaucoma

Sir,—We read with great interest the article by Pitts and Jay.1 We report the results of our study on this subject, which support their observation.

The association of Fuchs’s corneal dystrophy with angle closure glaucoma is usually attributed to the oedematous thick cornea crowding the anterior chamber angle.2,3 We have the impression that the acute angle closure glaucoma in patients with Fuchs’s dystrophy is unrelated to the corneal oedema, since it often occurs long before the process of corneal decompensation. In order to verify this observation we performed a retrospective analysis on our patients who underwent perforating keratoplasty because of Fuchs’s corneal dystrophy during the last 10 years. All the patients were re-examined and the following details were recorded: age, sex, refraction before surgery (from patients’ records or lensmeter readings of the patients’ oldest distance spectacles), history of angle closure glaucoma and peripheral iridectomy, anterior chamber, and anterior chamber depth before surgery.

Twenty-three patients (15 females and 8 males, 33 to 95 years old) were operated on. Eight of them had undergone an iridectomy because of an acute attack of angle closure glaucoma one to 30 years before the development of corneal oedema. An additional three patients had a very narrow angle when first examined by us, and therefore prophylactic iridectomy was performed during the kerato- plasty. Thirteen eyes had axial length shorter than 24.2 mm (mean 21.59, SD 0.80 mm) and anterior chamber depth less than 3.4 mm (mean 2.4, SD 0.24 mm). The means in the general population for axial length and anterior chamber depth are considered 24.2 mm and 3.4 mm respectively.4 Seven had hyperopic refraction (mean 2.25, SD 1.34 dioptr). Only one eye of each patient was considered in the calculations.

In summary, of the 23 patients 11 had problems related to angle closure glaucoma. The acute attack occurred in eight of them long before the clinical symptoms of Fuchs’s dystrophy. This rules out the possibility that the attack was caused by the oedematous cornea crowding the angle. The probability that the corneal damage was caused by the acute rise of pressure and/or the iridectomy seems unlikely. In six of the eight patients acute endothelial damage characteristic of Fuchs’s endothelial dystrophy was present in the fellow eye which had not sustained an acute attack of angle closure glaucoma.

It is obvious from Pitts and Jay’s series, and from the additional information presented in this report, that there is a high incidence of axial hypermetropia, shallow anterior chamber, and angle closure glaucoma in patients with Fuchs’s corneal dystrophy. The clinical evidence suggests that there is no causal rela- tionship in either direction, and it is likely that the association arises from a common factor leading to both conditions by genetic linkage.

ANAT LOEWENSTEIN
OKRA GEYER
DAPHNE HOURVITZ
MOSHE LAZAR
Department of Ophthalmology, Ichilov Hospital, 6 Weizmann Street, Tel Aviv 64239, Israel


Topical beta blockers and serum lipoproteins

Sir,—With reference to the paper by West and Longstaff,1 it was reassuring to read that topical timolol was found ‘to have no significant adverse effect on serum lipoprotein levels’. However, another recent paper came to a different conclusion that topical timolol significantly reduced plasma high-density lipoprotein (HDL) levels.2 The difference in the results of these two studies may be due to the following reasons:

(1) Coleman et al4 studied normal, healthy volunteers while West and Longstaff2 examined glaucoma patients. The effect of topical timolol on serum lipoproteins in elderly glaucoma patients (mean age 67.5 years) may be different from that in young, healthy subjects (mean age 35.2 years).

(2) It is well known that the compliance of patients with glaucoma drug therapy is often less than ideal.3 Since patient compliance with topical timolol appears not to have been objectively assessed in the study by West and Longstaff2, this may have affected the overall results.

(3) When testing for serum lipoproteins, two separate fasting blood samples, taken on different dates, have been recommended to take account of the large intraindividual variations.


Figure 1 T-2 dermato- tome herpes zoster.

Figure 2 T-2 dermato- tome herpes zoster.
in lipid values. The reliance on a single test value may have affected the results.

The authors use the serum triglyceride concentration to establish adequate study power to detect a clinically significant change in serum lipoprotein. It would be more helpful if they were to mention if their study had an adequate power to detect a clinically significant change in the HDL concentration, since it bears a well-established inverse relation to coronary heart disease, unlike serum triglycerides, the role of which is unclear.

We suggest that it is premature to rule out clinically important adverse effects on serum lipoprotein levels by topical beta blockers on the basis of the study by West and Longstaff. This is particularly so because the majority of patients with open angle glucoma throughout the world are commenced on topical beta-adrenergic antagonists. A significant number of them will have coexistent risk factors for coronary heart disease. In these patients even a small reduction in serum HDL concentration and/or increase in serum cholesterol may have a significant effect on the incidence of coronary heart disease. Hence a more comprehensive study of a larger number of glucoma patients involving different ophthalmic centres, if necessary, would be useful to establish the effects of various topical beta blockers on serum lipoprotein levels.

P W JOYCE P SUNDER RAJ
Department of Ophthalmology, District General Hospital, Southport
PR8 6NY


Reply

Sir,—We appreciate the interest shown by Raj and Joyce in our paper. We agree that the effects of topical timolol on serum lipoproteins in elderly patients may differ from that in young healthy subjects. However, as those people in the general population taking timolol therapy are glaucoma patients and not young healthy subjects, we felt it more relevant to study the effect in the former group.

Assessment of compliance with eye drop therapy is always difficult. The method used by Coleman et al. does provide a means of quantitating the number of drops expressed from each bottle, but does not take account of such factors as drops missing the eye and inadvertent or deliberate expression of drops from the bottle. Because our study was carried out over a short time period of 15 weeks on patients newly started on timolol, who had the importance of their therapy carefully explained, we would hope to have had good compliance.

In individual serum lipoprotein levels are known to suffer from a large coefficient of variation which can be reduced by increasing the number of samples from an individual. However, when comparing the mean of a group of individuals at a time with that of a group at another time intraindividual variation is accounted for in the statistical analysis. Our 95% confidence intervals for triglyceride levels at five weeks of −0.22 to +0.14 mmol/l allows us to say with 95% confidence that triglyceride levels do not rise by more than 9% at five weeks. From systematic studies one would expect the reduction in HDL levels to be half this (i.e., 4.5%), a relatively small reduction. However, as Raj and Joyce point out, even a small reduction in serum HDL levels may have a significant effect on the incidence of coronary heart disease.

A larger study demonstrating narrower confidence intervals would, we agree, be useful.

J WEST L JONGSTAFF
Department of Ophthalmology, Royal Hallamshire Hospital, Sheffield S10 2JF


Seeing stars

Sir,—My first few months as a first-year ophthalmology resident literally had me seeing stars. While attempting slit-lamp transillumination on a patient with pseudoxfoliation glaucoma I suddenly focused in on a perfectly symmetrical six-pointed star with a circular centre. Its location seemed to be somewhere between the corneal endothelium and the lens but was difficult to localise precisely.

After shaving the eye I was able to see the other residents in the clinic (who had never seen or heard of this star before) and extremely excited about the possibility of having this ophthalmological phenomenon named after myself. I spent the next two evenings searching through my newly purchased Duane’s textbook to make sure this star had not previously been described.

Finally, a not-so-convincing attending sat down at the slit-lamp and manipulated the settings until he was able to project the tiny image of the above described six-pointed star on to a piece of paper placed in front of the slit-lamp. Since then the sands of time have washed over Haag-Strauss and Bern slit-lamp service centre in New York which solved the mystery. Indeed, all of the newer models of Haag-Streit Bern slit-lamps incorporate a filter in the ‘s’ or small aperture setting which is described as a ‘target with fixation star particularly suitable for examining fixation in amblyopia.’ I suppose I will have to keep searching for an ophthalmological phenomenon to name after myself.

BRUCE ALAN MILLER
George Washington University Medical Center, Washington, DC, USA

Paget’s disease and angiod streaks: one complication less?

Sir,—I read with interest your editorial on the association of Paget’s disease in angiod streaks, and most particularly your comments regarding the article which appeared in the American Journal of Ophthalmology. Not only were the angiod streaks confirmed by colour fluorescein angiography, and then by careful histological serial sections which correlated the clinical appearance of the angiod streaks with linear breaks in Bruch’s membrane.

With regard to the association of angiod streaks with Paget’s disease, we performed a complete ophthalmological evaluation on 50 patients with clinically active Paget’s disease of the bone, and found angiod streaks in five of these patients. Interestingly, there was a higher incidence of angiod streaks in those patients who were clinically more severely affected.

The patients in our survey all had increased 24-hour production of urinary hydroxyproline and an alkaline phosphatase of at least twice normal. Therefore it is not surprising that a group of unselected and probably asymptomatic patients with Paget’s disease should demonstrate a lower incidence of angiod streaks. In addition, Dabb's and Skjodt only performed fluorescein angiography on selected patients. Angiod streaks may be difficult to detect on routine ophthalmoscopic examination.

We agree that the coexistence of pseudo-xanthoma elasticum may be difficult to rule out. However, the presence of even one patient in this unselected group of 70 patients chosen only because of their past exposure to canine distemper could be significant, particularly in the light of our findings that the patients with active Paget’s disease of the bone were more likely to show evidence of angiod streaks. It seems Terry’s original observation probably is correct. Our findings support Terry’s original observation. We assume that in 1934 those patients known to have Paget’s disease had obvious clinical manifestations of the disease, as opposed to the patients reviewed by Dabb and Skjodt, in whom the diagnosis of Paget’s disease may have been less obvious.

JOHN G CLARSKON
Department of Ophthalmology, University of Miami School of Medicine, Miami, USA


BOOK REVIEWS


The 1988 Year Book follows the traditional format of this popular series. Fifty three ophthalmic and specialty-related journals were reviewed, and the editorial selection article which reported the most interesting and innovative material published in 1988. The book is divided into 11 chapters dealing with all the ophthalmic clinical subspecialties as well as basic sciences. The chapters are introduced by short review articles by well known authorities in their fields. The reviews are followed by abstracts of 20 to 30 original papers. After each abstract there is a short (and often amusing) comment by Dr Deutsch.

As all Year Books, the 1988 edition makes