CORRESPONDENCE

*y* T cells in aqueous humour from untreated idiopathic uveitis patients

EDITOR—It is now well established that many lymphocytes are present in the anterior chamber secondary to a blood-ocular barrier breakdown, that most of them are of the T cell lineage,1 and that in some instances they are activated, as shown by the expression of membrane bound high affinity interleukin 2 receptors.2 To the best of our knowledge, however, no studies have determined whether cells bearing the y* T cell receptor heterodimer populate the anterior intracellular fluid in both normal and pathological conditions. By using an immunofluorescence staining technique and two direct enumerating monoclonal antibodies (mAbs) (a phycoerythrin conjugated anti-CD3 mAb (BD); Coulter Immunology, Hialeah, FL) and a fluorescein conjugated panreactive y* T cell reagent (anti-TCR a; T Cell Sciences, Cambridge, MA) we carried out two colour cytofluorimetric analysis on aqueous humour from 20 untreated patients with idiopathic anterior uveitis and in eight patients with idiopathic panuveitis. Ocular diagnoses were made on the basis of history, clinical examinations, and results of routine laboratory tests. The diagnosis was confirmed by no clinical evidence of uveitis syndromes, or conspicuous laboratory abnormalities. Aqueous samples for y* T cell quantitation were obtained by aqueous paracentesis using a plastic tulipan syringe and a 27 gauge needle. The percentage of circulating y* T lymphocytes calculated after density gradient centrifugation of heparinised venous blood from 12 of our patients, as well as from 10 healthy control subjects was assessed in parallel and used for comparison in statistical analyses. Despite similar proportions of CD3+ lymphocytes (data not shown), the number of cells bearing the y* T cell receptor for antigen (CD3+ y* TCR y* 1+) was significantly higher in aqueous humour, in either the autologous or heterologous bloodstream (Table 1). Although the biological significance of y* T cells in aqueous fluids during the clinical course of idiopathic uveitis remains unclear, increased levels in the blood of subjects with some infectious diseases and autoimmune disorders, as well as in the vitreous from a patient with acute sympathetic ophthalmia3 suggests these cells may be involved in immune surveillance and/or autoreactivity. Both y* T cells and uveitis are treated with local or systemic steroid therapy. We have recently demonstrated that the y* T lymphocytes are strongly susceptible to apoptosis induced by glucocorticoids.4 5 If intracellular y* T cells play a role in the pathogenesis of idiopathic uveitis, apoptotic signals may be one of the mechanisms by which these drugs lead to partial or complete remission of the symptoms. Supported by this work and the occasional observations in three patients with ocular complications of toxoplasmosis (two cases) and syphilis (one case) showing that y* T lymphocytes were virtually absent in their ocular fluids.

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History of ophthalmology

EDITOR—I have for some time now been very much enjoying the series ‘History of ophthalmology’ which appears in your journal, written by the estimable Fiona Roman. While not going so far as to say that it is the best thing in your columns, it certainly comes close to this and I am constantly amazed and diverted by the extraordinary pieces of information Ms Roman manages to dig up and provide to your readers.

Is it possible for us to know a little more about Ms Roman? Who is she? Is she a historian or an ophthalmologist (or both) and may we at some point hope to see some of her articles in a more permanent form such as a book?

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EDITOR—I thank John P Lee for his comments. I myself am fascinated by the detailed reports which can be found on all aspects of medical history, particularly where they give a hint of the personalities and attitudes behind them.

Xerophthalmia in Rwandan refugees

EDITOR—I, in July 1994 the influx of Rwandan refugees into the Ngara district of Tanzania created an emergency situation. The newcomers were in worse general condition than previous waves of refugees. During the same period a case of...
corneal ulcer was detected. These events prompted a xerophthalmia survey in the six feeding centres of the camps. In each feeding centre all children present on the day of the survey were examined clinically by the author. Risk factors were also investigated. One thousand five hundred and six children were examined. Bitot’s spot was seen in four children, conjunctival xerosis in one, and corneal ulcer in two. The children attending the two feeding centres serving the population of newcomers had a significantly higher risk of xerophthalmia (prevalence ratio 4.8, p<0.01), as did the children with history of diarrhoea in the previous month (prevalence ratio 1.7, p<0.05).

The 0.5% prevalence of Bitot’s spot and 0.2% temporary corneal lesions indicate that this condition is an important health problem in the refugee population investigated, not only because of the risk of blindness but also because vitamin A deficiency is associated with increased mortality mainly from diarrhoea,1,2 and because vitamin A deficiency is the major cause of death in the camps. The absence of cases of corneal scar suggests that xerophthalmia is a recent event in this population associated with refugee life, otherwise this permanent lesion would be about five times more frequent than the temporary corneal lesions, because of accumulation.3 Therefore, the prevalence of this condition should increase over time unless corrective measures are taken. Vitamin A distribution associated with vaccination and immunization was strengthened. The history of risk factors for this condition was introduced in the feeding centres for newly admitted children and the at risk population examined for signs of vitamin A deficiency. The Njara refugee camps are well regularly organised, but xerophthalmia may well affect other refugees and displaced Rwandan people in other camps.

Serious eye injury caused by rotating wire brushes

EDITOR,—Hassett1 reported a series of cases highlighting the risk of ocular injury from rotating wire brushes. We wish to emphasise this risk and also remind colleagues that such injuries may lead to serious intraocular infection.

A 37-year-old man sustained a corneoscleral perforation by a piece of wire from a rotating brush. He immediately removed the 3 cm long fragment before presenting to the casualty department. At presentation, visual acuity (VA) was 6/12 in the affected eye. Fundoscopy and x ray investigations revealed no evidence of retained intraocular foreign body (IOFB). The anterior chamber (AC) was formed, and slit-lamp examination showed a microhyphaema. The wound was not self sealing and primary repair was performed within 24 hours of the injury.

On the first postoperative day a hypopyon on developed, accompanied by a fibrinous pupillary membrane and intense AC activity. Vitreous biopsy and AC tap provided samples from which a heavy growth of coagulase negative Staphylococcus aureus was shown. An intraocular injection of vancomycin and gentamicin was given, and intensive topical administration of these antibiotics commenced. The signs of infection settled, but a mature cataract developed after 2 weeks.

Six weeks after the injury, he underwent further surgery, combining complete vitrectomy with cataract extraction and capsulotomy with posterior chamber lens implantation. After 8 weeks the visual acuity had returned to 6/36.

The overall rate of endophthalmitis secondary to infection following penetrating injury is less than 10%.2 Penetrating eye injuries caused by high velocity projectiles such as hardened steel fragments from steel shiels are thought to carry a low risk of infection. This is because the particles attain very high temperatures before penetration and therefore sterilise themselves. Wire brushes are not necessarily at a high temperature when they disintegrate and, as demonstrated in our case, may introduce infection. Therefore we recommend that injuries resulting from the use of rotating wire brushes should be treated with caution, as there is a definite risk of subsequent endophthalmitis.

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Ophtalmolososcopic sign of early keratoconus

EDITOR,—I read with interest the recent letter by Pathmanathan et al,1 about the ophthalmoscopic sign of early keratoconus.

This sign is known to some ophthalmologists and optometrists who use it regularly in the assessment of suspected cases of keratoconus at Moorfields Eye Hospital, London, as the ‘old droop sign’, because of the disruption of the reflex by a circular, dark or reddish-brown central shadow which looks like an oil drop.

The sign is best seen through a dilated pupil with a +5 lens in the direct ophthalmoscope held at 33–50 cm from the observer to the patient’s eye2 and is almost diagnostic of keratoconus.

Since, however, changes in the refractive index of the lens or early nuclear cataract can be confused with keratoconus when the sign is elicited, it would be desirable to evaluate the cornea from the temporal side also, to eliminate any reflex coming from the lens.

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