CORRESPONDENCE

Fundal white dots: observations from a murine model of congenital ocular toxoplasmosis

EDITOR,—In the BJO Ben Ezra and Forrester have reviewed extensively current knowledge concerning the phenomenon of multiple evanescent white dot syndrome (MEWDS), a specific clinicopathological entity which is characterised by the presence of evanescent or permanent, small, discrete subretinal lesions, which occur in the absence of vitreous inflammation.1 Multiple pale lesions2–3 (Fig 1) can be observed in the outer retinas of a small proportion of mice with experimentally induced congenital ocular toxoplasmosis.4 These focal lesions, which closely resemble MEWDS in humans, were clearly seen in the peripheral retina using indirect microscopy, and imparted an orange hue to the underlying choroid. The lesions were observed in the eyes of approximately 16% of congenitally infected mice and occurred either unilaterally or bilaterally. They were only observed from 3 weeks postpartum.

Histopathological examination of these refractive disturbances3–5 within the otherwise transparent murine retina revealed that they consisted of focal elevations of the photoreceptor layer (Fig 2). Clusters of inflammatory cells in the form of lymphocytes and macrophages were a consistent feature and were located between the photoreceptor and pigment epithelial layers of the disorganised retina. Immunocytochemical labelling consistently failed to reveal Toxoplasma antigen within or in apposition to these lesions.6–8 Three dimensional reconstructions (Fig 3) of these lesions using serial sections of resin embedded tissue and standard SAS GRAPH routines, revealed that they were consistently dome-shaped, and did not result from artefactual folds in the tissue consequent upon processing or cutting of the tissue.

It is our contention that the focal cellular infiltrates are liable to impinge upon the intrinsic processing mechanisms for removal of photoreceptor elements with the result that focal elevations of the photoreceptors appear in the retina; these are then perceived clinically, under direct illumination, as small discrete spots in the various conditions we reviewed. We believe that they are confusing atrophic retinal patches and/or diffusive aberrations in the mouse model of congenital ocular toxoplasmosis with the evanescent (choroidal - retinal pigment epithelium) white dots we have described in our paper. We, therefore, would not concur with their assumption that the patches they observe are comparable with the white dots observed in humans.

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Natural outcomes of stage 1, 2, 3, and 4 idiopathic macular holes

EDITOR.—In order to evaluate the results of surgical intervention in the treatment of macular holes it is essential that we understand the natural history and visual prognosis of all four stages of macular holes. The paper by Hikichi et al in the BJOG1 was heralded in the accompanying editorial as providing just such an insight into the natural history of idiopathic macular holes. However, I would like to highlight some striking similarities between their data on stage 2 macular holes presented in this study and those reported by the same authors in an article entitled ‘The natural history of stage 2 macular holes’, published in the American Journal of Ophthalmology2 (Table 1).

The authors may suggest that publication of such studies is justified as this study is the first to document the natural history of all four stages of macular holes in a single article. However, the data on stage 3 and 4 holes do little more than echo the authors’ findings in


Reply

EDITOR,—Hay and Dutton refer to ‘multiple pale lesions’ in the model of congenital ocular toxoplasmolysis in mice and believe that they are similar to the ‘white dots’ observed in humans in the various conditions we reviewed. We believe that they are confusing atrophic retinal patches and/or diffusive aberrations in the mouse model of congenital ocular toxoplasmolysis with the evanescent (choroidal – retinal pigment epithelium) white dots we have described in our paper. We, therefore, would not concur with their assumption that the patches they observe are comparable with the white dots observed in humans.
their 1993 American Journal of Ophthalmology article entitled 'Effect of the vitreous on the prognosis of full-thickness idiopathic macular holes' (Table 2).

If the data on stage 2 holes in these studies have been derived from the same cohort of patients I would suggest to the authors that this duplication may detract from the scientific merit of the individual articles.

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Reply

EDITOR—We thank Dr Ferris for his interest in our recently published articles,1,2 and we appreciate his comments. In 1992, when we began to study the natural history of idiopathic macular holes, only patients with stages 3 and 4 macular holes underwent vitreous surgery.4 In 1994, the results of vitrectomy to treat stage 2 macular holes were reported, which resulted in more favourable visual outcomes than those achieved with stage 3 and 4 holes.5 Patients with all stages of full thickness holes – that is, 2, 3, and 4, now undergo surgical management. The efficacy of the surgery has been documented in numerous articles; however, most of them have provided a compilation of data on all stages rather than by individual stage,6 even though the natural outcomes of the macular holes vary by stage. Because macular holes currently are receiving much attention and many new findings have been reported, the background of our studies also has changed, and thus each series1–4 has a different background, aims, and conclusion. Although there is some patient overlap in our studies, we believe that the individual articles have scientific merit and contribute significantly to the body of knowledge about management of idiopathic macular holes.

NOTICES

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

The following meetings (beginning at 5 pm) are open to RSM members and their guests only.

TOXOPLASMOSIS, 8 February 1996

Epidemiology of toxoplasmosis; Representing people affected by toxoplasmosis; The diagnosis of ocular toxoplasmosis; Clinical aspects of toxoplasmosis.

LANG LECTURE, 14 March 1996

New perspective; corean grafting Professor David Easty.

NEW HORIZONS IN THERAPEUTICS, 9 May 1996

The identification of human tumour antigens: a strategy for developing tumour vaccines; New developments in the management of CMV retinitis; The development of ophthalmic drugs; The challenge of gene therapy in the context of eye diseases.

Further details: Alyson Taylor, Sections Officer, Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE. (Tel: 0171 290 2985; fax: 0171 290 2989.)

REGISTRARS’ MEETING, 13 June 1996, 2 pm

For registrars to present research work and case reports. Papers to be considered for publication. Abstracts should be submitted (max. 200 words) for the attention of P Murray, The Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE. (Tel: 0171 290 2985; fax: 0171 290 2989.)

XI TÜBINGEN DETACHMENT COURSE: RETINAL AND VITREOUS SURGERY

The XI Tübingen Detachment course on retinal and vitreous surgery will be held on 11–12 April 1996 at Kurfürstendamm, 10787 Berlin, Germany. Further details: Professor Dr D Kreissig, Univ-Augenklinik, Schönbergstrasse 7, 70207 Tübingen, Germany.

Further details: Dr Amed Gupta, Organising Secretary, Department of Ophthalmology, Postgraduate Institute of Medical Education and Research, Chandigarh – 160 012, India.

Office of Continuing Medical Education

The 18th Annual Wilmer Institute’s Current Concepts in Ophthalmology will be held on 11–16 February 1996 at the Hyatt Regency Cerromar Beach Hotel, Dorado, Puerto Rico.

The 13th Annual Wilmer Institute’s Current Concepts in Ophthalmology will be held on 10–15 March 1996 at the Manor Vail Lodge, Vail, Colorado, USA. Further details: Conference Coordinator, Office of Continuing Medical Education, Johns Hopkins Medical Institutions, Turner 2, 720 Rutland Avenue, Baltimore, MD 21205–2195, USA. (Tel: (410) 955–2959; Fax: (410) 955–0807.)

First European Forum on Quality Improvement in Health Care

The first European forum on quality in health care will be held on 7–9 March 1996 at the QEII Conference Centre, London. Further details: Clare Moloney, BMA Conference Unit, BMA House, Tavistock Square, London WC1H 9JP. (Tel: 0171–383 6663; Fax: 0171–383 6478.)

XIth Symposium of the International Society for Genetic Eye Disease and VIIIth Symposium of the Retinoblastoma Society

The XIth Symposium of the International Society for Genetic Eye Disease and VIIIth Symposium of the Retinoblastoma Society will be held on 10–14 March 1996 at the West Point Convention Centre, Hobart, Tasmania, Australia. Further details: ISGED Secretariat, PO Box 309, Rosny Park, Tas 7018, Australia. (Fax: +61 2 34 44 64.)

Paediatric Week – Alder Hey Education Centre

A national study day on screening for visual defects in children – present evidence and future prospects, will be held at the Royal Liverpool Children’s NHS Trust Alder Hey on Wednesday 13 March 1996. Further details: Education Centre, Royal Liverpool Children’s NHS Trust Alder Hey, Eaton Road, Liverpool L12 2AP. (Tel: 0151 252 5218; Fax: 0151 252 5153.)

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