Presumed ocular larva migrans presenting with features of diffuse unilateral subacute neuroretinitis

EDITOR.—Ocular infection by *Toxocara* typically presents as diffuse endophthalmitis or a granuloma; less frequent are anterior uveitis, pars planitis, optic neuritis, conjunctivitis, keratitis, or lens involvement. Rarely, a motile larva may be visualised.

We present a patient with positive aqueous serology to *Toxocara* species associated with features consistent with diffuse unilateral subacute neuroretinitis (DUSN).

CASE REPORT

A 49-year-old man presented with a 3 day history of blurred vision of the right eye, associated with a floater and a right sided headache. Two weeks previously right corneal trauma had resulted in a red eye which had failed to respond to topical chloramphenicol.

At presentation, the anterior chamber exhibited circumcorneal injection, aqueous cells, and flare with multiple posterior synechiae. A hazy fundus view revealed perivascular 'white patches' and macular oedema. Prednisolone acetate 1% hourly, atropine 1% three times daily, and flurbiprofen 100 mg twice daily were prescribed initially. A 2 mm hypopyon formed 24 hours later but disappeared the following day. Numerous keratic precipitates were present inferiorly. A white 'string of pearls' lesion appeared adjacent to the optic disc (Fig 1A). Intense photophobia impaired fluorescein angiography and initial fundus photography. There was an end systolic aortic murmur. Endogenous endophthalmitis was thus initially thought to be the probable diagnosis. Serological tests excluded systemic candidosis; antimicrobials were withheld pending laboratory findings.

Temporal progression of the disease is illustrated in Figure 2. Cytomegalovirus, herpes simplex virus, varicella zoster virus, *Toxoplasma*, *Treponema pallidum*, *Mycobacterium tuberculosis*, and sarcoid were excluded. Over the following 7 days visual acuity improved and the anterior chamber became less inflamed. The retinal changes continued to evolve. Venous sheathing associated with a branch vein retinal occlusion developed with crops of perivenuous white plaques; this resembled DUSN (Fig 1B). Raised serum IgE and eosinophilia suggested allergy or helminth infection. The stool yielded both *Entamoeba coli* cysts and *Enterobius vermicularis* ova.

Over the following 2 weeks the retinitis became quiescent, although elevated serum IgE and eosinophilia remained. Despite the atypical clinical appearance ocular larva migrans was suspected. Enzyme linked immunosorbent assay (ELISA) on paired serum and aqueous samples demonstrated significantly elevated aqueous antibody activity against *Toxocara* spp; there was no evidence of acute visceral larva migrans.

The active inflammation subsided; treatment was discontinued after 1 month. Slight preretal fibrosis developed around the optic disc (Fig 1C). Right visual acuity remained at 6/18 for the ensuing 3 months.

COMMENT

DUSN is a clinical syndrome. Early features include visual loss, indocytis, vitritis, papillitis, and retinal vasculitis with recurrent crops of grey-white retinal lesions. There is progressive visual loss, optic atrophy, and diffuse pigment epithelial degeneration. The aetiology of
DUSN is undetermined, but has been attributed to subretinal migration by nematodes, including *Toxocara*. The clinical appearances described here are compatible with early DUSN.

Antibody activity against *Toxocara* spp was demonstrated in serum at 1:100 and 1:800 in aqueous. The ELISA measured all antibody classes against diagnostic antigens from shed larval cuticle. The clinically apparent retinitis may have been due to initiation of an immune response to previously shed antigens. Despite detailed biomicroscopy no intact larvae were observed, although this cannot be discounted.

Uveitis has been described in patients with *Entamoeba* colitis reaction, this is likely to be an epiphenomenon.

Another hypothesis is that previous *Toxocara* infection could have sensitised the retina to subsequent inflammatory episodes. Many cases of posterior uveitis are thought to be 'endogenous' being linked to MHC II autoantigen expression and autoimmunity. Such autoimmune posterior uveitis may produce vitritis, retinochoroidal infiltrates, retinal vasculitis, and macular oedema. These were present in this patient.

Trauma, and infection of the anterior eye, can induce non-specific posterior inflammation. Autoantibodies against corneal antigens may cross-react with uveal tissue; indeed uveitis is associated with elevated levels of antiretinal autoantibodies. Thus, antecedent trauma related anterior inflammation may have initiated an autoimmune reaction to retinal constituents if this was a previously sensitised patient.

Further, the eosinophilia and elevated serum IgE could have produced a type I hypersensitivity reaction by degranulation of choroidal mast cells. Local recruitment of immune cells with lymphokine release may induce, for example, vasodilatation and increased expression of 'adhesion' molecules and MHC II, producing breakdown of the blood-retinal barrier and enhanced immune reactivity. This could also have contributed to initiation of an immune response against retinal autoantigens or *Toxocara*. Cells in the aqueous were a feature with this patient. Aqueous cytology and cosinophilic may have proved useful. Eosinophilia is a feature of a number of ocular diseases. Limited quantity of sample, however, precluded such investigation as it was considered more appropriate to investigate the anti-*Toxocara* antibody titre. In summary, the patient described presented with panuveitis. The major feature was retinal vasculitis. Despite the atypical clinical appearance and a close but negative response to Toxocara antigen was demonstrated in the aqueous. Uveitis may have occurred in response to previously deposited *Toxocara* antigens or as a consequence of sensitisation to an autoimmun process.

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*Figure 1* Fundus photograph of left eye posterior pole showing typical *Candida* lesions.

*Figure 2* Fundus photograph of right eye showing peripheral lesion at presentation.

An uncomplicated left pars plana vitrectomy was performed with intracamerall injection of 5 μg amphotericin B in 100 μl volume into both vitreous cavities. Intravenous fluconazole (200 mg daily) was commenced. *Candida albicans* was isolated from vitreous samples and was found to be sensitive to amphotericin B but resistant to fluconazole, so systemic therapy was changed to intravenous miconazole (600 mg twice daily) and continued for the subsequent 6 weeks. Over this period, the left visual acuity steadily improved to 6/9 at which level it has remained.

Despite an initial slight response in the right eye, the peripheral lesion enlarged and developed a localised shallow exudative retinal detachment. He also developed a severe maculopapillary rash secondary to miconazole, which was stopped; further systemic therapy was considered unnecessary since he had already had 6 weeks of treatment. A right pars plana vitrectomy was then performed with injection of a further 5 μg amphotericin B. No retinal breaks were identified at vitrectomy, and the subretinal fluid resolved over the next month, leaving a pigmented scar. One year after his initial presentation, both eyes were quiet with no sign of disease activity, vision was 6/6 right and 6/9 left, and there was an inferonasal chorioretinal scar in the right eye.

**Comment**

This patient illustrates some of the considerable difficulties which can be encountered in managing *Candida* chorioretinitis. Although the clinical appearances of this disease are pathognomonic, delay in adequate treatment may still occur for a variety of reasons, which allows the opportunity for sight threatening complications such as retinal detachment or epiretinal membranes to
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