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.

![Fig IA](image1)

**Fig IA.** Initial presentation with 'string of pearls' lesion. (B) Crops of perivenous white plaques present during first week after presentation. (C) Slight preretinal fibrosis 1 month after presentation.

Figure 1 (A) Initial presentation with 'string of pearls' lesion. (B) Crops of perivenous white plaques present during first week after presentation. (C) Slight preretinal fibrosis 1 month after presentation.

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 syctic 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 perivenous 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 *Entodinium vermiculare* 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.

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

![Fig IC](image2)

**Fig IC.** Clinical and laboratory observations. *IgE levels against house dust mite, grass pollen, cat and dog danders were negative or only slightly raised (Pharmacia CAP System). Specific IgE against *Toxocara* spp was not detected; parasitic helminth infection may cause polyclonal stimulation of IgE producing B lymphocytes. *Anti-Ascaris IgE was minimally raised at 0.35 U/ml (Pharmacia CAP System).* Mebendazole 100 mg was administered on day 15; *E vermiculare* was absent from second stool sample, day 59.

COMMENT
DUSN is a clinical syndrome. Early features include visual loss, indocyclitis, 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.

Autoantibody 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 Toxocara oculi infection,3 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 viritis, retinalchoroidal infiltrates, retinal vasculitis, and macular oedema. These were features present in this patient.

Trauma,6 infection of the anterior eye can induce non-specific posterior inflammation. Autoantibodies against corneal antigens may also cross-react with uveal tissue; indeed uveitis is associated with elevated levels of antiretinal autoantibodies.7 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-retina 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 cytokines and eosinophilia may have proved useful. Eosinophilia is a feature of a number of ocular diseases.8 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, a immune 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 auto-immune process.

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Candida endophthalmitis

EDITOR.—Candida chorioretinitis is the most common cause of endogenous endophthalmitis with a characteristic clinical ocular appearance.1 Well known risk factors include major infections,2 surgery,3 especially on the gastrointestinal tract, indwelling intravenous catheters,4 intra- venous hyperalimentation,5 intravenous drug abuse,6 7 and systemic antibiotic therapy.8 Typical lesions can be found in both eyes in two thirds of symptomatic patients and the lesions are often multiple.9 If recognised early and before vision threatening retinal complications have developed, treatment with intravitreal amphotericin B and imidazoles is highly effective in eradicating the organism and preserving vision.10

CASE REPORT
A 26-year-old white male presented with a 2½ week history of left blurred vision. Three weeks previously he had undergone cholecystectomy for chronic cholecystitis, and 2 days postoperatively it had become pyrexial. Five years earlier he had required almost complete resection of a small bowel, as a result of Crohn's disease. A strangled volvulus, necessitating total perenteral nutrition via a Hickman cannula. The postoperative fever was found to be due to Candida albicans infection of his feeding can nula which was removed with resolution of this fever. No systemic antifungal therapy was given and drug sensitivities were not obtained. A new Hickman cannula was reinserted 8 days later.

Examination revealed a afebrile, healthy young man with no external signs of infection. The corrected visual acuities were 6/5 right and 6/60 left. There was a mild anterior uveitis in the left eye, the right anterior segment was normal, and the intraocular pressures were normal. Examination of the left fundus showed the presence of fluffy white preretinal and intraretinal infiltrates at the posterior pole extending into the posterior vitreous (Fig 1). The right posterior pole was healthy but there was a single white lesion in the inferonasal retina extending to the vitreous base (Fig 2). The clinical appearances in both eyes were consistent with the diagnosis of typical of Candida endophthalmitis and management by left pars plana vitrectomy, bilateral intravitreal amphotericin B injections, and intravenous fluconazole was undertaken.

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 intracameral 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 maculopapular 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 vitreectomy, and the subretinal fluid resorbed 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 6/6 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.