DUSN is undetermined, but has been attributed to subretinal migration by nematodes, including *Toxocara*.² 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.³ 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 coli* infection⁴; 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.⁵ Such autoimmune posterior uveitis may produce vitritis, retinochoroidal infiltrates, retinal vasculitis, and macular oedema. These were features present in this patient.

Trauma to,⁶ and infection of, the anterior eye can induce non-specific posterior inflammation. Autoantibodies against corneal antigens produced by local trauma can cross react with uveal tissue; indeed uveitis is associated with elevated levels of anticorneal 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 1 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 immunoreactivity. 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 to assess 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, an 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 autoimmune 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 surgery,² especially on the gastrointestinal tract, indwelling intravenous catheters,² intravenous hyperalimentation,³ intravenous drug abuse,^{4,5} and systemic antibiotic therapy.¹ Typical lesions can be found in both eyes in two thirds of symptomatic patients and the lesions are often multiple.¹ If recognised early and before vision threatening retinal complications have developed, treatment with intravitreal⁶ amphotericin B and imidazoles is highly effective in eradicating the organism and preserving vision.⁷

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 had become pyrexial. Five years earlier he had required almost complete excision of his small bowel as a result of a strangulated volvulus, necessitating total parenteral nutrition via a Hickman cannula. The postoperative fever was found to be due to *Candida albicans* infection of his feeding cannula which was removed with resolution of his fever. No systemic antifungal therapy was given and drug sensitivities were not obtained. A new Hickman cannula was reinserted 8 days later.

Examination revealed an afebrile, healthy young man with no external signs of infection. The best 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 considered 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 vitrectomy, 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 6/9 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

develop. Treatment of susceptible organisms early in the disease with a combination of intravitreal amphotericin and oral imidazoles, such as fluconazole or ketoconazole, may, however, be curative and reduce or prevent the need for surgical intervention.⁷

Even in those eyes with a vigorous vitritis or anterior uveitis, the number of organisms is low^{1,2} and microbiological confirmation of infection by needle biopsy of the vitreous has a low success rate,⁸ in contrast with that in bacterial endophthalmitis. Thus, there is a tendency to treat these eyes empirically avoiding vitrectomy, often to good effect, but the patient described above illustrates the value of isolating the infecting organism and determining its drug sensitivity. Because of his dependency on intravenous feeding, oral imidazoles could not be given, and of the available intravenous preparations, the organism was sensitive only to miconazole.

Although intravenous miconazole can penetrate the eye and achieve adequate therapeutic levels, this was not effective in this patient as demonstrated by the clinical appearance of progression of the lesion in the right eye. In eyes that are not very inflamed, the blood-retinal barrier may remain intact and systemic drug penetration of the eye may not be adequate. Intravenous amphotericin B has poor ocular penetration even in inflamed eyes but may be effective if used in sufficiently high dosage,⁹ or after vitrectomy when the blood-retinal barrier has been compromised.¹⁰ The overall effect of vitrectomy on the therapeutic efficacy of intracameral amphotericin B is difficult to gauge since ocular clearance of the drug is also enhanced.¹⁰

Alternative choices of therapy for this patient included repeated intravitreal injections of amphotericin B at weekly intervals or intravitreal injection of miconazole; in view of the limited therapeutic options, progressive retinal changes, and good outcome following surgery in the left eye, right pars plana vitrectomy and repeat intravitreal injection of amphotericin B were undertaken. This allowed debridement of the lesion and resulted in an excellent clinical outcome. In *Candida* endophthalmitis, progression of lesions in spite of medical treatment is an indication for vitrectomy to ensure that appropriate drug therapy is given and to reduce the risk of sight threatening complications.

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Congenital stationary nightblindness in a patient with osteopetrosis

EDITOR.—Osteopetrosis, which is characterised by increased skeletal mass due to failure of bone absorption by osteoclasts, is classified into two categories. The juvenile onset type is an autosomal recessive disorder, marked by frequent fractures, anaemia, blindness, low resistance to infection, and early death.¹ Adult onset type, inherited as an autosomal dominant trait, takes a more benign course; frequently asymptomatic and found radiologically.²

Ocular anomalies associated with osteopetrosis are optic atrophy, papilloedema, nystagmus, strabismus, impairment of extraocular motility, ptosis, and exophthalmos.³ However, there has been no report of association between congenital stationary nightblindness (CSNB) and osteopetrosis. We examined electrophysiologically a patient with adult onset osteopetrosis who complained of nightblindness.

CASE REPORT

The patient was a 36-year-old woman whose parents were first cousins. She and two of her three siblings had had nightblindness and high myopia since childhood, although we had no chance to examine her siblings. A chest x ray showed increased bone density and diffusely sclerotic bone structures. Computed tomography scan showed diffuse thickening and increased density of the skull bones (Fig 1). Narrowing of the optic canals was not observed radiologically. She was diagnosed as having adult onset osteopetrosis.

Her visual acuity was 20/25 with a correction of -8.0 D in both eyes. No abnormality was observed in eye movement or in the pupils. Funduscopically, myopic changes were observed. The margins of the optic discs were clear and the colour was good, although the discs were tilted (Fig 2). The dark adaptation curve showed absent rod-dark adaptation. The light peak/dark trough ratios on electro-oculogram were 2.2.

The electroretinogram (ERG) was recorded using contact lens electrodes (Kyoto-Contact-Lens, AE, Kyoto, Japan) as active electrodes. The light source was a stroboscopic light (Nihon-Kohden, SLS3100, Tokyo). After 30 minutes of dark adaptation,

Fig 1A

Fig 1B

Figure 1 (A) Chest x ray showed increased bone density and diffusely sclerotic bone structures indicative of osteopetrosis. (B) Computed tomographic scan of the head showed diffuse thickening and increased density of the skull bones.

scotopic b-wave, photopic flickering, and a single white flash responses were recorded with a blue dim light, 30 Hz flickering light, and a white flash light of 40 J, respectively. Single flash ERGs showed negative-type ERG. Scotopic ERGs were extinguished (Fig 2C).

COMMENT

CSNB has been classified into CSNB with abnormal fundi (Oguchi's disease and fundus albipunctatus) and CSNB with normal fundi.⁴ In CSNB with normal fundi, the fundus appearance is essentially normal, although some patients show changes associated with high myopia⁵ or optic disc changes (tilted, pale, or dysplastic).⁶ CSNB with normal fundi, which is inherited as an autosomal recessive, autosomal dominant, or X linked recessive trait, is classified into two types. Type I shows a markedly reduced ERG. Type II shows a negative type ERG; a normal a-wave with a markedly reduced or