Figure 3 The left fundus showing an extensive, multi-loculated subretinal abscess with patchy intra-retinal haemorrhage. The vitreous was clear.

acuity of 1/36. Further ophthalmic surveillance was performed, but two years later she succumbed to a different infection and died.

Comment

Nocardia are filamentous actinomycete bacteria. *N. asteroides* or *N. farcinica*¹ infection is well-recorded in patients who are immunodeficient, and may cause endophthalmitis or subretinal abscess,² the latter being rare, although *Nocardia* may be its most common bacterial cause.³

Nocardia infection may present with ocular involvement, but at least one other site is invariably affected,⁴ often lung or brain. Sub-retinal biopsy was not performed for our patient as the diagnosis was rapidly clarified by tissue sampling elsewhere, and resolution followed systemic antibiotic treatment.

The differential diagnosis of sub-retinal abscess includes infection by *Klebsiella* spp.,³ *Pseudomonas* spp.,⁵ *M. tuberculosis*, *Staph. Aureus* and *Strep. viridans*. Microscopy of *Nocardia* requires Ziehl-Neelson or Gomori methanamine silver staining, but identification may be difficult. Culture requires 10% carbon dioxide. Antibiotic sensitivity is variable, but staples include cotrimoxazole, amikacin, ampicillin, cefotaxime, vancomycin and linezolid. Combination treatment is usual, continuing for one year to avoid recrudescence.

Unusual intraocular infection is a feature of the immunodeficient patient and this may present acutely with endophthalmitis, or sub-acutely with a sub-retinal focus. *Nocardia* should be part of this differential diagnosis.

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Sir, Bilateral papillitis in ocular toxoplasmosis

Optic nerve involvement is well described in ocular toxoplasmosis, occurring in different situations: (1) associated to an active juxtapapillary retinochoroiditis lesion, (2) papillitis, associated to a healed toxoplasmic lesion, (3) neuroretinitis, associated to a swollen disc with serous macular detachment, and (4) swollen optic disc in the presence of a distant active lesion.¹ We report a case of ocular toxoplasmosis presenting with bilateral swollen discs and no associated retinochoroidal involvement, initially mistaken as a possible manifestation of intracranial hypertension.

Case report

A 64-year-old woman, with no past medical history presented with acute, isolated, painless, bilateral visual loss. Best-corrected visual acuity was 6/18 OU, with relative central scotoma in both eyes and colour vision deficits on Ishihara testing (6/20 errors OD, 3/20 errors OS). The remainder of the examination was normal, except bilateral, asymmetrical optic disc swelling with splinter haemorrhages (Figure 1), and mild vitritis. There were no other signs of intraocular inflammation, confirmed by fluorescein angiography. A general and neurological examination was normal. Routine laboratory investigations were normal, as well as ANA and ACE levels. A cerebral and orbital MRI with contrast was normal, and lumbar puncture showed normal cerebrospinal fluid biochemistry, and normal opening pressure. Serologies were negative for HIV and syphilis.

Serum ELISA IgM and IgG were positive for toxoplasmosis (titres IgG >300 IU/ml, IgM >5.54 AE/ml, VIDAS, BioMérieux, Marcy l'Etoile, France). The IgG-avidity ratio was 0.026, the Sabin–Feldmans dye test titre was 1:1250, and the ISAGA IgM (ImmunoSorbent Agglutination Assay; BioMérieux, Marcy l'Etoile, France) was 12 arbitrary units, suggesting a recent toxoplasmosis infection. Enzyme-linked immunosorbent assay (ELISA) was performed, using polymorphic polypeptides, specific to three clonal lineages, GRA6 II, GRA6 I/III,

Figure 1 Funduscopy at the initial presentation disclosing bilateral papillitis, which completely resolved after antibiotic treatment.

GRA7 I, and GRA7 III. Sera from the patient reacted exclusively to GRA6 II, showing that she most probably was infected with a genotype GRA6 type II *Toxoplasma gondii*. Oral antibiotic treatment was started, using pyrimetamine and sulphadiazine, during 14 days, supplemented with folinic acid, and resulting in dramatic clinical improvement. No corticosteroids were added. Two months after the initial signs, visual acuity improved to 6/4.5 and 6/6 in the right and left eyes, respectively. Colour vision and visual fields recovered completely, whereas funduscopy disclosed neither papillitis nor vitritis. The toxoplasmosis serological tests were performed 6 weeks later, confirming the diagnosis (IgG > 300 IU/ml, IgM 4.20 AE/ml, IgG-avidity ratio 0.035, the Sabin-Feldmans dye test 1 : 1250, and ISAGA IgM 12).

Comment

The serological profile of this patient presenting with bilateral papillitis and vitritis with no peripheral retinal involvement indicated a recent, acquired infection with *T. gondii*, with high *T. gondii*-specific IgG and IgM titres and a low IgG-avidity ratio. The normal cerebrospinal fluid composition and opening pressure, as well as the normal brain imaging ruled out a concomitant condition or central nervous system toxoplasmosis infection.

Isolated primary involvement of the optic nerve is unusual in toxoplasmosis, although infection of neural tissue may occur in congenital toxoplasmosis and in immunocompromised adults.² An inflammatory response secondary to only a few microorganisms has been shown to be the major cause of damage to the optic nerve in histopathological studies of toxoplasmosis,³ and this could have been the case in our patient. Although papillitis can be the result of parapapillary or optic nerve infection,⁴ we believe that the relatively preserved visual function in our patients pleads against such a hypothesis.

In conclusion, bilateral optic disc swelling associated to vitritis can be caused by toxoplasmosis, even when peripheral retinal scars are absent. In selected, atypical cases, intraocular toxoplasmosis should be included as a differential diagnosis of bilateral disc swelling.

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