Sir,

Pars plana vitrectomy with internal limiting membrane removal for a macular hole associated with Behçet's disease

The natural course and prognosis of macular hole in Behçet's disease are poor.[1] Here, we report about one patient with a macular hole and associated Behçet's disease who was successfully treated by vitrectomy and internal limiting membrane (ILM) peeling. The patient's vision improved subsequently.

Case report

A 42-year-old man with bilateral uveitis presented to our department with a complaint of blurred vision in the left eye (OS) for 1 year. He had a 2-year history of recurrent oral and genital ulcers. On presentation, the patient's best-corrected visual acuity was 6/15 in the right eye (OD) and 3/60 OS. Slit-lamp biomicroscopy revealed trace cells in the anterior chambers of both eyes (OU), clear lens, and 3+ cells in the vitreous OU. Indirect ophthalmoscopy revealed 1+ vitreous opacity OD, 2+ vitreous opacity OS, a mottling change of retinal pigment epithelium of macular OU, and a full-thickness macular hole OS (Figure 1a). Optic coherence tomography (OCT) showed a full-thickness macular hole and cystic oedema of the retina (Figure 1b). Behçet's disease was diagnosed based on the International Study Group for Behçet's Disease criteria.[2] Treatment with corticosteroids and cyclosporin A (200 mg/day) was initiated. Pars plana vitrectomy combined with removal of the epiretinal membrane and ILM with 0.1% indocyanine green was performed. Perfluoropropane (C_3F_8, 16%) was injected as a gas tamponade.

Postoperatively, the macular hole was anatomically closed (Figure 1c). OCT confirmed successful closure and demonstrated the disappearance of the intraretinal cystoid space (Figure 1d). The visual acuity of the patient's OS had improved from 3/60 to 6/30 at 6-month follow-up.

Comments

The development of a macular hole is a rare complication of panuveitis in Behçet's disease and can ultimately lead to severe loss of vision.[1] Because idiopathic macular holes rarely occur before the age of 55 years[3]—although we could not dispute the idiopathic origin of our case—we speculate that vitreous inflammation causing vitreous gel shrinkage and tangential traction in combination with macular oedema could cause a macular hole. The present case indicates that pars plana vitrectomy combined with ILM peeling might lead

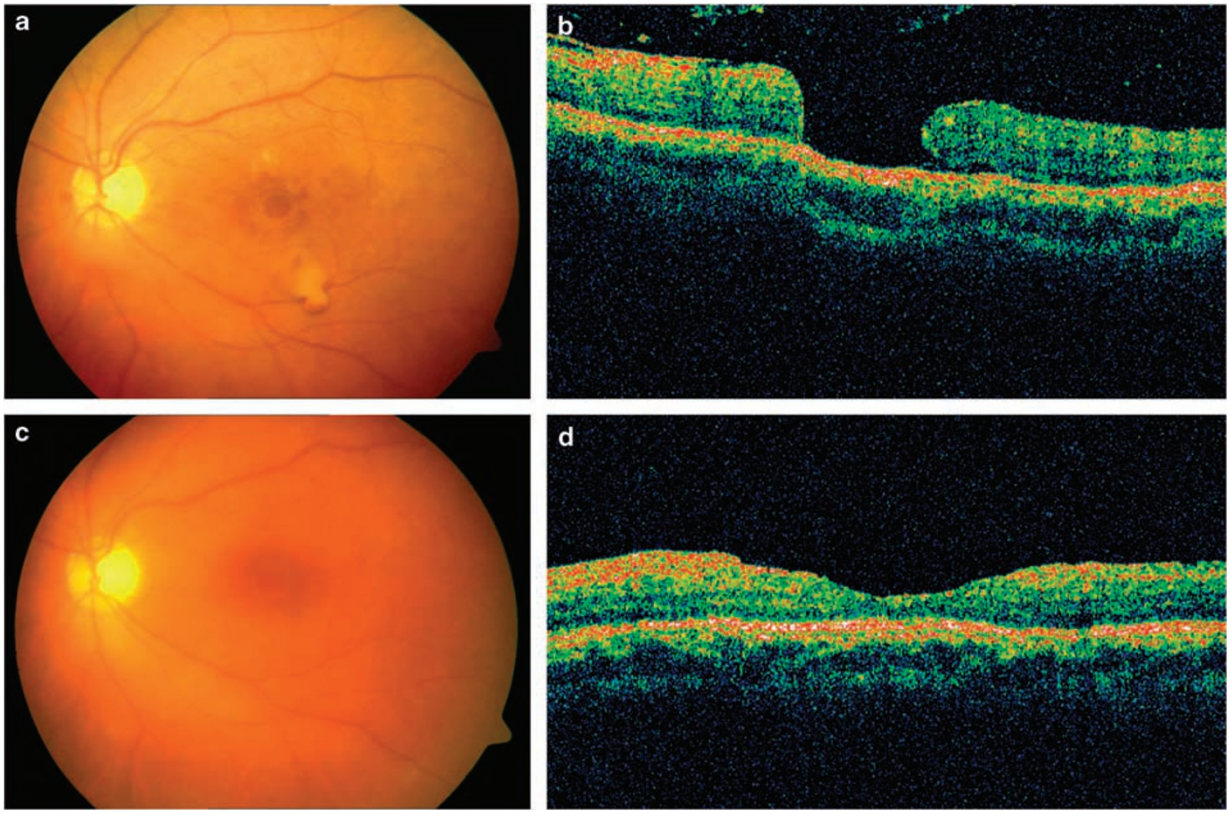

Figure 1 OCT images and fundus photographs of the patient's OS. (a and b) Preoperative fundus photograph and OCT image demonstrated a full-thickness macular hole and cystic oedema of retina. (c and d) A fundus photograph and OCT image taken 3 months postoperatively demonstrated that the macular hole was closed.

to macular hole closure with improvement in visual acuity.

References

1 Sheu SJ, Yang CA. Macular hole in Behcet's disease. *Kaohsiung J Med Sci* 2004; **20**: 558–562.
2 Wechsler B, Davatchi F, Mizushima Y, Hamza M, Dilsen N, Kansu E *et al*. Criteria for diagnosis of Behcet's disease. International Study Group for Behcet's disease. *Lancet* 1990; **335**: 1078–1080.
3 Yannuzzi LA, Seddon JM, Burton TC, Farber MD, Blair N, Haller J *et al*. Risk factors for idiopathic macular holes. The Eye Disease Case–Control Study group. *Am J Ophthalmol* 1994; **118**(6): 754–761.

T-T Wu[1,2,3] and M-C Hong[1,2]

[1]Department of Ophthalmology, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan
[2]Department of Ophthalmology, National Yang-Ming University, School of Medicine, Taipei, Taiwan
[3]Department of Nursing, Shu Zen College of Medicine and Management, Kaohsiung, Taiwan
E-mail: ttwu@isca.vghks.gov.tw

Eye (2009) **23,** 1606–1607; doi:10.1038/eye.2008.243; published online 1 August 2008

Sir,

Neuroretinitis secondary to concurrent infection with cat scratch disease and lyme disease

Neuroretinitis is an infectious or inflammatory process of the optic nerve characterized by optic disc oedema and macular star. The aetiology of neuroretinitis is typically infectious, and cat scratch disease (CSD) is the most common identifiable cause.[1] We report a unique case of neuroretinitis due to parallel infection with *Bartonella henselae* and *Borrelia burgdorferi*, the causative agents of CSD and Lyme disease (LD), respectively.

Case report

A 51-year-old woman presented with sudden decreased vision in the right eye with associated pain with eye movements. Several months before vision loss, she had constitutional symptoms of undetermined aetiology. On examination, visual acuity was 20/400 OD and 20/20 OS. A right relative afferent pupillary defect was present. Slit-lamp examination revealed mild cell and flare reactions in the anterior chamber OD. Funduscopic examination revealed posterior vitreous cells, swollen optic nerve, serous macular detachment, and macular star OD; left eye was normal.

Laboratory investigation revealed white blood count of $10.4 \times 10^3/mm^3$. Syphilis serology was negative. Serum indirect fluorescent antibody titres were positive for *B. henselae* (IgM and IgG >1:1024). *B. burgdorferi* western blot was positive for two of three IgM bands (23 and 41 kD) and 2 of 10 IgG bands (41 and 45 kD). Treatment for CSD was initiated with oral azithromycin and rifampin. She continued to have persistent symptoms; 1 week later, cerebrospinal fluid analysis retuned positive for Lyme IgM antibodies. She was then additionally started on a 1-month course of intravenous ceftriaxone for treatment of LD. Eight days later, systemic symptoms markedly improved. The disc oedema and macular star resolved over 4 months; vision improved to 20/30 OD.

Comment

Concomitant coinfection with LD and CSD should be considered in patients with neuroretinits and may be a reason for continued visual and systemic symptoms despite perceived adequate antimicrobial therapy. Also of note is the speculated mode of transmission of both infections. The *Ixodes ricinus* tick infests deer, dogs, and humans, and is a known vector for LD. Recently, however, *Bartonella* species have been isolated in the *I. ricinus* tick with concurrent presence of *B. burgdorferi* species.[2,3] Identification of coinfected ticks suggests a possible new transmission mechanism of CSD and LD and should be considered when evaluating a patient with neuroretinitis.

References

1 Ray S, Gragoudas E. Neuroretinitis. *Int Ophthalmol Clin* 2001; **41**: 83–102.
2 Schouls LM, Van De Pol I, Rijpkema SGT, Schot CS. Detection and identification of Ehrlichia, Borrelia burgdorferi sensu lato, and Bartonella species in Dutch Ixodes ricinus ticks. *J Clin Microbiol* 1999; **37**: 2215.
3 Halos L, Jamal T, Maillard R, Beugnet F, Le Menach A, Boulouis HJ *et al*. Evidence of Bartonella sp. in questing adult and nymphal Ixodes ricinus ticks from France and co-infection with Borrelia burgdorferi sensu lato and Babesia sp. *Vet Res* 2005; **36**: 79–87.

PK Gupta[1], R Patel[2] and MT Bhatti[1,3]

[1]Department of Ophthalmology, Duke University Eye Center and Duke University Medical Center, Durham, NC, USA
[2]Department of Ophthalmology, Medical College of Georgia, Augusta, GA, USA
[3]Department of Medicine, Division of Neurology, Duke University Medical Center, Durham, NC, USA
E-mail: tariq.bhatti@duke.edu

Conflict of interest: None
Funding: None

Eye (2009) **23,** 1607; doi:10.1038/eye.2008.288; published online 12 September 2008