- 10 Rennie CA, Newman DK, Snead MP, Flanagan DW. Nd: YAG laser treatment for premacular subhyaloid haemorrhage. *Eye* 2001; **15** (Part 4): 519–524.

AS Ioannidis, PG Tranos and M Harris

Department of Ophthalmology
Royal Free Hospital Medical School
Pond Street, London NW3 2QG, UK

Correspondence: AS Ioannidis
Tel: +44 7970 742846
E-mail: alexioannidis@hotmail.com

Sir,

Extensive peripapillary exudation secondary to cat-scratch disease

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Cat-scratch disease is a worldwide zoonotic infection, with the reservoir of infection being domestic cats. It became recognized as an important human pathogen in the mid-1990s.¹ In the United States, it has been estimated to affect approximately 22 000 people annually; its prevalence elsewhere is unknown.¹ Ocular involvement occurs in 5–10% of patients with cat-scratch disease.¹

Case report

A 42-year-old healthy female presented with a 5-month history of blurred vision in her left eye. On examination, vision was 6/6 right and 6/9 left. Visual fields to confrontation demonstrated a central scotoma. Pupillary reactions and colour vision were normal. Anterior segment examination was unremarkable. A few vitreous cells were noted, but the most remarkable finding was optic disc swelling with adjacent retinal folds. Right eye was normal. The patient had no family history of any eye problems. She had not recently travelled abroad and did not keep any pets. A number of blood tests including FBP, ESR, vasculitic, and autoimmune screen, were normal. Plain chest X-ray was also normal.

After 2 months, a yellowish deposit was accumulating around the optic nerve (Figure 1a). A fluorescein angiogram confirmed a swollen optic disc with prominent leakage. Further screening tests for infective agents including toxocara, toxoplasmosis, leptospirosis, and luetic disease were all negative. A CT scan of orbits,

optic nerve, and brain showed no abnormality. The patient was referred to haematology to exclude an infiltrative process. MRI scan was normal and bone marrow biopsy showed no evidence of lymphoproliferative disease. At the next clinic visit, vision was 6/36 and the yellowish deposit previously noted at the left optic disc had increased in size with an associated serous detachment of the macula (Figure 1b).

Bartonella titres were sent to rule out cat-scratch disease. These returned strongly positive (*B. henselae* IgG 256, IgM <20; *B. quintana* IgG 128, IgM <20). The patient then remembered that a feral cat had scratched her 12 months before the start of her visual symptoms, although at no time following this event did she recollect feeling unwell.

The patient was commenced on a 6-week course of rifampicin 300 mg b.d. and doxycycline 100 mg b.d. After 1 month, a definite decrease in the density of the lesion was noted. This improvement continued over the ensuing 12 months. The true extent of the substantial change in the prominence of the exudation can only be appreciated stereoscopically (Figure 2). Unfortunately, vision remained poor at 6/60 from structural foveal changes.

Figure 1 The pictures clearly show the increase in peripapillary exudation.

Figure 2 Post-treatment photograph – the density and prominence of the exudation has decreased. The exudation extended further outwards from the disc as absorption occurred leaving choriorretinal atrophy at the edges.

Comment

Cat-scratch disease is characterized by regional lymphadenopathy secondary to primary cutaneous lesions, usually cat scratches, or the bite of infected fleas. It is often associated with systemic symptoms. The visual complications sometimes coincide with, but more often follow, the systemic manifestations.² Ocular cat-scratch occurs in two forms.³

1. Unilateral granulomatous conjunctivitis.
2. Intraocular inflammation characterized by unilateral or bilateral optic neuritis, retinitis, and vitritis. Other findings include peripapillary and diffuse intraretinal haemorrhages, vaso-occlusive events, white intraretinal infiltrates, yellow-orange subretinal, peripapillary and perifoveal lesions, focal choroiditis, and necrotizing retinitis.

Neuroretinitis is the commonest posterior segment manifestation.¹ The optic disc oedema is often accompanied by peripapillary subretinal fluid, which usually predates the formation of a macular star by 2–4 weeks. However, some patients develop widespread subretinal and intraretinal exudates.¹ This exudation is secondary to abnormally permeable capillaries deep within the disc, which leak lipid-rich exudate into the surrounding retina.²

There is a paucity of information on the natural history of ocular *B. henselae*.⁴ The absence of *Bartonella*-specific IgM in this case along with the information from the patient suggests that ocular problems occurred late in the course of infection. Many reported cases of ocular *B. henselae* start relatively soon after the systemic symptoms and resolve without sequelae over several months.^{5,6} However, as this case demonstrates, ocular changes

secondary to *Bartonella* infection are not always self-limited. Peripapillary exudation started months after her initial presentation and showed no signs of regression until treatment was instituted.

The ideal treatment for ocular Bartonellosis is not yet known; however, it has been suggested that doxycycline or erythromycin with or without rifampicin could be used when infections are severe or sight threatening. The duration of treatment is usually 2–4 weeks in immunocompetent patients and 4 months for immunocompromised patients.^{1,5,7} There are studies showing that the use of these drugs can hasten recovery.⁵ On the other hand, the prognosis from ocular cat-scratch disease is good, with many patients achieving preinfection visual acuity without treatment.⁶

In conclusion, *B. henselae* screening is recommended for patients with papillitis especially if there are other signs of ocular inflammation. Treatment should then be commenced, as ocular cat-scratch disease may not always have a benign course.

References

- 1 Cunningham Jr ET, Kohler JE. Perspective ocular bartonellosis. *Am J Ophthalmol* 2000; **130**: 340–349.
- 2 Solley WA, Martin DF, Newman NJ, King R, Callanan DG, Zacchei T *et al*. Cat scratch disease posterior segment manifestations. *Ophthalmology* 1999; **106**(8): 1546–1543.
- 3 Jones DB. *Cat Scratch Disease. Ocular Infection and Immunity*. St Louis Mosby Year Book. Mosby: St Louis, MO, 1996: pp 1389–1397.
- 4 Kerkoff FT, Ossewaarde JM, de Loos WS, Rothova A. Presumed ocular bartonellosis. *Br J Ophthalmol* 1999; **83**: 270–275.
- 5 Reed JB, Scales DK, Wong MT, Lattuada CP, Dolan MJ, Schwab IR. *Bartonella henselae* neuroretinitis in cat scratch disease. Diagnosis, management, and sequelae. *Ophthalmology* 1998; **105**(3): 459–466.
- 6 Wade NK, Levi L, Jones MR, Bhisitkul R, Fine L, Cunningham ET. Optic disc oedema associated with peripapillary retinal detachment: an early sign of systemic *B. henselae* infection. *Am J Ophthalmol* 2000; **130**(3): 327–334.
- 7 Ormerod LD, Dailey JP. Ocular manifestations of cat-scratch disease. *Curr Opin Ophthalmol* 1999; **10**: 209–216.

CE McAvoy, J Best and JA Sharkey

Department of Ophthalmology, Royal Victoria Hospital, Grosvenor Road, Belfast BT12 6BA, Northern Ireland, UK

Correspondence: CE McAvoy
Tel +44 2890 240 503 x 3822
Fax +44 2890 330 744
E-mail: claramcavoy@doctors.org.uk