tissue spanning the schisis gap. These can clearly be seen in the OCT images. The fact that there are fewer of these strands in the right eye and that the inner layer of the schisis cavity is much thinner may indicate a more advanced stage of the condition and could explain the slightly lower visual acuity in this eye despite the smaller affected area.

Results from the conventional ERG are shown in Fig. 2. The b/a ratios were 1.4 for the right eye and 1.0 for the left (normal range: 1.3–2.9). The maximal b-wave response was clearly reduced in the left eye but just within the normal limits for the right. Cone b-wave responses fell just within the normal limits for both eyes. Implicit times were within the normal range for the maximal responses but slightly delayed in the cone response.

The mfERG showed a marked reduction in the P1 component of the mfERG waveforms in a number of areas in both eyes (Fig. 2). This reduction was most marked for the central hexagons and a larger area of abnormal function was observed in the left eye. P1 amplitudes of the central response for right and left eyes were 41 and 44 nV respectively (normal range: 80–190 nV); P1 latencies were 50 ms and 49 ms respectively (normal range: 38–41 ms); and P1/N1 ratios were 1.5 and 1.6 respectively (normal range: 2.3–2.8).

Comment

The reduced maximal b-wave amplitude observed in the left eye of this patient is characteristic of patients with XLR;[4] however, there has also been a report of a patient known to have XLR but still retaining a normal scotopic b-wave,[5] as seen in the right eye of our patient. The conventional ERG is a measure of the response from the whole retina and in this case did not show an abnormal response in the right eye. The mfERG is capable of eliciting responses from localised areas of the retina and clearly demonstrated that there was abnormal retinal function in a number of macular areas in the right eye despite the normal result from conventional ERG. Thus the mfERG is a better tool for demonstrating the extent of this condition and may be very useful in diagnosing cases of XLR. There have been no publications on mfERG in XLR, and thus further work on a larger cohort of patients is required to establish the characteristics of mfERG recordings from this group of patients. Our findings from OCT are similar to those reported by other groups.[6,7]

To date there have been very few pathological studies on XLR,[8,9] since this condition rarely results in enucleation. Although the resolution currently achievable on OCT scans is not as high as that from microscope images, OCT nevertheless sheds light on the anatomical features of XLR and gives information which cannot be obtained from fundus photography.

References

1. Inoue Y, Yamamoto S, Okada M, *et al*. X-linked retinoschisis with point mutation in the XLRS1 gene. Arch Ophthalmol 2000;118:93–6.
2. Parks SW, Keating D, Evans AL. Wide field functional imaging of the retina. IEEE Med Appl Signal Processing 1999;107:91–6.
3. Condon GP, Brownstein S, Wang NG, *et al*. Congenital hereditary (juvenile X-linked) retinoschisis: histopathological and ultrastructural findings in three eyes. Arch Ophthalmol 1986;104:576–83.
4. Peachey NS, Fishman GA, Derlacki DJ, *et al*. Psychophysical and electroretinographic findings in X-linked juvenile retinoschisis. Arch Ophthalmol 1987;105:513–6.
5. Sieving PA, Bingham EL, Kemp J, *et al*. Juvenile X-linked retinoschisis from XLRS1 Arg213Trp mutation with preservation of the electroretinogram scotopic b-wave. Am J Ophthalmol 1999;128:179–84.
6. Azzolini C, Pierrot M, Codenotti M, Brancato R. OCT images and surgery of juvenile macular retinoschisis. Eur J Ophthalmol 1997;7:196–200.
7. Stanga PE, Chong NH, Reck AC, *et al*. Optical coherence tomography and electrophysiology in X-linked juvenile retinoschisis associated with a novel mutation in the XLRS1 gene. Retina 2001;21:78–80.
8. Manschot WA. Pathology of hereditary juvenile retinoschisis. Arch Ophthalmolol 1972;88:131–8.
9. Yanoff M, Kertesz E, Zimmerman LE. Histopathology of juvenile retinoschisis. Arch Ophthalmol 1968;79:49–53.

S. Muscat[1,2]
B. Fahad[1]
S. Parks[1]
D. Keating[1,2]
[1]Electrodiagnostic Imaging Unit
Tennent Institute of Ophthalmology
Glasgow, UK
[2]Department of Clinical Physics and Bio-Engineering
University of Glasgow
Glasgow, UK

Dr D. Keating ✉
Electrodiagnostic Imaging Unit
Tennent Institute of Ophthalmology
Gartnavel General Hospital
1053 Great Western Road
Glasgow G12 0YN, UK

e-mail: d.keating@clinmed.gla.ac.uk

Sir,

Bilateral papilloedema with concomitant neuroretinitis in a 7-year-old girl with Lyme disease

Lyme disease is on the increase in the UK. It is becoming regarded as the new 'great imitator'.[1] The diversity of ophthalmic manifestations combined with the need for prompt identification and treatment makes an increased awareness amongst ophthalmologists essential. We describe a patient who presented with disc swelling apparently secondary to raised intracranial pressure. However, an ophthalmic assessment subsequently recognised features suggesting an additional neuroretinitis. This prompted further serological tests resulting in a diagnosis of Lyme disease.

Case report

A 7-year-old female presented initially to her general practitioner (GP) with a right Bell's palsy. She was given a 2 week course of systemic steroids resulting in complete resolution of her symptoms after 5 weeks. One month later she presented once again to the GP complaining of frontal headaches, backache and morning vomiting. She was given a short course of oral antibiotics with no effect. A subsequent referral was made to the neurosurgeons when she developed acute visual deterioration associated with distressed and agitated behaviour. Her visual acuities were recorded at 6/36 in the right eye and counting fingers in the left. Fundoscopy findings were documented as 'severe papilloedema'. An MRI scan was performed and reported as 'No space-occupying lesion; high signal changes in periventricular white matter'. This introduced the possibility of demyelination and so the patient was then transferred to the paediatric neurologists who performed a lumbar puncture. The opening pressure was measured at greater than 40 cm H_2O. The cerebrospinal fluid (CSF) was sent for further biochemical analysis and cultures. All these results were found to be within normal limits including absent oligoclonal bands.

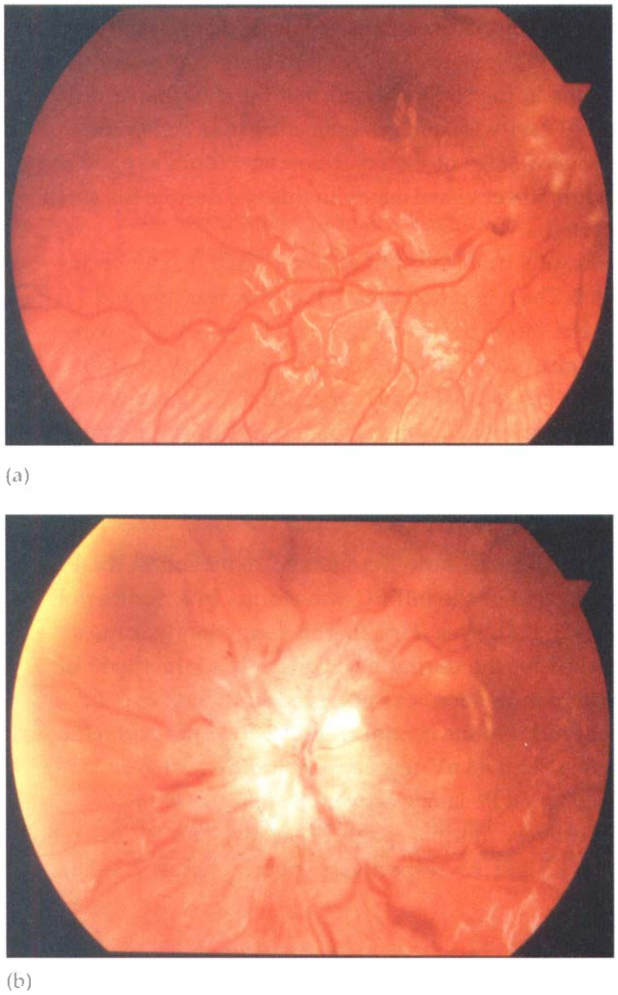
She was then reviewed in the eye clinic. The right (Fig. 1a) and left (Fig. 1b) fundus appearances were of florid bilateral disc swelling with macular exudation. There was no vitritis and both anterior segments were normal. A left relative afferent pupillary defect was noted and visual field testing indicated enlarged blind spots. The conclusion was that the disc appearance was compatible with raised intracranial pressure; however, there were features suggestive of an additional optic neuritis or more specifically neuroretinitis.

The initial working diagnosis was benign intracranial hypertension. Serology was, however, requested to exclude recognised associations of neuroretinitis. The patient was managed with therapeutic lumbar punctures, intravenous methylprednisolone and oral acetazolamide. The visual acuities improved to 6/18 on the right and 6/36 on the left. There was a marked improvement in behaviour.

Serology was returned as negative for *Mycoplasma*, *Chlamydia*, *Coxiella*, *Leptospira*, syphilis, *Bartonella* and herpesviruses. The serology for Lyme disease was returned as positive, i.e. *Borrelia burgdorferi* IgG/M positive and CSF antibodies and PCR positive for *Borrelia*. Intravenous ceftriaxone was commenced. The visual acuities continued to improve to 6/9 on the right and 6/12 on the left. There was complete resolution of the macular oedema and exudates. However, early consecutive optic atrophy was noted.

Comment

The UK incidence of Lyme disease is currently 20–30 new cases per year amongst children (≅ 1 in 500 000/year), and increasing. These cases are currently clustered in southern Britain but this picture may change with the current trend in climatic change. *Borrelia burgdorferi* is a spirochaete transmitted by tick bites. A reservoir exists in voles and dormice. The disease process has three distinct stages reminiscent of syphilis.



(a)

(b)

Fig. 1. *Fundus appearance of (a) the right eye and (b) the left eye.*

Stage 1 (2–30 days) presents with a 'flu-like syndrome and possibly erythema migrans (bull's eye skin lesions). Stage 2 (2 weeks–6 months) has features including cranial (VII, VI, III) and peripheral neuropathies, meningitis and Lyme carditis. Stage 3 (2 months–2 years) may consist of oligo-arthritis and chronic neuropsychiatric sequelae.[1] The antibiotics of choice are intravenous penicillin G or third-generation cephalosporins.[2]

The ophthalmic manifestations of Lyme disease are extremely varied.[3] They include anterior segment manifestations (more common in stage 1), posterior segment involvement (more common in stage 2) and neuro-ophthalmic features (in stages 2 and 3). Anterior segment manifestations may include a non-specific follicular conjunctivitis, a nummular non-staining keratitis, episcleritis and uveitis. These features will typically respond to topical steroids. Posterior segment involvement may include vitritis and optic neuropathy in the form of disc swelling, optic neuritis or neuroretinitis.[4] It should be remembered that systemic steroids should not be used without concomitant antibiotics.[5]

The differential diagnosis for neuroretinitis includes: spirochaetes (syphilis, Lyme disease, leptospirosis (Weil's disease)), viral, *Bartonella* (cat scratch fever) and idiopathic (Leber's idiopathic stellate neuroretinitis). It should, however, be considered that some other conditions can occasionally mimic the disc swelling and peripapillary/macular exudation of neuroretinitis. These include sarcoidosis, toxoplasmosis (Jensen's choroiditis), posterior scleritis and malignant hypertension.

References

1. Pachner AR. Neurologic manifestations of Lyme disease, the new 'great imitator'. Rev Infect Dis 1989;11(Suppl 6):s1482–6.
2. Christen HJ. Lyme neuroborreliosis in children. Ann Med 1996;28:235–40.
3. Zaidman GW. The ocular manifestations of Lyme disease. Int Ophthalmol Clin 1993;33:9–22.
4. Lesser RL. Ocular manifestations of Lyme disease. Am J Med 1995;98(4A):S60–2.
5. Zaidman GW. The ocular manifestations of Lyme disease. Int Ophthalmol Clin 1997;37:13–28.

J. Lochhead ✉
G.M. Thompson
St George's Hospital
Blackshaw Road
London SW17 0QT, UK

Sir,

Post-traumatic endophthalmitis caused by *Xanthomonas maltophilia*

Xanthomonas maltophilia is a Gram-negative bacillus with many similarities to *Pseudomonas* species.[1] It is becoming increasingly recognised as a nosocomial pathogen, particularly in the immunocompromised.[1] Reports of intraocular infection are rare. We report a case of post-traumatic endophthalmitis caused by *Xanthomonas maltophilia* in an immunocompetent individual. To our knowledge, this organism has not been implicated previously as a cause of post-traumatic endophthalmitis.

Case report

A 45-year-old man attended eye casualty complaining of a foreign body sensation and reduced vision in his right eye since hammering metal earlier in the day. He had not worn protective eye goggles. On examination, visual acuity was recorded as 6/6 in the right eye and 6/4 in the left. In the right eye there was a small entry wound 1 mm posterior to the limbus nasally, and a localised cataract. Dilated fundal examination showed a quiet vitreous and a small metallic intraocular foreign body (IOFB) lying inferiorly within the vitreous cavity. The patient was commenced on topical g. cefuroxime 1% and g. gentamicin 1% hourly and reviewed the next day.

The following day, vision remained at 6/6 and ocular examination was unchanged. Surgical removal of the IOFB was planned for the next day. Over the following 24 h, the patient developed increasingly severe pain and reduced vision. The following morning, visual acuity in the right eye was hand movements (HM). The conjunctiva was chemosed and injected and the cornea was oedematous. A 2 mm hypopyon was present and there was an intense vitritis precluding any fundal view. Post-traumatic endophthalmitis was diagnosed, and the patient was taken to theatre urgently for removal of the IOFB combined with lensectomy and vitrectomy. An anterior chamber aspirate and vitreous sample were taken for urgent microscopy, culture and sensitivity, and intravitreal antibiotics (1 mg vancomycin and 0.2 mg amikacin) were administered. Microscopy of the vitreous sample revealed +++ pus cells, but no organisms were seen.

Over the following days the eye gradually became less injected. Culture of both the vitreous and the anterior chamber aspirates grew Gram-negative bacilli, later identified as *Xanthomonas maltophilia*. The bacteria were sensitive to co-trimoxazole, gentamicin, chloramphenicol and cefuroxime, but resistant to ciprofloxaxin. On identification of the causative organism, the patient was commenced on systemic co-trimoxazole 960 mg b.d.

Four weeks later the patient developed a giant retinal tear and underwent retinal detachment surgery including silicone oil injection and band encirclement. One year later the silicone oil was removed, but the visual acuity remained at HM.

Comment

Xanthomonas maltophilia is recognised primarily as a nosocomial pathogen, which can cause potentially life-threatening infections in the immunocompromised.[2] Endophthalmitis due to *Xanthomonas maltophilia* has been described before in a patient with acquired immunodeficiency syndrome (AIDS) and transmission was through a sustained-release ganciclovir implant.[3] The only reported cases of ocular infection due to this pathogen occurring in immunocompetent individuals are a single case occurring after cataract extraction[4] and a small series of 4 cases that all occurred after cataract surgery.[2]

A feature of all these cases was the development of recurrent endophthalmitis and resistance of the pathogen to multiple antimicrobial agents. Of the 4 cases of endophthalmitis described by Chaudhry *et al.*,[2] all were sensitive to polymixin B, 3 were sensitive to co-trimoxazole, 3 were sensitive to ciprofloxacin and all were resistant to imipenem and gentamicin. This is in contrast to our patient, who demonstrated resistance only to ciprofloxacin. This illustrates the wide variety of antimicrobial resistance exhibited by *Xanthomonas maltophilia*. Resistance is thought to be due to the production of β-lactamases and low permeability of the organism to antimicrobial agents, including quinolone antibiotics.[5] However, the organism is usually sensitive to third-generation cephalosporins.[1]

An important aspect of this case was the delay in the removal of the IOFB. Removal of retained IOFBs and administration of prophylactic intravitreal antibiotics within 24 h of the injury reduces the risk of endophthalmitis and also the risk of proliferative