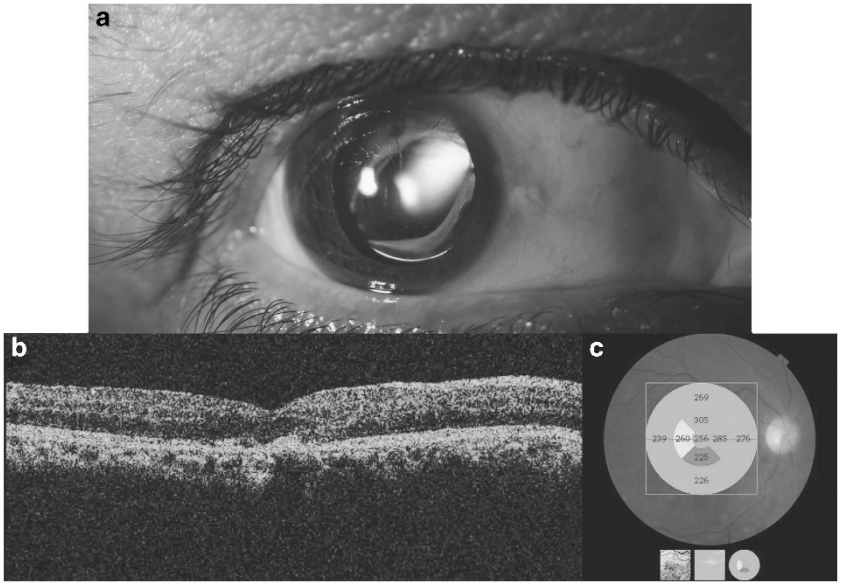

Figure 2 (a) Anterior segment photograph showing posterior chamber intraocular lens implant placed in the sulcus. (b) OCT scan demonstrating dry macula with almost complete resolution of fluid and presence of good foveal contour. (c) OCT map overlays on the colour fundus photograph confirming significant reduction in central retinal thickness. Note disc and retinal haemorrhages have also resolved in just over 3 months.

side effects of treatment were no worse than that reported in the Geneva study.[4,5] Although the implant landed in the crystalline lens and potentially contributed to earlier manifestations of known side effects, watchful and conservative management was prudent and outcomes, both in terms of VA and anatomical improvement of macular oedema, were good.

This case also highlights the need to ensure patient education, particularly keeping in mind the language barriers. Extra time with the patient to explain the procedure, with the help of an interpreter if required, may calm and acclimatise the patient.

Conflict of interest

The authors declare no conflict of interest.

References

1 Malclès A, Janin-Manificat H, Yhuel Y. Anterior chamber migration of intravitreal dexamethasone implant (OZURDEX™). *J Fr Ophtalmol* 2013; **36**(4): 362–367.
2 Bansal R, Bansal P, Kulkarni P, Gupta V, Sharma A, Gupta A. Wandering OZURDEX™ implant. *J Ophthalmic Inflamm Infect* 2012; **2**(1): 1–5.
3 Vela JI, Crespí J, Andreu D. Repositioning of dexamethasone intravitreal implant (Ozurdex) migrated into the anterior chamber. *Int Ophthalmol* 2012; **32**(6): 583–584.
4 Haller JA, Bandello F, Belfort R, Blumenkranz MS, Gillies M, Heier J *et al*. Randomized, Sham-controlled trial of dexamethasone intravitreal implant in patients with macular oedema due to retinal vein occlusion. *Ophthalmology* 2010; **117**: 1134–1146.
5 The Royal College of Ophthalmologists. Interim Guidelines for Management of Retinal Vein Occlusion 2010. Available at: http://www.rcophth.ac.uk/page.asp?section=451.

R Chhabra, K Kopsidas and S Mahmood

Department of Ophthalmology, Manchester Royal Eye Hospital, Manchester, UK
E-mail: romichhabra@gmail.com

Eye (2014) **28,** 624–625; doi:10.1038/eye.2014.18; published online 21 February 2014

Sir,
A case of intermediate uveitis as a precursor to acute disseminated encephalomyelitis (ADEM) in a teenager

Uveitis has been described as a precursor to acute disseminated encephalomyelitis (ADEM) in adults.[1] Here, we describe a case in a teenager. ADEM is a rare post-infectious encephalomyelitis, with characteristic MRI signs and subsequent recovery.[2]

Case report

A 17-year-old Caucasian girl, with past medical history of hypermetropia, mild amblyopia, squint surgery, and long-standing headaches, presented with an episode of pain, watering, and sensitivity to light in the left eye, as an emergency. Her best-corrected visual acuity was RE 6/5, LE 6/18. This was diagnosed as severe anterior uveitis and treated with topical steroids. After 10 days, she was found to have mild anterior chamber activity in

the right eye as well. During her follow-up, she was also found to have moderate amount of vitritis, snowballs, and cystoid macular oedema (CMO) on OCT (Figure 1). No afferent pupil defect was found. A fundus fluorescein angiogram did not show any evidence of vasculitis (Figure 2).

Four months after the onset of uveitis, she presented to the Emergency Department with an acute confusional state, vomiting, decreased consciousness, and floppy right arm. There was no preceding febrile illness, or history of vaccination before this episode. She was intubated, ventilated, commenced on cefotaxime, and transferred to the regional paediatric intensive care unit, where she was started on aciclovir. An urgent MRI of her brain was obtained, showing diffuse oedema and multiple areas of demyelination consistent with ADEM (Figures 3 and 4). A lumbar puncture showed no evidence of infection on culture or PCR for herpes simplex. CSF examination showed 0 WBC, 0 RBC, protein 0.31 g/l, glucose 5.5 mmol/l, and lactate 1.6 mmol/l. Oligoclonal bands were negative. In addition to Behcets and juvenile idiopathic arthritis (JIA),

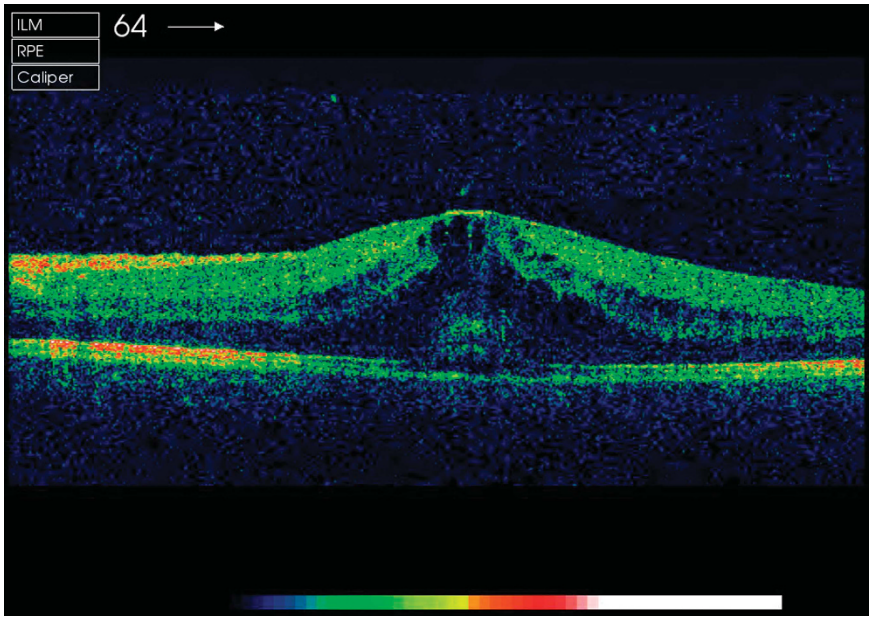

Figure 1 OCT of the left eye showing cystoid macular oedema.

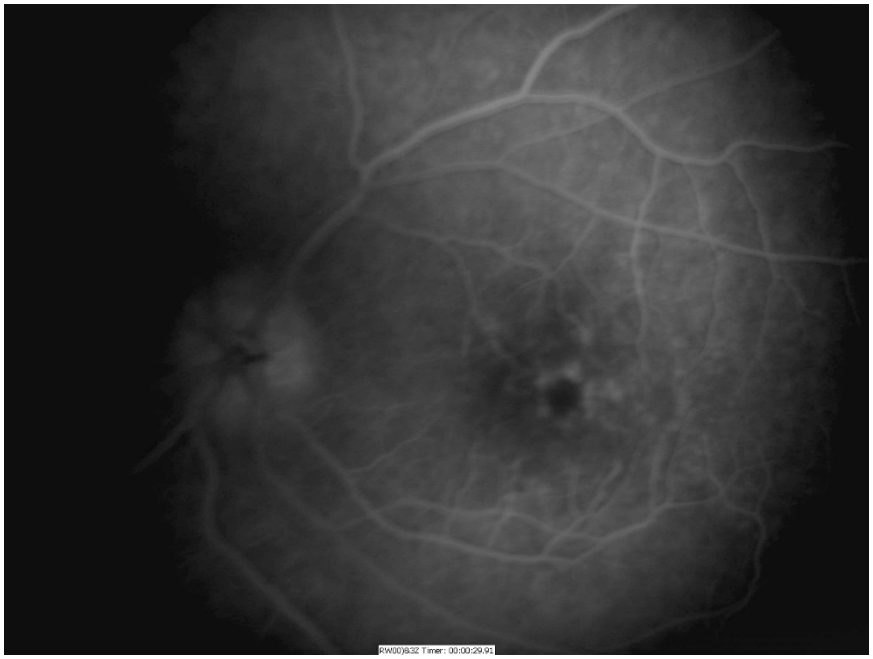

Figure 2 FFA of the left eye, cystoid macular oedema, and no evidence of vasculitis.

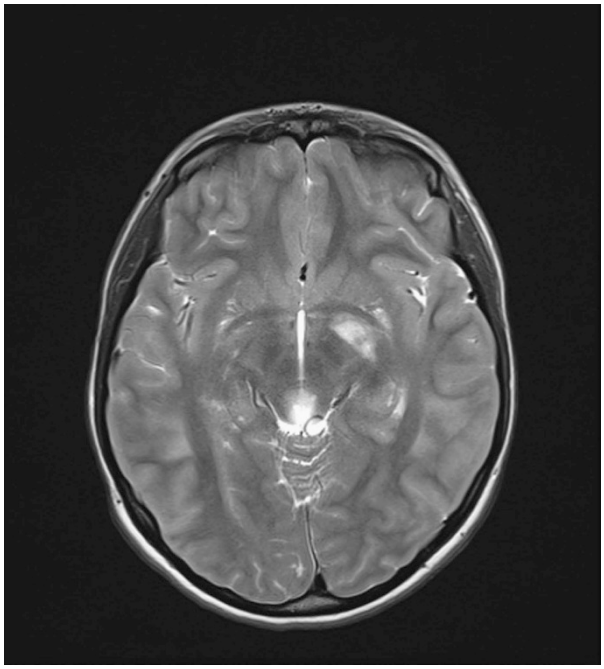

Figure 3 MRI showing high signal in the region of left internal capsule, explaining patient's hemiparetic weakness.

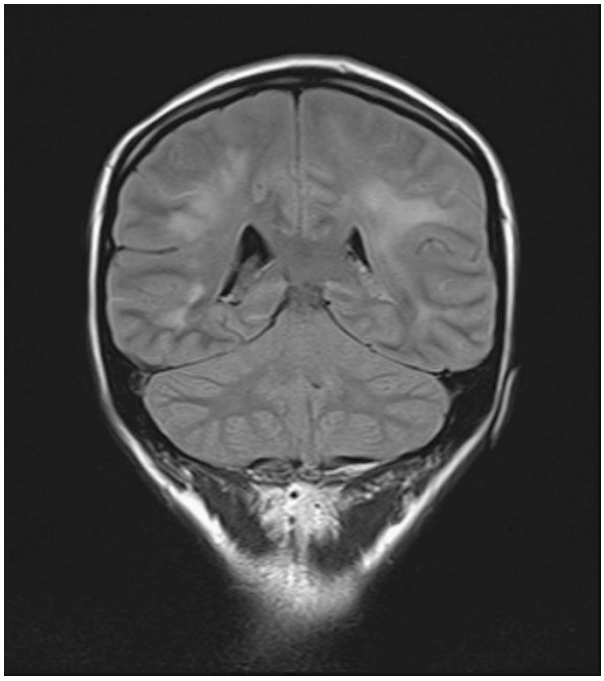

Figure 4 MRI coronal view showing bi-hemispheric widespread white matter high signal change indicative of ADEM.

sarcoidosis was considered and serum ACE was 37 U/l, and autoantibody screen including dsDNA was normal. Only NMDA receptor antibodies were carried out and they were negative. Neuromyelitis optica antibodies (AQP4) and myelin oligodendrocyte glycoprotein (MOG) were not carried out. ESR was 19 mm/h.

Once infection was excluded, she was started on intravenous methylprednisolone followed by a reducing course of oral prednisolone. She was ventilated for 3 days and went on to make a full recovery. Visual acuity improved at this stage to 6/6 in the left eye, after resolution of CMO.

Comment

Multiple sclerosis is associated with ocular inflammatory disease.[3] ADEM is a related, rare condition with diagnostic criteria of polysymptomatic onset encephalopathy, subsequent recovery, and specific MRI changes. Tsutsumi *et al*[1] previously reported a 69-year-old woman who had recurrent uveitis preceding ADEM. Our case presented with intermediate uveitis preceding encephalopathy features typical of ADEM in a teenager. Uveitis resolution occurred after complete resolution of ADEM features.

Although rare, this diagnosis should be considered with neurological features, especially in the presence of headache,[4] optic neuritis,[5,6] diplopia,[5] optic disc haemorrhages,[6] and uveitis.[1]

Conflict of interest

The authors declare no conflict of interest.

References

1 Tsutsumi R, Fukushima A, Hayashi N, Nishino K, Koura Y, Komatsu T *et al*. A case of uveitis followed by acute disseminated encephalomyelitis. [Japanese]. *Jpn J Clin Ophthalmol* 2005; **59**(4): 471–474.
2 Palace J. Acute disseminated encephalomyelitis and its place amongst other acute inflammatory demyelinating CNS disorders. *J Neurol Sci* 2011; **306**(1-2): 188–191.
3 Chen L, Gordon LK. Ocular manifestations of multiple sclerosis. *Curr Opin Ophthalmol* 2005; **16**(5): 315–320.
4 La Mantia L, Erbetta A. Headache and inflammatory disorders of the central nervous system. *Neurol Sci* 2004; **25**(Suppl 3): S148–S153.
5 Odusanya K, Walters BC, Moore W, Liasis A, Nischal KK. Ophthalmic manifestations of demyelinating disease in children. *J AAPOS* 2009; **13**(1): e24.
6 Chan WH, Lloyd IC, Ashworth JL, Jain S, May K, Hughes I *et al*. Acute disseminated encephalomyelitis associated with optic neuritis and marked peri-papillary hemorrhages. *Eye (Lond)* 2011; **25**(12): 1658–1659.

PY Tan[1], J Furness[2], AS Sohal[3], V Ramesh[3] and S Haider[1]

[1]Department of Ophthalmology, County Durham and Darlington Foundation Trust Hospitals, Darlington, UK
[2]Department of Paediatrics, County Durham and Darlington Foundation Trust Hospitals, Darlington, UK
[3]Department of Paediatrics, Royal Victoria Infirmary, Darlington, UK
E-mail: sajjad.haider@cddft,nhs.uk

Eye (2014) **28,** 625–627; doi:10.1038/eye.2014.30; published online 7 March 2014