Kaiser *et al*[5] have described images of the vitreoretinal interface in the context of a clinically evident TTPH showing a partial posterior vitreous separation on OCT, apparently indicating vitreoretinal traction. They hypothesise that vitrectomy may be beneficial in this group of patients because it relieves traction and allows resolution of a shallow traction retinal detachment. Duguid *et al*[8] and Patel *et al*[7] have reported patients with a similar pattern on OCT without a clinically evident TTPH. The patient that we present has responded well to vitrectomy. Preoperatively there was no clinically detectable TTPH, and OCT revealed a dome-shaped elevation with partial vitreoretinal separation (white arrows), suggesting that this OCT pattern may be useful in identifying patients who will potentially benefit from surgery regardless of the clinical appearance of the premacular posterior hyaloid.

Vitrectomy for macular oedema is not universally efficacious, and further investigation is clearly required into the efficacy of surgery in patients with DDMO. We are currently conducting a randomised controlled trial to evaluate the benefit of surgery in such patients. This case shows that patients with no clinical evidence of a TTPH may achieve and sustain anatomical and visual improvement following surgery. Further investigation into the OCT vitreoretinal interface pattern as a potential predictor of surgical outcome is warranted.

Acknowledgement

Proprietary interests: None.

References

1 Gandorfer A, Messmer EM, Ulbig MW, Kampik A. Resolution of diabetic macular edema after surgical removal of the posterior hyaloid and the inner limiting membrane. *Retina* 2000; **20**: 126–133.
2 Harbour JW, Smiddy WE, Flynn Jr HW, Rubsamen PE. Vitrectomy for diabetic macular edema associated with a thickened and taut posterior hyaloid membrane. *Am J Ophthalmol* 1996; **121**: 405–413.
3 Lewis H, Abrams GW, Blumenkranz MS, Campo RV. Vitrectomy for diabetic macular traction and edema associated with posterior hyaloidal traction. *Ophthalmology* 1992; **99**: 753–759.
4 Pendergast SD, Hassan TS, Williams GA, Cox MS, Margherio RR, Ferrone PJ *et al*. Vitrectomy for diffuse diabetic macular edema associated with a taut premacular posterior hyaloid. *Am J Ophthalmol* 2000; **130**: 178–186.
5 Kaiser PK, Riemann CD, Sears JE, Lewis H. Macular traction detachment and diabetic macular edema associated with posterior hyaloidal traction. *Am J Ophthalmol* 2001; **131**: 44–49.
6 Thomas D, Moorman C, Laidlaw DAH. How prevalent is a taut thickened posterior hyaloid? [abstract] 2002 Annual Meeting Abstract and Programme Planner [on CD-ROM]. Association for Research in Vision and Ophthalmology. Abstract 3467.
7 Patel JI, Cree IA, Gregor ZJ, Boulton ME, Hykin PG. Vascular endothelial growth factor and diabetic macular oedema [ARVO Abstract]. *Invest Ophthalmol Vis Sci* 2001; **42**: Abstract no. 3974.
8 Duguid G, Massin P, Houchine B, Erginay A, Gaudric A. Vitrectomy for diabetic macular edema: the effect on visual acuity and optical coherence tomography [ARVO abstract]. *Invest Ophthalmol Vis Sci* 2000; **41**: Abstract no. 585.
9 Otani T, Kishi S, Maruyama Y. Patterns of diabetic macular edema with optical coherence tomography. *Am J Ophthalmol* 1999; **127**: 688–693.
10 Giovannini A, Amato G, Mariotti C, Scassellati-Sforzolini B. Optical coherence tomography findings in diabetic macular edema before and after vitrectomy. *Ophthalmic Surg Lasers* 2000; **31**: 187–191.

D Thomas and DAH Laidlaw

The Vitreoretinal Unit
Department of Ophthalmology
St Thomas' Hospital Lambeth Palace Road
London SE1 7EH, UK

Correspondence: D Thomas
Tel: +44 207 928 9292x3239
Fax: +44 207 922 8165
E-mail: Dhanes.Thomas@gstt.sthames.nhs.uk

Sir,

Acute posterior multifocal placoid pigment epitheliopathy associated with adenovirus infection
Eye (2003) **17,** 542–544. doi:10.1038/sj.eye.6700389

The aetiology of acute posterior multifocal placoid pigment epitheliopathy (APMPPE) remains a subject of debate. Our patient represents the second reported case of APMPPE associated with adenoviral infection.[1]

Case report

A 37-year-old Caucasian male presented with acute bilateral blurring of vision. He also reported a 3-week period of prodromal general malaise with symptoms of myalgia, arthralgia, headache and nonproductive cough. He had no significant past history and was not taking any regular medication. Family history was negative.

Visual acuities at presentation were right eye (RE) 6/24 and left eye (LE) 6/36. Anterior and vitreous chambers were quiet. Fundoscopy showed subtle perifoveal pallor. The next day visual acuities had fallen to 6/60 in both eyes. Ocular examination revealed slightly injected bulbar conjunctivae and <1+ anterior chamber cells

without vitritis. Multiple, pale, greyish fundal lesions were now present in the postequatorial regions, densest at the posterior poles. These lesions were up to one disc diameter in size with mild overlying retinal oedema. A clinical diagnosis of APMPPE was made.

Plasma viscosity was 1.77 mPa s (normal range 1.5–1.72) and C-reactive protein, 18 mg/l (normal <5). Serum angiotensin converting enzyme, antinuclear antibody, antineutrophil cytoplasmic antibody and chest X-ray were normal or negative. No treatment was prescribed.

By day 4, visual acuities were RE CF and LE 6/60. Intravenous fundal fluorescein angiography demonstrated changes typical of APMPPE, namely early hypofluorescence followed by late staining of the fundal lesions (Figure 1). In view of the severity of visual loss, the patient was commenced on a 7-day course of oral prednisolone 40 mg daily. Visual acuities subsequently improved, RE 6/60 and LE 6/18 on day 7.

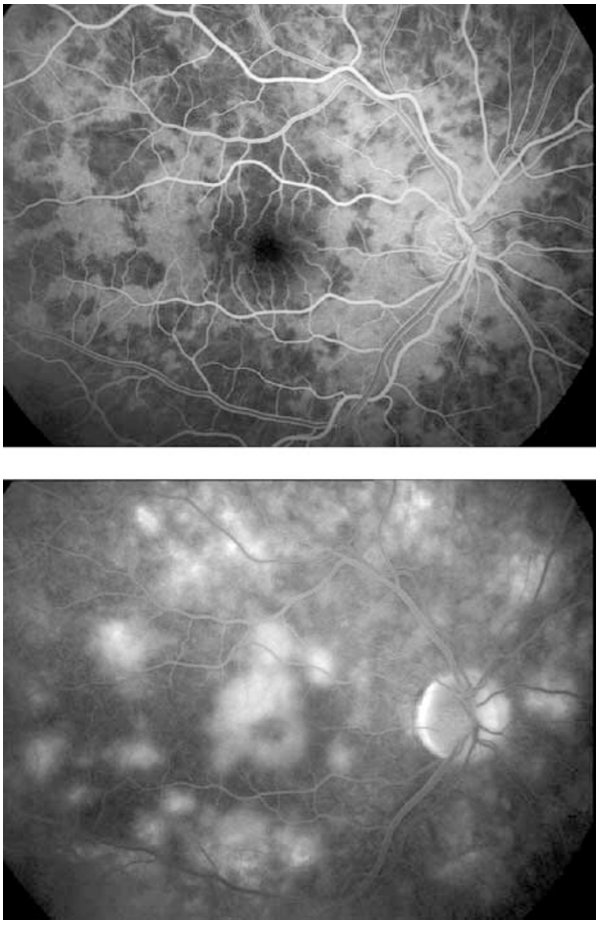

Figure 1 Fluorescein angiography, day 4. Top: early arteriovenous phase, showing hypofluorescence of placoid lesions. Bottom: late staining of placoid lesions at 6 min.

On day 12, he presented with a marked bilateral follicular conjunctivitis. Viral conjunctivitis was diagnosed and no topical treatment was given. Conjunctival swabs taken at this stage later identified adenovirus (direct immunofluorescence of cultured cell suspension). Bacterial and chlamydial swabs were negative. Later, the conjunctivitis worsened with the formation of conjunctival pseudomembrane, bilateral punctate corneal epitheliopathy and eyelid oedema. Subepithelial corneal opacities were noted on day 27.

On day 18, visual acuity in the LE had improved to 6/12 but in the RE, it remained 6/60. Fundal lesions were fading, with early pigmentary deposition. By day 39, retinal pigmented epithelium scars had formed including foveal involvement in the RE. Visual recovery continued in the LE with a final visual acuity of 6/5 on day 140, while the RE remained limited at 6/60.

Comment

This is the second reported case of proven adenovirus occurring simultaneously with the acute phase of APMPPE and lends support to a respiratory virus being implicated in the aetiology of this rare condition.[1] Our patient's prodromal symptoms suggest preceding systemic infection, a feature previously noted in APMPPE[2]. Following the onset of APMPPE, administration of systemic steroid from days 4 to 11 may have promoted resolution of pigmentary epitheliopathy; however, it may also have contributed to the manifestation of adenoviral infection in a further site, that is, keratoconjunctivitis.

To date there is no histology available on an eye with active APMPPE and the aetiology remains a subject of debate. APMPPE can follow systemic triggers such as bacterial[3] or viral[1,4] infection, vaccination,[5] and use of antimicrobial agents,[6] suggesting an immune-mediated mechanism. An immunogenetic predisposition for developing APMPPE is supported by the association of HLA types B2 and DR7[7]. Abnormalities of choroidal blood flow, demonstrated by indocyanine green angiography,[8] suggest a choroidal vasculitis, and there are reports of associated systemic vasculitides.[9,10]

Gass[11] originally described APMPPE as a benign disease with a good visual prognosis. More recently, it has been suggested that lack of visual recovery may be predicted by the presence of atypical features including age over 60 years, recurrent disease and unilateral involvement.[12] In contrast to this, our 36-year-old patient had profoundly different visual outcomes in each eye following new, bilateral disease. This case illustrates that the prognosis in APMPPE with foveal involvement should be guarded.

Acknowledgement

Proprietary interests declared: None.

References

1 Azar P, Gohd RS, Waltman D, Gitter KA. Acute posterior multifocal placoid pigment epitheliopathy associated with an adenovirus type 5 infection. *Am J Ophthalmol* 1975; **80**: 1003–1005.
2 Holt WS, Regan CD, Trempe C. Acute posterior multifocal placoid pigment epitheliopathy. *Am J Ophthalmol* 1976; **81**(4): 403–412.
3 Lowder CY, Foster RE, Gordon SM, Gutman FA. Acute posterior multifocal placoid pigment epitheliopathy after group A streptococcal infection. *Am J Ophthalmol* 1996; **122**(1): 115–117.
4 Borruat FX, Piguet B, Herbort CP. Acute posterior multifocal placoid pigment epitheliopathy following mumps. *Ocular Immunol Inflamm* 1998; **6**(3): 189–193.
5 Brezin AP, Massin-Korobelnik P, Boudin M, Gaudric A, LeHoang P. Acute posterior multifocal placoid pigment epitheliopathy after hepatitis B vaccine. *Arch Ophthalmol* 1995; **113**(3): 297–300.
6 Lyness AL, Bird AC. Recurrences of acute posterior multifocal placoid pigment epitheliopathy. *Am J Ophthalmol* 1984; **98**(2): 203–207.
7 Wolf MD, Folk JC, Panknen CA, Goeken NE. HLA-B7 and HLA-DR2 antigens and acute posterior multifocal placoid pigment epitheliopathy. *Arch Ophthalmol* 1990; **108**(5): 698–700.
8 Dhaliwal RS, Maguire AM, Flower RW, Arribas NP. Acute posterior multifocal placoid pigment epitheliopathy. An indocyanine green angiographic study. *Retina* 1993; **13**: 317–325.
9 Laatikainen LT, Immonen IJ. Acute posterior multifocal placoid pigment epitheliopathy in connection with acute nephritis. *Retina* 1988; **8**(2): 122–124.
10 Wilson CA, Choromokos EA, Sheppard R. Acute posterior multifocal placoid pigment epitheliopathy and cerebral vasculitis. *Arch Ophthalmol* 1998; **106**(6): 796–800.
11 Gass JD. Acute posterior multifocal placoid pigment epitheliopathy. *Arch Ophthalmol* 1968; **80**(2): 177–185.
12 Pagliarini S, Piguet B, Ffytche TJ, Bird AC. Foveal involvement and lack of visual recovery in APMPPE associated with uncommon features. *Eye* 1995; **9**(1): 42–47.

SPS Thomson[1] and STD Roxburgh[2]

[1]Ophthalmology Department
Royal Bournemouth Hospital
Castle Lane East
Bournemouth, BH7 7DW, UK

[2]Ophthalmology Department, Ninewells Hospital
Dundee DD1 9SY, UK

Correspondence: SPS Thomson
Tel: +44 1202 303626
Fax: +44 1202 704367
E-mail: st2@whsmithnet.co.uk