appropriate antibiotics. A significant amount of bacterial debris and potentially toxic products remain to account for treatment failure. The treatment of bacterial endophthalmitis ideally should be directed to the simultaneous control of infections and inflammation.⁷ Hence, we prefer to inject intravitreal dexamethasone with antibiotic.

In our patient, despite treatment, the vitreous remained culture positive, and the same organism with the same sensitivity pattern was grown. This could probably be due to phenotypic changes in the organisms within the eye, thereby inhibiting the effect of antibiotics⁸ and causing recurrent infections. This ultimately resulted in phthisis bulbi.

In conclusion, endophthalmitis due to *B. cepacia* is potentially difficult to cure. Patients need close follow-up due to risk of recurrence of infection.

Acknowledgements

This work was supported by Hyderabad Eye Research Foundation, Hyderabad.

References

- 1 Speert DP. Advances in *Burkholderia cepacia* complex. *Pediatr Respir Rev* 2002; **3**(3): 230–235.
- 2 Piero ED, Pennet M, Leopold I. *Pseudomonas cepacia* endophthalmitis. *Ann Ophthalmol* 1985; **17**: 753–756.
- 3 Irvine WD, Flynn HW, Miller D, Pflugfelder SC. Endophthalmitis caused by gram negative organisms. *Arch Ophthalmol* 1992; **110**: 1450–1454.
- 4 Simpson IN, Finlay J, Winstanley DJ, Dewhurst N, Nelson JW, Butler SL *et al.* *J Antimicrob Chemother* 1994; **34**(3): 353–361.
- 5 Joklik WK, Willet HP, Amos DB, Wilfert CM (eds). *Zinsser Microbiology*, 19th ed. Conn. Appleton & Lange: East Norwalk, 1988, pp 488–489.
- 6 Stern GA, Engel HM, Driebe WT. Recurrent postoperative endophthalmitis. *Cornea* 1990; **9**: 102–107.
- 7 Das T, Jalali S, Gothwal VK, Sharma S, Naduvilath TJ. Intravitreal dexamethasone in exogenous bacterial endophthalmitis. Results of a prospective randomized study. *Br J Ophthalmol* 1999; **83**: 1050–1055.
- 8 Davey PG, Barza M, Stuart M. Dose response of experimental *Pseudomonas* endophthalmitis of ciprofloxacin, gentamicin and imipenem: evidence for resistance to 'late' treatment of infections. *J Infect Dis* 1987; **155**: 518–523.

A Pathengay¹, B Raju¹, S Sharma², T Das¹ and Endophthalmitis Research Group

¹Smt. Kanuri Santhamma Retina Vitreous Center
LV Prasad Eye Institute, Hyderabad, India

²Jhaveri Microbiology Center
LV Prasad Eye Institute Hyderabad, India

Correspondence: A Pathengay
Smt Kanuri Santhamma Retina Vitreous Center
LV Prasad Eye Institute, LV Prasad Marg
Banjara Hills, Hyderabad 500 034, India
Tel: + 91 040 23608262
Fax: + 91 040 23548271
E-mail: avinash@lvpei.org

Support: Hyderabad Eye Research Foundation
Hyderabad

Eye (2005) **19**, 358–359. doi:10.1038/sj.eye.6701501
Published online 30 July 2004

Sir, Purtscher-like retinopathy as an initial presentation of thrombotic thrombocytopenic purpura: a case report

Thrombotic thrombocytopenic purpura (TTP) is a rare haematological disorder in which there is microvascular thrombosis with increased platelet consumption.¹ Ocular manifestations of TTP includes retinal haemorrhages, oculomotor nerve palsies, pupil abnormalities, and choroidal and retinal vascular occlusions.² We report a case of TTP, which presented with a severe bilateral ischaemic Purtscher-like retinopathy several days before any haematological abnormalities were detected. The patient has been left with permanent visual loss.

Case report

A 24-year-old Nigerian woman, presented with a 2-week history of left-sided pleuritic chest pain. Clinical examination and chest X-ray demonstrated left lower lobe consolidation. Her blood test showed neutrophilia and eosinophilia. C-reactive protein measured 105.

After 2 days, the patient complained of bilateral loss of vision and was referred for an ophthalmic opinion. Her visual acuities were 6/24 in the right eye and 6/60 in the left best corrected. Dilated fundal examination revealed bilateral Purtscher's retinopathy with macular oedema (Figure 1). Fundus fluorescein angiography demonstrated areas of capillary drop out and retinal nonperfusion at the posterior poles (Figure 2).

A provisional diagnosis of a systemic vasculitis was made, and treatment with intravenous methylprednisolone 1 g/day was commenced. After

Figure 1 Dilated fundal examination revealed extensive bilateral peripapillary cotton wool spots and intraretinal haemorrhages, together with macular oedema.

Figure 2 Fundus fluorescein angiography demonstrated areas of capillary drop out and retinal non-perfusion at the posterior poles.

5 days, the patient developed acute renal failure, and was subsequently, transferred to the intensive care unit following a cardiac arrest. Autoimmune screen and test for parasitic, viral infections including HIV were negative. Renal biopsy showed evidence of microangiopathy. Blood test revealed anaemia, thrombocytopenia, and deranged clotting function. A blood film showed fragmentation of red blood cells. A diagnosis of TTP was made, and treatment with plasmapheresis, haemodialysis, and intravenous hydrocortisone was commenced. The patient recovered without the need for further dialysis.

Discussion

The ophthalmic findings in this case are consistent with a Purtscher's retinopathy. There are a small number of reported cases of this rare ocular manifestation of TTP in the literature.^{3,4} However, what is unusual about this case

is that the patient presented with retinopathy with no associated systemic abnormality, but soon began to deteriorate and became rapidly ill within a few days. The presence of this Purtscher-like retinopathy should alert ophthalmologists and physicians to the presence of an underlying systemic microangiopathy with multiple organ involvement. Complete haematologic and metabolic workup should be initiated promptly. Renal biopsy and early treatment with steroids or immunosuppression may be useful even in the absence of systemic illness, and may reduce mortality from this rapidly fatal condition.

References

- 1 Moschowitz E. An acute pleiomorphic anaemia with hyaline thrombosis of the terminal arterioles and capillaries. An undescribed disease. *Arch Intern Med* 1925; **36**: 89–93.

- 2 Jellie HG, Gonder JR, Canny CLB, Arce FP, Kaufmann JC. Ocular involvement in thrombotic thrombocytopenic purpura; the angiographic and histopathological features. *Can J Ophthalmol* 1984; **19**: 279–283.
- 3 Power MH, Regillo MC, Custis PH. Thrombotic thrombocytopenic purpura associated with purtscher retinopathy. *Arch Ophthalmol* 1997; **115**(1): 128–129.
- 4 Patel MR, Bains AK, O'Hara JP, Kallab AM, Marcus DM. Purtscher retinopathy as the initial sign of thrombotic thrombocytopenic purpura/hemolytic uremic syndrome. *Arch Ophthalmol* 2001; **119**(9): 1388–1389.

T Ong, W Nolan and J Jagger

Ophthalmology Department, Royal Free Hospital
London NW3, UK

Correspondence: T Ong
Tel: +44 77 60273918
Fax: +44 208 692 9111
E-mail: tuyen.ong@doctors.org.uk

Eye (2005) **19**, 359–361. doi:10.1038/sj.eye.6701491
Published online 1 October 2004

Sir,
Calcification of Aqua-Sense intraocular lenses

We read with interest the article by Izak and co-workers, reporting clinical and pathological features of hydrophilic acrylic intraocular lenses (IOLs) of three major designs explanted because of late postoperative opacification.¹

These authors analysed eight Aqua-Sense IOLs (Ophthalmic Innovations International, OII). Five of them had been explanted in South Africa, two in the UK, and one in Brazil. According to the paper, a total of 23 cases of postoperative opacification of the Aqua-Sense lenses were observed by Dr Troskie in South Africa, and the manufacturer of the Aqua-Sense lens has apparently reported 12 similar cases to the authors in a personal communication in September 2001.¹

In the University Hospital Aintree, Liverpool, UK, we have exchanged 25 opacified Aqua-Sense lenses between August 2001 and July 2003. The initial Aqua-Sense IOL implantations were performed in the year 2000 and early 2001. We summarised our experience with the exchange of these lenses in a paper that we submitted recently for consideration of publication in the *Journal of Cataract and Refractive Surgery*.

A further nine Aqua-Sense lenses developed severe late opacification but have not been exchanged so far for various reasons, bringing the total number of opacified Aqua-Sense lenses observed in our department to 34. Thus, the overall number of opacified Aqua-Sense lenses might be higher than suggested in the article and we are wondering whether other colleagues have exchanged or observed opacified Aqua-Sense lenses.

Acknowledgements

Proprietary interests: Research funding: none.

References

- 1 Izak AM, Werner L, Pandey SK, Apple DJ. Calcification of modern foldable hydrogel intraocular lens designs. *Eye* 2003; **17**: 393–406.

E Dages, MA Khan, GM Kyle and D Clark

Department of Ophthalmology, University Hospital
Aintree, Liverpool, UK

Correspondence: E Dages
Tel: +44 7811 405836
Fax: +44 1515 294283
E-mail: edages@yahoo.com

Eye (2005) **19**, 361. doi:10.1038/sj.eye.6701502
Published online 30 July 2004

Sir,
Bilateral, multiple choroidal effusions after vomiting

Choroidal effusions occur secondary to local changes such as hypotony, surgery, or inflammation. Primary uveal effusion may also occur with nanophthalmos or scleral abnormalities.¹ Acute choroidal effusion has been described in association with local or systemic predisposing factors.^{2–6} We present a patient with acute, bilateral choroidal effusions related to an episode of vomiting alone.

A 64-year-old man presented with a 2-day history of painless visual loss in the left eye noticed the morning after an episode of vomiting. Other than type II diabetes mellitus, his past medical and ocular histories were unremarkable. Visual acuity with low myopic correction was 6/6 in the right eye and 6/36 in the left. Examination of the ocular adnexae, anterior segments, and intraocular pressure was normal. Dilated fundal examination revealed multiple, shallow choroidal effusions in the