Subsequently, *Stenotrophomonas maltophilia* was grown, and after consultation with the infectious diseases unit, ceftazidime 2 mg was given intravitreally. Intravenous sulphamethoxazole 240 mg/trimethoprim 1.2 g t.d.s. was substituted for the vancomycin when the culture and sensitivities were completed.

Post-operatively, the retina remained flat with the aqueous cells clearing and the vitreous gradually becoming less hazy. At about 1 month post-operatively the patient was discharged back to the provincial centre. At this stage, the macula looked healthy with no oedema and visual acuity in the right eye was count fingers. Six months later, best corrected visual acuity in the right eye had improved to 6/36.

Comment

Stenotrophomonas maltophilia is primarily a free-living, aerobic plant pathogen that is also isolated in soil and water. Of particular note is its isolation on contact lenses and lens care systems.[2] Previously known as *Pseudomonas maltophilia* and then *Xanthomonas maltophilia*, it has been documented as the causative agent in conjunctivitis, corneal ulceration in contact lens wearers, keratitis (including in a corneal graft), infected scleral buckle, orbital cellulitis, dacryocystitis, preseptal cellulitis and endophthalmitis.[2–4]

In ocular infections and elsewhere this organism is becoming a more important nosocomial pathogen, particularly in the debilitated and immunosuppressed. Difficulty in distinguishing between colonisation and infection by *S. maltophilia* fostered the belief, particularly in the early days after its discovery, that it had limited pathogenicity.[2] Since its first description in 1960, it has shown a steady increase in its isolation rate in diagnostic laboratories, now being the second most common pseudomonad (after *Pseudomonas aeruginosa*) found in clinical specimens.[2]

Intralenticular abscess is an extremely uncommon clinical infection. The route by which the organism gained access to the lens capsule is obscure; however, risk factors would be the previous iritis, and the bacteraemia associated with recent childbirth. The patient denied and there was no evidence of ocular penetration or any intraocular injection of drugs.

Penicillins and cephalosporins are generally agreed to exhibit poor activity against *S. maltophilia*; however, ceftazidime has been shown to possess reasonable activity against it.[1] Susceptibility of this organism to fluoroquinolones is variable.[2] Trimethoprim–sulphamethoxazole has been found to be active against most strains of the bacterium, and in some cases may be an adequate treatment for this type of infection. Choice of antibiotic should be strongly based on culture and sensitivity results on a case-by-case basis. The authors were fortunate to be able to consult infectious diseases colleagues in order to individualise treatment for this organism with highly variable antimicrobial susceptibilities.

This case highlights the increasing context of this opportunistic organism and patterns of resistance that can make treatment difficult.

References

1. Denton M, Kerr KG. Microbiological and clinical aspects of infection associated with *Stenotrophomonas maltophilia*. Clin Microbiol Rev 1998;11:57–80.
2. Penland RL, Wilhelmus KR. *Stenotrophomonas maltophilia* ocular infections. Arch Ophthalmol 1996;114:433–6.
3. Khater TT, Jones DB, Wilhelmus KR. Infectious crystalline keratopathy caused by gram-negative bacteria. Am J Ophthalmol 1997;124:19–23.
4. Chen S, Stroh EM, Wald K, Jalkh A. *Xanthomonas maltophilia* endophthalmitis after implantation of sustained-release ganciclovir. Am J Ophthalmol 1992;114:772–3.

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Sir,

Transient phacodonesis after circling buckle procedure for rhegmatogenous retinal detachment

The pathomechanisms involved in producing phacodonesis have not all been firmly established. We present a patient who was admitted to our department due to severe ocular pain 9 days after a circling buckling procedure for rhegmatogenous retinal detachment in that eye. Ocular examination revealed severe phocodonesis, with ciliary body effusion on ultrasound biomicroscopy. Systemic steroid therapy resulted in prompt resolution of ocular pain with reabsorption of ciliary effusion as well as disappearance of the phacodonesis.

Case report

A 35-year-old highly myopic (–11.00 dioptre) male was evaluated due to visual deterioration in his right eye, which had a corrected visual acuity of counting fingers. The anterior segment was normal. Funduscopy revealed inferior macula-off retinal detachment.

The right eye underwent a circling buckling procedure of 360° and drainage of subretinal fluid, after which visual acuity was 1/36 and intraocular pressure was 10 mmHg, with no other change in the anterior segment. Funduscopy revealed a high 360° buckle indentation and an attached retina. The patient was discharged with topical steroids and atropine 1% twice daily.

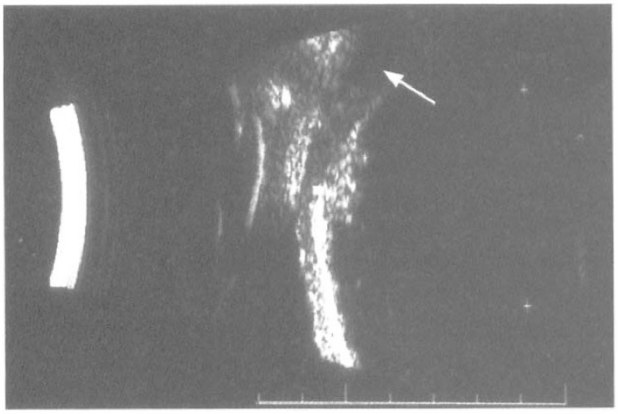

Fig. 1. *UBM showing ciliary body effusion (arrow).*

Nine days post-operatively, he experienced pain in his right eye and was readmitted 3 days later. Visual acuity was 1/36 and the intraocular pressure was 16 mmHg. The anterior chamber was deep and severe phacodonesis was noted for the first time. Funduscopy revealed an attached retina and no choroidal detachment. Ultrasound biomicroscopy (UBM) (Fig. 1) showed ciliary body effusion but with no pupillary block. Oral prednisolone 100 mg treatment was initiated. Four days later there was complete relief of ocular pain and disappearance of the phacodonesis. UBM showed resolution of the ciliary body effusion (Fig. 2). The dosage of corticosteroids was gradually tapered, with no recurrence of pain.

Comment

We present a patient in whom severe pain and severe phacodonesis with ciliary body effusion, which occurred 9 days after a circling buckling procedure for rhegmatogenous retinal detachment, completely resolved with systemic steroid treatment.

Ciliary effusion and ciliary body thickening may be seen after scleral buckling procedures and can cause angle closure by anterior iris rotation and pupillary block.[1,2] A possible pathogenic mechanism is congestion and swelling of the ciliary body due to a temporary interference with venous drainage by the scleral buckle.[3] Nagahara *et al.*[4] showed recently that buckling procedures with encircling elements decreased tissue blood velocity in the choroid and retina. Maruyama *et al.*[5] evaluated changes in the ciliary body and anterior choroid after retinal detachment surgery, using an ultrasound biomicroscope. They found that ciliary detachment occurred frequently after scleral buckling procedures, that it lasted for at least 2 weeks and resolved within 2 months. Topilow and Ackerman[6] described three aphakic patients who underwent successful scleral buckling surgery for unilateral rhegmatogenous retinal detachment who developed severe ocular pain with massive exudative retinal and choroidal detachments within 2 weeks post-operatively. Prednisone therapy led to resolution of ocular pain and reabsorption of subretinal and suprachoroidal fluid.

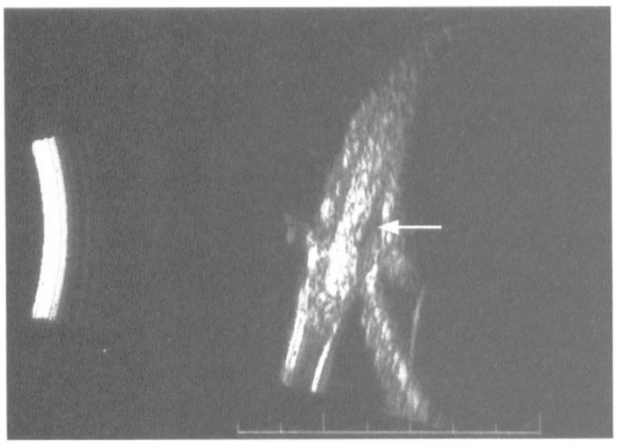

Fig. 2. *UBM showing resolution of ciliary body effusion (arrow).*

Our patient suffered severe ocular pain 9 days after a buckling procedure. There was no visual deterioration, but significant phacodonesis was noted and ciliary body effusion without choroidal effusion in the operated eye was revealed by UBM. We interpret our findings to support Pavlin *et al.*,[1] that reduced venous drainage with congestion of the ciliary body may cause ciliary body detachment with stretching of zonules and lead to phacodonesis. The severe ocular pain could possibly be explained by anterior segment ischaemia, although there were no specific clinical signs and the rapid resolution of symptoms is uncommon. We believe that systemic steroids may play a role in reducing this ciliary congestion with prompt resolution of phacodonesis.

References

1. Pavlin CJ, Rutnin SS, Devenyi R, Wand M, Foster FS. Supraciliary effusions and ciliary body thickening after scleral buckling procedures. Ophthalmology 1997;104:433–8.
2. Berler DK, Goldstein B. Scleral buckles and rotation of the ciliary body. Arch Ophthalmol 1979;97:1518–21.
3. Perez RN, Phelps CD, Burton TC. Angle-closure glaucoma following scleral buckling operations. Trans Am Acad Ophthalmol Otolaryngol 1976;81:247–52.
4. Nagahara M, Tamaki Y, Araie M, Eguchi S. Effects of scleral buckling and encircling procedures on human optic nerve and retinochoroidal circulation. Br J Ophthalmol 2000;84:31–6.
5. Maruyama Y, Yuuki T, Kimura Y, Kishi S, Shimizu K. Ciliary detachment after retinal detachment surgery. Retina 1997;17:7–11.
6. Topilow HW, Ackerman AL. Massive exudative retinal and choroidal detachments following scleral buckling surgery. Ophthalmology 1983;90:143–7

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