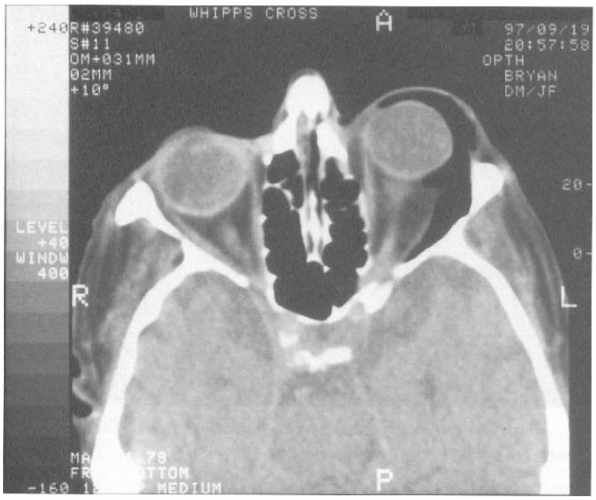

Fig. 1. *Axial CT scan through the orbits showing left-sided proptosis with elongation of the optic nerve, intraorbital emphysema and fracture of the left nasal bone.*

orbital compartment to forward movement of the globe. As the limits of spontaneous decompression are surpassed, the pressure is transmitted directly to the orbital structures. The small vessels supplying the optic nerve are more sensitive to orbital pressure than the larger central retinal artery, but may withstand longer compression without permanent visual loss.[6] This mechanism probably accounts for the reduced vision in our case, where no signs of retinal ischaemia were observed.

Management of cases with significant discomfort or visual impairment includes an urgent orbital CT scan both to confirm the diagnosis and to localise the air mass. The air does not stay predictably at any single site within the orbit, but its position does correlate well with the location of the orbital fracture.[7] Air that enters the orbit but remains subperiosteal can have a more transient effect on vision, most likely due to the lack of ball-valve effect from the orbital fat.[8]

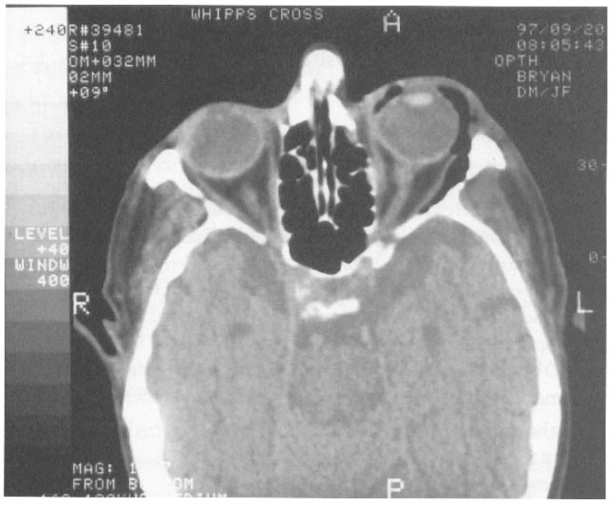

Fig. 2. *Axial CT scan at the same level 10 h later showing reduced proptosis and air mass.*

Systemic absorption of an intraorbital air mass can take up to 7 days[9] and patients with impaired visual function therefore require immediate orbital decompression. Treatment using a needle-coupled syringe is effective and relatively safe. A modification to this technique, however, is the addition of normal saline to an open syringe which then allows the monitoring of air bubbles released and avoids potential suction of orbital structures into the needle tip.[7] In cases with severe visual loss a lateral canthotomy and cantholysis may be appropriate.[4]

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Sir,

Successful treatment of saccular endophthalmitis with clarithromycin

A 78-year-old patient underwent extracapsular cataract extraction in 1995 when an intra-ocular lens (6.5 mm PMMA optic and PMMA type of haptic) was implanted. There were no post-operative complications and visual acuity was maintained at 6/6.

A trabeculectomy was performed in that eye in November 1997 for open angle glaucoma as the intraocular pressure could not be controlled with dipivefrine and timolol drops. The anterior chamber had become flat post-operatively and this was managed with a pressure pad. On the fifth post-operative day the anterior chamber was still shallow and there was a suspicion of uveal tissue seen through the thin superficial

scleral flap. Donor sclera was arranged. The patient was taken to theatre on the seventh post-operative day and the wound was explored. It was found that the superficial scleral flap had given way. This was reinforced with a donor scleral flap.

After 5 days there was fibrin in the anterior chamber both anterior and posterior to the intra-ocular lens. This was thought to be due to infection and a regime of oral ciprofloxacin 500 mg b.d. and topical gentamicin 1.5% was initiated. In addition, topical prednisone forte was given hourly with systemic prednisolone 60 mg per day. After 1 week there was still a prolific fibrinous response with no reduction in inflammation. The remaining lens capsule around the intraocular lens appeared thickened and white (Fig. 1) and a clinical diagnosis of saccular endophthalmitis was made.[1–3]

Treatment was commenced with clarithromycin (Klaricid, Abbott) 500 mg b.d. by mouth for 1 week. Within 3 days the visual acuity had increased from hand movements to 6/60. However, the intraocular pressure increased to 36 mmHg as the patient was found to be a 'steroid-responder'. This required stopping both systemic and topical corticosteroids and commencing latanoprost drops o.d. at night. The patient continued on clarithromycin 250 mg b.d. for a total of 4 weeks. The patient made good progress, the corneal oedema reduced and the visual acuity returned to 6/6 over the following 6 weeks. No recurrence of inflammation has occurred 4 months later and the pressure has been controlled with latanoprost.

At the end of clarithromycin therapy, an anterior chamber tap was performed with a syringe and needle, by a technique that was as sterile as possible, and 0.1 ml was removed into a sterile bottle that was immediately frozen to −20 °C. This was dispatched to Dr C. R. Lohmann (Eye Clinic) and Drs L. Naumann and U. Reischl (Institute of Hygiene) of the University of Regensburg, Germany, for testing for the presence of bacteria by the polymerase chain reaction (PCR). They carried out PCR-based amplifications with different sets of primers and performed DNA sequencing of the amplification products. Their PCR test for fungal DNA was negative but that for bacteria was positive. Their subsequent DNA sequencing suggested a degree of homology with 16S rDNA of *Pseudomonas putida* but this is considered an unlikely pattern as that organism is resistant to clarithromycin.

Comment

This case demonstrates that systemic clarithromycin, by the oral route, penetrates satisfactorily into the inflamed anterior chamber to treat acute saccular endophthalmitis. This drug is further potentiated in its anti-bacterial action by being concentrated intra-cellularly within polymorphonuclear cells and macrophages – an important means of combating infection that does not occur with the penicillins (beta-lactams) or aminoglycosides.[3] This is especially important in

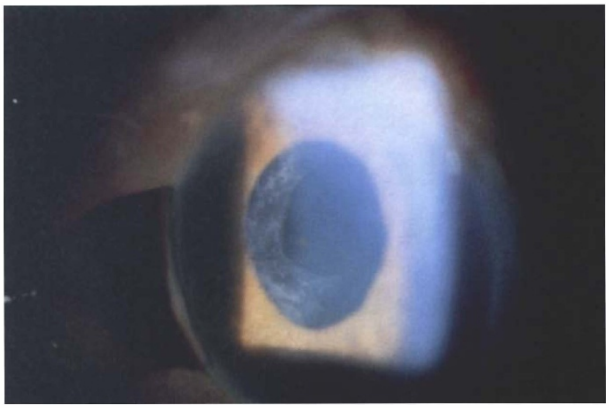

Fig. 1. *Fibrin both anterior and posterior to the intraocular lens and thickened capsule around the implant.*

saccular endophthalmitis where there is good evidence, at least in chronic inflammatory disease, that this can be due to macrophage-associated bacteria.[2]

Future management of saccular endophthalmitis, whether acute or chronic, should involve an anterior chamber tap under sterile conditions when 0.1 ml of aqueous can be safely removed. One drop (0.025 ml) should be instilled into a fluid enrichment medium, such as brain–heart infusion broth, for bacterial and fungal culture with prolonged incubation for 4 weeks, and the other 0.075 ml should be frozen at −20 °C for future molecular-based investigations such as those carried out in Regensburg. Either of the two new azilide derivatives of erthyromycin – clarithromycin or azithromycin – should be started for a 'trial-of-therapy' at the maximum oral systemic dose, since saccular endophthalmitis associated with an intraocular lens is usually due to Gram-positive bacteria.[3] A failure to respond should raise the rare possibility that the infection is due to a Gram-negative bacterium. Enrichment culture is not always successful when the infection has become chronic and been suppressed but not eradicated by corticosteroids, as the bacteria will have become intracellular.[2] It is in this situation that PCR-based molecular diagnosis becomes particularly useful.

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Sir,

Post-operative saccular endophthalmitis caused by macrophage-associated staphylococci

An 83-year-old healthy man underwent extracapsular cataract extraction with placement of an intraocular lens (IOL) implant (PMMA) into the capsular bag. Pre-operative preparation included topical aqueous chlorhexidine. The incision was in the superior limbus, of size 14 mm. Surgery lasted 30 min. The patient received no antibiotic prophylaxis except Maxitrol drops (Alcon; dexamethasone 0.1%, neomycin 0.35% and polymyxin B 6000 U/ml) post-operatively.

On the first post-operative day the visual acuity (VA) was 6/60, which improved to 6/12 with a pinhole. The anterior chamber was quiet when a good view of the posterior pole was obtained. By the tenth day VA was 6/60, which improved to only 6/36 with a pinhole. There were a few cells in the anterior chamber suggesting mild anterior uveitis.

On the twenty-seventh day the patient complained of pain and impaired vision. VA was 6/36 with pinhole. There were keratic precipitates, cells and fibrin in the anterior chamber. Deposits on the IOL indicated an increase in the severity of the uveitis. The patient received topical dexamethasone 0.1% hourly and cyclopentolate 0.5% t.d.s.

After 49 days VA had dropped to < 6/60 with thickening of the capsular bag. There was fibrinous exudate in the anterior chamber but the fundal glow was reasonable. The patient was treated additionally with oral ciprofloxacin 500 mg b.d. for 2 weeks. The anterior chamber reaction reduced but VA did not recover because of capsular thickening. Vitreous tap failed to yield any bacteria.

Further inflammation occurred at 20 weeks, with VA reduced to perception of light, with increased keratic precipitates. Again the patient was treated with corticosteroids which resulted in reduced inflammation but no improvement in the VA.

The patient developed severe pain with hypopyon at 32 weeks (Fig. 1A). Faint fundal glow was visible. There was no relative afferent pupillary defect. Ultrasound examination showed clear vitreous with no evidence of retinal detachment. Anterior chamber tap was performed twice for investigation (Gram stain with culture) but no organisms were identified. A clinical diagnosis of saccular (within the 'bag') endophthalmitis was made and removal of the IOL was planned. The patient was given intravitreal vancomycin 1 mg together with oral co-trimoxazole, topical cefuroxime and further corticosteroids.

At 38 weeks, following resolution of the hyopyon with corticosteroids, the IOL and capsular bag were removed. Post-operatively the patient made a good recovery with a quiet, non-inflamed eye but retained little vision, due to a retinal detachment involving the macula.

Ocular pathological findings

The capsular fragment (Fig. 1B) was fixed in 1% glutaraldehyde for staining with haematoxylin and eosin (H&E) and Gram's stain and for electron microscopy. The IOL and another piece of the capsular fragment were placed in thioglycollate broth for culture.

Light and electron microscopic findings

The low-power view of the capsular fragment stained with PAS confirms the presence of lens material and the surrounding capsule (Fig. 1C). The high-power view (H&E) demonstrates many viable macrophages within the capsular fragment (Fig. 1D).

The low-power view stained with Gram's stain shows the presence of uniformly staining Gram-positive cocci within the macrophages but not extracellularly (Fig. 1E). The high-power view demonstrates morphology suggestive of actively multiplying coagulase-negative staphylococci (CNS) due to the large numbers of bacteria present within the cells (Fig. 1F).

Transmission electron microscopy (TEM) revealed macrophages within the capsular fragment (Fig. 1G) and, at high power, multiple intracellular bacteria with coccal morphology (Fig. 1H).

Comment

Late-onset pseudophakic endophthalmitis typically presents several months after cataract surgery, often as a hypopyon uveitis that has failed to respond to corticosteroids.[1] The term 'saccular endophthalmitis' denotes chronic uveitis associated with thickening of the lens capsule around the plastic IOL. It has been proposed (J. Dart, personal communication) that the causative bacteria are in a biofilm around the IOL and are consequently resistant to antibiotics. Progress of the disease may be responsive to treatment with high-dose cephalosporins given intravenously or intravitreally but often there is need for aggressive adjunctive surgery with removal of the IOL as well as excision of the remaining capsule.[1] The bacterium recorded most often is *Propionibacterium acnes*, but CNS have also been isolated. Inexplicably, however, bacteria have not been isolated on approximately 50% of occasions when the polymerase chain reaction (PCR) test has demonstrated their presence.[2,3] This typical clinical case, requiring removal of the IOL and capsular fragment, failed to yield bacterial growth from three taps (two anterior chamber and one vitreous).