PostScript

LETTERS

Cyclodiode treatment of neovascular glaucoma secondary to Coats' disease

Coats' disease is characterised by an idiopathic retinal telangiectasia and exudative retinopathy, which may lead to retinal detachment.¹⁻³ In 10% of eyes, neovascular glaucoma may occur and these eyes have been treated with primary enucleation.^{2,4} Although enucleation is an effective means of relieving severe ocular pain, it may remove an eye with some visual potential and postoperative complications occur in approximately 50% of cases.^{5,6} We report the first use of cyclodiode laser treatment in three consecutive patients with neovascular glaucoma secondary to advanced Coats' disease.

Method

Three patients with neovascular glaucoma secondary to Coats' disease presented to the Glaucoma Service at Moorfields Eye Hospital, London, UK, between 2004 and 2005. Table 1 summarises the characteristics of the patients.

Cyclodiode technique

All three patients underwent trans-scleral diode laser cyclophotocoagulation (cyclodiode) using the contact G-probe (Iris Medical instruments, Mountain View, California, USA) under general anaesthesia. The ciliary body was identified by transillumination and the cyclodiode probe placed 1 mm behind the ciliary body. The treatment consisted of 30-40 applications of 1000-1500 mW energy applied for 1.5 s over 270° sparing the 3 and 9 o'clock regions. Each treatment was started with 1500 mW; if a "pop" was heard during laser application, the power was reduced in 100 mW steps. The standard application was 40 shots with 10 shots in each quadrant; one patient had only 30 shots as the eye was small in size. Subconjunctival betamethasone was given immediately after laser treatment, and the patients were given 1% prednisolone and 0.5% chloramphenicol four times a day. All patients were reviewed at 1 week, 1 month and 3 months after treatment, and drugs for glaucoma were withdrawn.

Results

The intraocular pressure (IOP) was lowered successfully in all three patients by a single treatment of trans-scleral cyclodiode, with a

mean follow-up time of 8 months and there were no complications of hypotony, uveitis, hyphaema or vitreous haemorrhage (table 1).

Discussion

Coats' disease is asymptomatic in the early stages and 50% of cases⁷ may progress with subretinal lipid deposition leading to retinal detachment.^{2,4} Neovascular glaucoma develops in approximately 10% of eyes and is a poor prognostic sign.^{2,3} The principal treatment of neovascular glaucoma in Coats' disease has been enucleation.⁴ Historically, patients underwent enucleation as retinoblastoma could not be excluded; however, modern imaging techniques, including ultrasonography, CT and MRI, make this unnecessary.⁸ Although enucleation is effective at eliminating pain, it may remove an eye with some visual potential and has a complication rate of 50%, including extrusion of the implant, socket contraction and marked socket discharge.^{5,6}

Cyclodiode laser has been established as a relatively safe treatment for refractory glaucoma. The technique is safer than other cyclodestructive procedures such as YAG (yttrium-aluminum-garnet) laser cyclophotocoagulation and cyclocryotherapy. Single cyclodiode treatment is effective in 60-90% of eyes, achieving an IOP of 5-21 mm Hg or a 30% lowering of IOP,^{9,10} although the response to a single treatment may be unpredictable, and multiple treatments may be required in the long term.⁹ The three patients we reported remained medication free at 6-12 months of follow-up; however, further treatments may be required in the long term. Cyclodiode laser has been reported to have adverse events of hypotony and phthisis in 10% of treatments; however, with low-energy treatments these complications may be reduced.

The conventional treatment of neovascular glaucoma secondary to Coats' disease with enucleation is invasive and associated with postsurgical complications. Cyclodiode laser was effective in lowering IOP in our three consecutive cases, and we propose its use as an effective and relatively safe technique.

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doi: 10.1136/bjo.2006.097121

Accepted 27 October 2006

Competing interests: None declared.

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Table 1 Summary of patients' characteristics

	Patient 1	Patient 2	Patient 3
Age	6 months	17 months	6 years
Ethnicity	Asian	Caucasian	Asian
Sex	Male	Female	Male
Visual acuity	No fixation	Poor fixation	No fixation
Leukocoria	Yes	No	No
Strabismus	No	Small left exotropia	No
Anterior chamber	Normal	Normal	Small hyphaema
Retinal vessels	Extensive retinal telangiectasia	Extensive retinal telangiectasia	No retinal view
Retinal exudation	Extensive macular exudation	Subretinal macular exudation	
Retinal detachment	Inferotemporal and macular retinal detachment	Total retinal detachment	Total retinal detachment
IOP	71	72	52
Gonioscopy	Neovascular glaucoma, secondary narrow angle	Neovascular glaucoma, secondary narrow angle	Neovascular glaucoma
Timing of neovascular glaucoma/months after presentation	4	10	0
Coats stage ⁴	Stage 3A2: subtotal exudative retinal detachment with foveal elevation	Stage 3B: total exudative retinal detachment	Stage 4: total retinal detachment
Cryotherapy	No	1x cryotherapy of two quadrants	No
Ultrasonography	Posterior inferotemporal retinal detachment	Thickened + fibrotic total retinal detachment	Thickened funnel-shaped retinal detachment
Cyclodiode	30 burns, 1000-1500 mW, 1500 ms	40 burns, 1000-1500 mW, 1500 ms	40 burns, 1200-1500 mW, 1500 ms
Post-Cyclodiode	IOP 15	IOP 11	IOP 18
Follow-up period	12 months	6 months	6 months
Drugs for glaucoma medications	0	0	0

IOP, intraocular pressure.

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Clostridium novyi keratitis

Anaerobic bacteria are rarely implicated in corneal infections. In our laboratory, only 3 of a series of 432 bacterial isolates from the cornea were anaerobes (*Clostridium perfringens*, *Peptostreptococcus anaerobius*, *Propionibacterium acnes*). This fraction is not unusual. The value of anaerobic culture for corneal specimens may be open to question, but we recently had a patient in whom the diagnosis would not otherwise have been made.

Case report

A 73-year-old man presented to a general practitioner. He had received a corneal laceration when struck by an Australian magpie. He was given chloramphenicol 1% ointment, but experienced increasing pain, redness and visual loss over the next 2 days. The patient was referred to Flinders Medical Centre, South Australia, Australia. A partial thickness, 4-mm-long shelving wound was seen across the visual axis. It was associated with a large grey infiltrate. Corneal oedema, Descemet's folds, and cells in the anterior chamber were also present. Visual acuity was perception of hand motions.

Scrapings were taken, and media were inoculated for bacterial, fungal and viral investigations. Chloramphenicol was discontinued, and cefazolin 5% and gentamicin 1% drops were given hourly, as is our standard practice.¹

Gram stain showed polymorphonuclear cells and Gram-positive bacilli. The organism appeared on blood agar after 48 h of incubation at 35°C in an anaerobic chamber. A clostridial species was suspected, but a reverse-CAMP test suggested that it was something other than *C. perfringens*, the species isolated most commonly from tissue.² Tests for fungi and herpes viruses were negative.

The patient's condition stabilised and then improved steadily. The drops were reduced to 4-hourly after 5 days, and gentamicin was stopped 3 days later. After another 3 days, the patient's visual acuity was 6/24, and the treatment was changed to chloramphenicol ointment and fluorometholone 0.1% drops twice daily. One month after referral, the patient had a residual scar with minimal shelving. Treatment was stopped.

The isolate swarmed over the agar, was a strict anaerobe and had cells with subterminal swellings. In speciation tests, lecithinase and lipase activities were detected. No reactions were seen in a rapid ID32A strip (BioMérieux, Marcy l'Etoile, France), so DNA sequencing was performed. Cells were incubated in lyso-

zyme (1 mg/ml) for 1 h at 37°C, and DNA was purified using a Wizard Genomic Kit (Promega, Annandale, New South Wales, Australia). The 5' region of the 16S rRNA gene was amplified using universal primers 27F³ and 787r.⁴ The reaction mixture included 2 µl of DNA and 0.4 µM of each primer in a final volume of 50 µl. Amplification conditions were 94°C for 3 min, 30 cycles of 94°C for 30 s, 60°C for 45 s and 72°C for 1 min, and a final extension at 72°C for 5 min. The product (707 bases) was purified using GeneClean (QBIogene, Carlsbad, California, USA), sequenced using BigDye Terminator V.3.1 in an ABI Prism 3100 Analyzer (Applied Biosystems, Foster City, California, USA) and compared with sequences in GenBank using FASTA.⁵ It was identical to four strains of *C. novyi* from three laboratories, including the type strain of *C. novyi* type A (ATCC 17861).⁶ The next closest matches, from strains of *C. botulinum*, differed by 14 bases.

Comment

The Australian magpie (*Gymnorhina tibicen*) is a common bird whose carolling is evocative of Australian life. Although generally well regarded, they become aggressive to humans when nesting. The eye is involved in 20% of presentations to hospitals after attacks.^{7,8} *C. novyi* has been cultured from soil and animals,⁶ and we suspect that the magpie was the source of infection in our patient.

C. novyi is an agent of gas gangrene. With respect to the eye, it has been isolated from conjunctival discharge in a girl with anophthalmia.⁹ Corneal infection has not been reported. Type A strains produce γ -toxin (lecithinase), δ -toxin, ϵ -toxin (lipase), and the lethal α -toxin.¹⁰

In general, clostridia are susceptible to chloramphenicol, have variable susceptibility to cephalosporins and are resistant to aminoglycosides. The high antibiotic concentrations achievable in the cornea with fortified drops may have contributed to the good outcome here. Other antibiotics that can be given topically and may be useful in clostridial keratitis include penicillin G and clindamycin.²

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doi: 10.1136/bjo.2006.097774

Accepted 19 July 2006

Competing interests: None.

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Drug-induced cicatrising granulomatous conjunctivitis

Ocular toxicity resulting from chemicals in eye drops is common. Preservatives such as benzalkonium chloride (BAC) and related quaternary ammonium salts have been shown to play an important part in these reactions.¹ The symptoms can range from mild injection and itching to severe conjunctival reactions. The term pseudophemphigoid has been applied to drug-induced cicatrising conjunctivitis.²⁻⁵ We present a patient who developed drug-induced granulomatous cicatrising conjunctivitis secondary to glaucoma drops.

Case report

A 60-year-old man presented to the eye clinic for ocular irritation. His ocular history was positive for glaucoma, for which he was using latanoprost (Xalatan, Pfizer, New York, New York, USA), and brimonidine 0.2% (a generic preparation). Examination showed severe conjunctival injection bilaterally, as well as symblephara in the lower eyelid (fig 1A). A conjunctival biopsy showed non-caseating granulomatous inflammation within the substantia propria (fig 1B). The differential diagnosis for granulomatous conjunctivitis includes sarcoidosis and infections.⁶ Sarcoid investigation by rheumatology was negative, and the patient did not have a history or symptoms of an infection.

He returned to clinic, at which time an allergic reaction was suspected. Initially, brimonidine was discontinued, followed by latanoprost when the injection did not resolve. The patient was treated with prednisolone acetate 1%, which brought about a resolution of the conjunctival injection and irritation.

To control his intraocular pressure, different glaucoma drops were tried, but the conjunctival injection and irritation quickly resumed each time. These drops included timolol 0.5%, brinzolamide 1% (Azopt, Alcon, Fort Worth, Texas, USA), dorzolamide 2% (Trusopt, Merck, Whitehouse Station, New Jersey, USA), bimatoprost 0.03% (Lumigan, Allergan, Irvine, California, USA) and pilocarpine 1%. Each time, discontinuing the eye drops and prescrib-