she had an inferior corneal scar sparing the visual axis and an early cataract. Final visual acuities were right 6/9 and left 6/12.

Comment

Worldwide, an estimated 260 000 children have severe visual impairment from corneal causes. This is the most important cause of avoidable worldwide childhood blindness.[2] Most of this occurs in 'low-income countries', in particular, those in Sub-Saharan Africa, India, and other parts of Asia There are, however, case reports of vitamin A deficiency presenting in developed communities. Inadequate diet is one mechanism. In one case a 7-year-old vegetarian British girl presented with Bitot's spots.[3] In another case, a 6-year-old child presented with xerophthalmia secondary to anorexia from severe gastro-oesophageal reflux and oesophagitis.[4] Malabsorption syndromes, particularly those affecting the terminal small intestine where vitamin A is absorbed, can also result in vitamin A deficiency. In one case, a child with short bowel syndrome presented with xerophthalmia.[5] Large intestine pathology is a less likely cause, although it can occur and has been reported in a patient who underwent a haemicolectomy.[6] In our cases, the patient's chosen diet was the cause of the deficiency. They presented with xerophthalmia complicated by microbial keratitis.

Both patients' ERG s showed evidence of significant retinal involvement with subsequent partial recovery after vitamin A supplementation. In keeping with other studies,[7] the rod-mediated ERG was more affected, but implicit times were unchanged. This reduction in ERG amplitude is said to correlate with reduced levels of the rod pigment, rhodopsin, resulting from the reduction in blood retinol.

It is important to consider vitamin A deficiency in the differential diagnosis of any 'ocular surface presentation' in developed communities with features of surface drying. In both cases, the patients presented with microbial keratitis, without any underlying ocular associations. Poor dietary history combined with conjunctival and corneal drying, in theses cases, suggested the diagnosis of vitamin A deficiency. Nyctalopia is usually the earliest manifestation of the disease, although it does not occur in all cases.[6] Prompt recognition and treatment of vitamin A deficiency with oral supplements combined with treatment for microbial keratitis brought about an effective recovery.

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BJ Connell[1], AB Tullo[1], NRA Parry[1], L Brown[2], A Osman[3] and M Edwards[2]

[1]Manchester Royal Eye Hospital, Oxford Road, Manchester, UK

[2]Royal Hallamshire Hospital, Glossop Road, Sheffield, UK

[3]Manchester Royal Infirmary, Oxford Road, Manchester, UK

Correspondence: BJ Connell, Ophthalmology, Manchester Royal Eye Hospital, Oxford Road, Manchester M13 9WH, UK
Tel: +44 161 276 1234;
Fax: +44 161 276 5217.
E-mail: connellb@netspace.net.au

Eye (2006) **20,** 623–625. doi:10.1038/sj.eye.6701943; published online 3 June 2005

Sir,

Suprachoroidal haemorrhage: a rare complication of cyclodiode laser therapy

Suprachoroidal haemorrhage (SCH) is a severe complication of intraocular surgery. We report the first case of delayed SCH following cyclodiode laser therapy for refractory glaucoma.

Case report

The subject was a 72-year-old man with bilateral aphakia and secondary glaucoma. He had undergone multiple penetrating keratoplasties and trabeculectomies in both eyes. Previous cyclodidode laser therapy in the left

eye resulted in hypotony and a choroidal detachment, which resolved after 4 weeks. He had no significant past medical history and was not on any systemic medication.

Visual acuity was 6/12 in the right eye and count fingers in the left eye. Examination revealed extensive peripheral anterior synechiae, cupped discs (0.95), and advanced field loss in both eyes. Despite maximal medical therapy of latanoprost (Xalatan, Pfizer Inc.), dorzolamide hydrochloride-timolol maleate ophthalmic solution (Cosopt, Merck & Co., Inc.), and acetazolamide 250 mg (Diamox, Wyeth) bid, intraocular pressure (IOP) in the right eye remained at 24 mmHg. It was felt that IOP in the right eye had to be urgently lowered and cyclodiode laser (60 shots each of 2.0 W and 2.0 s to 360°) was performed under retrobulbar anaesthesia. Immediately postoperative, the subject was comfortable and discharged. However, a week later, the subject stated that pain and reduced vision was experienced in the right eye 4 days postlaser. There was blood in the anterior chamber with no view of the fundus and the IOP was 5 mmHg. B-scan ultrasound revealed a SCH (Figure 1). The subject was admitted for bed rest and topical steroids. No further haemorrhage occurred and the eye became comfortable again. At last follow-up, he remained blind in the right eye.

Comment

SCH has been associated with cataract extraction, penetrating keratoplasty, glaucoma procedures, and vitreoretinal surgery. Intraoperative or expulsive SCH can cause massive bleeding, resulting in the expulsion of intraocular contents. It has an incidence of 0.03% with phacoemulsification[1] and 0.15% in glaucoma filtering surgery.[2] Delayed or postoperative SCH occurs in a closed system and does not usually result in the expulsion of contents. The reported incidence of SCH is 1.6% after filtering surgery[3] and 6% after non-valved (Molteno) tube implantation.[4]

We report the first occurrence of delayed SCH following cyclodiode laser treatment. Risk factors for the patient were advanced age, glaucoma, aphakia and a previous episode of hypotony following cyclodiode laser in the contralateral eye. Other risk factors not present in this subject include hypertension, diabetes, myopia, pseudophakia, intraoperative hypertension with excessive drop in IOP, and ocular inflammation. Currently, there is no optimal treatment protocol for cyclodiode therapy and the dose–response is unpredictable. However, a recent study has suggested that high mean energy per treatment session may be associated with hypotony.[5] In this case, the higher dose of cyclodiode laser used may have increased the risk of hypotony and hence SCH.

The precise mechanism of SCH is unclear but it is believed that hypotony leads to a choroidal effusion, which then stretches and tears the short or long posterior ciliary arteries.[6]

Cyclodiode laser is increasingly used to treat refractory glaucoma. While it is considered a relatively safe procedure, our case shows that severe and devastating complications such as SCH can still arise. The use of lower total energy levels should be considered in the treatment of high-risk cases.

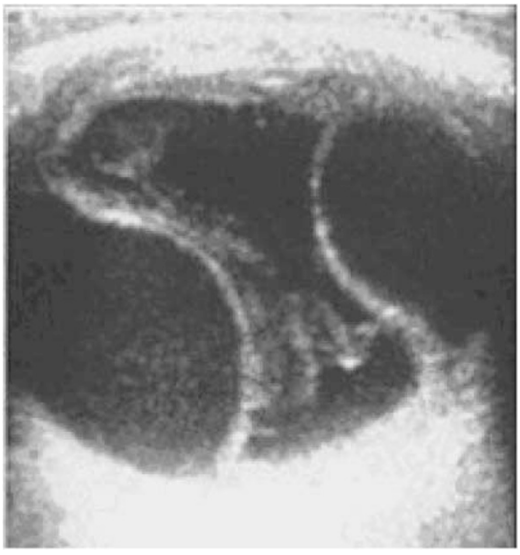

Figure 1 B scan showing bullous temporal SCH and bullous nasal choroidal effusion in the right eye. A posterior vitreous detachment with intragel opacities is present.

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E Tay[1], T Aung[1,2] and I Murdoch[1]

[1]Moorfields Eye Hospital, London, UK

[2]Singapore National Eye Centre, Singapore, Singapore

Correspondence: E Tay,
Tel: +44 207 253 3411.
E-mail: dr_eugenetay@yahoo.com

Meeting presentation: Presented at SERI-ARVO, Singapore, February 16–19, 2005.
Disclosure: None for all authors.
Conflict of interest: None for all authors.

Eye (2006) **20,** 625–627. doi:10.1038/sj.eye.6701948; published online 3 June 2005

Sir,

With regards to 'Endophthalmitis' (Editorial, Eye 2004, 18, 555–556)

Contemporary early cataract extraction for glare, blurring, and 'nuisance' vision, etc. together with the advent of clear lens replacement for high hypermetropia and hypermetropic with presbyopia (RSM Meeting 2003) makes the consequences of endophthalmitis even more important for the individual patient. Ms Seward pertinently remarks 'prevention remains the most important factor' and as described, the statistically valid evidence base for prophylaxis is still developing.

In the meantime an experience based and rational approach may contribute:

Use of the unique vancomycin as a prophylactic, risks resistance development in a potentially MRSA Hospital environment. In the eyes of nonophthalmologists, it may reduce ophthalmology to a rogue speciality. All prophylaxis transgresses one of medicines most accepted principles: 'treat effectively in dosage and duration to avoid resistance.'

The evidence would have to be extremely strong to justify its routine use. It is not.

Conjunctival sac iodine and intracameral cephuroxine, etc. may well reduce the incidence of endophthalmitis but how long do their effects last?

One of three eyes lost in the first 30 years of cataract surgery (AK) was due to *Streptococcal faecalis* in a padded patient who had soiled the bed from top to bottom overnight. Postoperative exogenous infection certainly occurs and not only from blepharitis, natural lid flora or a vulnerable conjuntival sac with a blocked tear duct: in the days of routine use of sutures many surgeons had occasion to witness abscess tracking through suture leading to endopthalmitis.

Nowadays, entrance of organisims through the iatrogenic portal of entry can occur until epithelialisation is complete. As this occurs mainly in the first 24 h it is worth considering the protective value of postoperative ointment in the eye together with subconjuctival antibiotic and a pad.

OCC chloramphenicol would induce bacteriostasis and entrapment of organisms. With subconjunctival antibiotics, a significant part of the vulnerable entrance period would coexist with a bacteriolytic concentration of antibiotic in the anterior chamber. Also, any organism carried in with the inert, organism proliferation enhancing foreign body implant, would be exposed to a sustained hostile bacteriolytic environment, while in their weakest numbers, and before any protective host exudative reaction occurs.

Many eyes may well be thus saved, while an evidence base is secured for each of the several precautions necessary for each of the several risk factors.

It is interesting that the evidence base for prophylactic antibiotics (local and systemic) is extremely thin, when joints are replaced. They are, however, routinely used by our Orthopaedic colleagues.

If prophylaxis with subconjunctival antibiotics is accepted on the rationale above there are strong theoretical reasons to suggest a 'Kanski cocktail' approach, not only for the range of cover but also with the overlap to remove adaptation and resistance to individual antibiotics.

The magnitude of the catastrophe of endophthalmitis is not only in numbers. The loss of normal or slightly abnormal eyes in contemporary surgery has to be compared with 'mature' cataract and 6/24 eyes of previous eras.

Finally, it may also be interesting to ascertain whether the routine use of postoperative steroids in modern avascular surgery increases the incidence of endophthalmisks from usually nonpathogenic organisms.

HA Khatib, AG Karseras

MDA Clinic, 65 Cardiff Road, Llandaff, Cardiff CF5 2AA, UK

Correspondence: AG Karseras,
Tel: +44 29 20 56 3004;
Fax: +44 29 20 56 7846.
E-mail: laservision@mdaclinic.co.uk

Eye (2006) **20,** 627. doi:10.1038/sj.eye.6701980; published online 1 July 2005