Penetrating ocular injuries in previously injured blind eyes: should we consider primary enucleation?

We read with great interest the paper by Kilmartin et al in which the authors noted that most of the newly diagnosed cases of sympathetic ophthalmia (SO) in their prospective study occurred in eyes that had sustained multiple injuries, either via trauma or intraocular surgery, and that enucleation following the onset of SO in these eyes was not related to a better visual outcome in the fellow eye. We have recently encountered two cases of ocular trauma in which, with the aforementioned two points in mind, we found it reasonable to consider and offer primary enucleation in an attempt to decrease the risk of SO.

The first case was a 46 year old man who had suffered a previous penetrating injury to his left eye as a child and now presented with a ruptured pre-phthlial globe after striking his left eye with his hand. The patient had previously undergone cataract extraction and trabeculectomy but had no light perception in that eye before the second injury, because of advanced glaucoma. The second patient was a 49 year old man who presented with a large scleral rupture in the left eye after being struck with a metal wrench. Seven years earlier, the patient had sustained a similar injury that had left him with no light perception in that eye. Given that both patients had no light perception in these previously injured eyes, we considered primary enucleation as a way to minimise the risk of SO. Both patients declined primary enucleation and have not developed any signs of ocular inflammation more than 6 months following repair of their second penetrating ocular injuries.

Sympathetic ophthalmia, a rare bilateral granulomatous panuveitis, presumably arises following penetrating ocular injury and surgery as a result of lymphatic exposure to a previously sequestered antigen. The risk of SO following a penetrating ocular injury ranges from 0.1% to 0.3%. Although the risk of SO with multiple penetrating injuries has not been defined, increased antigen release with repeat uveal exposure probably carries an additive risk. This is supported by the finding that most of the newly diagnosed cases of SO in the study by Kilmartin et al had experienced multiple penetrating ocular events.

In addition, as Kilmartin et al have shown, once SO develops, secondary enucleation of the exciting eye to reduce inflammation in the sympathising eye does not necessarily lead to a better visual outcome or to a reduced need for anti-inflammatory treatment. Secondary enucleation is often performed within 14 days of injury as protection against SO for repaired ruptured globes that demonstrate no potential for functional vision. Unfortunately, the time frame necessary to perform prophylactic secondary enucleation remains uncertain, as SO has been reported with secondary enucleation performed as early as 5 days following a penetrating ocular injury that had left him with no light perception in these eyes, however, changes the context in which a subsequent penetrating ocular injury is managed. In this setting, repairing the injury in order to assess for visual potential is futile, and primary enucleation may offer the best prophylaxis against SO.

In many instances, individuals who sustain multiple episodes of trauma either have poor access to health care or are non-compliant with prescribed drugs. Should SO develop in such patients, delayed presentation to an ophthalmologist may lead to an unfavourable outcome, as improved results have been shown with prompt and aggressive anti-inflammatory therapy. Even patients who present early in the course of the disease may be committed to a lifetime of immunosuppressive therapy in order to salvage vision in their only seeing eye.

While primary enucleation is not typically recommended in open globe injuries, it may have a role in select cases of penetrating ocular injuries, such as those involving previously injured blind eyes. In these situations, the ophthalmologist and the patient must carefully assess and discuss the potentially increased risk of SO associated with preserving a disorganised, possibly painful, sightless eye.

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References


Can antiseptic scrubs between cataract surgeries reduce bacterial load on surgical gloves to safe levels?

Although previous studies have reported on aspiration cultures after cataract surgery, 2,3 the minimum strength of inoculum causing endophthalmitis or the route through which the pathogen enters the eye remains unclear. A recent study reported the use of operative face masks to have a significant effect on the bacterial load falling on the operative site.4 Although cataract extraction and lens implant is a procedure of relatively short duration, airborne bacterial contamination of surgical gloves is still possible from the environment in the operating theatre. We designed a study to determine if antiseptic scrubs of surgical gloves between cataract surgeries would reduce the bacterial load to levels equivalent to a new pair of sterile gloves.

We obtained approval of the ethics committee of the institute before initiating the study, and obtained necessary consent from participating subjects, Cataract surgery with intraocular lens implant was carried out through a self sealing scleral tunnel incision and manual expression of the lens. We randomly selected operating surgeons for the study. Each operating surgeon operated on a patient; a preoperative hand scrub repeated twice using chlorhexidine 0.5% solution for 5 minutes. Each operating surgeon wore a face mask, and donned a sterile surgical gown before gloving. Surgeons used commercially available pre-packed sterile powdered latex surgical gloves in the operating room. Each surgeon used 70% isopropyl alcohol solution applied for 30 seconds to scrub gloves between surgeries. None of the surgeons left the operating area in between surgeries. We used sterile cotton moistened with saline to take swabs from the gloves. Swabs were taken from the pre-packed sterile gloves after opening the pack in the operating room, and streaked across a blood agar plate. Swabs were collected from the fingertips and webs of fingers of both hands at random intervals after the first surgery, and streaked across independent blood agar plates. Thirty-six surgeons also streaked separate blood agar plates with a random fingertip of both hands at the conclusion of their operating list before removal and disposal of the gloves. Surgical gloves once removed were not used again. Additional swabs were collected from the surgical gown over the elbows and abdominal
to change of gloves in between cataract surgeries. Antiseptic scrubs of gloves can be an alternative to preventing contamination leading to endophthalmitis. We would have been greatly assisted in this process if they had been informed, at an early stage, about local blind associations, eligibility for financial benefits, and the availability and efficacy of low vision equipment. Additionally, close working between relevant professionals can make it more likely that relevant information is routinely made available. To this end, specialist workers from local social services sensory impairment teams and relevant voluntary sector agencies could usefully join forces to provide an “outreach” information/advice service within eye clinics. In parallel with these initiatives, and to further bolster a more proactive approach to meeting the “social care” needs associated with late onset sight loss, ophthalmic units may additionally benefit from the regular input of relevant case study material and outcomes, presented at clinical meetings by practice and district nurses, rehabilitation workers, and specialist social services staff. Such an exchange of professional experience and knowledge offers the opportunity for developing a more holistic and integrated approach to improving the services available within eye clinics.

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References
Sorsby fundus dystrophy presenting with choroidal neovascularisation showing good response to steroid treatment

Sorsby fundus dystrophy (SFD) is a dominantly inherited macular dystrophy in which bilateral central visual loss occurs in the fifth decade of life. Duke-Elder and Perkins described it as pseudo-inflammatory macular dystrophy. It is now known to be caused by mutations in exon 5 of the gene for tissue inhibitor of metalloproteinases-3 (TIMP3). The disorder has variable expressivity and signs vary depending on age at review, but common fundus characteristics include diffuse fine yellow-white deposits, macular choroidal neovascularisation (CNV), and delayed choroidal filling and retinal pigment epithelial (RPE) mottling on fluorescein angiography (FFA). Later stages include atrophy of the macula and periphery.1,2 Punctate inner choroidopathy (PIC) is characterised by multiple yellow opacities at the level of the inner choroid of the posterior pole and mid periphery without other evidence of inflammation. CNV is a frequent complication which responds to steroid treatment.3

We describe a patient who presented with clinical signs consistent with PIC complicated by bilateral CNV. The CNV was treated over a period of six years using either oral or sub-Tenon steroids with improvement in vision. She was subsequently found to have SFD.

**CASE REPORT**

A 36 year old woman presented with a two month history of difficulty reading with the left eye. Past ocular and medical history were unremarkable except for mild myopia (-2.00 dioptres). On examination, visual acuities (VAs) were 6/5 in the right and 6/18 in the left eye with contact lenses. Fundoscopy revealed a retinal haemorrhage encroaching on the centre of the left fovea and a small chorioretinal scar temporal to the right fovea, which did not appear treatable on fluorescein angiography (FFA). Four months later, she became symptomatic in the right eye with distortion and blurring of vision (fig 1). The RVA was 6/24 and the LVA had stabilised at 6/12 with an old chorioretinal scar in the inferotemporal macula and a small white lesion superior to the fovea. There were several mid-peripheral chorio-retinal scars in both eyes (fig 2). Repeat FFA demonstrated right subfoveal CNV that was not amenable to laser treatment.

Investigations including full blood count, erythrocyte sedimentation rate, electrolytes, renal function, glucose, and chest x ray were all normal. Toxoplasma serology was negative. Serum angiotensin converting enzyme was mildly elevated at 65 U/l (20–54 U/l). A diagnosis of punctate inner choroidopathy was made based on the clinical signs and the patient was started on 60 mg of daily oral prednisolone, reducing to 5 mg daily by six weeks. This was increased to 30 mg after her RVA deteriorated to 6/60. She remained on a low dose for another three months, by which time her vision returned to a RVA of 6/9 and LVA of 6/6 with glasses.

Three weeks later, she reported a large central scotoma affecting the right eye. RVA was 6/12 and LVA was 6/6. Fundoscopy revealed a small right foveal haemorrhage associated with subretinal fluid. Following a sub-Tenon injection of 20 mg dexamethasone, the RVA improved with a subjective reduction in the size of the central scotoma. For the next two years, her vision remained stable. Her left vision deteriorated again. Fundoscopy revealed areas of CNV superior and adjacent to the old scar in the left eye and FFA confirmed active CNV. She was started on a reducing course of 30 mg oral steroids daily. However, three more episodes of recurrent CNV occurred during oral steroid dose reduction, and were treated by a transient increase in oral steroid dose followed by two sub-Tenon injections of 40 mg triamcinolone. Her LVA improved to 6/9 and remained at 6/12 in the right with resolution of the oedema and scarring. She eventually stopped her steroids for 18 months.

Five years after initial presentation, her left vision deteriorated again. Fundoscopy revealed areas of CNV superior and adjacent to the old scar in the left eye and FFA confirmed active CNV. She was started on a reducing course of 30 mg oral steroids daily. However, three more episodes of recurrent CNV occurred during oral steroid dose reduction, and were treated by a transient increase in oral steroid dose followed by two sub-Tenon injections of 40 mg triamcinolone. Her LVA improved to 6/9 and remained at 6/12 in the right with resolution of the oedema and scarring. She eventually stopped her steroids for 18 months.

Five years after presentation, a paternal cousin was diagnosed with SFD. Although her family history at presentation was unremarkable, subsequent enquiries revealed that her paternal aunt had been diagnosed with age related macular degeneration (AMD) in her forties. Her father died at the age of 39. She was consequently screened for mutations in the TIMP3 gene which confirmed that she had the same Ser181Cys mutation as her cousin.

In the following presentation, she has developed the characteristic yellow-white deposits originally described by Sorsby in both eyes.

**COMMENT**

This case highlights two main points. Firstly, the fundus features at presentation mimicked those of PIC and SFD, but the characteristic yellow-white deposits of SFD were not obvious until six years later. Although it is possible that this patient may have had a combination of PIC and SFD, she may just illustrate the phenotypic variability of SFD. Secondly, a clear objective improvement in vision was demonstrated after systemic and peri-ocular steroid treatment. This is new experimental and clinical evidence for using steroids in the treatment of choroidal neovascularisation and thus potentially for SFD. Angiostatic steroids inhibit experimental neovascularisation of the choroid5 and have been used successfully in patients with CNV secondary to ocular histoplasmosis6 and AMD.7 In SFD, mutations in the TIMP3 gene result in the characteristic accumulation of extracellular matrix (ECM) degradation by matrix metalloproteinases (MMPs) and therefore play a key role in a range of physiological processes that include angiogenesis. In the homozygous Timp3-null mouse, absent TIMP-3 inhibitory activity leads to the unscheduled activation of MMPs.8 The resultant breakdown of the ECM would allow endothelial cells to initiate the process of sprouting or release sequestered angiogenic factors such as vascular endothelial growth factor and/or basic fibrilast growth factor, which promote endothelial cell proliferation and migration. A third possibility is the exposure of an angiogenic cryptic site upon proteolytic cleavage of ECM molecules such as collagen type IV9 This may account for the reported inhibitory effect of TIMP-3 on angiogenesis10 although mutant TIMP-3 may not have this property.

Interestingly, MMP expression is significantly attenuated by high concentrations of corticosteroids in the eye11 which have also been shown to stimulate TIMP-3 expression.12 Both actions would have the predicted effect of reducing ECM breakdown and inhibiting angiogenesis. It is therefore not unexpected that steroid treatment in our patient had the effect of improving or stabilising vision with suppression of growth of CNV.

It has been noted that laser treatment in SFD is generally unhelpful,13 although the role of photodynamic therapy has yet to be fully evaluated. Therefore, early intervention with steroids, whether orally, peri-ocularly, or even by intravitreal injection, may be a useful treatment in this condition with a notoriously poor natural history.

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HIV/AIDS and the eye
The latest issue of Community Eye Health (No 47) discusses the impact of the HIV/AIDS epidemic on prevention of blindness programmes. For further information, please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email: Anita.Shah@lshtm.ac.uk; website: www.jch.co.uk). Annual subscription (4 issues) UK£28/US$45. Free to developing country applicants.

Elimination of avoidable blindness
The 56th World Health Assembly (WHA) considered the report on the elimination of avoidable blindness (doc A56/26) and urged Member States to: (1) Commit themselves to supporting the Global Initiative for the Elimination of Avoidable Blindness by setting up a national Vision 2020 plan by 2005; (2) Establish a national coordinating committee for Vision 2020, or a national blindness prevention committee to help implement the plan; (3) Implement the plan by 2007; (4) Include effective monitoring and evaluation of the plan with the aim of showing a reduction in the magnitude of avoidable blindness by 2010; (5) Support the mobilisation of resources for eliminating avoidable blindness. The WHA also urged the Director-General to maintain and strengthen WHO’s collaboration with Member States and the partners of the Global Initiative for the Elimination of Avoidable Blindness as well as aid in the coordination and support of national capability.

5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT)
The 5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT) will take place 11–14 March 2004, in Monte Carlo, Monaco. Please visit our website for details of the scientific programme, registration, and accommodation. To receive a copy of the Call for Abstracts and registration brochure, please submit your full mailing details to http://www.kenes.com/isopt/interest.htm. Further details: ISOPT Secretariat (website: www.kenes.com/isopt).

XVth Meeting of the International Neuro-Ophthalmology Society
The XVth Meeting of the International Neuro-Ophthalmology Society will take place 18–22 July 2004, in Geneva, Switzerland. Further details: Prof. A. Safran, University Hospital Geneva, c/o SYMPORG SA, Geneva (fax: +41 22 839 8484; email: info@symprop.ch; website: www.symprop.ch).

4th International Congress on Autoimmunity
The 4th International Congress on Autoimmunity will take place 1–3 November 2004 in Budapest, Hungary. The deadline for the receipt of abstracts is 20 June 2004. Further details: Kennes International Global Congress Organisers and Association Management Services, 17 Rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland (tel: +41 22 908 0488; fax: +41 22 732 2850; email: autoimm04@kenes.com; website: www.kenes.com/autoimm2004).

4th Meeting of the British Oculoplastic Surgery Society (BOPPS)
The British Oculoplastic Surgery Society (BOPPS) will be holding a meeting in Manchester on 9 & 10 May 2004 (immediately before the College Congress). The organisers are: Sal Ataullah, Brian Leatherbarrow, Claire McAvoy and Mark Halliwell. The social programme begins on the evening of Saturday 8 May. The meeting dinner will be held on Sunday 9 May. The key note invited speaker is Alan McNab, Melbourne. The deadline for abstracts is 27 February 2004. Further information is available from www.bopps.org or by telephone voice mail, on 07092 104 769.