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A novel technique to treat traumatic corneal perforation in a case of presumed brittle cornea syndrome

The management of corneal perforation can be difficult. We describe a novel technique to manage corneal perforation in brittle cornea syndrome (BCS).

Case report

A 14-year-old daughter of consanguineous Pakistani parents presented with a history of a bottle cap having struck her left eye. She had a history of multiple trauma to both eyes since childhood.

At presentation, there was limbus-to-limbus corneal perforation in the left eye. The right eye had a failed corneal graft (for extensive corneal opacities). An examination under general anaesthesia showed a collapsed left eye with rolled-in corneal edges, without any scleral injury. There was partial aniridia, aphakia and prolapsed vitreous, with no obvious retinal detachment. She was also noted to have hypermobility of the small joints of her hands, with bilateral camptodactyly of the fifth fingers and over-riding toes.

During primary repair, the cornea was noted to be soft, with cheese-wiring of 10/0 nylon sutures. At review on past-operative day 1, she had a flat anterior chamber, necessitating a second operation. During this operation, suture track leaks were observed with the use of fluorescein. These were successfully tamponaded with air. To allow sufficient duration of tamponade, a non-expansile (14%) perfluoropropane (C_3F_8) gas exchange was performed after transcorneal three port vitrectomy to gain maximal gas fill. Postoperatively, the cornea was Siedel negative. The patient was kept supine for 10 days, at the end of which the globe remained formed (fig 1).

A month later, the patient received a penetrating corneal injury to the right eye while not using her protective goggles. She underwent primary surgical repair similar to the left eye using C_3F_8 gas. The final visual acuity was hand movement by both eyes.

Discussion

The patient's history suggested an underlying connective tissue disorder affecting the eyes.

BCS is a generalised connective tissue disorder characterised by corneal rupture, after a minor trauma, or spontaneously.[1] Other features include keratoconus or keratoglobus, blue sclera, red hair, hyperelasticity of the skin without excessive fragility, and hypermobility of the joints.[2] BCS has been reported mainly in Middle Eastern consanguineous families, although no underlying genetic defect has been identified to date.[3]

A differential diagnosis is Ehlers–Danlos syndrome type VI, which is associated with kyphoscoliosis and thin sclera (with rupture after trivial trauma). It is characterised by the absence or mutation of the procollagen lysyl hydroxylase gene on chromosome 1, causing a deficiency of the enzyme lysyl-hydroxylase.[4] This leads to a build-up of urinary hydroxylysyl-pyridinoline. In contrast, BCS has normal total urinary pyridinoline ratios. The urinary test results of this patient showed a normal ratio of total lysyl-pyridinoline to total hydroxylysyl-pyridinoline, suggesting a diagnosis of BCS.

Repair of corneal perforations using tissue adhesives and viscoelastic agents,[5] and onlay epikeratoplasty with a donor corneoscleral button to repair a ruptured keratoglobus,[6] has been reported previously. Air tamponade is also commonplace, but the intraocular gas tamponade we used is a novel technique. Prolonged C_3F_8 gas contact with the corneal wound prevented aqueous egress, allowing sufficient wound integrity while keeping the globe formed. This is the first case report of the use of a non-expansile volume of C_3F_8 gas to repair the brittle cornea in BCS, which could be generally applied to fragile leaking corneas.

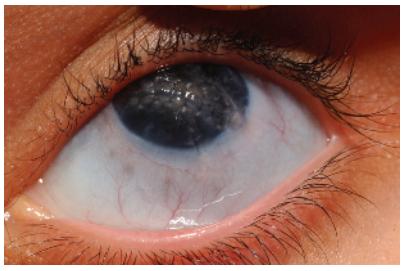

Figure 1 The left eye after the corneal perforation was repaired. Informed consent was received for publication of this figure.

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Sympathetic ophthalmia after ruthenium plaque brachytherapy

Sympathetic ophthalmia is a rare, bilateral inflammatory process with an incidence of 0.03/100 000 in the UK and Ireland.[1] It usually follows either penetrating eye injury or intraocular surgery. Although sympathetic ophthalmia has previously been described after irradiation of ocular melanoma,[2 3] it has never been reported after simple ^{106}Ru plaque brachytherapy. We present a case which confirms that there is a risk of developing sympathetic ophthalmia after charged-particle therapy in the absence of a penetrating injury of the uveal tract.

Case report

A 41-year-old lady was referred by her optician for a slow-growing iris lesion. Fine needle aspiration biopsy confirmed the diagnosis of ciliary body malignant melanoma. Systemic investigations for metastatic disease were negative. The patient underwent routine surgery for the insertion and subsequent removal of a ^{106}Ru plaque. Postoperative recovery was uneventful.

At 6 months after surgery, the patient presented with a 1-week history of reduced vision in both eyes to 6/24 OD and 6/36 OS. This was accompanied by photophobia, soreness and redness of both eyes.

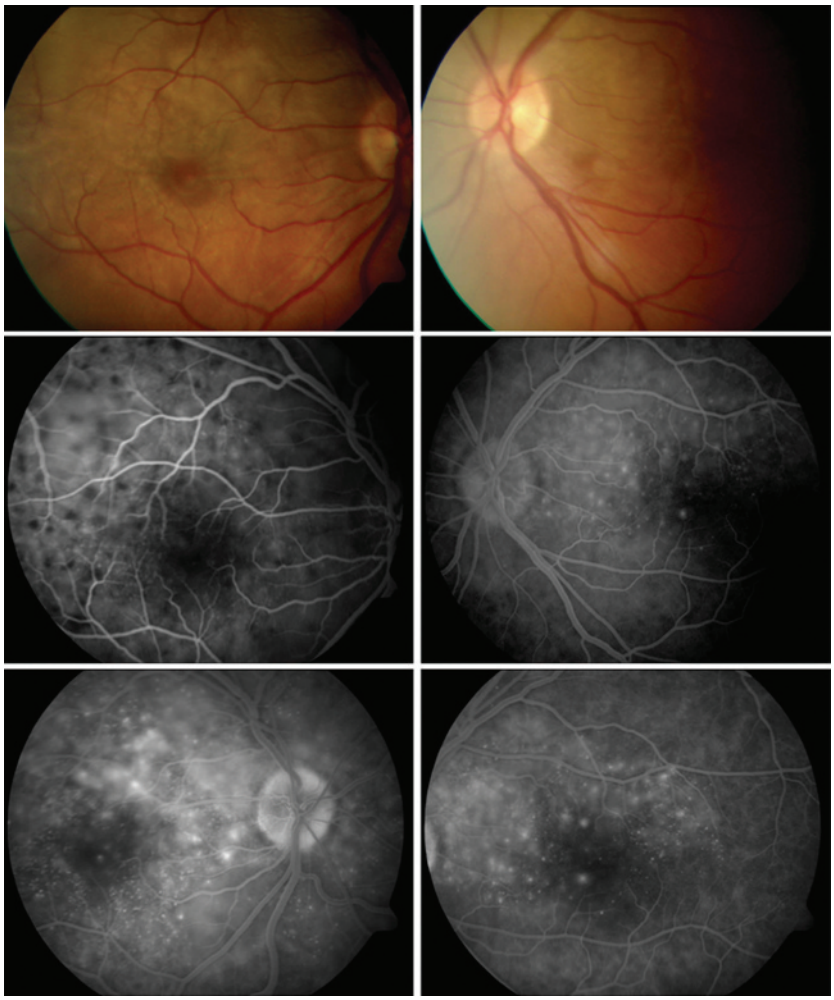

Figure 1 Colour fundus photographs and fluoroscein angiography showing yellowish sub retinal spots at the pasterior pole along with nellro sensory detachments at the macula giving rise to retinal state. FFA shows early hypofluoresence followed by late staining of the yellow spots.

Examination showed moderate anterior uveitis with mutton fat keratic precipitates in the left eye. There was no vitritis. Both fundi showed macular neuroretinal detachments with faint yellowish subretinal spots. Fluorescein angiography showed multiple hypofluorescent spots in the fundus with neuroretinal detachments, which was also confirmed on optical coherence tomography (figs 1 and 2).

A diagnosis of sympathetic ophthalmia was made, and the patient was treated with intensive topical and systemic steroids. She responded well to treatment and was discharged on a tapering dose of systemic steroids. After 6 months, her vision had improved to 6/5 OU, with no evidence of recurrent inflammation.

Comment

The pathogenesis of sympathetic ophthalmia consists of an autoimmune hypersensitivity response directed against exposed uveal tissue in the traumatised (exciting) eye, which is followed by a similar response against the fellow (sympathising) eye. If left untreated, it follows a chronic, relapsing and progressive course. Various surgical causes previously reported include complicated cataract surgery,[4] cyclocryotherapy,[5] laser cyclophotocoagulation,[6–8] proton beam and helium ion irradiation,[2 3] and vitrectomy.[9 10]

The disruption of uveal tissue that set off the chain of events in this case is thought to have resulted from the radioactive effect of the treatment. Fine needle aspiration biopsy is unlikely to have caused sympathetic ophthalmia, as the biopsy was taken through clear cornea under direct visualisation and avoiding choroidal tissue. Perforation at the time of suturing during surgery was not noted peroperatively, and is unlikely given the very anterior location of the tumour, which made surgery technically easy at an area with relatively thick sclera.

This case shows a rare delayed complication of Ruthenium brachytherapy that has never been reported previously. The most likely differential diagnosis is Vogt Koyanagi Harada syndrome, which may present in a similar manner.

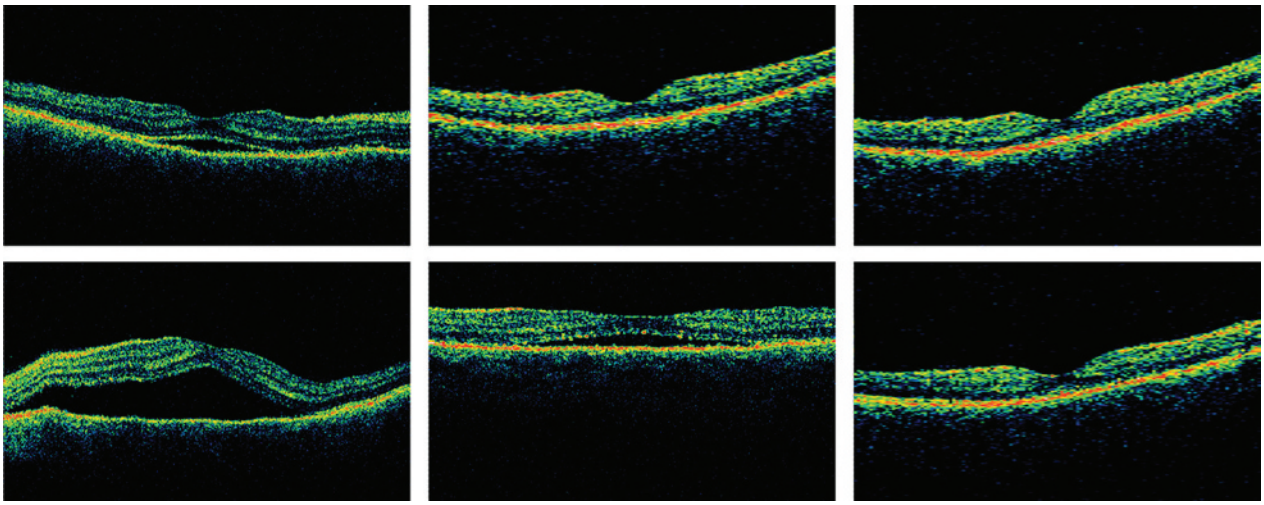

Figure 2 Sequential OCT scans of the right (top) and left (bottom) eyes shaving extensive neurosensory retinal detachments and the resolution of SRF over a 3 week period of treatment with steroids.

However, Vogt Koyanagi Harada syndrome follows a relapsing remitting course, often with systemic symptoms despite treatment with immune suppression, unlike sympathetic ophthalmia, which is a potentially curable disease if managed properly in acute stages. An absence of recurrence of disease process on stoppage of therapy, as well as the clinical history of our patient, makes the diagnosis of sympathetic ophthalmia more likely.

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Frequency of non-arteritic anterior ischaemic optic neuropathy in adult Chinese: The Beijing Eye Study

Non-arteritic anterior ischaemic optic neuropathy (NAION) is an acute optic neuropathy occurring predominantly in small optic nerve heads in elderly patients.[1–3] It has been debated which factors, other than old age and a small optic disc size, predispose to the disease. Data on the prevalence of NAION in the general population has so far been scarce.[4 5] They have been completely missing for the Chinese. Therefore, the aim of this study was to evaluate the prevalence of optic nerve damage with the features of a preceding NAION in the Beijing Eye Study.

The Beijing Eye Study is a population-based cohort study in Northern China.[6] The Medical Ethics Committee of the Beijing Tongren Hospital approved the study protocol and all participants gave their informed consent, according to the Declaration of Helsinki. Of 5324 individuals aged ⩾40 years residing in the study area, 4439 individuals (2505 women) participated in the eye examination (response rate, 83.4%). This study included 4027 (90.7%) subjects for whom readable optic disc photographs were available. The mean (standard deviation (SD)) age was 55.2 (10) (median, 55; range, 40–101) years. The participants underwent an ophthalmic examination including photographs of the optic disc and macula (Fundus camera CR6-45NM, Canon, Lake Success, New York, USA) and frequency doubling perimetry (screening program C-20-1; Zeiss-Humphrey, Dublin, California, USA) as described in detail previously.[6] For all eyes with visual acuity <0.6 and for all eyes with any visual field loss, the photographs of the macula and optic disc were assessed twice by a panel that included several ophthalmologists (YW, LX, JBJ). The diagnostic criteria for NAION were a small optic disc with a cup/disc diameter ratio of ⩽0.4, segmental pallor, segmental loss of the retinal nerve fibre layer and segmental visual field loss.[1–3 7]

Of the 8876 eyes included in the study, only one eye fulfilled the definition of NAION. The age of the male subject with suspected previous NAION was 71 years, his vision was 0.30, and he showed visual field defects in the inferior hemisphere. His optic disc, with a size of 2.0 mm^2, did not have cupping and exhibited a pale neuroretinal rim in its superior half. The visibility of the retinal nerve fibre layer was markedly reduced in the temporal superior region, and the temporal superior artery was markedly thinner than the temporal inferior artery. In a mathematical sense, the calculated mean (SD) prevalence of NAION in the whole population was 0.02 (1.6%) (95% confidence interval −0.02 to –0.07), or about 1 in 4500 subjects.

Comment

The prevalence rate of 1 in about 4500 Chinese with an age of ⩾40 years may be at the lower end of the frequency range reported and calculated from previous studies on Caucasian populations in which about 2.3–10.3 patients per 100 000 inhabitants >50 years were affected with NAION in the US per year.[4 5] Despite its marked statistical limitation due to the low prevalence rate of NAION, this study may, therefore, support the finding from previous investigations that whites may be affected by NAION more commonly than population groups of another ethnic background such as Chinese.[4 5] One may consider that the interethnic differences in optic disc size, with the smallest optic discs found in Caucasians, medium-sized optic discs in Asians and largest discs in Afro-Americans, might be responsible for the interethnic differences in the frequency of NAION, which occurs predominantly in small optic nerve heads. The major limitation of this study is the low prevalence rate of NAION so that the prevalence rate for NAION as calculated in the present investigation may have to be confirmed in even larger population-based studies with >10 000 subjects included. One may conclude that NAION-associated optic nerve damage is present in 1 out of about 4500 adult Chinese. The figure will be around 100 000 patients with NAION-induced optic nerve damage in the whole of China.

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Frequency of spontaneous pulsations of the central retinal vein

The central retinal vein is the only structure in the body which can be examined non-invasively, runs through the cerebrospinal fluid space, and has a shape that depends on the relationship between its internal pressure and the pressure in the space surrounding it. Estimation of central retinal vein pressure is, therefore, helpful in the assessment of cerebrospinal fluid pressure—that is, the intracranial pressure.[1–4] Central retinal vein pressure may be assessed by determining the external pressure at which the central retinal vein starts to pulsate. This method of assessment is similar to Riva-Rocci's method of indirect measurement of arterial blood presure. For the central retinal vein, the external pressure is the intraocular pressure. The purpose of the present study was to find out the proportion percentage of subjects in whom the central retinal vein shows spontaneous pulsations, indicating that the pressure in the vein is lower than the intraocular pressure. The assessment of spontaneous retinal venous

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