

Trauma as a cause of CRAO has been reported as a sole factor or in association with other systemic pathology such as haemoglobinopathies and coagulation abnormalities.^{4,5} Trauma can be directed to the eyeball or the head. The possible mechanisms that cause closed-eye CRAO can be compression of the central retinal artery induced by a haematoma,³ by air in case of orbital emphysema,⁶ or raised intraorbital pressure resulting from swelling of orbital soft tissue. Direct compression after surgical repair of orbital blow-out fracture with a Teflon plate has been reported.⁷ Common to these conditions is damage to the endothelial cells of the artery with exposure of the underlying collagen, which stimulates platelet aggregation and thrombus formation.^{2,8} If CT/MRI scans show a significant haematoma, the patient requires an urgent neurosurgical referral for decompression.

Severe reflex vaso-spasm initiated as a direct response to concussion injury to the arterial wall smooth muscle is another mechanism.⁹ As there was no evidence of severe ecchymosis or proptosis, we can only postulate that this was the possible mechanism in our patient. Some authors have suggested stellate ganglion block to relieve the spasm if the patient is seen within 4 h of injury.⁹

Blunt ocular trauma can lead to commotio-retinae, a relatively benign and self-limiting condition, which does not require any specific treatment. Trauma can also produce CRAO. Rarely, as in our case, the two can coexist with commotio-retinae masking the clinical appearance of CRAO. A patient with such a history but with profound visual loss and RAPD should raise suspicion of CRAO. If optic nerve contusion and compression are ruled out, then spasm of the retinal artery is the likely mechanism for CRAO. If seen within 4 h of injury, aggressive measures may be undertaken to alleviate spasm.

References

- 1 Henkind P, Chambers JK. Arterial occlusive disease of the retina. In: Duanes TD (ed). *Clinical Ophthalmology*, Vol 3, Chap 14. Harper and Row: Philadelphia, 1987, pp 10–14.
- 2 Brown GC, Magargal LE, Shields JA *et al*. Retinal artery obstruction in children and young adults. *Ophthalmology* 1981; **88**: 18–25.
- 3 Chawala JC. Traumatic central retinal artery occlusion. *Trans Ophthalmol Soc UK*. 1972; **92**: 777–784.
- 4 Sorr EM, Goldberg RE. Traumatic retinal artery occlusion with sickle cell trait. *Am J Ophthalmol* 1975; **80**: 648–652.
- 5 Michaelson PE, Phaffenbach D. Retinal artery occlusion following ocular trauma in youths with sickle-cell trait haemoglobinopathy. *Am J Ophthalmol* 1972; **74**: 494–497.
- 6 Linbreg JV. Orbital emphysema complicated by acute central retinal artery occlusion: case report and treatment. *Ann Ophthalmol* 1982; **14**: 747–749.
- 7 Emery JM, Huff JD, Justice JJ. Central retinal artery occlusion after blow-out fracture repair. *Am J Ophthalmol* 1974; **78**: 538–540.
- 8 Moake JL, Levine JD: Thrombotic disorders. *Ciba-Geigy Clin Symp USA* 1985; **37**: 3–32.
- 9 Chawla JC: Traumatic central retinal artery occlusion. *Trans Ophthalmol Soc UK* 1972; **92**: 777–784.

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Sir,

Dacryocystectomy as treatment of chronic dacryocystitis in a frail, elderly patient

Eye (2004) **18**, 334–336. doi:10.1038/sj.eye.6700662

Chronic dacryocystitis develops secondary to obstruction of the nasolacrimal duct (NLD) caused by infection or inflammation. The patient presents with epiphora, a mucocoele that can become secondarily infected due to tear stasis (acute or chronic dacryocystitis) or a chronic discharging fistula to the skin. The treatment of chronic dacryocystitis is usually with an initial course of broad-spectrum oral antibiotics followed by external dacryocystorhinostomy (DCR) and intubation. However, prior to the development of DCR surgery in 1904, chronic dacryocystitis was managed by dacryocystectomy (DCT), whereby the lacrimal sac and any fistulae present were excised.¹ We report a case of a frail, elderly patient in whom DCT was preferred to DCR surgery and resolved a chronic dacryocystitis that had been resistant to antibiotic treatment.

Case report

A frail 71-year-old man with chronic epiphora and discharge of several months duration presented to the eye casualty department with a 2-day history of a localised tender, erythematous swelling over the right infero-medial canthal area with associated preseptal cellulitis. There was also mucopurulent discharge from a fistula over the lacrimal sac and acute dacryocystitis. He

had undergone a dacryocystogram to investigate his persistent right-sided epiphora just 3 days earlier, which showed total obstruction of the NLD on the right. He had pseudobulbar palsy secondary to brainstem cerebrovascular disease resulting in expressive dysphasia and difficulty swallowing. He was on treatment for hypertension and duodenal ulceration.

He was admitted and treated with intravenous cefuroxime and metronidazole and topical chloramphenicol. He was discharged 3 days later on Augmentin when the preseptal cellulitis was much improved. Unfortunately, he represented 4 days later with recurrence of a localised abscess and purulent discharge from the fistula, but no preseptal cellulitis (Figure 1). The abscess was drained and packed with povidone iodine-soaked ribbon gauze and he was referred for urgent surgical treatment.

In view of his poor medical condition, DCT was performed rapidly under local anaesthesia rather than OCR. During surgery, the lacrimal sac was excised in one piece (Figure 2a). The proximal end of the nasolacrimal duct was cauterised. Probing of the canaliculi revealed an occluded superior canaliculus and a patent inferior canaliculus, which was cauterised. The fistula was also excised. A pressure dressing was left *in situ* for 3 days and he was discharged home on oral Augmentin and topical chloramphenicol. The skin sutures were removed at 1 week and he has had no further recurrence of dacryocystitis or fistula formation (Figure 2b). The histology report confirmed severe chronic inflammatory changes but no evidence of dysplasia or malignancy.

Comment

DCT was first described by Woolhouse in 1724 as a treatment for recurrent dacryocystitis secondary to

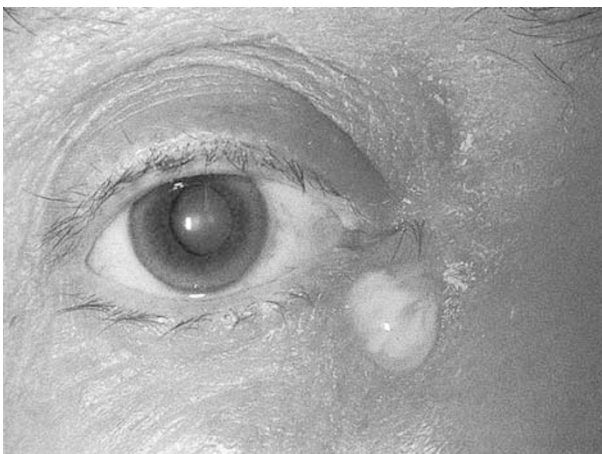


Figure 1 Preoperative photograph showing discharging fistula over the lacrimal sac area but no preseptal cellulitis.

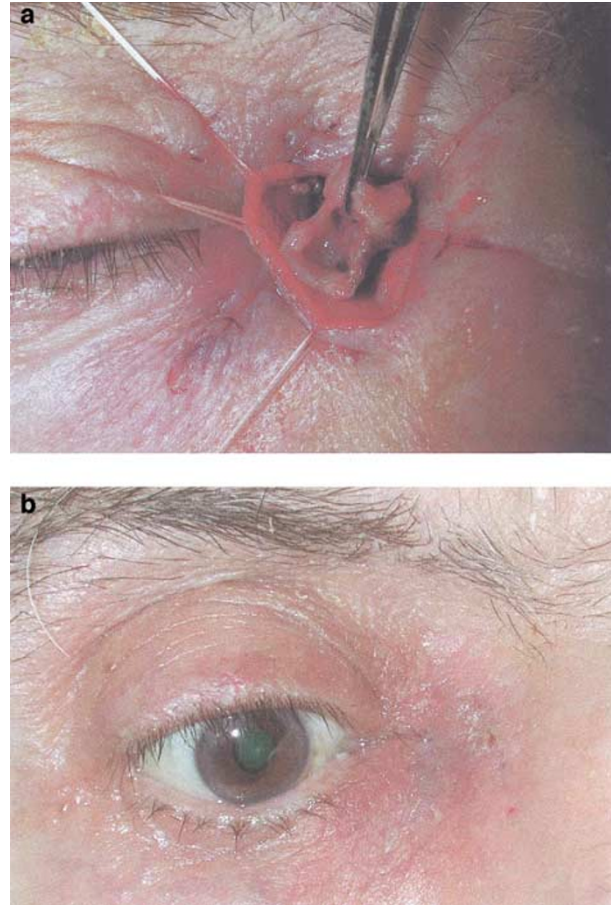


Figure 2 (a) Intraoperative photograph showing lacrimal sac excision. The sac is opened to show the interior and is orientated with the nasolacrimal duct on the right. (b) Postoperative appearance showing resolution of fistula and healed incision site.

acquired nasolacrimal duct obstruction.¹ However, after the introduction of DCR surgery, the use of DCT declined. At present, the main indication for DCT is excision of lacrimal sac tumours. However, other less common indications are recurrent dacryocystitis due to inflammatory causes such as Wegener's granulomatosis when there is a risk of subsequent nasalcutaneous fistula formation following DCR surgery² or recurrent dacryocystitis without epiphora.^{3,4}

DCT differs from DCR surgery in that there is no osteotomy or breaching of the nasal mucosa and hence there is less risk of aspiration pneumonia due to intraoperative nasal haemorrhage.⁵ Secondly, DCT is a safer procedure to perform on a frail, elderly patient than DCR as the surgical time is much shorter than that of external DCR surgery and the type of local anaesthesia required is safer in DCT. In DCR surgery under local anaesthesia, it is necessary to pack the nose with either cocaine or local anaesthetic and nasal decongestant

and/or vasoconstrictive agent to prevent haemorrhage as well as infiltrate the lacrimal fossa with local anaesthetic and a vasoconstrictor. These agents can have significant systemic effects on frail, elderly patients, with exacerbation of systemic hypertension, tachycardia, dysrhythmia, and a risk of myocardial toxicity due to their sympathomimetic action.^{6,7} DCT can be performed with standard local infiltration of the medial canthal area with lidocaine and adrenaline alone without the need for nasal packing.

Other authors have advocated the use of DCT in the management of chronic dacryocystitis when there is a dry eye.^{3,4} However, in this case the patient initially presented with epiphora and subsequently went on to develop acute dacryocystitis, then chronic dacryocystitis with fistula formation. The surgical choice of DCT over DCR was guided principally by the ill health of the patient. This patient has performed well, with no recurrence of dacryocystitis, fistula, or epiphora. We therefore advocate that surgeons consider DCT in some frail, elderly patients with chronic dacryocystitis as a safe alternative to DCR.

References

- 1 Duke-Elder S, MacFaul PA. The ocular adnexa. In: Duke-Elder S (ed). *System of Ophthalmology, Vol 13. Part II. Lacrimal, Orbital and Para-orbital Diseases*. Henry Kimpton: London, 1974, pp 715–718.
- 2 Holds JB, Anderson RL, Wolin MJ. Dacryocystectomy for the treatment of dacryocystitis patients with Wegener's granulomatosis. *Ophthalmic Surg* 1989; **20**(6): 443–444.
- 3 Boynton JR, Anawis MA. Role of dacryocystectomy in the management of failed dacryocystorhinostomy associated with chronic dacryocystitis. *Ophthalmic Surg Lasers* 1996; **27**(2): 133–136.
- 4 Mauriello JA, Vadehra VK. Dacryocystectomy: surgical indications and results in 25 patients. *Ophthalmic Plastic Reconstruct Surg* 1997; **13**(3): 216–220.
- 5 Bartley GH, Nichols WL. Hemorrhage associated with dacryocystorhinostomy and the adjunctive use of Desmopressin in selected patients. *Ophthalmology* 1991; **98**: 1864.
- 6 Meyers EF. Cocaine toxicity during dacryocystorhinostomy. *Arch Ophthalmol* 1980; **98**: 842–843.
- 7 Meyer DR. Comparison of oxymetazoline and lidocaine versus cocaine for outpatient dacryocystorhinostomy. *Ophthalmic Plastic Reconstruct Surg* 2000; **16**(3): 201–205.

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Competing interests: None

Sir,

Chorioretinal arterial and venous anastomoses as a result of blunt trauma

Eye (2004) **18**, 336–338. doi:10.1038/sj.eye.6700663

Venous Chorioretinal anastomosis has been induced using a variety of techniques in the treatment of retinal vein occlusions. This process can also occur in a number of infective, inflammatory, or traumatic processes. We describe the formation and angiographic appearance of Chorioretinal venous and arterial anastomoses following a nonpenetrating eye injury.

Case report

A 40-year-old pseudophakic white male patient presented following a blunt injury to the right eye, reportedly caused by a punch. This man had had bilateral cataract extractions using a phacoemulsification technique with intraocular lens insertion 2 years previously for idiopathic presenile cataract.

On initial examination, visual acuity was 6/18 right eye and 6/5 left eye. On the right side, a partial dislocation of his posterior chamber intraocular lens into the anterior chamber was present. The intraocular pressure was 13 mmHg on both sides by applanation tonometry. On dilated examination, a vitreous haemorrhage and an inferior Chorioretinal rupture could be seen.

The Chorioretinal rupture measured approximately five disc diameters in length and originated one and a half disc diameters inferior to the optic nerve head. The rupture passed inferotemporally, following a curvilinear course that transected the inferior temporal retinal arteriole and vein. The left eye examination was unremarkable.

Following initial treatment with topical steroid and antibiotic drops, visual acuity improved to 6/9 within 8 weeks of the injury. Anterior uveitis with secondary glaucoma continued to intermittently cause symptoms.

On review at 5 months following the injury, it was noted that although the inferior temporal retinal arteriole proximal to the chorioretinal rupture was stenosed and did not appear to have significant blood flow the arteriole proximal to the rupture was of normal calibre and appearance (Figure 1). The inferotemporal retina