Table 1 Causes of sphenocavernous or orbital apex syndrome

Differential diagnosis of sphenocavernous or orbital apex syndrome

Inflammatory

- 1 Sarcoidosis
- 2 Tolosa-Hunt syndrome
- 3 Systemic lupus erythematosus
- 4 Churg-Strauss syndrome
- 5 Wegener granulomatosis
- 6 Giant cell arteritis
- 7 Orbital inflammatory pseudotumour
- 8 Thyroid orbitopathy

Infection

- 1 Fungi: aspergillosis, mucormycosis
- 2 Bacteria: *Strep* spp, *Staph* spp, *Actinomyces*, Gram negative bacilli, anaerobes, *M tuberculosis*
- 3 Spirochaetes: *Treponema pallidum*
- 4 Viruses: herpes zoster

Neoplasia

- 1 Neural tumours: meningioma, neurofibroma, schwannoma, ciliary neurinoma
- 2 Endocrine tumours: Pituitary with or without apoplexy
- 3 Head and neck tumours: pituitary, naso/craniopharyngeal, squamous cell, adenoid cystic carcinomas
- 4 Metastatic tumours: lung, breast, renal cell carcinoma, malignant melanoma
- 5 Haematological: non-Hodgkin's lymphoma, Burkitt lymphoma, leukaemia
- 6 Perineural invasion of cutaneous malignancy

Trauma/iatrogenic

- 1 Traumatic: penetrating, non-penetrating injury, orbital apex fracture, retained foreign body
- 2 Iatrogenic: orbital/facial, sinonasal, surgery

Vascular

- 1 Carotid cavernous aneurysm or fistula
- 2 Cavernous sinus thrombosis
- 4 Sickle cell anaemia

Other

- A Mucocoele

and may present to a variety of specialists, including ophthalmologists, as illustrated by our case. A high index of suspicion, rapid diagnosis, and multidisciplinary care are essential when managing women of child-bearing age with neurological or orbital symptoms, as early treatment is curable.

Multiple cranial nerves palsies, particularly 3, 4, 6, and 5 divisions V1 and V2, while sparing optic nerve function are strongly suggestive of cavernous sinus pathology.^{4,5} Common neoplastic causes of sphenocavernous syndrome include nasopharyngeal carcinoma, lymphoma, meningioma, lung and breast metastases (see table 1 for other aetiologies); however, metastatic choriocarcinoma has, to our knowledge, not been reported.

Although a less favourable prognosis is associated with central nervous system (CNS) metastasis,³ newer intensive chemotherapy regimens such as high dose EMA-CO (etoposide, methotrexate, actinomycin-cyclophosphamide, and vincristine), have encouraging results,⁶ as highlighted by this patient. In sufficiently chemosensitive tumours, the blood-brain barrier does not prevent treatment from eliminating CNS

disease.⁶ Additional modalities for treating cerebral metastases include intrathecal chemotherapy and neurosurgery.¹⁻³ Cranial irradiation may be used in selective cases; however, there is no consensus regarding its role.^{3,6}

E Sharkawi, K Tumuluri, J M Olver

Department of Ophthalmology, Charing Cross Hospital, Fulham Palace Road, London, UK

Correspondence to: Eamon Sharkawi, MBBS, BSc, MRCP, MRCOphth, Department of Ophthalmology, Charing Cross Hospital, Fulham Palace Road, London W6 8RF, UK; eamonsark@doctors.org.uk

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Primary posterior continuous capsulorhexis; a new technique

Posterior capsule opacification (PCO) is by far the most common complication of cataract surgery, with a reported incidence of 10% to 50%.^{1,2} Primary posterior continuous curvilinear capsulorhexis (PCCC) has been employed to prevent PCO,³⁻⁶ based on the theory that removing the scaffold for the migration of lens epithelial cells will provide a permanently clear optical zone.

Case reports

We prospectively recruited 48 emmetropic patients with senile fibrous posterior capsular cataracts, but the decision to perform PCCC was made intraoperatively after attempts to remove the fibrotic plaques were unsuccessful. All patients gave informed consent and ethics committee approval was obtained for the study. The mean age of the patients was 73 years. All patients had negative ocular and systemic history. Complete ophthalmic examination and central macular thickness (CMT) was recorded on optical coherence tomography (OCT) scans (six radial lines, OCT 2, Carl Zeiss Meditec AG, Germany) through best possible fixation preoperatively and postoperatively at 1 week, 1 month, and 3 months.

Routine phacoemulsification was performed. The posterior capsular fibrous plaque was successfully removed in 18 patients. In the remaining 30 patients, a PCCC was performed. After intraocular lens (IOL) implantation in the capsular bag a thin film of viscoelastic was spread inferior and superior to the IOL, causing the IOL to have

Figure 1 Size of PCCC is smaller than the size of intraocular lens optic (white arrows).

anterior tilt superiorly. Under high magnification, a flap of posterior capsule was raised with the tip of standard 25 gauge cystotome and a PCCC was performed smaller than the optic diameter (fig 1) with capsulorhexis forceps. After PCCC, viscoelastic was washed out with standard irrigation/aspiration (IA) handpiece. All patients were given a routine postoperative course of combined topical drops of dexamethasone 0.1%, neomycin sulphate 0.35%, and polymyxin B sulphate 6000 IU for 4 weeks.

In two eyes, PCCC was poorly controlled. These eyes exhibited positive vitreous pressure manifested by convex posterior capsule during phacoemulsification. At the point when it extended towards the capsular equator, attempts to complete it were abandoned. It was sufficient to clear the central visual axis. No other intraoperative and postoperative complications were encountered.

Preoperative CMT (mean (SD)) was 170.4 (5.6) μ m compared to 174.6 (4.7), 179.1 (5.0), 172.6 (5.7) μ m at 1 week, 1 month, and 3 months respectively. IOP remained normal. All patients achieved best corrected visual acuity of 6/6 6 weeks postoperatively and maintained it throughout the follow up.

Comment

Posterior capsule opacification (PCO) continues to stimulate important work towards understanding its causes, prevention and effectively treating it. It is visually debilitating if the posterior capsular plaque was unsuccessfully removed preoperatively.

In 1996, Galand *et al* suggested primary posterior capsulorhexis as a routine procedure during cataract surgery in adults. In their retrospective series of 319 eyes, 12 intraoperative complications occurred. Postoperatively, three of 49 eyes showed cystoid macular oedema on fundal fluorescein angiography, anterior vitreous prolapse occurred in three eyes and one retinal detachment was noted at 4 weeks. In 2001, Hugkultstone reported the incidence of intraoperative complications and visual results of suction posterior capsulorhexis. In his elective 86 eyes requiring suction posterior capsulorhexis two eyes had intraoperative vitreous prolapse during IOL insertion and viscoelastic removal.

The lack of complications in this series of patients is attributed to two factors; firstly, PCCC is performed in a closed chamber only

following IOL implantation and thus compression or decompression of anterior chamber is unlikely to occur. Secondly, it is important to apply posterior pressure on the IOL with an IA hand piece to temporade the PCCC margins, ensuring that anterior hyaloid face remains intact.

In this study we used OCT to identify the presence of CMO. No significant changes in macular thickness were found for the duration of follow up, paralleled by a corresponding good visual acuity and no patient had CMO on clinical examination. These results suggest that the incidence of preoperative and postoperative complications associated with PCCC is possibly less with our technique.

S K Gibran, S Jungkim, B Patil, P E Cleary
Department of Ophthalmology, Cork University Hospital, Cork, Republic of Ireland

Correspondence to: Mr S K Gibran, Department of Ophthalmology, Cork University Hospital, Cork, Republic of Ireland; syedgibran@yahoo.com

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Infectious crystalline keratopathy associated with intravitreal and posterior sub-Tenon triamcinolone acetonide injections

Infectious crystalline keratopathy (ICK) is a rare, progressive microbial infection characterised by branching, intrastromal crystalline opacities and minimal ocular inflammation. Predisposing factors most commonly include topical corticosteroids, previous corneal surgery, herpetic keratitis, neurotrophic keratopathy, and topical anaesthetic abuse.^{1–3} We describe a patient who developed ICK following intravitreal and posterior sub-Tenon (PST) triamcinolone acetonide injections despite being off topical corticosteroids for 6 months.

Case report

A 75 year old man with diabetes mellitus developed cystoid macular oedema (CMO) in the left eye following cataract extraction complicated by anterior vitrectomy, anterior

chamber intraocular lens placement (ACIOL) and subsequent ACIOL exchange. The CMO resolved with topical prednisolone acetate 1% and a posterior sub-Tenon injection (PST) of triamcinolone acetonide (40 mg). After being off topical corticosteroids for 6 months, the CMO recurred and the patient was treated with repeat PST of triamcinolone acetonide (40 mg) followed 1 month later by intravitreal triamcinolone acetonide (4 mg) injection using sterile techniques. The patient's peripheral cornea had become oedematous following multiple intraocular surgeries, yet there was no history of a persistent epithelial defect.

Six weeks after the intravitreal injection, the patient developed pain in the left eye. His ocular medications included timolol maleate 0.5% twice daily and ketorolac tromethamine four times daily in the left eye. Corrected visual acuity was 20/20 in the right eye and 20/70 in the left eye with normal corneal sensation in both eyes. A tri-lobed anterior stromal crystalline infiltrate was present on the left cornea, with an overlying epithelial defect, measuring 3 mm (vertical) × 2 mm (horizontal) (fig 1). Additionally, peripheral microcystic oedema was present on the left cornea with several epithelial bullae. Cultures of corneal scrapings performed on presentation were positive for *Streptococcus viridans*. He was treated with vancomycin (50 mg/ml) drops every 1–2 hours for 1 week, and subsequently switched because of ocular toxicity to topical moxifloxacin 0.5%, to which the organism was also sensitive. The epithelial defect gradually healed and the crystals resolved with 2 months of topical treatment. The cornea has subsequently decompensated requiring a penetrating keratoplasty.

Comment

Infectious crystalline keratopathy is a chronic infection which is often difficult to eradicate owing to the presence of a glycocalyx (biofilm) produced by the infecting organism.² Factors related to both the organism and host may play a part in the pathogenesis of the crystalline architecture. *Streptococcus viridans* is most commonly associated with ICK, though reports have also implicated *Staphylococcus epidermidis*, *Pseudomonas*, *Haemophilus*, *Enterococcus*, *Mycobacterium*, and *Candida*.^{4–7} Lack of epithelial integrity and a deficient immune response, typically secondary to topical corticosteroids, may allow for the inoculation and proliferation of the organism. In this case, a ruptured epithelial

Figure 1 Slit lamp photograph of left cornea on initial presentation with tri-lobed crystalline corneal infiltrate.

bullae may have provided entry for colonisation.

Despite the absence of topical corticosteroids in our patient for 6 months, the successive periocular and intraocular corticosteroid injections probably contributed to a localised immune suppression, predisposing to the development of ICK. ICK has been associated with both systemic immune suppression⁸ and subconjunctival 5-fluorouracil.⁹ Animal studies of bacterial endophthalmitis suggest that intravitreal triamcinolone acetonide injection may lead to a greater susceptibility to infection.¹⁰ Furthermore, anterior segment concentrations of triamcinolone may have been higher than usual in this unicameral eye after anterior vitrectomy. Though the patient had used ketorolac tromethamine for less than 1 month, the use of a topical non-steroidal anti-inflammatory drug (NSAID) may have also contributed to his condition. With the increasing use of intravitreal corticosteroid injections for retinal diseases, it should be recognised that periocular and intravitreal corticosteroids may predispose to the development of ICK, especially in compromised corneas.

D A Hollander, E L Clay, Y Sidikaro

Jules Stein Eye Institute, University of California, Los Angeles, CA, USA

Correspondence to: David A Hollander, MD, MBA, Cornea, External-Ocular Disease and Uveitis Division, Jules Stein Eye Institute, UCLA, 100 Stein Plaza, Los Angeles, CA 90095, USA; hollandar@jsei.ucla.edu

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