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.

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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.

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