CASE REPORT

Infectious crystalline keratopathy after Descemet's stripping endothelial keratoplasty

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Accepted 30 May 2017

SUMMARY

A 68-year-old woman presented with infectious crystalline keratopathy 4 months after she underwent a combined phacoemulsification and Descemet's stripping endothelial keratoplasty for Fuch's endothelial dystrophy in her left eye. After 5 months of topical moxifloxacin 1%, the infiltrate responded well but had not completely resolved, with the resulting endothelial failure requiring a penetrating keratoplasty 9 months after the initial operation. Microbiology identified *Enterococcus faecalis* with the histopathology demonstrating bacterial colonies within the graft interface. Postoperatively she developed endophthalmitis, needing vitrectomy and intravitreal antibiotics. The infection settled with no recurrence, with topical and oral antibiotics continued for 2 months. A sutured toric piggyback intraocular lens was performed 18 months postvitrectomy for graft astigmatism, achieving a best-corrected vision of 6/15.

Infectious crystalline keratopathy can occur following Descemet's stripping endothelial keratoplasty, requiring long-term topical therapy and potentially leading to graft failure with the necessity for further keratoplasty.

BACKGROUND

Infectious crystalline keratopathy (ICK) is a well-reported indolent corneal infection characterised by insidious discrete branching grey or white needle-like opacities, with minimal associated inflammation in adjacent ocular tissue. We present a previously unreported case of ICK developing after Descemet's stripping endothelial keratoplasty (DSEK) requiring a penetrating keratoplasty and subsequent vitrectomy to resolve the infection.

CASE PRESENTATION

A 68-year-old female was referred with Fuch's endothelial dystrophy and significant nuclear sclerosis. Other relevant medical history included polymyalgia rheumatica and rheumatoid arthritis, for which she was taking oral prednisone 1.5 mg daily and methotrexate 10 mg weekly. She underwent uncomplicated bilateral combined phacoemulsification and DSEK procedures: first in the right eye and 6 months later in the left eye. Postoperatively, topical chloramphenicol 0.5% and topical prednisolone acetate with phenylephrine 0.12/1% six times daily were administered, with the antibiotics ceased after 3 weeks and the corticosteroid reduced to four times daily. Best-corrected visual acuity improved from 6/120 to 6/12 in the left eye.

At 2 months, the left superior corneal sutures were removed, and the graft was clear. Two months later, the patient re-presented with a decrease in visual acuity associated with ocular pain and epiphora with a non-suppurative branching white opacity typical of ICK, located deep in the left temporal midperipheral stromal interface (figure 1). There were no signs of inflammation in the surrounding ocular tissue or anterior chamber. Hourly topical moxifloxacin 1% was commenced, and the prednisolone acetate/phenylephrine 0.12/1% continued four times daily. She responded well to treatment (figure 2A), and the moxifloxacin dose was decreased as the infiltrate reduced over the next 5 months. During this time, the endothelium developed signs of rejection with graft haze and an epithelial defect (figure 2B). Despite increasing the topical corticosteroid to hourly and the graft initially clearing, the endothelium failed requiring a penetrating keratoplasty with an 8.5 mm trephine that was undertaken 9 months after the first operation (figure 2C). Histopathology demonstrated ovoid basophilic aggregates, consistent with bacterial colonies at the endothelial graft host interface, with minimal inflammatory infiltrate (figure 2D). Cultures identified *Enterococcus faecalis* with sensitivities to amoxicillin, chloramphenicol, penicillin, tetracycline and gentamicin. Postoperatively, the patient was continued on moxifloxacin 0.5% and prednisolone acetate with phenylephrine 0.12/1%, six times daily. By day 10, the corneal graft was clear; however, a vitreous haze had developed, with debris evident on the B-scan. A vitrectomy with administration of intravitreal vancomycin 1 mg in 0.1 mL and ceftazidime 2.5 mg in 0.1 mL. Postoperatively, topical moxifloxacin 1% and prednisolone acetate/phenylephrine 0.12/1% were continued four times daily, with oral ciprofloxacin 750 mg and cefalexin 500 mg twice daily. The vitreous humour culture returned positive for *E. faecalis*.

OUTCOME AND FOLLOW-UP

The antibiotics were stopped over the next month, with no recurrence of the ICK or the endophthalmitis. A sutured toric piggyback intraocular lens was performed 2 years later to improve graft astigmatism, with a resulting best-corrected visual acuity of 6/15.

DISCUSSION

ICK occurring after penetrating keratoplasty accounts for most of the reported cases. Risk factors

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To cite: Porter AJ, Lee GA, Whitehead K. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-220464

Figure 1 Slit lamp photomicrograph showing a non-suppurative branching white opacity deep in the left temporal stromal-graft interface.

include a break occurring in the epithelium over the graft as well as the sutures acting as a nidus for infection. Topical and oral immunosuppression with corticosteroids can exacerbate the infection and may also contribute to the quiescent nature of ICK with limited inflammatory response.¹ Although ICK has been reported in lamellar keratoplasty,² there have been no previously documented ICK following DSEK.

Progression to endothelial failure may have been exacerbated by the latent colonisation in the interface. Moura *et al* investigated the postoperative complications in a phaco-DSEK group and reported 4 of 47 eyes (8.5%) developed a haze in the interface, in addition to seven eyes (14.8%) that developed secondary graft failure.³ ICK is commonly caused by alpha-haemolytic *Streptococcus* although the list of pathogens has grown considerably since first being described. It predominantly affects the anterior stroma; however, ICK involving the posterior

stroma has also been reported.^{4,5} *E. faecalis* has been associated with ICK previously.^{6,7} Enterococcus is a Gram-positive cocci of intestinal origin that forms short to medium chains and is commonly associated with nosocomial infections.⁸ It is a facultative anaerobe, which may account for its ability to survive in the posterior cornea at the graft-host interface as occurred in this case.

The use of topical moxifloxacin appeared to improve the infiltrate, and this guided the decision to reduce treatment. Evidence of bacterial colonies in the histopathology, despite 5 months of antibiotic treatment indicate the difficulty in fully resolving ICK. Immunosuppression has been shown to contribute to bacterial colony biofilm formation and is thought to contribute to the appearance of ICK.⁹ Cessation of immunosuppression in this patient was not possible with a history of polymyalgia rheumatica and rheumatoid arthritis.

Christakopoulos *et al* investigated the difference between infectious keratitis and ICK, and identified an impairment in complement mediated phagocytosis and inflammatory cell recruitment in ICK cases.¹⁰ This was thought to be attributable to a biofilm produced by the microorganisms in ICK. The use of adjunctive treatment, such as Nd:YAG laser or intrastromal antibiotics to target the biofilm may have been considered in this case. Potentially, the laser would disrupt the biofilm, thereby increasing the exposure of the bacteria to the antibiotic. Intrastromal antibiotics could also be injected directly in the zone of the infiltrate, improving local availability of the antibiotic. Both modalities carry risk to the endothelium either by direct trauma or causing inflammation with potential risk of rejection. Inadvertent injection of antibiotics into the endothelial graft-host interface could also possibly result in graft dehiscence. The development of endophthalmitis in this case is unusual but has been previously reported with ICK cases.¹¹⁻¹³ The posterior location of the infiltrate may have contributed to involvement of the anterior chamber.

In summary, ICK can complicate DSEK and may present a significant challenge in management. Intensive topical antibiotics remain the first line of management; however, treatment duration needs to be prolonged and as occurred in this case, may require redo keratoplasty.

Figure 2 (A-D) Slit lamp photomicrographs of 1 month postcommencement of topical antibiotics showing reduced size of the branching infiltrate. (B) Three months postcommencement of topical antibiotics with the endothelium showed signs of rejection with graft haze and an epithelial defect. (C) Penetrating keratoplasty with 16 interrupted 10-0 nylon sutures. (D) Histopathology showing ovoid basophilic aggregates, consistent with bacterial colonies at the endothelial graft-host interface, with minimal inflammatory infiltrate (H&E staining. Original magnification 300×).

Learning points

- ▶ Infectious crystalline keratopathy can occur after Descemet's stripping endothelial keratoplasty.
- ▶ Initial treatment with aggressive medical therapy initially is necessary; however, surgical excision of the infection may still be required.
- ▶ Endophthalmitis can occur due to the posterior location of the infectious crystalline keratopathy and potential communication between the endothelial graft-stromal interface and the anterior chamber.

Contributors All authors meet the criteria for authorship. GAL was involved in the conceptual design, drafting and editing the article. AJP was involved in case data acquisition and drafting the article. KW was involved in case data interpretation. All authors were in agreement for approval of the article for publishing and to ensure that the article is accurate.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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