

# Delayed postoperative interface keratitis due to *Enterococcus faecalis* after Descemet membrane endothelial keratoplasty

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## SUMMARY

To report the clinical course and management of interface keratitis due to *Enterococcus faecalis* after Descemet membrane endothelial keratoplasty (DMEK). A 64-year-old man underwent DMEK, with unevenful immediate postoperative course, with a visual recovery of 20/30 at 2 weeks. At 3 months of clinical visit, interface keratitis was noted. DMEK graft removal with stromal bed scrapings was performed. A diagnosis of *E. faecalis* interface keratitis was made. The patient responded favourably to antibiotic susceptibility-guided intensive treatment with vancomycin 5% with complete resolution of infection. After 2 months of graft removal, Descemet stripping endothelial keratoplasty (DSEK) was performed. The corneal clarity was restored and the best corrected visual acuity was 20/40 at last follow-up of 1 year. *E. faecalis* should be kept as a differential in delayed onset interface keratitis after DMEK. After microbiological cure with antibiotic therapy, visual rehabilitation with DSEK restores corneal clarity and results in favourable visual outcome.

## BACKGROUND

Descemet membrane endothelial keratoplasty (DMEK) involves selective replacement of diseased endothelium with healthy endothelium-Descemet membrane. In Descemet stripping endothelial keratoplasty (DSEK), the diseased endothelium and Descemet membrane of host is replaced with posterior corneal stroma, Descemet membrane and endothelium of donor. In DMEK absence of posterior stromal lamella in scroll, reduces posterior astigmatism, hyperopic shift and altered higher order optical aberrations caused by lenticule in DSEK.<sup>1</sup> Similar to DSEK, DMEK also creates a graft host interface that can harbour microorganisms in the sequestered space leading to peculiar complication of interface keratitis.

Interface keratitis after any type of lamellar keratoplasty (LK) is challenging to diagnose and manage due to an impaired access to the site of infiltration for microbiological analysis and antibiotic penetration.<sup>2,3</sup> Often the treatment is governed by an empirical approach and indirect evidence such as cultures from the donor rim. Several organisms have been implicated in interface keratitis after DMEK,<sup>2-8</sup> of which fungus is the most common aetiological agent reported.

Herein, we report the clinical course and management of a case of delayed interface keratitis after DMEK with *Enterococcus faecalis*

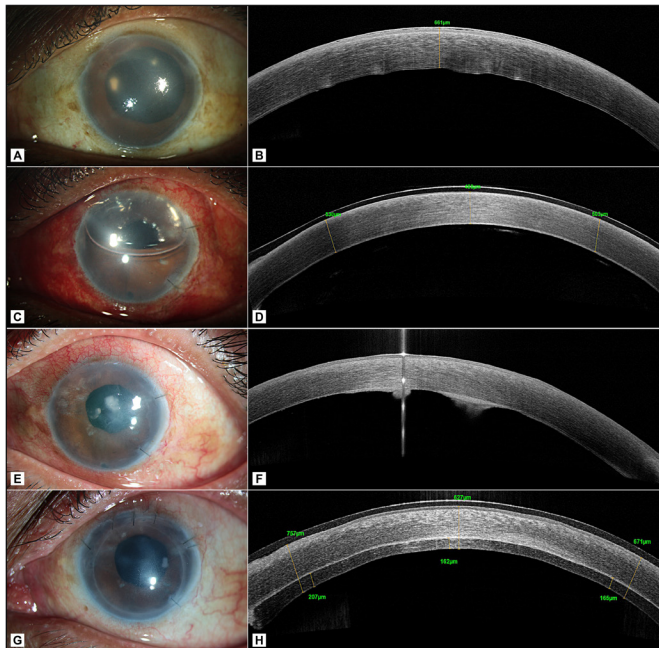
## CASE PRESENTATION

A 64-year-old man underwent DMEK for pseudophakic corneal oedema in the left eye (figure 1A,B). A subconjunctival injection of 0.3% moxifloxacin was administered at the conclusion of the surgery. The DMEK graft was prepared immediately prior to the surgery in the operating room from a 72-year-old phakic, male donor, collected from a hospital source, with an endothelial cell density of 2600 cells/mm<sup>2</sup>. The death to preservation time was 4 hours and preservation to utilisation time in MaCarey Kaufman medium (MK medium) containing gentamicin was 70 hours. Postoperative day 1, the graft was attached well (figure 1C,D). At 2-week review visit after surgery, the cornea was clear with a best corrected visual acuity (BCVA) of 20/30. After 3 months, he presented to the clinic with reports of poor vision, redness and discomfort in the operated eye. Further, on enquiry, he revealed that the decrease in vision began at 1 month after the surgery and was gradually progressive. However, he was unable to visit the clinic then. The visual acuity at this visit had reduced to 20/600. Slit-lamp examination showed mild congestion, yellowish-white infiltrate in the interface (figure 1E). High-resolution optical coherence tomography corroborated with the clinical findings and revealed that the infiltrate was localised at the interface level (figure 1F). DMEK graft removal was done by scoring with reverse sinsky hook and interface scraping was performed. The recipient bed was scraped with a blunt cannula and aspirated using a 3 mL syringe attached to it. The aspirate was plated and inoculated on the culture media. Intracameral amphotericin B 25 mg/0.1 mL was administered at the time of the surgery. The Descemet membrane removed was bisected at the site of the infiltrate; one half was sent in 10% formalin for histological evaluation and the second half was plated on chocolate agar for microbiological evaluation. The gram stained smears showed the presence of gram-positive cocci in groups and culture media grew *E. faecalis* (figure 2A,B) that was identified by VITEK 2 and found to be sensitive only to vancomycin and resistant to cefazoline, ciprofloxacin, ofloxacin, gatifloxacin, moxifloxacin, clindamycin, tetracycline, chloramphenicol and gentamycin. The histology sample showed the Descemet membrane with colonies of gram-positive cocci on gram stain (figure 2C). As a part of our eye bank's protocol of investigation of an adverse event after keratoplasty, the details of donor history and the mate pair of



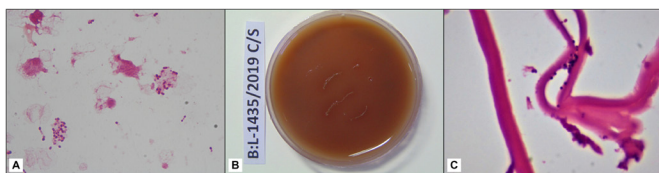
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**Figure 1** (A and B) Preoperative slit lamp and OCT image of the left eye; (C and D) slit lamp photograph and OCT image on postoperative day 1 after DMEK; (E and F) slit lamp photograph and OCT at 3 months visit showing 2 foci of interface infection; (G and H) slit lamp photograph and OCT image at 1 month post DSEK. DMEK, Descemet membrane endothelial keratoplasty; DSEK, Descemet stripping endothelial keratoplasty; OCT, Optical coherence tomography.

the donor cornea used for DMEK were reviewed. The mate pair of this donor was pseudophakic with unrecordable endothelial cell density and a pterygium scar and hence was not used for transplantation. The practice at our eye bank is to preserve the corneoscleral rim after keratoplasty for a week after surgery, beyond which these are discarded. Hence, the corneoscleral rim was unavailable for microbiological culture at 3 months when the patient was seen in the clinic. The patient was treated with vancomycin 5% eye drops every 1 hour for 1 week, every 2 hours thereafter for the next 2 weeks and atropine sulphate eye drops two times per day for 2 weeks. After 1 week of intensive antibiotic treatment, prednisolone acetate 1% and sodium chloride 5% were prescribed every 4 hours. The patient responded favourably to the treatment and there was resolution of infiltrate in 5 weeks duration. The B scan ultrasonography performed at the time of diagnosis of interface keratitis, a week, and 5 weeks after DMEK graft removal was echo free.



**Figure 2** (A) Direct smear examination of interface scrapings showing polymorphonuclear cells 0–4/oil immersion field (OIF), and purple to deep pink cocci in pairs and short chains 0–10/OIF ( $\times 1000$ ); (B) growth of confluent tiny, smooth cream bacterial colonies on the streaked regions on chocolate agar (incubation 37°C, 3 days); (C) photomicrograph of Descemet membrane showing gram-positive cocci in small clusters (Gram stain,  $\times 1000$ ).

The cornea was oedematous and developed paracentral haze. At 2 months after graft removal, DSEK was performed in view of relatively poorer visualisation of the anterior segment compared with the earlier when he underwent DMEK.

## OUTCOME AND FOLLOW-UP

The postoperative course was uneventful after DSEK and visual acuity was improved to 20/80 at 1 month postoperative time (figure 1G,H). The cornea clarity recovered over the next 3 months when BCVA was 20/40. At the last follow-up of 1 year, the patient's cornea had cleared, with a BCVA of 20/30.

## DISCUSSION

Interface keratitis is a rare complication after LK. With the growing popularity of DMEK, there has been an increased reporting of interface keratitis after DMEK. Although majority of reports of the interface keratitis after DMEK were due to *Candida* sp, there are isolated case reports of interface keratitis caused by other organisms such as *Mycobacterium chelonae* and *Nocardia asteroides*. We report *E. faecalis* as another cause of interface keratitis after DMEK.

*E. faecalis* is a Gram-positive, commensal bacterium in the gastrointestinal tracts of humans. D'Oria *et al*<sup>9</sup> reported a case of microbiologically proven *E. faecalis* interface keratitis post deep anterior lamellar keratoplasty in which therapeutic penetrating keratoplasty (PK) was performed with good outcome. Hannush *et al*<sup>10</sup> reported *E. faecalis* deep stromal abscess post DSEK with venting incisions treated with medical management and PK post infection resolution with fair visual outcome. A case of infectious crystalline keratopathy (ICK) after DSEK caused by *E. faecalis* was reported by Porter *et al* and treated with PK and vitrectomy to resolve infection.<sup>11</sup> Our case presented in the interface between posterior stroma and Descemet membrane, while the case of ICK reported by Porter *et al* was in the stroma–stroma interface.

Managing interface keratitis can be difficult unless the diagnosis can be made with certainty. There are several challenges faced in appropriate diagnosis. The deep-seated nature of the infiltrate makes microbiological assays inaccessible. In eyes with early onset infection, the rim cultures may aid in making a diagnosis. However, in those cases presenting with delayed onset of infection, the corneoscleral rim may not be available (as was the case here) and hence other methods of microbiological diagnosis are required. The rim cultures cannot be relied on for the diagnosis of postoperative problems and they are a poor substitute for specimens from the site of infection. Our patient had symptoms of gradual decrease in vision from 1 month after DMEK. There was no evidence of infection at 2 weeks of clinical visit postsurgery, when the interface was clear and vision was 20/30. He presented to the clinic only after 3 months when his vision had dropped significantly. Considering the delayed onset and indolent course of the interface infection, although *Candida* interface infection was suspected and kept as a differential diagnosis, a microbiological confirmation was clearly needed. In view of this, our approach involved DMEK graft extraction along with stromal bed scrapings for microbiological and histological diagnoses.

The patient responded to intensive treatment with vancomycin 5%. In view of graft removal, there was significant corneal oedema and some degree of stromal haze ensued, that precluded repeated DMEK due to visualisation concerns; hence, DSEK was performed instead. There was gradual and satisfactory improvement in corneal clarity and visual acuity over a period of 1 year.

Although the source of infection in this case could not be ascertained, we believe it is likely due to donor contamination. The donor cornea was harvested from hospital source and the *Enterococcus* spp isolated was resistant to several antibiotics. There is an increasing antimicrobial resistance in hospital setting and corneas harvested from donors with hospitalisation can have microbial contamination that can potentially cause donor-related infection, especially if the organism is found resistant to the antibiotic in the corneal preservation medium like gentamycin-enriched MK medium in our case.<sup>12</sup>

The case highlights the importance of diagnostic intervention in cases of delayed interface keratitis after DMEK. A definitive diagnosis is required to initiate a specific antibiotic therapy to achieve eradication of infection, following which visual rehabilitation should be considered.

### Learning points

- ▶ Interface infectious keratitis (IIK) is a known complication after lamellar keratoplasty (LK) warranting surgical intervention for infected graft removal and for microbiology testing.
- ▶ Culture sensitivity-guided medical management for LK results in infection resolution and secondary surgical intervention results in optimal outcome.
- ▶ Fungus (*Candida*) is the most common reported aetiology for IIK, other causes like *Enterococcus*, *Nocardia* and *Mycobacterium* should be considered and appropriately evaluated.

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