adopted or translated into a virtual event. Limitations to this study include the small number of participants as well as the inherent bias of a survey design. Additionally, as Olympics is an annual event, resident confidence may increase year to year. Future studies will be helpful to better understand the long-term impact of such a course.

Ophthalmology Olympics is a training course focusing on acute ophthalmologic procedures that are often overlooked in surgical training. Early exposure to surgical procedures decreases resident anxiety and increases surgical confidence.

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References

1. Blackmore C, Austin J, Lopushinsky SR, Donnon T. Effects of postgraduate medical education "Boot Camps" on clinical skills, knowledge, and confidence: a meta-analysis. J Grad Med Educ 2014;6:643–52.
2. Rogers G, Oetting TA, Lee AG. Impact of a structured surgical curriculum on ophthalmic resident cataract surgery complication rates. J Cataract Refract Surg 2009;35:1956–60.
3. Grantcharov TP, Kristiansen VB, Bendix J, Bardram L, Rosenberg J, Funch-Jensen P. Randomized clinical trial of virtual reality simulation for laparoscopic skills training. British Journal of Surgery 2004;91:146–50.
4. Gedde SJ, Volpe NJ, Feuer WJ, Binenbaum G. Ophthalmology resident surgical competence: a survey of program directors. Ophthalmology 2020;127:1123–5.
5. Fisher JB, Binenbaum G, Tapino P, Volpe NJ. Development and face and content validity of an eye surgical skills assessment test for ophthalmology residents. Ophthalmology 2006;113:2364–70.
6. Bommer C, Sullivan S, Campbell K, et al. Pre-simulation orientation for medical trainees: An approach to decrease anxiety and improve confidence and performance. Am J Surg 2018;215:266–71.

Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this article.

Late-onset recurrent *Acremonium* fungal keratitis after therapeutic penetrating keratoplasty

Fungal keratitis is a prominent cause of blindness worldwide. Therapeutic penetrating keratoplasty (TPK) has been demonstrated as an effective treatment for fungal keratitis that does not respond to antifungal medications. Recurrent fungal infection after TPK is uncommon but occurs typically within 2 weeks after surgery.[1]

Acremonium species are a group of filamentous fungi isolated from vegetation matter and are a rare cause of fungal keratitis. We present here an unusual, delayed case of recurrent fungal keratitis with *Acremonium* species that recurred over 2 months after successful treatment with TPK.

Case Report

An 86-year-old woman presented with increased pain and foreign body sensation in her right eye for several weeks. Her past medical history was significant for Sjögren syndrome, for which she had been taking azathioprine 50 mg daily for many years. She had no history of previous eye trauma. Past ocular history was significant for wet age-related macular degeneration for which she was undergoing regular treatment with intravitreal bevacizumab. On presentation, visual acuity in the right eye was light perception. Slit lamp examination showed moderate conjunctival injection with a subtotal corneal ulcer overlying a corneal infiltrate located paracentrally, with 50% corneal thinning centrally and 80% corneal thinning inferotemporally. The anterior chamber was deep with a 1-mm hypopyon (Fig. 1A).

Corneal scraping revealed presence of oval conidia noted on Gram stain, and the patient was diagnosed with fungal keratitis. She was started on topical antifungal amphotericin B 0.15% every 1−2 hours in the right eye. Two weeks later, the microbiological report of the culture from a national mycology reference laboratory identified the presence of *Acremonium* species. The patient started a tapering course of topical voriconazole 1% along with the amphotericin B 0.15% and progressively improved (Fig. 1B), with ultimate healing of the ulcer in 3 months' time. Topical loteprednol 0.5% was used sparingly for control of inflammation only upon disappearance of the ulcer and infiltrate.

Four months after her initial presentation, upon suspension of the antifungal medications, the patient presented with large, recurrent multifocal infiltrates and corneal ulcer (Fig. 1C), with precipitous worsening of the clinical picture (Fig. 1D) and rapid evolution towards perforation. Uncomplicated TPK was performed. Postoperatively, she continued topical amphotericin B 0.15% daily and topical voriconazole 1% daily hourly for 1 week and then 4 times daily, along with topical tacrolimus 0.1% 4 times daily to prevent graft rejection (Fig. 1E). No topical or systemic steroids were used to reduce the chance of recurrence of the infection. While still on antifungal medications, the patient developed

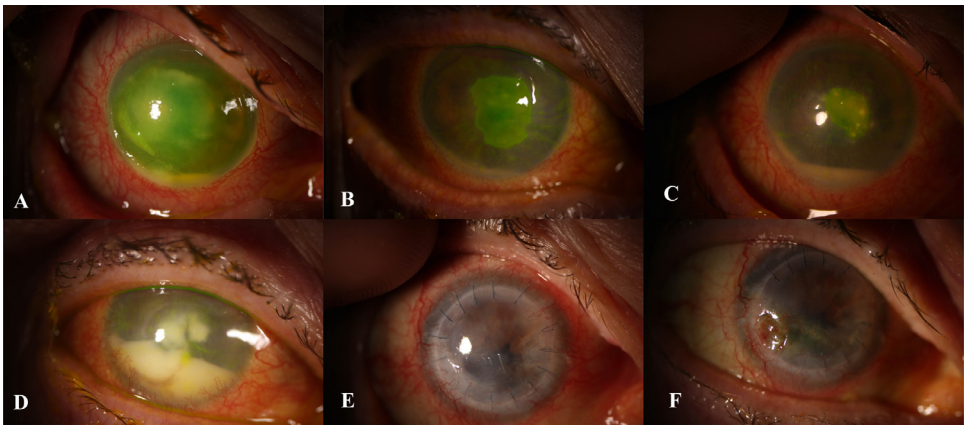

Fig. 1—(A) Patient at presentation, with multifocal diffuse corneal infiltrates, large corneal ulceration, and hypopyon. (B) Progressive resolution of the infiltrates, ulcer, and hypopyon. (C) Recurrence of the multifocal infiltrates, ulceration, and hypopyon upon suspension of antifungal medications. (D) Rapid worsening of the clinical picture, with increase in size of corneal infiltrates and hypopyon. (E) Patient 1 month after therapeutic penetrating keratoplasty. Note peripheral neovascularisation with loosening of interrupted sutures secondary to withheld use of topical steroids. Central cornea remained relatively transparent. (F) Recurrence of inferotemporal ulcer with superficial infiltrates at the graft-host junction of therapeutic penetrating keratoplasty graft.

endothelial rejection with corneal edema and neovascularization 2 months postoperatively. Topical prednisolone 1% was added 6 times daily, with resolution of the rejection in 1 week. The topical antifungals were suspended 1 week after starting topical steroids because there were no signs of any active fungal infection at that time. The patient remained on tacrolimus 4 times daily throughout this period.

Two weeks after starting steroid medication and 76 days after the TPK, a 1-mm epithelial defect was noted on the patient's temporal cornea. Two days later, the epithelial defect increased in size, and an ulcer was noted on the patient's temporal corneal (Fig. 1F). A positive culture confirmed the recurrence of *Acremonium* species. The patient was started again on topical amphotericin B 0.15% and topical voriconazole 1% 6 times daily. Prednisolone eyedrops were reduced to twice daily. These medications were continued for 1 month, at which point the epithelial defect healed. The amphotericin B was discontinued, and the tacrolimus and voriconazole were eventually discontinued over the next 2 months, with the prednisolone reduced to once daily. One year after discontinuing all antifungal medication and 21 months after her initial presentation, there continued to be no recurrence of a fungal infection.

Discussion

Fungal keratitis can unfortunately recur after TPK. A retrospective analysis of 1148 cases of fungal keratitis treated with TPK reported that recurrence typically appeared within 2 weeks after surgery in 7.7% of eyes,[1] consistent with previous reports.

It has also been shown that recurrence rates for fungal keratitis in patients who have been given steroids before surgery are significantly higher than those who were not treated with any steroids prior to surgery.[2] To note, the patient in our case had been administering loteprednol sparingly to manage corneal edema a week before her TPK. Additionally, recurrence rates are higher for patients with a preoperative hypopyon,[2] corneal perforation,[2] and larger infiltrates[3]—all of which were present in our patient. Our patient had also been taking azathioprine, a known immunosuppressant, for many years, which could have played a role in the recurrence of the fungal infection. This medication was not changed during the course of her fungal keratitis treatment. A common postoperative treatment after fungal keratitis outlined by Shi et al. involves 0.5% fluconazole and 0.25% amphotericin B or 5% natamycin. If no signs of recurrence are present for 2 weeks, low concentration topical steroids are administered twice daily.[2]

In our case, tacrolimus 0.1% was started as an immunosuppressive medication to prevent graft rejection on postoperative day 1, although steroids were withheld until 2 months postoperatively and only when corneal edema and neovascularization owing to presumed graft rejection were noted. Our patient succumbed to a recurrence of *Acremonium* keratitis more than 2 months after TPK upon initiation of topical steroid treatment and without any prior signs of lingering or recalcitrant infection. In retrospect, perhaps it would have been beneficial to maintain the antifungal treatment for a longer period once the steroids were started. Another option would have been to use topical cyclosporin drops, which also have an antifungal effect.[4]

This case reports the longest latency between TPK and recurrent fungal infection described to date, with recurrence on the 76th day after TPK. The second longest delay in recurrence we could find in the literature was the 66th day after TPK.[5] Our case highlights the particular difficulty in treating *Acremonium* keratitis, demonstrating its ability to remain quiescent in the cornea and reactivate months after

TPK. One previous similar case has been documented, occurring in a 65-year-old man.[6] In this case, the patient underwent a corneoscleral keratoplasty and began prednisolone 1% acetate drops 2 weeks after the procedure. Seven weeks after the corneoscleral keratoplasty, the patient was diagnosed with recurrent *Acremonium* keratitis and ultimately required a second TPK.

In summary, our present case demonstrates the potential for *Acremonium* keratitis to reoccur months after TPK in the absence of clinical signs of persistent infection and encourages clinicians to exercise caution in their use of immunosuppressive medication during postoperative management of this fungal species.

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References

1. Gong Y, Xin M. Incidence of recurrent fungal keratitis after primary keratoplasty and visual outcome and prognosis after intervention for the recurrence. Medicine 2019;98:e15910.
2. Shi W, Wang T, Xie L, et al. Risk Factors, clinical features, and outcomes of recurrent fungal keratitis after corneal transplantation. Ophthalmology 2010 May;117:890–6.
3. Mundra J, Dhakal R, Mohamed A, et al. Outcomes of therapeutic penetrating keratoplasty in 198 eyes with fungal keratitis. Indian J Ophthalmol 2019;67:1599.
4. Perry HD, Doshi SJ, Donnenfeld ED, Bai GS. Topical cyclosporin A in the management of therapeutic keratoplasty for mycotic keratitis. Cornea 2002;21:161–3.
5. Chatterjee S, Agrawal D. Recurrence of infection in corneal grafts after therapeutic penetrating keratoplasty for microbial keratitis. Cornea 2020;39:39–44.
6. Wang MX, Shen DJ, Liu JC, Pflugfelder SC, Alfonso EC, Forster RK. Recurrent fungal keratitis and endophthalmitis. Cornea 2000;19:558–60.

Footnotes and Disclosure

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Late-onset bilateral epithelial ingrowth following rapid corneal decompensation owing to amantadine

Epithelial ingrowth (EI) can occur at the interface between the flap and the stromal bed of the patient's cornea following laser assisted in situ keratomileusis (LASIK) surgery**.** The introduction of corneal epithelial cells into the interface can happen during surgery or after the procedure owing to loss of contact inhibition and the presence of a pathway to the interface. Epithelial ingrowth is a relatively uncommon complication, with a reported incidence of 3.9% following primary LASIK and up to 12.8% following enhancement with a flap lift.[1] While usually asymptomatic, EI may lead to decreased vision owing to direct intrusion of cells into the visual axis, irregular corneal astigmatism, or melt of the overlying flap. Risk factors for EI have been described; however, systemic drug intake is not reported. We report a case of late-onset bilateral EI induced by acute corneal edema 6 years following primary LASIK in a patient taking amantadine, botulinum toxin (BT) injections, and natalizumab for multiple sclerosis.

In April 2019, a 49-year-old white female patient was referred for acute bilateral corneal edema unresponsive to topical corticosteroids. Her past medical ocular history was notable for uncomplicated bilateral LASIK in 2013, with uncorrected visual acuity (UCVA) of 20/15 in her right eye (OD) and monovision of J1 in her left eye (OS). (Best spectacle corrected visual acuity 20/15 OS with a −1.75 diopter [D] correction). No EI OU. Her past medical history was relevant for multiple sclerosis, diagnosed in 2012 and treated with 200 mg of oral amantadine daily since 2012, BT injections every 3 months for leg spasticity since 2013, and monthly intravenous natalizumab since 2017.

On examination, UCVA was 20/200 OD and 20/150 OS (J8 and best spectacle corrected visual acuity 20/80 OS). Anterior segment was unremarkable OU with only rare cells OS. Slit lamp examination showed moderate corneal edema, no keratic precipitates, normal LASIK flaps, no diffuse lamellar keratitis, no infiltrates, no EI, and no interface haze (Fig. 1A, B). Ultrasound pachymetry revealed a central corneal thickness of 666 microns (μm) OD and 763 μm OS. Digital and tonometer intraocular pressure was normal and fundus examination was unremarkable OU.

Hourly topical prednisolone acetate 1% was continued while sodium chloride 5% drops and ointment were added without improvement. The patient underwent sequential Descemet stripping endothelial keratoplasty (DMEK) grafts OU. Due to a mild head tremor, the patient required rebubbling OU for partially nonadherent grafts and a second DMEK following failed rebubbling OD.

Epithelial ingrowth appeared 20 weeks and 25 weeks after the initial decrease in vision for OD and OS respectively (Fig. 1C, D). The EI occurred before DMEK in OD and after DMEK in OS.