pass through Descemet membrane into the anterior chamber, similar to fungi. Recently, microsporidia have been phylogenetically shown to be fungi, and their tissue behavior akin to that of fungi would be expected.4

Our patient had clinical features of herpetic viral endophthalmitis at the initial visit, which improved initially with antiviral treatment. There is a possibility that the patient may have had viral keratitis initially and then became secondarily infected with microsporidium. Also, coinfection by microsporidia with herpes cannot be excluded.

Penetrating graft rather than lamellar keratoplasty is recommended for patients with deep stromal involvement to avoid any chance of recurrence in the lamellar bed.3 In our case, the presence of microsporidial spores in the endothelial exudates highlights the possible penetration of microsporidial spores through intact Descemet membrane, thereby emphasizing the need for penetrating keratoplasty in cases of deep stromal involvement, especially with endothelial exudates.

We conclude that endothelial exudates may manifest in microsporidial keratitis, and the spores may be present in the endothelial exudates.

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Actinomyces Infectious Crystalline Keratopathy

We report a difficult-to-treat, indolent infection following penetrating keratoplasty (PKP) with the clinical appearance of infectious crystalline keratopathy. The causative agent was not recovered from multiple corneal scrapings but morphologically resembles Actinomyces species histopathologically in the deep stroma of corneal tissue removed at repeat PKP.

Report of a Case. A 75-year-old woman underwent uncomplicated combined right PKP and lenticular phacoemulsification for Fuchs corneal dystrophy and cataract. Three months postoperatively, she developed peripheral stromal opacities associated with 2 loose sutures. These opacities grew despite topical treatment with tobramycin, and marked conjunctival injection and a moderate anterior chamber inflammatory reaction developed. She was referred to our service for treatment of corneal ulceration in the nasal and temporal portions of the graft (Figure 1A). Corneal scrapings revealed no organisms on Gram or Giemsa staining, and no organisms grew on aerobic, anaerobic, fungal, or viral cultures. She was treated with fortified cefazolin and gentamicin sulfate eyedrops but developed 2 new stromal infiltrates and required repeat PKP (Figure 1B). Histopathologic analysis of the removed tissue revealed full-thickness corneal edema and stromal scarring, consistent with healed keratitis. Gram staining at multiple levels failed to reveal the presence of organisms.

Four months postoperatively, the patient noted a foreign body sensation and was found to have an area of corneal thinning at the graft-host junction near the temporal site of previous ulceration. A loose suture in that area was sent for culture and the patient was treated for presumed herpetic keratitis. No organisms grew from the suture, and the patient developed stromal opacities similar to her preoperative appearance. Repeat corneal ulceration (Figure 1C and D) prompted another PKP with removal of a 1-mm-larger disc to encompass the previous graft and additional host cornea affected by ulceration and thinning. Histopathologic analysis of the removed cornea revealed acute and chronic keratitis associated with centrifugal intrastromal colonies of strongly and uniformly gram-positive bacteria with short, stubby branches typical of Actinomyces species (Figure 2). Ziehl-Neelsen acid-fast staining was negative, suggesting Actinomyces rather than Nocardia species. Cultures of stromal tissue again were negative.

Postoperatively, graft and host corneal clarity were maintained. After corneal suture removal, visual acuity of 20/20 was achieved with a rigid gas-permeable contact lens.

Comment. Actinomyces can be a rare cause of infectious crystalline keratopathy, especially in an immune-suppressed condition such as corneal graft. Infectious crystalline keratopathy is a unique corneal infection characterized by the slowly progressive development of needle-like opacities in the corneal stroma, most commonly caused by streptococcal species.1-2 To our knowledge, this is the first case of infectious crystalline keratopathy due to an organism with morphologic characteristics consistent with Actinomyces. There have been other rare reports of Actinomyces corneal infections. Severe corneal ulceration from Actinomyces has been reported,3 as has delayed-onset keratitis after laser in situ keratomileusis.4 In both of these cases, cure was achieved with debridement of the infected cornea and intensive treatment with topical antibiotics, as well as oral penicillin.
in the case of the corneal ulcer. Actinomyces has also been associated with chronic postoperative endophthalmitis that required surgical treatment. Actinomyces species are more commonly encountered in ophthalmology as a cause of canaliculitis. In these cases, Actinomyces is difficult to eradicate medically and usually requires surgical treatment.

Actinomyces can be a difficult organism to grow in culture and is difficult to treat even with aggressive medical treatment. The histopathologic appearance in this case is reminiscent of a biofilm with minimal associated inflammation. Similar to Actinomyces in canaliculitis, our case responded only to surgical excision with wide boundaries for complete removal of the causative agent.

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Figure 1. Clinical slitlamp photographs of Actinomyces infectious crystalline keratopathy at the initial visit (A), immediately following the first repeat penetrating keratoplasty (B), and at low (C) and high (D) magnification of recurrent corneal opacities 6 months later. Arrows indicate temporal opacity within the host cornea.

Figure 2. Histopathologic photomicrographs of intrastromal colonies of filamentous, branching, gram-positive bacteria consistent with Actinomyces species with minimal associated inflammation, showing staining with periodic acid–Schiff stain (original magnification ×100) (A), hematoxylin-eosin stain (original magnification ×1000) (B), and Brown-Hopps stain (original magnification ×1000) (C).
Endogenous Endophthalmitis With Brain Abscesses Caused by *Streptococcus constellatus*

We report a novel case of endogenous endophthalmitis presumably caused by *Streptococcus constellatus*. Concurrently, the patient developed severe lower-extremity weakness. Biopsy of brain abscesses yielded *S constellatus*.

**Report of a Case.** A 54-year-old man with uncontrolled diabetes mellitus had a 1-week history of progressively worsening floaters and poor vision in the left eye. He denied having eye pain. He reported a resolved febrile illness 2 weeks prior with productive cough, nausea, vomiting, and abdominal pain. He denied having eye trauma, recent indwelling catheters, recent intravenous treatment, or intravenous drug use. There was questionably a history of diverticulitis.

Best-corrected visual acuity measured 20/25 OD and light projection OS. No relative afferent pupillary defect was detected. Ocular motility was full and without pain. Intraocular pressures were within normal limits. The examination results of the right eye were unremarkable. Slit-lamp examination of the left eye revealed moderate conjunctival injection, nongranulomatous keratoprecipitates, 3+ anterior chamber cells, and a mild nuclear sclerotic cataract. Significant vitritis precluded a clear view of the retina in the left eye, but an elevated white macular lesion was noted (Figure 1).

Endogenous endophthalmitis was suspected. A diagnostic vitrectomy was performed, and vancomycin hydrochloride, cefazidine, and clindamycin phosphate were injected intravitreally. Vitreous cultures and polymerase chain reaction of vitreous washings for cytomegalovirus, herpes zoster virus, *Candida*, and *Toxoplasma* were negative. Tuberculin purified protein derivative and blood cultures were negative. Results from complete blood count, comprehensive metabolic panel, rapid plasma reagin, fluorescent treponemal antibody absorption, *Toxoplasma* serology, human immunodeficiency virus testing, angiotensin-converting enzyme level, serum lysozyme level, and urinalysis were normal, except for elevated serum and urine glucose levels. Postoperatively, best-corrected visual acuity was counting fingers OS. The macular lesion was soon less apparent and continued to fade postoperatively. Systemic workup to this point was unfruitful. Computed tomography of the chest showed only nonspecific lower-lobe scarring in the right lung.

The patient began to have progressive lower-extremity weakness and slurred speech. He was admitted to the intensive care unit. We consulted the infectious disease service. Magnetic resonance imaging of the brain and spine revealed multiple ring-enhancing lesions (Figure 2). A transesophageal echocardiogram revealed no evidence of shunts or vegetations. Owing to the broad differential diagnosis, a biopsy of the frontal brain lesions was performed; this revealed intracranial abscesses that grew *S constellatus*. On further questioning, the patient revealed a history of tooth extraction 2 months prior to his initial visit. With intravenous treat-