vere trachoma and specifically included the variation among villages in the analysis. Furthermore, our analysis assessed the prevalence of clinically active trachoma in a village as opposed to its presence in an individual. A village-level analysis is most useful for trachoma program managers, who make treatment decisions based on the prevalence of trachoma in a village. Our findings suggest that in areas with hyperendemic trachoma treated through mass distribution of azithromycin, the signs of active trachoma can be expected to resolve slowly, even if antibiotic distributions are quite successful in reducing the prevalence of ocular chlamydia.

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**Intraocular Invasion by Microsporidial Spores in a Case of Stromal Keratitis**

Microbial keratitis is a visually disabling condition caused by a variety of infectious organisms. Although uncommon, microsporidial stromal keratitis has recently been reported as an emerging cause of stromal keratitis. Microbial keratitis is a visually disabling condition caused by a variety of infectious organisms. Although uncommon, microsporidial stromal keratitis has recently been reported as an emerging cause of stromal keratitis. Intraocular microsporidiosis causing endophthalmitis and sclerouveitis with clear cornea has been reported, although there was no evidence of contiguous anterior chamber involvement. We report a rare case of microsporidial stromal keratitis in which microsporidial spores were detected from corneal scraping, corneal tissue, and endothelial exudates.

**Report of a Case.** A 40-year-old woman had intermittent pain, redness, photophobia, and decreased vision in her right eye for 1 year. She had trauma with a leaf 1 year before her initial visit to us. Since then, she had been treated irregularly with topical antiviral medication (acyclovir, 3%) by various health care providers.

On examination, her best-corrected visual acuity was 20/70 OD. Slitlamp biomicroscopic examination of the right eye revealed conjunctival congestion, central stromal edema with Descemet folds, and keratic precipitates (Figure A). Her intraocular pressure was normal. Based on results of the clinical evaluation, a diagnosis of herpetic viral endotheliitis was made. She began treatment with systemic acyclovir, 400 mg 5 times daily, and topical corticosteroid (prednisolone acetate 1%). Although her visual acuity and clinical picture improved, she revisited us 4.5 months after the initial visit with a decrease in vision, tearing, and pain that had lasted 15 days. She was using topical steroid once every other day. Her visual acuity was light perception with projection OD. There was a 3.8×2.7-mm central full-thickness stromal infiltrate. The endothelium showed exudates arranged as a sheet. The corneal scrapings examined in potassium hydroxide and calcofluor white mount (Figure, D). She began intensive treatment with topical polyhexamethylene biguanide, 0.02%, eyedrops. Owing to progressive thinning and descemetocoele formation (Figure, D), she underwent tissue adhesive application 4 days later. After a week with no response to medical therapy, she underwent therapeutic penetrating keratoplasty. Histopathologic examination of the corneal tissue showed multiple microsporidial spores within the stroma as well as in the subendothelial exudates with intact Descemet membrane (Figure, E and F). The postoperative period was uneventful. At the last follow-up, 6 months postoperatively, the graft was clear with visual acuity of 20/200 (Figure, G).

**Comment.** Stromal keratitis due to microsporidium is less common than superficial keratoconjunctivitis. It is frequently misdiagnosed as herpes simplex keratitis. Pre-
vious reports have described various clinical pictures of stromal keratitis such as stromal edema with anterior to mid and/or deep stromal involvement with or without endothelial exudates and corneal opacity. However, to our knowledge, there are no reports documenting microsporidial spores in the endothelial exudates. The finding of intact Descemet membrane in histopathologic examination suggests that microsporidial spores may indeed

Figure. Clinical presentation and microbiological and histopathologic evidence of microsporidial stromal keratitis. A, Slitlamp picture showing corneal edema with Descemet folds during the initial visit. B, Clump of oval spores in the corneal scraping smear stained with calcofluor white and seen with a fluorescent microscope (original magnification ×400). C, Gram staining of corneal scraping showing intracellular and extracellular, oval, stippled, gram-positive, well-defined spores of microsporidia (original magnification ×1000). D, Slitlamp picture showing full-thickness stromal infiltrate with severe thinning. E, Section of the cornea showing subtle inflammation of the stroma with a focus of exudates posterior to Descemet membrane, suggestive of keratic precipitates (arrow) (hematoxylin-eosin, original magnification ×400). F, The exudates showing a single focus of microsporidia (arrow) (1% acid-fast stain, original magnification ×1000). G, Slitlamp picture showing clear corneal graft without any recurrence.
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 endothe-litis 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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Actinomycoses 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 Actinomycosis 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 phaco-emulsification 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 ceftazolin 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-Neelsson 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 a 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 debriement of the infected cornea and intensive treatment with topical antibiotics, as well as oral penicillin.