Trachoma, caused by infection with ocular strains of chlamydia, is the leading infectious cause of blindness worldwide. The World Health Organization recommends that in districts where the prevalence of clinically active trachoma exceeds 10% in children aged 1 to 9 years, communities should receive 3 annual mass antibiotic distributions followed by clinical reassessment; any communities with persistent trachoma should continue receiving annual mass antibiotic treatments until the prevalence of clinically active trachoma in children aged 1 to 9 years falls below 5%.

Although trachoma treatment decisions are based on the prevalence of clinically active trachoma, it is unclear how quickly the clinical signs of trachoma resolve once infection has been cleared, especially in areas with severe trachoma. We recently performed a series of cluster-randomized clinical trials for trachoma in an area of Ethiopia with hyperendemic trachoma. In these trials, infection was brought to a low level in 24 villages randomized to receive mass azithromycin treatments every 6 months. This provided an opportunity to determine the rate of resolution of the clinical signs of trachoma given little to no chlamydial reinfection.

**Methods.** Twenty-four randomly selected villages received mass azithromycin treatment every 6 months for 18 months. In these villages, we monitored the prevalence of ocular chlamydia and clinically active trachoma in all children aged 1 to 5 years 2 months after baseline and then every 6 months after baseline for 24 months. Monitoring preceded mass antibiotic treatment at each of the biannual visits. Ocular chlamydial prevalence was assessed using maximum likelihood estimations from pooled chlamydial polymerase chain reaction (Amplicor; Roche Diagnostics, Branchburg, New Jersey) as described previously. Clinically active trachoma was defined as follicular trachoma and/or intense inflammatory trachoma according to the World Health Organization’s simplified grading system. Clinical examiners were trained and validated against gold-standard examiners as described previously.

The primary outcome was the resolution rate of clinically active trachoma at the village level. We used a generalized estimating equation to predict the log prevalence of clinically active trachoma during biannual monitoring visits, using time and the log baseline prevalence of clinically active trachoma as fixed effects, village as a random effect, a first-order autoregressive correlation structure, and the Huber-White sandwich estimate of variance. Use of the log-transformed outcome was decided a priori and was supported by plots of the residual vs fitted values. Analyses were performed with Stata version 10.0 statistical software (StataCorp LP, College Station, Texas).

**Results.** The mean prevalence of ocular chlamydial infection in children before the baseline mass azithromycin treatment was 52.9% (95% confidence interval, 44.2%-61.3%) (Figure). Mass azithromycin distribution subsequently occurred at months 0, 6, 12, and 18. The mean prevalence of ocular chlamydia in children during posttreatment biannual monitoring visits was 5.1% (95% confidence interval, 3.6%-6.6%). During these 24 months, the prevalence of clinically active trachoma decreased by 33.3% (95% confidence interval, 28.6%-38.0%) per year as estimated from the generalized estimating equation (Figure).

**Comment.** In a hyperendemic area, repeated biannual mass antibiotic treatments reduced the level of ocular chlamydial infection in children to a low level. The prevalence of the clinical signs of active trachoma declined at a much slower rate of 33.3% per year. This rate of resolution is somewhat lower than that reported elsewhere. However, prior reports have been conducted in fewer villages, with less severe trachoma. In contrast, this study was conducted in many villages in an area with se-
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.1 Intraocular microsporidiosis causing endophthalmitis and sclerouveitis with clear cornea has been reported,2,3 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 intraocar 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, B) showed multiple oval microsporidial spores. The spores were also seen in Gram staining of the corneal scraping (Figure, C). She began intensive treatment with topical polyhexamethylene biguanide, 0.02%, eyedrops. Owing to progressive thinning and descemetocele 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-