corneal abrasion. Chloramphenicol ointment was prescribed, and the patient was advised to tape her left eyelid shut at night. Four days later, corneal exposure with a persistent corneal abrasion was still evident. Surgical tarsorrhaphy was refused; however, the patient consented to tarsorrhaphy with Indermil. The tarsorrhaphy lasted 7 days, during which the corneal abrasion healed. Function of the left facial muscles was restored during this period, and the exposure keratopathy resolved.

Case 3. A 40-year-old man was seen in the eye casualty with a 4-day history of facial nerve palsy secondary to herpes zoster infection. Corneal exposure with a central abrasion was evident. There was 5 mm of lagophthalmos. He was prescribed chloramphenicol ointment and advised to tape his eyelids shut at night. He subsequently developed a skin allergy to the tape and discontinued its use. A subsequent examination found a persistent central corneal abrasion. The patient refused surgical tarsorrhaphy but was agreeable to lateral tarsorrhaphy with Indermil. The first tarsorrhaphy lasted 6 days but his lower eyelid function remained poor, and worsening of the exposure keratitis occurred over the next 5 days. Glue tarsorrhaphy with Indermil was performed on 2 other occasions, each lasting 7 days. At the end of the third application, the corneal defect had healed and eyelid function recovered satisfactorily.

Comment. Exposure keratitis is a complication of facial nerve palsy. Without treatment, this may lead to corneal ulceration with severe visual loss from scarring and infection. In mild cases, exposure keratopathy can be managed conservatively with copious lubricants and eyelid taping, but in severely affected corneas, eyelid closure may be required to maintain corneal integrity. Surgical tarsorrhaphy and botulinum toxin–induced ptosis are 2 well-recognized methods of providing corneal protection. Both are effective but have their disadvantages.

Surgical tarsorrhaphy may be divided into suture tarsorrhaphy over bolsters (for short-term use) and reversible permanent eyelid adhesion tarsorrhaphy (for longer-term use). The former is commonly accepted as the gold standard for temporary eyelid closure. However, both forms of surgical tarsorrhaphy are time consuming, and there may be a risk of permanent scarring to the eyelids from surgery. In addition, patients often refuse surgical tarsorrhaphy for cosmetic reasons. On the other hand, botulinum toxin may not be available universally because of constraints of cost and expertise. Moreover, the induced ptosis is variable in its onset and duration, and there are risks associated with the injection.

An alternative approach is to perform glue-assisted tarsorrhaphy. Indermil is a tissue adhesive (N-butyl-2-cyanoacrylate monomer) that is widely used in surgery for the closure of skin wounds and internal wounds without the need for suturing.1 The use of Indermil has also been described in obstetric and gynecologic procedures, otolaryngologic procedures, hand surgery, and plastic and reconstructive surgery. The use of cyanoacrylate glue has been described previously in the management of corneal epithelial defects2 and other ocular problems.3 However, there has been little documentation to date on the successful use of licensed medical preparations of tissue glue for tarsorrhaphy.

Indermil-assisted tarsorrhaphy lasts for about a week and can easily be repeated when necessary. With regard to safety, a previous case series has suggested that there is no long-term morbidity from superglue contact with the eye.4 The technique is not a replacement for surgical tarsorrhaphy; however, it may be considered as an alternative in certain situations. First, the technique can be used to provide short-term corneal protection prior to recovery of facial nerve palsy. Second, it may serve as a temporary measure for exposure keratopathy while awaiting more definitive treatment. Third, it is of value in patients who refuse surgical intervention.

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The diagnosis of central serous choriotereinopathy (CSC) was considered, and the oral prednisone was gradually reduced.

On June 13, 2002, the patient’s visual acuity was 20/60 OU. The patient stated that he had fainted the day before and was not feeling well. He was receiving 5 mg of prednisone daily and 25 mg of methotrexate weekly. The prednisone was increased to 10 mg daily, and he was scheduled to see a neurologist.

On June 17, 2002, the patient sought care at the emergency department with the worst headache he had ever had. He reported loss of consciousness and a possible seizure. He received phenytoin sodium and dexamethasone intravenously. Magnetic resonance imaging showed multifocal cystic lesions in the lateral ventricles bilaterally, with gross dilation of the lateral and third ventricles consistent with obstructive hydrocephalus (Figure 2). Neurocysticercosis was diagnosed and treated with 400 mg of albendazole twice daily for 8 days, phenytoin, and a ventriculoperitoneal shunt. Subsequently, his visual acuity improved to 20/40 OD and 20/30 OS, and his serous retinal detachments resolved. Phenytoin remained his sole treatment.

Comment. This case was a confusing one of a Mexican American man with serous retinal detachments, neck stiffness, and headaches mimicking Vogt-Koyanagi-Harada syndrome, although there was no inflammation in the vitreous. We now believe he had CSC causing visual symptoms and neurocysticercosis causing neck stiffness and headache. We do not know if the increased intracranial pressure and its stress with possible concomitant glucocorticoid production played a role in his severe CSC. Glucocorticoid use has been associated with subretinal fibrin in CSC.

The oral prednisone and periocular and intraocular triamcinolone did not improve the CSC and ultimately worsened it. The correct diagnosis with tapering of corticosteroids caused resolution of the serous fluid but worsened the symptoms of hydrocephalus from the neurocysticercosis.

This case shows that it is important to make the distinction between CSC and inflammatory causes of serous retinal detachments because corticosteroids will make CSC worse. Another learning point is that serous fluid due to Vogt-Koyanagi-Harada syndrome should promptly resolve after treatment.

Figure 1. A, Fundus photographs obtained in May 2002 show multiple serous detachments with fibrinous exudates in each eye. B, Corresponding fluorescein angiograms obtained in each eye. The image at 50 seconds (left) reveals early hyperfluorescence in the right eye, which shows late leakage at 329 seconds (middle). At 603.5 seconds (right), the left eye shows late leakage.
Diagnosis of Microsporidia Keratitis by Polymerase Chain Reaction

Report of a Case. A 33-year-old man positive for human immunodeficiency virus had a 6-month history of bilateral blurred vision, tearing, and photophobia, with associated conjunctival hyperemia. The patient had been previously treated with topical antibiotic drops, topical steroids, and topical nonsteroidal anti-inflammatory drops for several months without resolution of his symptoms. He was being highly active antiretroviral therapy as well as trimethoprim-sulfamethoxazole prophylaxis; his CD4 cell count was 3. His medical history included weight loss and diarrhea. His ocular history was otherwise unremarkable. His uncorrected visual acuity was 20/70 OD and 20/50 OS with pinhole to 20/50 OD and 20/30 OS. The conjunctiva had 2+ hyperemia with a moderate papillary reaction bilaterally. The corneas exhibited diffusely distributed small, gray intracellular lesions without associated stromal infiltrate (Figure 1). There was no anterior chamber reaction, and the posterior segments were unremarkable.

The patient underwent a conjunctival biopsy and cytologic smear examination. Gram stain, bacterial culture, fungal culture, chlamydial testing, and viral culture were negative. Cytologic examination was negative for microsporidia or typical viral or chlamydial inclusion bodies. A fine-needle corneal epithelial scraping was performed. The scraped sample was suspended in 50 µL of phosphate-buffered saline and boiled for 15 minutes. Polymerase chain reaction (PCR) for microsporidia was performed using a protocol adapted from Muller et al,1 which is capable of identifying several Enterocytozoon and Encephalitozoon species of microsporidia. Briefly, 5 µL of the sample was subjected to 35 cycles of thermocycling using forward primer V1 (5’-CACCAAGGTATTCT-GCCTGAC-3’) and reverse primer PMP2 (5’-CCTCTCCGGAAAC-CAAACCGTT-3’) with 1-minute denaturation at 94°C, 2-minute annealing at 60°C, and 3-minute extension at 72°C. A single ~270-base pair fragment was observed on agarose gel electrophoresis and ethidium bromide staining of the PCR-amplified patient sample, but not from the phosphate-buffered saline-only control sample (Figure 2A). The PCR product was directly sequenced. A BLAST search of the National Center for Biotechnology Information database revealed a near-perfect alignment with the ribosomal RNA small unit gene of Encephalitozoon hellum (Figure 2B).

The patient was prescribed hourly 1% topical clotrimazole and 100 mg of oral itraconazole 2 times daily, and showed gradual improvement in his symptoms and clinical findings during a 2-week period.

Comment. Microsporidia are a group of at least 6 genera of intracellular protozoa that are frequent opportunistic pathogens in immunocompromised patients, particularly infecting the gut. Diagnosis is