macular edema was the result of RPE dysfunction, since the leakage observed on FFA appeared to come from RPE and responded well to oral acetzolamide therapy. Furthermore, diffuse RPE changes were present on FFA and no abnormalities on the retinal vessels were seen.

Evidence suggests that retinoids are directly involved in the formation and accumulation of lipofuscin in the RPE that, in its turn, could compromise RPE function. However, the mechanism by which acitretin could have caused the RPE changes observed in this patient remains uncertain. Although a dysfunction in the fluid-pumping mechanism of the RPE appeared to be present in this patient, the alteration of the RPE was not severe enough to compromise the supporting role of the RPE on photoreceptor cell function and, thus, to impair the PERG. This is, however, not surprising. Salzman et al found that 47% of patients with aphakic macular edema had a normal PERG. Furthermore, diffuse RPE abnormalities on fundus autofluorescence images have been detected in some patients with a normal PERG (N. L. and G. E. Holder, PhD, unpublished data, 1998).

Because symptoms, VA, and macular abnormalities appeared months after initiating acitretin therapy and have continued to improve since its discontinuation, it is possible that this drug may have played a role in their occurrence. However, this could be confirmed only if the above anatomical and functional changes reappear following reinitiation of the therapy. Because it is not yet clear whether these changes are totally reversible, treatment with acitretin has not been reestablished.

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Figure 2. Fluorescein angiogram obtained 3 days after the initial examination. Although diffuse hyperfluorescence at the level of the retinal pigment epithelium was still observed, cystoid macular edema had completely resolved.


Self-induced, Bilateral Retinal Detachment in Tourette Syndrome

In 1885, the French neurologist Georges Gilles de la Tourette described 9 patients with childhood-onset tics accompanied in some by uncontrollable noises and utterances, as well as hyperactivity and obsessive-compulsive behavior. The current diagnosis of Tourette syndrome, according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), involves multiple motor tics and at least 1 vocal tic, which occur many times a day, nearly every day, or intermittently for more than 1 year. Tics must begin before age 18 years. The average age of onset is 7 years, and boys are more commonly affected than girls. Motor tics are characterized by involuntary movements such as facial grimacing, frequent eye blinking, blepharospasm, spitting, and arm jerking. Vocal tics often have an aggressive or sexual component, such as gruntin, barking, echolalia, and coprolalia (uncontrolled swearing). The condition often results in deleterious social consequences. We report a case of self-induced bilateral retinal detachment in a young man with Tourette syndrome who was

initially referred for monocular hyphema.

**Report of a Case.** A 25-year-old white man was initially seen with a 1-week history of floaters and decreased vision in his left eye. The patient had been diagnosed with Tourette syndrome at age 7 years, obsessive-compulsive disorder at age 11 years, and depression at age 24 years. His motor tics involved excessive blinking, blepharospasm, clapping, jabbing his fingers into his eyes, and punching himself in the periorbital area. The patient was taking buspirone hydrochloride (10 mg twice a day) and clomipramine hydrochloride (25 mg twice a day). On examination, the patient was alert and oriented, and he had no evidence of cognitive impairment. Visual acuity was 20/200 OD and hand motion OS. There was no afferent pupillary defect. Intraocular pressures were 18 OD and 16 OS. Slit-lamp examination findings of the right eye demonstrated pigment deposits on the corneal endothelium, moderate (2+) aqueous pigmented cells, and posterior subcapsular cataract. The left eye had a less than 1-mm hyphema and many (4+) circulating red blood cells in the anterior chamber, as well as a dense posterior subcapsular cataract. Funduscopy results revealed a retinal dialysis from the 1:30 to the 4:30 clock position with a macula-on-retinal detachment in the right eye. Vitreous hemorrhage was present centrally in the left eye, and there were nasal and temporal giant retinal tears. The right eye was repaired with a scleral buckling procedure. The left eye underwent anterior segment washout, pars plana lensectomy, pars plana vitrectomy, endolaser, and silicone oil injection. Intraoperatively, the giant retinal tears were found to extend from the 12:30 to the 4:30 clock position with 4 long radial extensions to the temporal macula and from the 6-o’clock to the 11-o’clock position with 1 long radial extension to the optic disc. There was an additional radially oriented posterior retinal break. Postoperatively, the retinas were attached in both eyes. One month later, the left eye developed proliferative vitreoretinopathy with retinal detachment and underwent reoperation. At 6 months, the retinas remained attached and the visual acuity was 20/100 OU.

**Comment.** Ophthalmic manifestations of Tourette syndrome include frequent blinking and blepharospasm, gaze deviations and abnormal saccades, and accidental and self-inflicted ocular injuries.1-3 The retinal detachments in our patient were most likely the result of repeated, self-induced finger jabbing to the eyes since the patient had no other risk factors for retinal detachment. In patients with retinal detachment, factors suggesting a traumatic etiology typically include unilateral vitreoretinal findings, retinal dialysis or giant retinal tear, and age younger than 40 years.4-5 However, in patients with self-induced or repeated trauma, the vitreoretinal pathologic features may be bilateral, as demonstrated by our patient. To prevent further self-injury, patients should wear protective polycarbonate goggles, and they should be monitored closely in conjunction with the psychiatry service. Treatment of the underlying disorder with behavior modification and pharmacotherapy is essential, and pharmacological agents that antagonize dopamine are most effective in reducing the severity of motor and vocal tics.

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**Correction**

**Error in Signature.** In the Clinicopathologic Reports, Case Reports, and Small Case Series titled “Leukocoria Caused by Intraocular Heterotopic Brain Tissue,” published in the March 2004 issue of the ARCHIVES (2004;122:390-393), an error occurred in the signature. On page 393, the signature should have appeared as follows: Sarit Patel, MD, Madison, Wis; Joanne Dondey, MD, Helen S. L. Chan, MB, BS, Elise Heon, MD, Susan Blaser, MD, Toronto, Ontario; Daniel Albert, MD, Madison; and Brenda L. Gallie, MD, Toronto. Drs Dondey and Patel contributed equally to this article. The journal regrets the error.