vitreous hemorrhage or a rhegmatogenous retinal detachment.1 The abnormal retinoschisis cavity is filled with 20% sulfur hexafluoride gas and the patient maintained face-down positioning for 1 week.

One week postoperatively, the retinal detachment had resolved, and the visual acuity improved to 20/200 OD. At the 1 month visit, the visual acuity remained 20/200 OD, and the macular contour had improved (Figure 3A). Optical coherence tomography showed foveal cysts, but the subretinal fluid was gone (Figure 3B).

Shortly thereafter, the patient complained of sudden vision loss in the left eye. The visual acuity was counting fingers at 2 ft OS. The clinical appearance was very similar to the initial examination of the right eye, showing a shallow posterior pole retinal detachment with outer retinal corrugations and inner retinal cysts, with no visible retinal breaks. The patient underwent a pars plana vitrectomy, mechanical separation of the hyaloid, and gas tamponade with 20% sulfur hexafluoride gas in this eye. One week later, the visual acuity had improved to 20/200, and the subretinal fluid had resorbed, but the foveal cysts remained.

At the 3-month postoperative visit for the left eye (4 months after surgery in the right eye), the visual acuity had improved to 20/100 OD and 20/60 OS. The retina remained flat and attached in both eyes, with optical coherence tomography showing an overall decrease in the amount of detachment and a decrease in the foveal cysts in the left eye.

Comment. Patients with XLR have a defect in the XLRS1 gene, which encodes retinoschisin, a protein that is believed to be essential to cellular adhesion. The abnormal retinoschisin may cause dysfunction of the Muller cells, which results in a schisis cavity.2 Most patients with XLR have mild to moderate vision loss due to foveal schisis, and this can gradually worsen during adulthood.3 Cases of severe vision loss are usually due to vitreous hemorrhage or a rhegmatogenous retinal detachment. Up to 22% of patients develop a rhegmatogenous retinal detachment attributable to peripheral retinal breaks.2

There are several reports detailing the surgical results of scleral buckling and vitrectomy for the repair of the rhegmatogenous retinal detachments in these patients.3,4,5 We are unaware of any previous reports in the literature describing nonrhegmatogenous macular retinal detachment as a cause of vision loss in XLR and could find no reference to it on a MEDLINE search.

We have described a patient with XLR who was initially seen with bilateral, sequential, macular retinal detachments, which were repaired via vitrectomy with short-acting gas tamponade. While pronounced corrugations suggest a rhegmatogenous origin, both clinical and intraoperative examinations failed to demonstrate retinal breaks, pigment, or hemorrhage in either eye. These could be exudative macular detachments. However, no other causes suggestive of an exudative process were identified, no leakage was present on the angiogram, and corrugations would not be expected. We believe that this case represents a variant of vitreomacular traction, which, when combined with the defective cellular adhesion of juvenile XLR, resulted in such a striking appearance.6 It is possible that the traction caused an enormous schisis cavity and relief of the traction improved the retinal contour. Gas tamponade was used to aid in the closure of a possible occult inner wall hole within the schisis cavity, but this may not have been necessary. Our experience suggests that these detachments may respond well to vitrectomy surgery with removal of the posterior hyaloid in combination with short-term gas tamponade.

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Infliximab-Associated Third Nerve Palsy

A third nerve palsy (TNP) may show gadolinium enhancement of the cisternal segment of the oculomotor nerve on magnetic resonance imaging. Causes include inflammation, infection, neoplasm, ophthalmoplegic migraine, and demyelination. Infliximab, a tumor necrosis factor (TNF) α inhibitor, may cause demyelination or increase relapses in patients with multiple sclerosis.1 We report a patient who developed a TNP associated with infliximab use.

Report of a Case. A 47-year-old man with rheumatoid arthritis received monthly infusions of 300 mg of infliximab since December 2002. In February 2004, he was initially seen with painless ptosis of his right upper eyelid along with double vision in left and upgaze.

On examination, he had minimal ptosis and limitation of elevation and adduction of the right eye. Pupils were equal in size and reactivity. Visual acuity, dilated fundus examination, neurologic examination, and review of systems were unremarkable. Other medications included 400 mg of hydroxychloroquine daily and 10 mg of methotrexate weekly. He took latanoprost and carteolol hydrochloride for glaucoma.

Results of rapid plasma reagin, fluorescent treponemal antibody
absorption, and angiotensin-converting enzyme tests; blood chemistry; chest computed tomography; and acetylcholine receptor antibody and Tensilon tests were normal. Lumbar puncture findings were unremarkable including cell counts, protein level, cytology, flow cytometry, VDRL test, oligoclonal bands, and fungal cultures. Brain magnetic resonance imaging showed gadolinium enhancement of the cisternal segment of the right oculomotor nerve. There were no white matter lesions or dural enhancement (Figure 1). After stopping infliximab administration, the diplopia and ptosis gradually resolved during 3 months. Repeat magnetic resonance imaging showed resolution of the oculomotor nerve enhancement (Figure 2). No new neurologic symptoms had developed after 16 months' follow-up.

Comment. Infliximab is a chimeric monoclonal antibody against TNF-α.

Figure 1. High-resolution axial (A) and coronal (B) fat-suppressed, gadolinium-enhanced, T1-weighted images demonstrate prominent enhancement of the cisternal segment of the right third cranial nerve (arrow).

Figure 2. A and B. Similar follow-up images in the same patient show resolution of the abnormal enhancement of the right third cranial nerve.
Early studies demonstrated that anti-TNF antibodies protected animals from developing experimental autoimmune encephalomyelitis. However, a double-blind, placebo-controlled study of 168 patients with multiple sclerosis showed that patients taking an anti–TNF-α agent received no benefit in the treatment group. Instead, significantly more relapses occurred than in the placebo group. Therefore, this class of drugs is now contraindicated in patients with multiple sclerosis. Postmarketing surveillance data in 2002 revealed 64 cases of demyelinating polyradiculoneuropathy associated with infliximab use in patients with multiple sclerosis.3 Although, to our knowledge, demyelinating TNP has not been reported previously, others have described peripheral, multifocal motor neuropathy occurring 3 to 24 months after initiation of infliximab therapy.

Several disorders may cause enhancement of the cisternal segment and palsy of the oculomotor nerve. The transient and isolated nature of the palsy described herein suggests demyelination. Evaluation did not reveal evidence of infection, inflammation, or migraine. Seven cases of demyelinating TNP associated with enhancement of the oculomotor nerve cisternal segment were reported in patients with multiple sclerosis. Although, to our knowledge, demyelinating TNP has not been reported previously, others have described peripheral, multifocal motor neuropathy occurring 3 to 24 months after initiation of infliximab therapy.

The complete effect of infliximab on the immune response is not entirely understood, and therefore, its long-term safety remains unknown. Infliximab may increase the risk of demyelination, which we believe caused the TNP in our patient. Although coincidence is a possibility, physicians should be aware of the potential association when confronted with a patient receiving a TNF-α inhibitor who develops a demyelinating event.

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### Choroidal Lesions Preceding Symptom Onset in Birdshot Chorioretinopathy

Birdshot chorioretinopathy was first characterized by Ryan and Maumenee1 as a chronic intraocular inflammatory condition with discrete, depigmented spots scattered throughout the fundus; mild vitritis; and vasculitis. A very strong association of this condition with the HLA-A29 gene and electrophysiologic abnormalities have since been described.2,3

In most cases, the patient's initial symptoms are blurred vision, floaters, and/or photopsia and ocular signs consistent with birdshot chorioretinopathy.4,5 A few reports have described the appearance of the classic fundal spots long after the onset of symptoms, inflammation, and vasculitis, but these seem to be in the minority.6 We are unaware of any report of the appearance of the classic fundal lesions of birdshot chorioretinopathy prior to the onset of symptoms. Herein we describe such a case.

**Report of a Case.** A 33-year-old white man was first seen with a 3-week history of bilateral floaters. There were no associated or preceding ocular or systemic symptoms. His history included moderate myopia and blunt left ocular trauma in early childhood, after which a small scar at the posterior pole associated with decreased visual acuity was noted. Follow-up after this trauma revealed no change in either the scar or visual acuity.

Initial examination findings revealed a best-corrected visual acuity of 6/5 OD and 6/12 OS. Neither eye had dilated conjunctival vessels. Anterior segment examination revealed a trace of cells bilaterally, very few fine inferior keratic precipitates, and no granulomatous changes. A mild to moderate vitritis was noted in both eyes. Fundal examination ([Figure 1](#fig1)) revealed large areas of pale, flat depigmented lesions typical of birdshot chorioretinopathy, associated with a few isolated areas of retinal vas-

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**Figure 1.** Fundal photographs taken at initial examination showing typical birdshot chorioretinal lesions (black arrows) in both eyes (A-C) and an old macular scar in the left eye (asterisk)(B). C, Areas of peripheral vasculitis (white arrow) are shown.

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