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.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 viretomaculation 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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Financial Disclosure: None reported.


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.
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 demyelination associated with infliximab use including central nervous system demyelination, chronic inflammatory demyelinating polyradiculoneuropathy, and “neuropathy.”

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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Financial Disclosure: None reported.

Funding/Support: This study was supported in part by an unrestricted grant from Research to Prevent Blindness, New York, NY (Dr Lee).

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.4,4.5 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 keratitic precipitates, and no granulomatous changes. A mild to moderate vitritis was noted in both eyes. Fundal examination (Figure 1) revealed large areas of pale, flat depigmented lesions typical of birdshot chorioretinopathy, associated with a few isolated areas of retinal vasculitis.