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 patients with multiple sclerosis.2002 revealed 64 cases of demyelination, chronic inflammatory demyelination, 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 peripherally, 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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Figure 1. Fundal photographs taken at initial examination showing typical birdshot choroidal 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.

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 revealed large areas of pale, flat depigmented lesions typical of birdshot chorioretinopathy, associated with a few isolated areas of retinal vas-
culitis and intraretinal hemorrhages bilaterally. An old atrophic scar was noted in the left macula, with the right macula appearing normal. There were no vitreal snow balls, snow banking, punched-out chorioretinal scars, nor any other complications.

Investigations included a full blood examination and film, calcium levels, syphilis serology, angiotensin-converting enzyme assay, chest radiography, and HLA-A29 typing. All results were normal except for the HLA-A29 typing, which was positive. Electrophysiology revealed delayed scotopic blue and photopic red b wave amplitudes. Fluorescein angiography revealed areas of vasculitis and typical late staining of the birdshot lesions.

Six months prior to the onset of symptoms, the patient, who works in an eye research institution, volunteered to have his fundi photographed for staff training purposes. Review of these photographs revealed his preexisting left macular scar and the presence of the typical depigmented birdshot chorioretinopathy lesions, retinal hemorrhages, and patchy retinal vasculitis (Figure 2).

Comment. Typically, the multiple depigmented lesions of birdshot chorioretinopathy are seen at the same time as the patient’s initial examination for onset of symptoms.1,4,5 This case appears to be a typical manifestation of birdshot chorioretinopathy. It was purely coincidental that fundus photographs were taken 6 months prior to the onset of symptoms. It is quite possible that the fundal lesions of birdshot chorioretinopathy precede the onset of symptoms by some months but remain undetected in an asymptomatic subject who has no reason to be seen by an ophthalmologist.

The pathogenesis of birdshot chorioretinopathy remains unknown but there are several theories.1,3,6 Some place the disease focus in the choroid, which is supported by indocyanine green angiography findings. However, fluorescein angiography and electrophysiologic findings implicate inner retinal dysfunction secondary to retinal vasculitis.3,5,7 Our patient is interesting in that he shows that both the retinal vasculitis and choroidal lesions can be present very early in the disease process.

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Tadalafil-Induced Subretinal and Choroidal Hemorrhage in a Patient With an Unsuspected Uveal (Choroidal and Ciliary Body) Melanoma

Tadalafil (Cialis; Lilly ICOS LLC, Bothell, Wash) is a Food and Drug Administration–approved phosphodiesterase type 5 (PDE5) enzyme inhibitor approved for the treatment of erectile dysfunction. Although preclinical testing of tadalafil and sildenafil citrate (Viagra; Pfizer Inc, New York, NY) included an extended eye examination, electoretinography, and postmortem histologic analysis and no adverse effects were seen, a variety of studies have subsequently highlighted ophthalmic problems with both agents. Transient changes in vision, transient and mild impairment in color discrimination, eye pain, eyelid swelling, electroretinographic abnormalities,1-3 abnormal histopathologic findings,7 pupil-sparing third nerve palsy,8 and central serous chorioidopathy have been reported.9

We recently observed a male patient who suddenly developed a painful red eye and loss of vision after taking tadalafil and was found to have ruptured blood vessels in and on the surface of an ocular melanoma. The acute bleeding may be related to the vasodilatory effects of the drug on the ocular circulation in a patient taking aspirin.

Report of a Case. A 63-year-old man with erectile dysfunction was awakened by severe pain in his left eye a few hours after taking a single tadalafil tablet (200 mg). He went back to sleep, but in the morning he awakened with limited sight in his left eye. His initial examination that morning by an ophthalmologist demonstrated a markedly red eye, and he was treated with topical ketorolac tromethamine eyedrops (Acular; Allergan, Irvine, Calif) for what was initially diagnosed as noninnfectious conjunctivitis. His medical his-