Milkowski2 reveals numerous cases to remove the glass IOFB. Gopal et al provides additional security. In this study that the "migration of intraocular glass is always from back forwards."2 A similar case with anterior migration is presented herein as case 3. However, late posterior segment migration as in cases 1 and 2, causing additional retinal pathologic conditions or symptoms must be rare and, to our knowledge, have not been previously reported.

We describe 3 eyes with glass IOFBs that migrated after they were deemed stable with partial encapsulation or entrapment in the vitreous base. In 2 cases, partially encapsulated glass IOFBs caused late posterior segment findings including retinal laceration and obstruction of macular vision. Though the late posterior migration of retained glass IOFB is considered rare, these cases highlight the need for close follow-up in such cases. Early intervention with glass IOFB removal must be weighed against the hazards of removal and the necessity of close follow-up.

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Central Serous Chorioretinopathy Associated With Periocular Corticosteroid Injection Treatment for HLA-B27-Associated Iritis

Central serous chorioretinopathy (CSCR) is characterized by accumulation of fluid under the neurosensory retina and/or retinal pigment epithelium, resulting in a serous detachment that often involves the macula. Although the exact mechanism producing CSCR is unknown, increased adrenergic stimulation has been hypothesized to play a role.1 Furthermore, elevated corticosteroid levels after systemic administration or secondary to endogenous sources have been implicated in the causation or exacerbation of CSCR.2 Subtenon local corticosteroid injection is effective in the treatment of certain forms of uveitis. This report details a case of CSCR that developed after a single local subtenon corticosteroid injection to treat HLA-B27–associated iritis and was confirmed by optical coherence tomography (OCT).

Report of a Case. A healthy 37-year-old man was examined because of a 10-day history of progressive blurred vision, photophobia, and floaters in the left eye. Medical and social histories were noncontributory. The patient had had an upper respiratory tract infection 2 weeks before the examination. A review of systems was negative for gastrointestinal, genitourinary, dermatological, or musculoskeletal symptoms, including joint or back pain.

Best-corrected visual acuity measured 20/20 OD and 20/50 OS. Intraocular pressure was 21 mm Hg OD and 16 mm Hg OS. Results of an examination of the anterior and posterior segment of the right eye were normal. However, the anterior segment of the left eye showed 2+ con-
Junctival injection and mild keratitis precipitates. There was a 2+ anterior-chamber cellular reaction with a 1-mm hypopyon, engorged iris vessels, and fibrinous iris posterior synechiae that were released after pupillary dilation. Binocular and indirect ophthalmoscopy of the left eye showed a normal optic nerve, macula, retinal vasculature, and periphery. There was no evidence of retinal or vitreous inflammation, vasculitis, or cystoid macular edema (CME). The fovea was well visualized after pupillary dilation, with a normal and distinct foveal reflex.

Although this was the first episode of iritis, systemic evaluation was performed because of the fibrinous anterior-chamber reaction and hypopyon. HLA-B27 iritis was suspected and subsequently confirmed with positive serotyping results. All other tests produced normal findings, including a complete blood cell count, an erythrocyte sedimentation rate, a chest x-ray, and fluorescent treponemal antibody absorption, angiotensin-converting enzyme, lysozyme, antinuclear antibody, and purified protein derivative of tuberculin tests. Treatment with 1% prednisolone acetate every hour and cycloplegic eye drops was commenced, and a 1.0-mL periocular injection of triamcinolone acetonide corticosteroid (40 mg/mL) was given in the subtenon space of the left eye. Within 1 week, there was marked therapeutic response with complete resolution of the hypopyon and fibrin deposition and partial improvement of acuity to 20/40 OS. The occasional residual anterior-chamber inflammatory cell (<1 per high-power field) was noted. The ocular media was clear. Macular biomicroscopy revealed the new development of subretinal fluid and a serious pigment epithelial detachment at the fovea. Fluorescein angiography confirmed an enlarging pinpoint spot of hyperfluorescence, as may be observed with CSCR (Figure 1). The fluorescein leakage was not petalloid and did not demonstrate CME. Optical coherence tomography confirmed the subretinal location of this fluid collection, which was consistent with the diagnosis of CSCR (Figure 2A). The topical corticosteroid drops were rapidly tapered and discontinued over 5 days, and the patient was followed up with serial clinical and OCT examinations. He showed progressive reduction in subretinal fluid, with gradual improvement in visual acuity. By 12 weeks after the initial examination, the fluid had resolved and visual acuity recovered to 20/20 OS. There were no sequelae of ocular inflammation, and OCT confirmed resolution of the foveal contour toward normal (Figure 2B).

Comment. Central serous chorioretinopathy is considered an idiopathic disorder and the exact pathogenesis is uncertain. Speculation regarding adrenergic stimulation as a mechanistic cause has been based on the association of CSCR with emotional stress, type A personality, and pregnancy. Hypercortisolism has also been implicated in the etiology, as increased endogenous cortisol levels and exogenous corticosteroid use have been associated with the development or exacerbation of CSCR. This mechanism is presumed to act via damage to the choroidal circulation. Such damage may result from increased catecholamine-mediated vasoconstriction in the choroid, or the choroid may become hyperpermeable because of increased capillary fragility caused by elevated cortisol levels. Alternatively, corticosteroids may damage retinal pigment epithelial cells directly through inhibition of extracellular matrix components and decreased fibroblastic activity. Although the exact sequence of events in this cause-effect relationship is not understood, evidence from multiple studies suggests that the role of corticosteroids in this disorder is not coincidental.

Exogenous corticosteroids that are inhaled or that are administered orally, intravenously, or by epidural or, most recently, intra-articular injection have been associated with development of CSCR. To our knowledge, this is
the initial report detailing the onset of CSCR following periorcular corticosteroid injection with confirmation by OCT imaging. Periorcular corticosteroids may be administered for the treatment of uveitis as well as postoperative CME. The presence of intraocular inflammation secondary to uveitis may affect the retinal pigment epithelial barrier and increase susceptibility to local periorcular corticosteroid effects. A similar mechanism has been proposed in a small series that reported CSCR following systemic corticosteroid therapy for uveitis that occurred secondary to bird-shot chorioretinopathy, Vogt-Koyanagi-Harada disease, and scleritis.

A variety of contributing factors may account for the paucity of reports linking CSCR and the common procedure of periorcular corticosteroid injection. In eyes with uveitis, clinical features such as synechiae, pupil constriction, or media opacity severe enough to warrant periorcular corticosteroid administration may preclude visualization of distinct macular details. Subtle fluid from CSCR that is related to a periorcular corticosteroid injection may be interpreted as CME or may be overshadowed by features of coexisting CME. Visual symptoms from secondary CSCR may be attributed to the primary diagnosis of uveitis, and thus the fundus examination that is required to detect this entity may be omitted. Optical coherence tomographic imaging may be especially helpful in this scenario to differentiate between the features of CSCR and CME. It is unlikely that the topical corticosteroid drops played a role in the development of CSCR because of the relatively brief time of administration and the lack of posterior-segment penetration, especially in this patient with phakic eyes. In contrast, periorcular corticosteroids are injected directly behind the globe to induce a posterior pole effect.

Because almost every other route of corticosteroid administration, including local intra-articular injection, has been linked with CSCR thus far, it appears logical that the periorcular depot corticosteroid injection would be associated with development of this disorder. The periorcular route should be included in the suggested etiologic association between corticosteroid therapy and development or exacerbation of CSCR.

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**Acitretin-Associated Maculopathy**

Isotretinoin and etretinate are synthetic retinoids commonly used to treat skin disorders. Ocular adverse effects have been reported after short- and long-term use of these drugs, with keratoconjunctivitis sicca being the most common one. Nictalopia and decreased dark adaptation have been described in patients treated with isotretinoin and etretinate. Furthermore, abnormalities in the electroretinogram (ERG), including reduced scotopic amplitudes and color vision, have also been reported.

The case of a 32-year-old man who noted decreased visual acuity (VA) after long-term use of acitretin (Neotigason), a metabolite of etretinate, is reported. Slitlamp biomicroscopy, fluorescein angiography (FFA), and electrophysiology findings are described.

**Report of a Case.** A 32-year-old white man was seen in the emergency department on July 26, 2002, reporting a 3-day history of blurred vision.