steroid therapy and led to further ocular complications and resultant poor visual acuity, whereas the cases reported herein responded well to topical corticosteroid therapy and had better visual outcomes.

One patient did develop a choroidal neovascular membrane formation 11 years after the initial diagnosis of VKH syndrome that has previously been reported to occur in 11% of affected eyes. It has been suggested that chronic AU may lead to inflammatory events in the retina and choroid that damage the retinal pigment epithelium and Bruch’s membrane, causing chorioretinal degeneration and choroidal neovascular membrane formation, based on the histopathologic condition. However, this was localized and unassociated with any other posterior segment changes.

The trigger for this recurrent AU remains unknown, but it cannot be purely a reflection of syndrome reactivation as the choroid and retina remained uninvolved in each case. Therefore, it seems probable that it may represent a secondary immune event. A similar phenomenon has been reported in patients with healed acute retinal necrosis, who may also demonstrate recurrent AU in the absence of posterior segment inflammation or recurrence of acute retinal necrosis. It has been suggested that the destruction of retinal tissue may release antigen that initiates an immune response to ocular tissue; similar to the sensitization to retinal S-antigen that follows panretinal photocoagulation. As this phenomenon does not occur in all patients with VKH syndrome, there may be the additional involvement of a genetic predisposition.

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Late Posterior Migration of Glass Intraocular Foreign Bodies

The treatment of intraocular foreign bodies (IOFBs) in the previtrectomy era was largely dependent on the source and composition of the material, associated ocular pathologic features, and intraocular location. With modern vitreoretinal surgical techniques, however, most glass IOFBs are removed despite their inert nature. Occasionally, the surgical complexity involved in their extraction must be weighed against the risks of leaving them in place. We describe 3 patients with retained glass IOFBs in which initially stable or encapsulated glass IOFBs subsequently migrated and induced further complications or visual symptoms. There are rare reports of glass IOFBs located initially in the posterior segment, migrating anteriorly, and producing additional anterior segment decompensation, such as illustrated in case 3 below. However, cases 1 and 2 are most unusual, as late migration caused additional retinal pathologic conditions.

We followed up 3 patients from 2 institutions with retained glass IOFBs for more than 6 months. The mechanism of injury, visual acuity, findings from the clinical examination, and fundus appearance were documented. Late migration necessitated surgical intervention in all 3 patients, although 1 patient refused surgery because of special circumstances.

Report of Cases and Results. Case 1. A 23-year-old man was struck with a glass beer bottle during an altercation and sustained a penetrating open globe injury with multiple glass IOFBs in the anterior and posterior segments of the left eye (Figure 1). The wound involved 7 mm of the cornea and an additional 22 mm on the scleral surface. All anterior segment glass IOFBs were removed (the largest measuring 7 × 3 × 3 mm) and the wound was repaired. One additional fragment with largest dimension measuring approximately 1 cm

Figure 1. Case 1. Preoperative axial computed tomographic scan obtained following initial injury, demonstrating 3 intraocular foreign bodies in the left eye.
remained on the inferior equatorial retinal surface. Owing to the risk of additional retinal injury with further glass IOFB manipulation, removal was not performed at the initial surgery.

Postoperatively, the glass IOFB became partially encapsulated and remained immobile on the inferior retinal surface. Retinal examination 3 months after injury revealed an immobile glass IOFB with some overlying gliosis (Figure 2). The patient obtained a visual acuity of 20/50 OS 10 months after the initial injury.

Twelve months after injury, the patient noted that, “I can see the glass moving.” Visual acuity was 20/60 OS. Ophthalmic examination revealed a mobile glass IOFB with a small underlying retinal laceration, moderate (3+) pigment cells in the anterior vitreous, and numerous small preretinal opacities (Figure 3). Owing to the mobility of the glass fragment and the potential for further retinal damage, pars plana vitrectomy, pars plana lensectomy, and IOFB removal were performed (Figure 4). Three weeks after surgery, a new round retinal hole was noted in the region of the preretinal fibrosis. Argon green laser retinopexy was performed but 1 week later, 4 additional open holes were noted along with a large inferior retinal detachment and early proliferative vitreoretinopathy. The patient underwent pars plana vitrectomy, membrane peeling, further endolaser treatment, 16% perfluoropropane gas tamponade, and scleral buckling. Five weeks after this intervention, the patient's retina remained attached, although he had anterior membranes and preretinal fibrosis. The patient was subsequently lost to follow-up.

Case 2. A 21-year-old man robbing a bank, leaped through a plateglass window and sustained lacerations to his face and eye. Immediately after capture, ophthalmologic examination revealed a pars plana entry wound; a glass IOFB, estimated to be 4.5 × 1.5 × 1.5 mm, was found lodged on the surface of the peripheral retina of his right eye. The glass IOFB was not removed at the initial surgery because of poor posterior visualization and the risk of retinal injury during the glass IOFB manipulation. The anterior segment was not damaged. Other than vitreous hemorrhage, no additional pathologic condition was found. The patient's visual acuity eventually returned to 20/20 OD. Fundus examination of the peripheral retina revealed a large immobile glass IOFB with localized fibrosis around its base, where it was in apposition to the retina. The patient was sentenced to life in prison for the robbery and the killing of a guard, but was examined periodically in prison and the glass IOFB re-
mained in place in its peripheral location. As no inflammation developed and because the right eye was quiet and stable with 20/20 visual acuity, subsequent removal of the glass IOFB was not felt to be necessary.

On awakening in his cell 6 years after the crime, he noted double vision and beautiful “rainbows” in his right eye when he lay supine in his cell. He was examined in the prison and the glass IOFB had dislodged from its original location and was now resting on the fovea when the patient was in the supine position (Figure 5). When sitting, the glass IOFB drifted inferiorly away from the macula and symptoms cleared.

Surgical removal of the glass IOFB through a pars plana vitrectomy was recommended and the risks of surgery were discussed. The inmate, however, insisted that if there were any complications whatsoever, he would see to it that his surgeon was killed. Further periodic follow-up was, therefore, offered under these unusual circumstances. The patient was seen again 6 months later with no change in his eye findings, but he was ultimately transferred to another high-security facility and was lost to follow-up.

Case 3. A 21-year-old man who was a passenger involved in a motor vehicle collision sustained a head injury with a severe scalp laceration and an open left globe. A glass IOFB lacerated the cornea and produced a cataract, necessitating cataract removal and corneal wound repair. The vitreous was not removed, as the cornea was cloudy during the wound repair. Visual acuity 1 week after the procedure was 20/400 OS from corneal edema. The fellow right eye was normal with uncorrected 20/25 visual acuity.

On funduscopic examination, the retina was not injured, but a 4 × 4 × 3-mm rectangular piece of glass was present in the inferior vitreous just posterior to the equator, as shown on computed tomography (Figure 6). It did not move with positioning and, after discussion, he decided to be examined periodically for possible fragment migration.

Visual acuity remained 20/400 OS, and the patient’s condition

Figure 4. Case 1. Intraoperative view obtained during pars plana vitrectomy and glass intraocular foreign body removal. A, Glass intraocular foreign body lying over the optic nerve with small pieces of lens cortex on the posterior pole. The glass fragment measured 9 × 6 × 3 mm. Standard pars plana vitrectomy techniques were used, and perfluorin liquid was used to mobilize the glass intraocular glass foreign body and protect the macula. B, The glass intraocular foreign body was then delivered through a limbal wound. Endolaser treatment was applied to the retinal laceration followed by introduction of 16% perfluoropropane gas tamponades.

Figure 5. Case 2. Six years after the initial injury, the glass intraocular foreign body migrated posteriorly over the macula and caused symptoms of positional visual obscuration.

Figure 6. Case 3. Preoperative computed tomographic scan of patient demonstrating glass intraocular foreign body in posterior chamber.
was monitored at monthly intervals for 6 months. However, almost 7 months after the injury, he was seen in the emergency department reporting pain and discomfort in the left eye. The glass IOFB had migrated through the pupil into the anterior chamber and was lodged inferiorly causing local corneal edema. Removal of the glass IOFB was performed through a keratotomy incision. The retina remained unaffected, except for some localized fibrosis at the former site of the glass IOFB. A few weeks thereafter, the cornea was clear, and visual acuity with a contact lens improved to 20/25 OS.

Comment. Glass IOFBs can pose special challenges. They are often large relative to the entry wound, and surgical removal can be challenging, particularly if they are of a large cuboidal or irregular shape. Grasping the glass fragment with foreign body forceps is often difficult and sharp edges can cause retinal or lenticular injury during extraction. In some cases, the level of surgical complexity required for extraction combined with their inert nature may dictate that a glass IOFB be retained in the posterior segment.

One of the largest series of glass IOFBs is provided by Gopal et al,1 in which they report on 51 eyes with glass IOFBs. In 8 eyes, glass pieces were left behind without any untoward events at an average of 31 months' follow-up. In none of these eyes was any late anterior or posterior segment complication encountered. The authors hypothesized that preretal fibrosis encapsulates the glass IOFB and provides additional security. In this same study, Gopal et al encountered iatrogenic retinal breaks in 13 eyes when attempts were made to remove the glass IOFB.

A review of the literature by Milkowski2 reveals numerous cases of retained glass IOFBs near the optic nerve, retina, vitreous, and lens where complications did not develop. This article also summarizes rare cases of migration of posterior IOFBs into the anterior chamber causing cataract, corneal edema, and iridocyclitis.2 A 1991 report by Saar et al3 describes the anterior migration of glass splinters from the vitreous to the anterior chamber causing corneal edema. They also stated in this article that the “migration of intraocular glass is always from back forwards.” 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 3 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-