pigment epithelial atrophy, the patient was managed conservatively. Follow-up at 4 months showed spontaneous resolution of the subretinal fluid on optical coherence tomography (Figure 1C); visual acuity had returned to 6/60 OS but the macular hole remained open. Four months later, the patient’s vision remained stable but the subretinal fluid had returned. An inverse pseudohypopyon was visible at the macula (Figure 2), and optical coherence tomography now demonstrated the presence of droplet-like particles suggestive of emulsified silicone oil (Figure 1D). The patient declined further intervention. Posturing did not appear to affect the distribution of the oil.

Comment. Silicone oil is a commonly used intraocular tamponade in the surgical management of giant retinal tear. Subretinal silicone oil is a rare but serious complication that can result in loss of internal tamponade and subsequent failure of retinal detachment repair. Silicone oil can migrate into any open retinal break, particularly if the surrounding retina is detached. The delayed presentation of subretinal silicone oil has been described in cases of retinal detachment associated with optic disc pit. We believe that this is the first description of emulsified submacular silicone oil presenting as an inverse pseudohypopyon. It is unclear when the macular hole formed and therefore whether the emulsified silicone oil passed through a newly formed macular hole into the subretinal space or whether subretinal silicone oil migrated to the posterior pole over time. It is possible that emulsification along the posterior oil interface may have allowed tiny droplets to enter the hole, thereby contributing to retinal detachment. Recent imaging studies have observed that silicone oil can pass through an open macular hole under certain postural conditions. It is interesting to note that the level of visual acuity is relatively well preserved despite the possibility that the oil might have been present in the subretinal space for many years.

We postulate that the spontaneous accumulation and resolution of submacular fluid suggest a dynamic process whereby the emulsified oil might act to intermittently close the macular hole, allowing the retinal pigment epithelial pump to clear the fluid when an effective seal is present. We do not have any images to confirm this. This case demonstrates a novel clinical entity that raises further questions about how silicone oil can behave within the eye.

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Traumatic Airbag Maculopathy

W ith the prevalence of motor vehicle crashes, airbag deployment is a significant source of ocular trauma. We describe a case of traumatic airbag maculopathy in which imaging studies document a constellation of interesting findings, including subretinal fluid with impending macular hole and persistent paracentral scotoma with underlying electrophysiologic disturbance despite anatomical recovery on optical coherence tomography (OCT).

Report of a Case. A 49-year-old woman was involved in a motor vehicle crash at 65 mph, hitting the center divide front-on with airbag deployment. She immediately noted blurry vision. The emergency department evaluation otherwise revealed no traumatic injuries beyond chest contusions. The patient had normal neurological examination findings and never lost consciousness, and no head imaging was indicated. She visited the retina service after having persistently blurry vision for 3 days. Her ocular history consisted of high myopia and retinal detachment in each eye leading to laser demarcation in the right eye. Examination of the right eye revealed visual acuity of 20/150, posterior vitreous detachment, numer-
ous cotton-wool spots surrounding the optic disc, and subretinal fluid (Figure 1A). Fluorescein angiography revealed a transmission defect in the inferior fovea as well as minimal diffuse leakage in the peripapillary retina. Fundus autofluorescence showed a diffuse area of increased autofluorescence around the fovea, extending superiorly toward the nerve (Figure 1B). Spectral-domain OCT findings were remarkable for a foveal detachment consistent with impending macular hole (Figure 2A).

Three weeks later, the cotton-wool spots and subretinal fluid had resolved, although some photoreceptor abnormalities remained on OCT (Figure 2B). After 5 months, repeated OCT revealed recovery of her baseline architecture (Figure 2C) with visual acuity improved to 20/25. However, even a year after injury, the patient described a central doughnut-shaped area of blurry vision. Given the absence of structural evidence on fundus examination or OCT that would completely account for her persistent visual complaints, the patient underwent multifocal electroretinography, which showed reduced signal throughout the macula with some temporal sparing (Figure 1C).

Discussion. Persistent airbag-associated scotoma is rarely described in the literature. We are aware of 2 other case reports of a persistent paracentral scotoma following airbag trauma. In one, the scotoma was attributed to a break in the Bruch membrane, while the other demonstrated focal thinning of the juxtafoveal neurosensory retina but had normal findings on multifocal electroretinography. In contrast, our case exhibited clear electrophysiological dysfunction on multifocal electroretinography, an outcome consistent with findings that central electroretinal activity remained depressed 6 months following acute traumatic maculopathy despite resolved OCT abnormalities. While no specific architectural disruption accounted for this patient's functional impairment, it is notable that the pattern of retinal injury suggested on fundus autofluorescence 3 days after the motor vehicle crash corresponded closely with the diminished signal distribution on multifocal electroretinography a year later. While the sudden shock on impact may have caused the photoreceptor injury described, the presence of subfoveal fluid with visual nadir 3 days after impact cannot be discounted. Serous retinopathy is a rare finding associated with trauma and may arise from localized concussive damage with retinal dehiscence on impact. Initially diffuse intraretinal edema has been shown to progress to outer retinal disruption. Alternatively, in the context of posterior vitreous detachment, tractional or concussive forces may have led to a fluid collection with impending macular hole, independently contributing to our patient's vision loss. Yamashita et al propose 2 mechanisms of traumatic macular hole formation: (1) immediate vision loss from primary foveal dehiscence due to concussive forces, and (2) delayed vision loss from continued vitreomacular traction. Both mechanisms may have been present in our patient. In this case, serial imaging

Figure 1. At presentation, several areas of retinal opacification around the disc on a fundus photograph (A) corresponded to foci of decreased autofluorescence on fundus autofluorescence (B). Notably, fundus autofluorescence also shows a diffuse area of increased autofluorescence around the fovea extending superiorly toward the nerve. This distribution of photoreceptor injury appears as a near total loss of electroretinographic signal in the same pattern a year later (C).

Figure 2. Optical coherence tomography suggested an impending macular hole at presentation (A). The retinal architecture recovered by 3 weeks (B), and at 5 months it continued to show preservation of the external limiting membrane, inner segment–outer segment junction, and foveal cone structure (C).
provided insight into the evolution of traumatic maculopathy and pathogenesis of traumatic macular hole formation. Further advanced imaging may improve our ability to prognosticate and intervene following ocular trauma.

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Corneal Graft Alterations After Descemet Stripping: Implications for Split Cornea Transplantation

In recent years, there has been tremendous progress in improving lamellar keratoplasty techniques such as deep anterior lamellar keratoplasty1,2 and Descemet membrane endothelial keratoplasty.3,4 Splitting of a single donor cornea into an anterior part (including epithelium, its basement membrane, Bowman layer, and stroma) for use in a deep anterior lamellar keratoplasty procedure in a patient with anterior stromal disease (eg, keratoconus) and into a posterior part (endothelium–Descemet membrane layer) for use in a Descemet membrane endothelial keratoplasty procedure in a patient with endothelial disease (eg, Fuchs endothelial dystrophy) can reduce the need and cost for corneal donor tissue by up to 47%.5,7 Since it is thus far unclear what time limits are acceptable for storing anterior and posterior grafts in split cornea transplantation, we investigated split corneal tissue for temporal morphologic alterations after Descemet stripping.

Methods. Eighteen pairs of healthy human donor corneas unsuitable for corneal transplantation were included in this experimental study performed in conformance with the tenets of the Declaration of Helsinki. All corneoscleral buttons were organ cultured in Dulbecco modified Eagle medium containing streptomycin, penicillin (Biochrom), and fetal calf serum (Linaris) at 34°C. The mean (SD) donor age was 60 (24) years, the mean (SD) postmortem time was 10 (3) hours, and the mean (SD) preservation time prior to splitting was 300 (181) hours.

Sixteen corneas were split into anterior and posterior lamellas by Descemet stripping2 and stored for 4 weeks in organ culture to assess the time course of central corneal thickness using slitlamp-adapted optical coherence tomography (Heidelberg Engineering) and endothelial cell count using phase-contrast microscopy (Olympus BX51).

For histologic and ultrastructural analyses, 10 split and 10 full-thickness buttons were compared after 1 and 3 weeks in culture using Mann-Whitney test. P < .05 was considered statistically significant.

Results. For anterior donor lamellas, the mean (SD) central corneal thickness increased nonlinearly from 770 (140) μm prior to splitting to 1057 (128) μm at 1 hour, 1122 (118) μm at 1 day, 1179 (131) μm at 1 week, and 1230 (139) μm at 4 weeks after stripping.

The mean (SD) endothelial cell count of posterior lenticules decreased linearly from 2683 (412) cells/mm² prior to splitting to 2517 (227) cells/mm² postoperatively, 2468 (238) cells/mm² at 1 week, 2336 (393) cells/mm² at 2 weeks, 2269 (366) cells/mm² at 3 weeks, and 2006 (293) cells/mm² at 4 weeks after stripping (ie, mean loss of 128 cells/mm² per week).

By light microscopy (Figure 1) and electron microscopy (Figure 2), corneal epithelium and stroma revealed significantly more edematous alterations after stripping than full-thickness corneas (P = .02), with a significant increase from 1 to 3 culture weeks (P = .02) and significant anterior keratocyte loss within 3 culture weeks (P = .02). Descemet membranes showed an intact and viable endothelium up to 3 culture weeks in split and non-split buttons.

Discussion. Longer storage of split donor tissue would simplify the logistics of split cornea transplantation. However, it was thus far unclear which (potentially irreversible) graft alterations occur after longer storage of the anterior and posterior lamellas.

In this study, anterior grafts revealed chronic edematous changes with anterior keratocyte loss starting after 1 week in culture, and posterior grafts showed a sharp increase of endothelial cell loss between 3 and 4 weeks in culture. These morphologic findings suggest that anterior lenticules can be stored reliably up to 1 week and posterior lenticules—depending on endothelial cell count prior to stripping—up to 3 weeks before grafting. However, no conclusions regarding the reversibility of those alterations can be drawn. Therefore, probably an even longer interval between splitting and grafting may be feasible. Further studies are necessary, especially to define the minimum tolerable donor endothelial cell count for Descemet membrane endothelial keratoplasty and the effect of different preservation media on graft alterations.

Nevertheless, our data support the safety of anterior donor tissue stored in organ culture up to 1 week as well as posterior donor tissue stored in organ culture up to 3 weeks for use in deep anterior lamellar keratoplasty and Descemet membrane endothelial keratoplasty surgery,