(Figure 2C). Results of OCT of the left eye appeared normal.

The patient underwent vitrectomy on the right eye to relieve the vitreous traction on the macula. Postoperatively, visual acuity was 20/40. The macula was flat, and the optic disc was less elevated (Figure 1D). The apparent vitreous bands were no longer seen by OCT, except for a thin band extending upward from the temporal disc edge (Figure 2D).

**Comment.** Vitreomacular traction syndrome has been well described in the literature. Vitreopapillary traction syndrome has also been described, but usually in conjunction with other manifestations of anomalous posterior vitreous detachment, which also includes rhegmatogenous retinal detachment, macular pucker, macular holes, and proliferative diabetic vitreoretinopathy. Vitreopapillary traction can occur in the absence of other forms of anomalous posterior vitreous detachment in diabetic vitreoretinopathy, but there are few articles describing tractional forces exerted by the posterior hyaloid on the optic nerve head in the absence of diabetes mellitus or other forms of retinovascular disease. Schepens described the histopathology of what he referred to as pseudopapillary edema with incomplete posterior vitreous detachment from the optic disc. Katz and Hoyt, using slit-lamp biomicroscopy and ultrasonography in a series of 8 patients, observed that vitreopapillary traction could produce intrapapillary and peripapillary hemorrhages. Wisotsky et al also described 2 patients with vitreopapillary traction causing optic nerve head elevation demonstrated by ultrasonography.

Optical coherence tomography is a valuable tool for illustrating vitreous traction on the optic nerve. Recently, Rumelt et al described OCT findings in 3 patients with optic disc traction as well as macular traction associated with central retinal vein occlusion. Our cases were referred for apparently isolated optic nerve head elevation, and OCT confirmed the presence of idiopathic vitreopapillary traction. One patient (case 1) subsequently developed vitreomacular traction, and the other (case 2) had concurrent vitreomacular traction. Evaluating patients with OCT in the setting of optic nerve head elevation may show vitreopapillary traction and can obviate the need for more extensive procedures, such as neuroimaging, or invasive procedures, such as lumbar puncture.

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**Ultra-High Resolution Optical Coherence Tomography of Retinal Pigment Epithelial Tear Following Blunt Trauma**

Tears of the retinal pigment epithelium (RPE) were first described in 1981 by Hoskin et al as a complication of detached pigment epithelium in patients with age-related macular degeneration. Since then, RPE tears have also been described in patients with chorioretinal scarring in retinal detachments, with subretinal neovascular membranes, following glaucoma surgery, and after laser photocoagulation of pigment epithelium detachments. We describe a patient who developed an RPE tear following blunt trauma to the eye. Ultra-high resolution optical coherence tomography was performed, and it provided unprecedented visualization.

**Report of a Case.** A 43-year-old woman reported falling and hitting her head and left eye on a wooden rail 1 week prior to her initial visit. After the swelling in her left eye subsided, she noticed decreased vision in that eye. At the time, the best-corrected visual acuities were 20/100 OD and 20/70 OS. Ocular history was significant for amblyopia in the right eye. Amsler grid testing of the left eye revealed areas of waveliness and a scotoma in the center of the grid. Intraocular pressures were 15 mm Hg OU. Dilated fundus examination results of the right eye were normal whereas the left eye showed a well-demarcated area of RPE loss in the macula that was elevated with fluid. A scroll of pigmented RPE was noted inferonasally (Figure 1A), and a horseshoe tear with surrounding subretinal fluid was noted superotemporally (located outside of the photographic field). Fluorescein angiography showed an early window defect from the lost RPE measuring several disc areas in size and involving the entire temporal and superior macula. A band of blocked fluorescence on the nasal margin was consistent with the scroll of RPE (arrow in Figure 1B). Ultra-high resolution optical coherence tomography was performed using a standard protocol. The horizontal temporonasal scan revealed a large area of subretinal fluid in the fovea. The area of RPE distorsion in the nasal region (arrow in Figure 2) corresponds to the clinical finding of scrolled RPE as a result of retraction and folding of the RPE following the tear.

Given the presence of the horseshoe tear with localized retinal detachment, the patient underwent laser treatment without any complications. She was informed that no successful therapy for RPE tears was available. Several options were discussed, including surgery to unroll the RPE and autologous iris pigment epithelial cell transplant. Surgery to drain the subretinal fluid was also considered.

Although the subretinal fluid had almost completely resolved sponta-
neously 5 weeks later, the patient perceived no improvement in vision. In fact, the best-corrected visual acuity had worsened to 20/300 OS. Ultra-high resolution optical coherence tomography revealed disruption of the inner and outer segments of the photoreceptors as well as thinning of the outer nuclear layer centrally. These findings may help explain the poor vision in this eye.

Comment. Retinal pigment epithelial tears are rarely caused by trauma. It is thought that the force applied must fit into an extremely narrow window. If the force is too small, no tears will occur. Only if the force is strong enough to tear the pigment epithelium but not strong enough to tear Bruch’s membrane will a traumatic RPE tear result. In experimental studies, Korte et al found that 1 week after an RPE tear, a layer of flattened, depigmented cells had replaced the area stripped of RPE. Although these cells may physically reconstitute the outer blood-retinal barrier, they may not be able to take the active role of normal, healthy RPE cells. Korte and colleagues have also shown that choriocapillaris death occurs as early as 11 weeks after RPE removal. Progressive deterioration in the visual acuity in our patient was noted as early as 8 weeks after the initial traumatic event, correlating well with experimental studies and other clinical reports.6

Figure 1. Following blunt trauma to the left eye, the patient underwent fundus photography and fluorescein angiography. A, Color fundus photograph of the left eye at the initial visit showing a well-demarcated hypopigmented area corresponding to loss of retinal pigment epithelium and exposed Bruch’s membrane. A hyperpigmented strip of rolled-up pigmented retinal pigment epithelium was noted infranasally. B, Late-stage fluorescein angiography (59 seconds) of the left eye showing mottled hyperfluorescence in the shape of a large crescent involving the macula and extending temporally. A band of blocked fluorescence (arrow) on the nasal margin corresponded to the scroll of retinal pigment epithelium found on clinical examination.

Figure 2. A 6-mm horizontal temporonasal scan with ultra-high resolution optical coherence tomography was performed through the center of the fovea, showing a large accumulation of subretinal fluid. The arrow indicates the distorted retinal pigment epithelium in the nasal region. This is thought to be due to the retraction and folding inward of the retinal pigment epithelium following the tear.

Spinal Fluid Leak After Chiropractic Manipulation of the Cervical Spine

Intracranial hypotension (ICH) caused by cerebrospinal fluid (CSF) leakage is a well-documented cause of severe headaches and neurologic deficits. Cerebrospinal fluid leaks most often occur as a complication of neurosurgical procedures, in particular lumbar puncture, or after accidental trauma.

Report of a Case. A 51-year-old woman had binocular horizontal diplopia for 4 weeks. She reported having headaches for several weeks, which had been treated by her chiropractor. After receiving cervical spinal manipulation on 3 separate occasions, she did not experience relief but instead escalation of the headache. One week after the last chiropractic treatment, she developed binocular horizontal diplopia prompting neuro-ophthalmic evaluation. Her visual function and ocular fundus were normal. Ocular motility testing revealed a right cranial nerve VI palsy with a 20–prism diptor (PD) esotropia (ET) in primary gaze; and 1-PD ET in right gaze; and 1-PD ET in left gaze. Magnetic resonance imaging showed diffuse enhancement of the meninges (Figure A), which was thought to be the causative lesion and treated with a blood patch. This led to prompt headache relief, but the diplopia, which had been worsening before intervention, did not resolve immediately. Ten days later, she had a 30-PD ET in primary gaze, increasing to 40 PD in right gaze with a 20% right abduction deficit. Since the patient was now headache free, she was followed up without further intervention. The ocular misalignment resolved completely over the ensuing 5 months.

Comment. Forceful flexion and distraction of the cervical spine is a well-established mechanism for causing dural tears and ICH. But even small insults, like Valsalva maneuvers, may lead to CSF leakage, usually in conjunction with a focal weakness of the thecal sac. There are only a few reports of dural tears with CSF leakage and ICH after chiropractic manipulation, none of which recount any neurologic symptoms besides headache. In our patient’s case, headache that worsened after each manipulation and the appearance of cranial nerve VI palsy shortly thereafter strongly suggest a cause-effect relationship between spinal manipulation and the radiologically proven CSF leak. Interestingly, the patient did not associate her escalating headache with the previous chiropractic treatment. This reflects a common public conception of the innocuous nature of chiropractic maneuvers. In fact, the estimated complication rate of chiropractic treatment ranges from 1.3 in 100 000 to 1 in 2 million manipulations with the most frequent serious complication being cerebral or cerebellar stroke caused by dissection or occlusion of the vertebral or internal carotid artery. Fortunately, unlike stroke, neurologic deficits caused by ICH usually resolve, albeit slowly, once normal intracranial pressure has been reestablished.

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