Case Report

Soccer player whiplash maculopathy

Abstract

A 17-year-old girl experienced a head-to-head collision on the soccer field and presented several hours later with central vision loss. Eye examination findings revealed macular edema consistent with whiplash maculopathy. Symptom resolution required 3 months with no intervention necessary. Whiplash maculopathy is a little-known disease pathology in which the proposed mechanism of action involves traction on the eye’s vitreous base. Rapid acceleration and deceleration forces cause focal areas of detachment at the retinal pigment epithelial junction and thus result in visual loss.

A 17-year-old girl presented to the emergency department after a head-to-head collision on the soccer field a few hours earlier, complaining of vision loss. While doing her homework, she realized she was having trouble reading the center of the page even though she could read the periphery easily. She denied loss of consciousness, persistent headache, or any other injury.

Physical examination revealed normal vitals and normal neurologic exam. Eye examination revealed normal visual acuity of 20/200 and peripheral visual acuity of 20/25 bilaterally. Pupillary light reflexes were normal, with full accommodation and no relative afferent pupillary defect. Slit lamp examination revealed normal conjunctivae, corneas, irises, and anterior chambers. Retinal examination under direct ophthalmoscopy revealed normal retinal vessels, optic cups, and discs. The maculae, however, were clearly affected with indistinct foveal borders and diffuse edema bilaterally (Figs. 1 and 2). These findings were consistent with a diagnosis of whiplash maculopathy.

The patient was observed, and over a period of 3 months, she gradually regained her central vision with corresponding resolution of the macular edema. No intervention was required.

Whiplash injury, consisting of rapid acceleration and deceleration forces, has been shown to cause significant injury to the head, neck, and trunk. Injuries to the eye appear less common but can involve oculomotor disturbances and minor decreases in visual acuity [1]. Duke-Elder [2] notes that whiplash injury can be severe enough to cause Horner syndrome, difficulties with accommodation, heterotropia, and even temporary abducens nerve palsy. The term whiplash maculopathy refers to an immediate reduction in visual acuity with corresponding development of a foveolar depression and increased thickness of the peripheral retina [3]. Occasionally, there can be small vitreous detachments as well.

The only pathologic description of whiplash maculopathy comes from a forensic pathology study of a young woman
who died of severe whiplash injury in a roller coaster accident in the UK [4]. Upon examination of the eyes, bilateral extracranial optic nerve sheath hemorrhages were found extending into the scleral tissues. Folding of the retina was also noted, extending from the temporal aspect of the optic disc through the macula and to the fovea. The fovea conspicuously had no pit or hole.

Histologic examination revealed disruption of the photoreceptors and local retinal pigment epithelium (RPE) detachment seen as folding of the epithelium with sub-RPE edema. The RPE itself was disrupted as well, with detachment from the Bruch membrane. Collections of sub-RPE fluid had developed in this new space, surrounding the macular and peripapillary areas. Interestingly, the small focal areas where the RPE detachment was found were concentrated in the periphery where the vitreous base is firmly attached. It is here that retinal splitting or retinoschisis is postulated to occur when shearing forces applied to the layers of the eye cause the retina to slide against the underlying structures including the RPE.

The case that we present above is an example of extensive whiplash injury in the setting of seemingly minor head injury. Even though whiplash maculopathy is a relatively uncommon disease pathology [3,5], it should not be ignored as a possibility in the process of differential diagnosis, especially in conjunction with head injury. Abrupt acceleration and deceleration forces can cause a range of macular pathology, with the most severe being local detachment of the retinal layers and the development of macular edema. Our patient’s case clearly demonstrates this pathology.

In this patient, the small retinal detachments and edema resolved without intervention and with full restoration of her visual acuity. However, it is plausible that patients with preexisting retinal disease would have poorer outcomes. We propose that a simple screening examination of visual acuity in at-risk patients would identify those that require ophthalmology referral for a full retinal examination and follow-up.