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Case of the Month – February 2019

Presented by Christian Sanfilippo, MD

A 12 year-old female was referred for evaluation of central vision loss following a soccer ball injury to the right eye the day prior. Her presenting visual acuity was 20/200 +2 and 20/25+2 in the right and left eyes, respectively. Intraocular pressures were normal. Slit lamp examination of the anterior segment was unremarkable. Dilated funduscopic examination of the right eye showed a normal appearing optic nerve, mottled appearance of the central retinal pigment epithelium, mild superior vitreous hemorrhage and an area of retinal whitening consistent with commotio retinae within the superior mid periphery and extending into the superior macula. Dilated examination of the left eye was unremarkable with the exception of two CHRPE lesions in the temporal periphery.

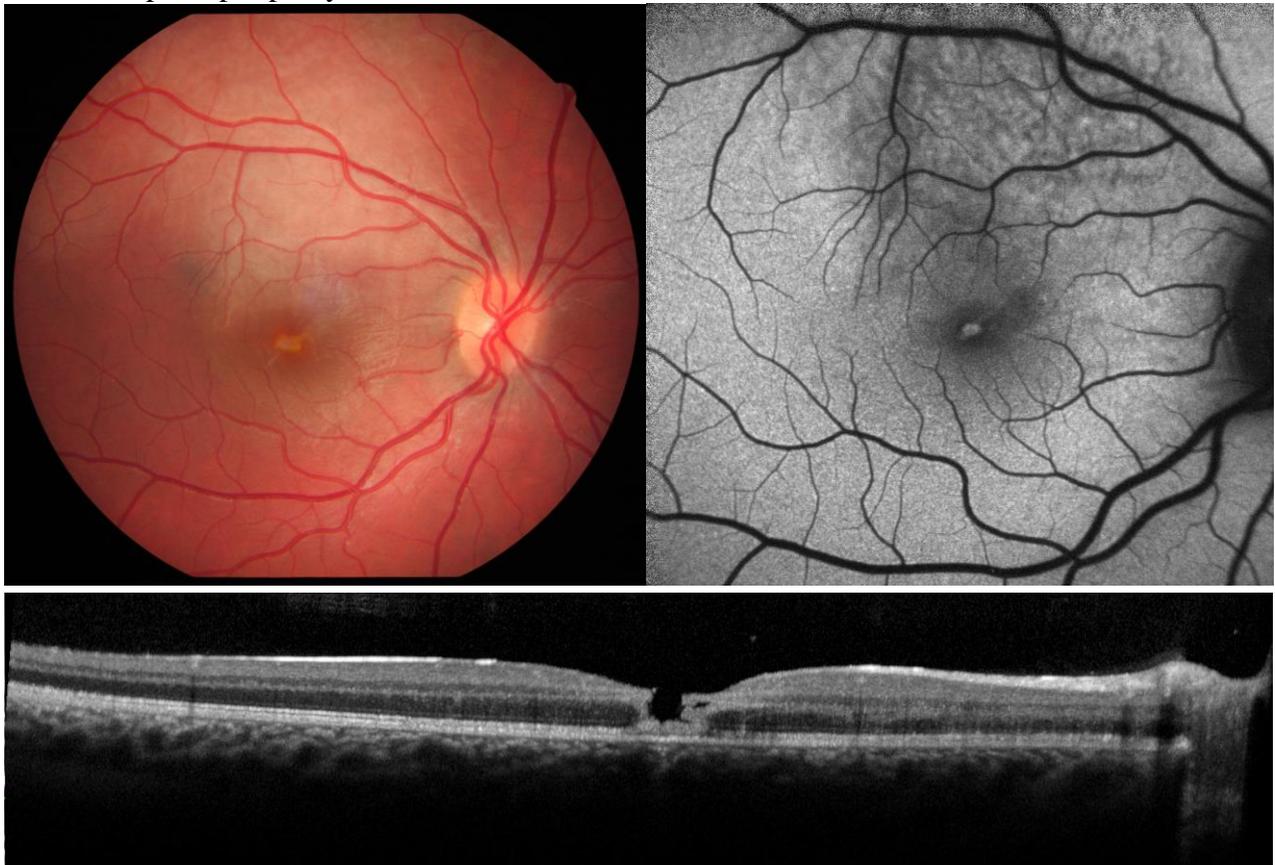


Figure 1: **A.** Color photograph of the right eye shows retinal whitening in the near periphery extending to the superior macula and a central yellow pigmentary alteration. **B.** Fundus autofluorescence highlights the area of commotio retinae within the superior macula. The central hyperfluorescence may be the result of photoreceptor unmasking from loss of inner retinal tissue, or an accumulation of photoreceptor pigments. **C.** OCT of the macula shows disruption of the inner retinal layers with an abnormal appearing and elongated photoreceptor layer remaining centrally.

Clinical Course:

Although the outer retina had not completely dehisced, there was high suspicion of an evolving traumatic macular hole. The patient was closely observed, and returned for repeat examination one week later. Evaluation on that visit showed evolution to a full thickness traumatic macular hole (Figure 2A). Because of the progression, surgery was scheduled for the following week with a plan to repeat an OCT one day prior to surgery to check once more for spontaneous closure. Unfortunately, the macular hole continued to enlarge (Figure 2B) and surgery was performed the following day with pars plana vitrectomy, internal limiting membrane peeling and gas tamponade.

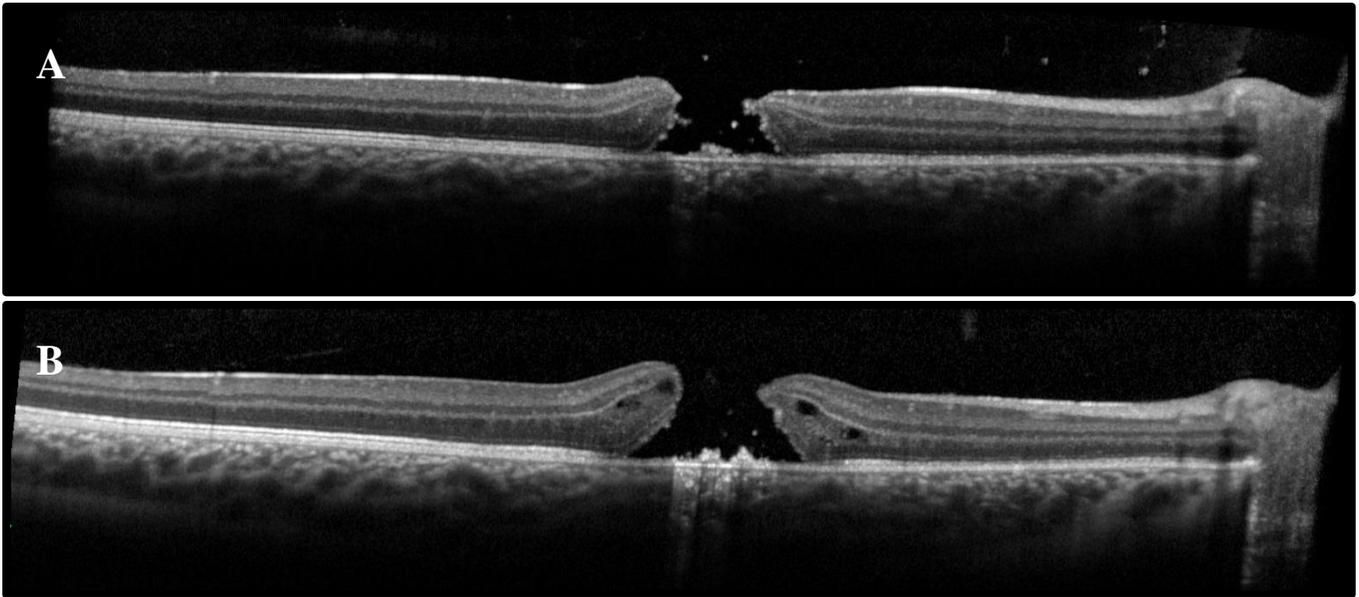


Figure 2: **A.** OCT of the macula performed 1 week after injury showing evolution to full thickness macular hole. **B.** OCT of the macula performed 2 weeks after injury, and 1 day prior to surgery showing increase in macular hole size.

The hole successfully closed with initial residual irregularity of the outer retina (Figure 3A). Her anatomy and vision slowly improved (Figure 3B). At her most recent follow up, 17 months post-surgery, her visual acuity had improved to 20/40+1 with the aid of pinholes.

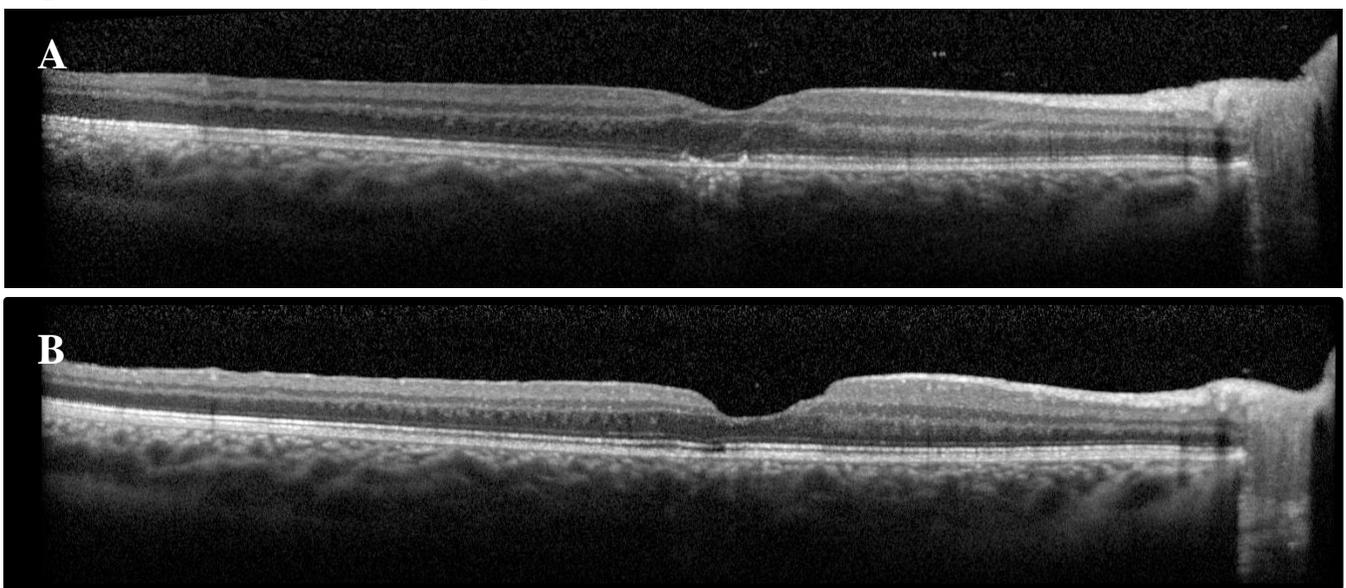


Figure 3: **A.** OCT of the macula performed 1 month after surgery showing closure of the macular hole with residual irregularity of the ellipsoid and RPE layer. **B.** OCT of the macula performed 17 months after surgery recovery of the ellipsoid band and normalization of the underlying RPE.

Discussion:

Traumatic macular holes are an uncommon complication of ocular trauma. They most frequently occur in pediatric patients and young male adults, which overlaps with the at-risk demographic for ocular trauma in general. Several theories have been proposed to explain their formation. The most widely accepted is the theory that traumatic macular holes form as the result of sudden equatorial expansion of the globe from blunt trauma. This force is transmitted tangentially to the fovea resulting in splitting at the foveal center. Other proposed theories include sudden anterior-posterior traction at the fovea with avulsion of the foveal tissue, and central macular necrosis as a result of coup-contrecoup injury. This is in contrast to the more common idiopathic macular hole encountered in older adults, which most frequently forms as a result of abnormal anterior-posterior vitreo-foveal traction as the posterior hyaloid face begins to detach from the posterior pole, or less commonly as a result of abnormal tangential traction from a taught ILM. In contrast to idiopathic macular holes, those associated with trauma rarely have concurrent posterior vitreous detachment. This lends further support to the beliefs that these are indeed separate entities.

Because traumatic macular holes occur in the setting of severe blunt trauma, other sequelae of ocular trauma often occur concurrently. Commotio retinae (as in our case), choroidal rupture, retinal tear, retinal dialysis, traumatic iritis, angle recession, lens dislocation and cataract must be ruled out and treated when indicated. The management of traumatic macular hole typically begins with a period of observation. Unlike idiopathic macular holes, which rarely spontaneously close, traumatic macular holes have been reported to spontaneously close at a rate of 10% to 67% over the course of a few weeks to months. In those cases which fail to close, or progress during the initial period of observation, as in our case, surgery is indicated. Surgical management is similar to idiopathic macular hole, with pars plana vitrectomy, peeling of the internal limiting membrane and gas tamponade being the procedure of choice. Special considerations must be made when planning vitreoretinal surgery in the pediatric age group. Specifically, surgical induction of a posterior vitreous detachment can be very challenging, and patient adherence to post-operative positioning may not be possible for children, necessitating a longer-acting tamponade agent. While the anatomical success rate of surgery is lower than for idiopathic macular hole, the majority of holes can be closed, with most studies reporting closure rates in the 80-90% range.

Management, however, does not stop with surgery. After successful closure, the patient must be monitored for other complications of trauma, many of which can present in a delayed fashion. A second case of ocular trauma is shown below which presented initially with choroidal rupture and secondary choroidal neovascularization. The patient was treated with intravitreal anti-vegf therapy and was stable for years when she developed a macular hole adjacent to the choroidal rupture. The macular hole was successfully repaired with pars plana vitrectomy, peeling of the internal limiting membrane and gas tamponade. She has required ongoing anti-vegf therapy for the choroidal neovascularization, but her vision remains 20/50+ at most recent follow up 5 years post-operatively despite expansion of the RPE atrophy overlying her choroidal rupture. This case exemplifies that good visual acuity can be maintained following multiple complications of ocular trauma with aggressive management.

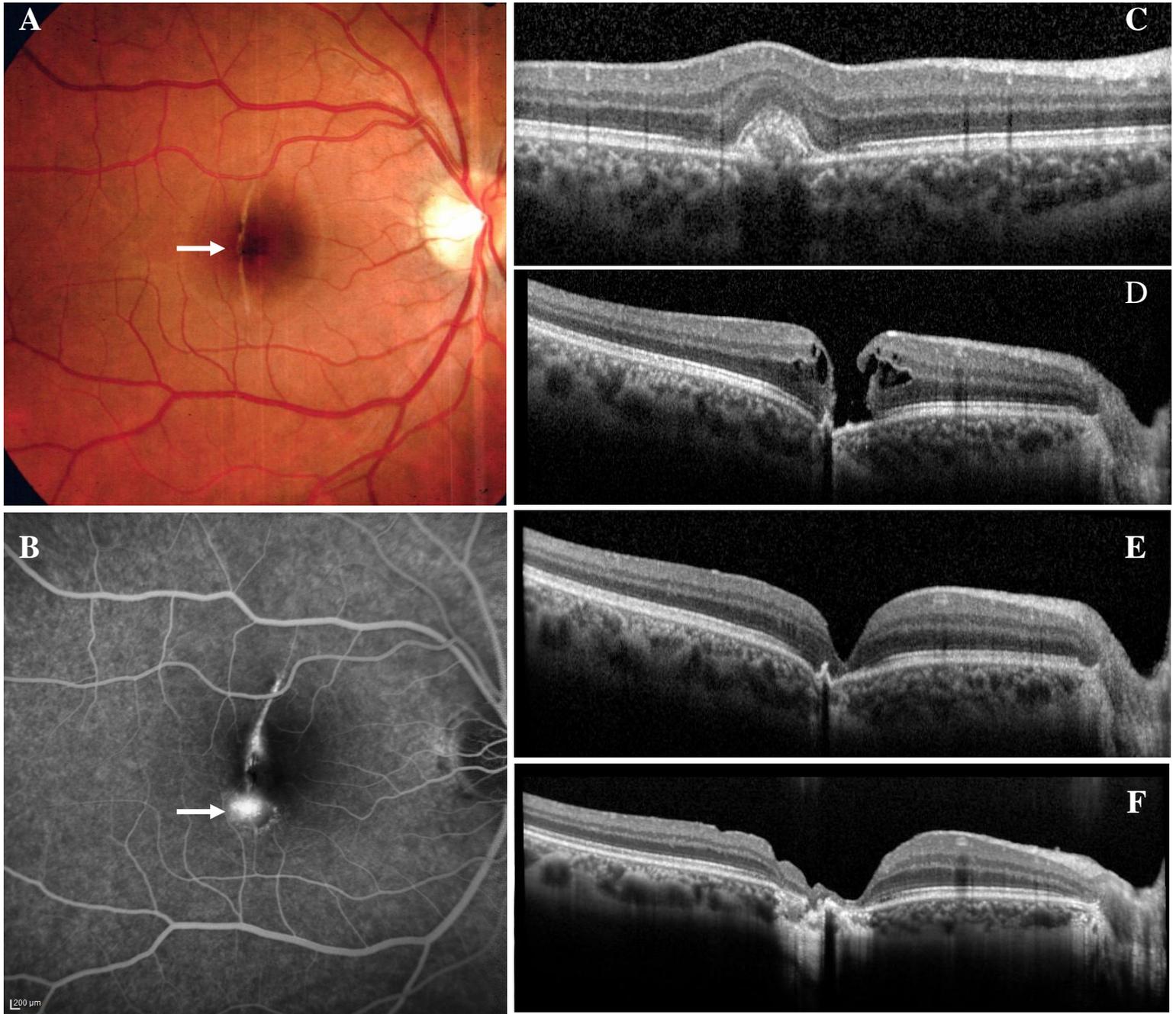


Figure 4: **A.** Color fundus image at presentation. Note the circumferential choroidal rupture through the fovea (Arrow). **B.** Late phase fluorescein angiogram showing choroidal neovascularization at the inferior edge of the choroidal rupture (Asterisk). **C.** OCT line scan through the choroidal neovascular membrane. **D.** OCT of the macula performed 3 years after presentation showing evolution to full thickness macular hole overlying the choroidal rupture. **E.** OCT of the macula 1 month after surgery showing closure of the macular hole. **F.** OCT of the macula 5 years after surgery showing expansion of RPE and retinal atrophy overlying the choroidal rupture

Take Home Points

- Traumatic macular holes represent a distinct subtype of macular hole with a unique pathophysiologic mechanism of formation.
- Traumatic macular holes may close spontaneously, and therefore a short period of initial observation is warranted.
- Serial detailed anterior and posterior examination must be performed in cases of traumatic macular hole to rule out other concurrent sequelae of trauma.
- Vitrectomy for traumatic macular hole can yield excellent anatomic and visual results when performed by an experienced surgeon
- Visual acuity can be maintained through many complications of ocular trauma if recognized and treated appropriately.



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