Macular tractional retinal detachment: A rare complication of blunt trauma

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Traumatic rhegmatogenous retinal detachment after blunt ocular trauma is a known entity. A tractional macular detachment occurring posttrauma without a retinal break is a unique presentation. A 25-year-old gentleman after blunt ocular trauma with a ball presented a week later with a vision of 20/800, large subretinal bleed and resolving vitreous hemorrhage in the right eye. Three weeks later, an extensive glial proliferation at the posterior pole and macular tractional retinal detachment was noted with the worsening of visual acuity. A vitrectomy, membrane peeling, and silicone oil tamponade with a subsequent silicone oil removal at 6 months stabilized the macula, and vision improved to 20/120. A tractional macular detachment post blunt trauma is rare albeit an interesting occurrence, which can be effectively managed with vitrectomy and allied procedures.

Key words: Blunt ocular trauma, glial proliferation, subretinal hemorrhage, traumatic tractional macular detachment, vitrectomy, vitreous hemorrhage

Blunt ocular trauma in sports is fairly common and is almost always preventable. The incidence of eye injuries is reported to be 3.5 eye injuries per 100,000 population, with males affected by 80% of open globe injuries.[1] Of these, 37%–52% are sports-related and mainly affecting young children.[2,3] Direct blunt trauma constricts the eye in the axis of the force significantly thereby elongating the tissues in a perpendicular plane. Indirect injury is caused by this elongation leaving several trauma-related complications ranging from trivial inflammation to severe complications like globe rupture and retinal detachment.

Traumatic retinal detachments account for 10%–40% of all detachments and are more common after closed globe injuries.[4] It is seen in up to 9% of contusion injuries but may take many years to develop and are mostly rhegmatogenous due to breaks and dialysis.[5] The macula also is extremely susceptible to trauma due to the vascular damage of the nerve fiber layer and direct mechanical forces that lead to photoreceptor and retinal pigment epithelium (RPE) destruction.[5] We report a rare case of tractional retinal detachment (TRD) involving the macula after a sports-related blunt trauma in a young patient with no evidence of retinal breaks and its further course.

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Case Report

A 25-year-old gentleman presented with complaints of blurred vision in the right eye (RE) after a history of blunt trauma with a cricket ball seven days prior. He had been started on anti-glaucoma drops and topical steroids by a local physician. His corrected distance visual acuity (CDVA) was 20/800 in the RE and 20/20 in the left eye (LE). The intraocular pressure on applanation tonometry was 17 and 16 mm Hg in the RE and LE, respectively. On ocular examination, a resolving hyphema and anterior chamber reaction were noted in the RE. Funduscopy revealed a subretinal bleed at the posterior pole and inferiorly with a resolving vitreous hemorrhage [Fig. 1]. There was no evidence of a rhegma or dialysis after a dilated indirect ophthalmoscopy with scleral indentation. The optical coherence scan (OCT) of the RE macula showed extensive subretinal scarring with gross disruption of the photoreceptor layer and decreased retinal thickness [Fig. 1]. The LE examination was within normal limits.

The patient was explained about the guarded vision prognosis and asked to review in a fortnight. On follow-up at three weeks, fundus examination revealed a thick epiretinal membrane, glial condensation with a taut posterior hyaloid face and a macular TRD [Fig. 2]. An OCT scan revealed a thick preretinal membrane bridging the disc and macula with a pucker like concave configuration [Fig. 2, below]. A thorough fundus examination again did not reveal any breaks or dialysis.

In view of the above development, a vitrectomy with membrane peeling and silicone oil infusion (for a longer tamponade) was done for the RE, following which the vision improved to 20/120 one-month post-surgery. There were poor outer retinal integrity and RPE clumping and scarring evident on both clinically and on the OCT scan [Fig. 3]. An intraoperative peripheral retinal evaluation also did not reveal any predisposing breaks or dialysis.

Silicone oil removal was done 6 months later, following which the vision remained stable at 20/120 and the clinical findings remained status quo [Fig. 4].

Discussion

Blunt ocular trauma can be a predisposing factor for several vision-threatening complications like subretinal and

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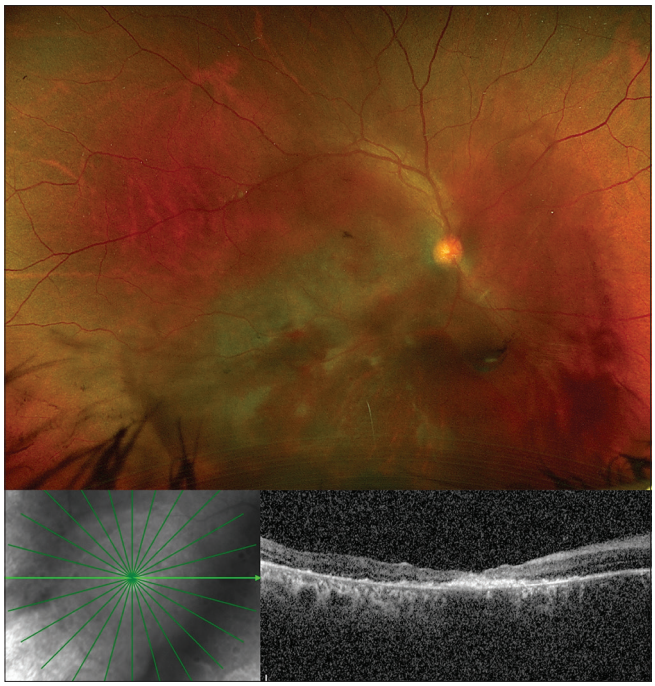

Figure 1: Right eye fundus image showing subretinal bleed at the posterior pole and inferiorly and a resolving vitreous hemorrhage (Top) and OCT macula scan showing retinal thinning with subretinal hyperreflectivity and outer retinal loss (Below)

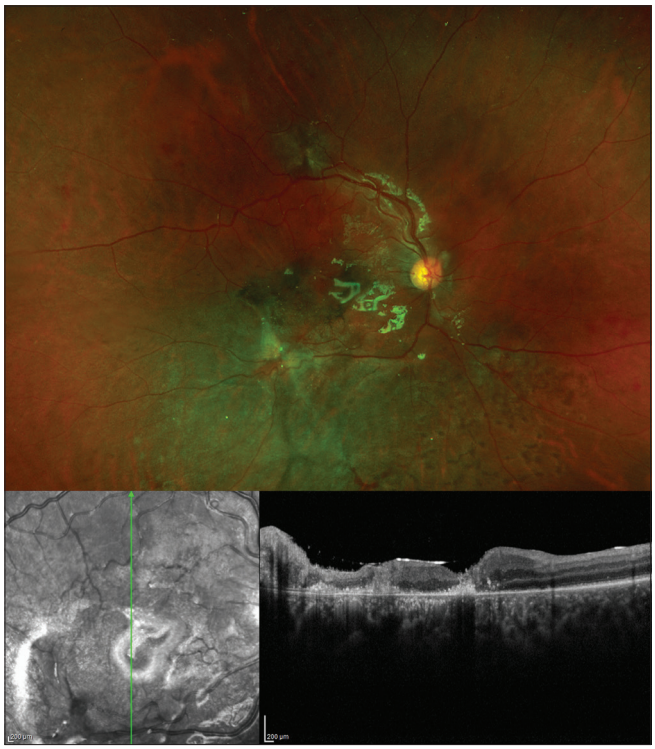

Figure 3: Right eye postoperative fundus image showing scarring and retinal pigment epithelium clumping at the macula with silicone oil in-situ (Top) and postoperative OCT scan showing an attached macula, subretinal scarring, retinal pigment epithelium clumping and outer retinal thinning (Below)

vitreous bleed, traumatic macular hole and rhegmatogenous retinal detachment complicated by proliferative

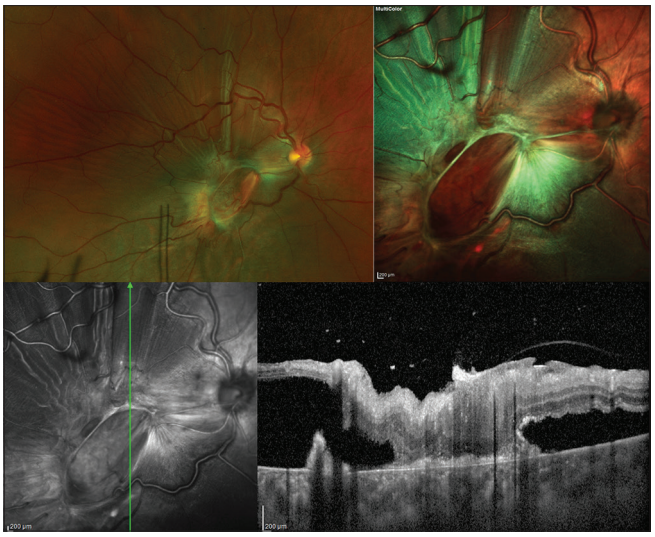

Figure 2: Right eye fundus image showing extensive premacular membranes and proliferation causing a macular pucker (Top left), multicolor image showing the extent of premacular membranes and traction (Top right) and OCT macula scan showing a thick premacular membrane and tractional retinal detachment (Below)

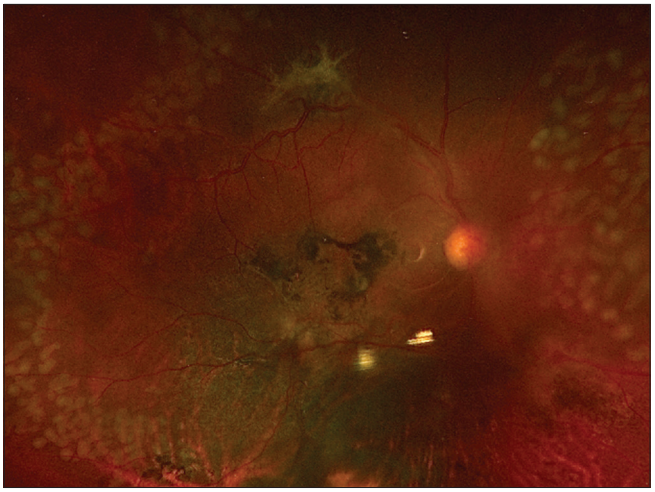

Figure 4: Right eye fundus image post silicone oil removal showing scarring at the macula

vitreoretinopathy (PVR).[4] A traumatic injury to the eye, whether closed or open globe, causes inflammation leading to breakdown of the blood-retinal barrier (BRB). This process includes chemotaxis of inflammatory cells like macrophages, lymphocytes, and polymorphonuclear cells leading to scar proliferation, tissue remodeling, and eventual restoration of retinal integrity.[6,7] An associated intraocular hemorrhage can lead to further leakage of fibrin and pro-inflammatory growth factors. These events get further accentuated with retinal breaks or retinal detachments due to RPE migration and eventual PVR.[6,7] Epiretinal membranes form when cells from within the retina, such as the RPE, Müller cells, and astrocytes, start proliferating and drifting onto the surface of the retina.[6] These cells grow through the internal limiting membrane and proliferate along the inner retinal surface forming more localized membranes with a collagen component that gives it a white appearance.[7,8] Once this scaffold forms, other cell

types present at the vitreoretinal interface, such as hyalocytes and macrophages, contribute to the ongoing proliferation.[6,7]

Despite a short history of trauma, our patient showed rapid progression with an extensive and rapid glial cell proliferation and contraction of the preretinal vitreous face. Machemer,[8] MacLeod and colleagues[9] in 1977 described an entity called "Massive Periretinal Proliferation" (MPP), a severe form of vitreous contraction that can complicate retinal detachments, especially with a rhegmatogenous element existing. Sarit *et al*. also showed that the proliferation rate is higher in membranes of shorter duration.[7] Previous studies of vitreous fluid from patients with PVR have shown increased thrombin activity, increased activation of proinflammatory and profibrotic pathways in the RPE cells.[10,11]

The severity of the blunt trauma could have precipitated a severe proliferation along the posterior hyaloid face leading to a macular TRD despite the absence of a rhegma in our patient. The possible mechanism could be the extensive RPE damage leading to a retinal remodeling and retinal glial tissue proliferation along the interface between the posterior hyaloid face and the retinal surface. A tangential contraction of these membranes can lead to the tractional component with the vitreous hemorrhage and inflammation further worsening the proliferation. The unbalanced action of growth factors and cytokines can also add to the element of fibrosis.Despite an early intervention, the visual acuity remained suboptimal in our case due to the primary outer retinal damage and scarring from the subretinal bleed.

Conclusion

While epiretinal membrane formation is reported, this case represents a rare, unusual and vision-threatening complication of blunt trauma which has not been reported in the literature so far. Hence, a close follow-up and timely management, even in the absence of a retinal tear or dialysis, is imperative in those with severe trauma.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflict of interest

There are no conflict of interest.

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