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Optical coherence tomography angiography-guided diagnosis of a traumatic choroidal rupture-associated choroidal neovascular membrane and its management with intravitreal ranibizumab

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Abstract:

A 25-year-old female presented with sudden onset diminution of vision in the right eye (oculus dextrus [OD]) following blunt trauma after a sports injury. Ocular examination revealed best-corrected visual acuity (BCVA) of 20/30 in OD and fundus revealed commotio retinae, localized preretinal bleed, and a large choroidal rupture (CR). She was managed conservatively at that moment. Three months following trauma, she returned with further deterioration of BCVA (20/80) in OD. Optical coherence tomography angiogram (OCTA) confirmed the presence of choroidal neovascular membrane (CNVM). She was treated with single intravitreal ranibizumab injection. Repeat OCTA after 6 weeks showed the regression of CNVM. Her BCVA improved to 20/30 at 6 months of follow-up. In the index report, we present a unique OCTA guided the diagnosis of posttraumatic CR-associated CNVM and its successful management with single intravitreal ranibizumab.

Keywords:

Anti-vascular endothelial growth factor, blunt ocular trauma, choroidal neovascular membrane, choroidal ruptures, optical coherence tomography angiogram

Introduction

Von Graefe first described choroidal rupture (CR) as a break in the choroid, Bruch's membrane (BM), and retinal pigment epithelium (RPE).^[1] It usually occurs secondary to closed-globe injuries but can also be observed in open-globe injuries.^[2] Sports injuries involving tennis ball, cricket, basketball, and badminton have especially been incriminated for the causation of closed globe blunt trauma to the eye. Choroidal neovascular membrane (CNVM) is an infrequent but visually significant

complication of traumatic CR. Intravitreal anti-vascular endothelial growth factor (VEGF) is considered as the mainstay of the management of CR-associated CNVM.^[3] Currently, optical coherence tomography angiography (OCTA) has emerged as a noninvasive imaging modality for the early detection of CNVM of variable etiology with good specificity.^[4] In the index case, we report OCTA-guided early diagnosis of a traumatic CR-associated CNVM in a young female following sports injury and demonstrate its successful regression with single intravitreal ranibizumab injection.

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Case Report

A 25-year-old female presented to the ophthalmology department with complaint of sudden diminution of vision in the right eye (oculus dextrus [OD]). She sustained a blunt trauma in OD with shuttlecock while playing badminton 3 days back. Examination revealed best-corrected visual acuity (BCVA) of 20/30 in OD and 20/20 in the left eye (oculus sinister [OS]). Intraocular pressure was 18 mmHg in OD and 16 mmHg in OS. Pupillary reflexes were normal in both eyes (oculus uterque [OU]). Detailed slit-lamp examination of the anterior segment of OU was unremarkable. Dilated fundus examination of OD [Figure 1a] revealed commotio retinae and a localized preretinal hemorrhage inferior and nasal to optic disc. A large crescentic CR was also noted extending from superonasal peripapillary area to approximately one disc-diameter inferior to fovea; middle extent of the CR was obscured by the presence of preretinal bleed. Binocular indirect ophthalmoscopy also showed mild inferior vitreous hemorrhage without any obvious retinal dialysis. Fundus examination of OS [Figure 1b] was unremarkable. Systemic examination was noncontributory. Spectral domain optical coherence tomography (SD-OCT) (Cirrus™, Carl Zeiss Meditec, Dublin, CA, USA) of OD at presentation [Figure 1c] showed disruption of RPE-BM complex with back-scattering corresponding to the inferotemporal extent of CR. Macular SD-OCT of OS [Figure 1d] showed normal foveal contour. She was advised baseline angiography, but she refused to give consent for any further evaluation at this point of time. She was managed conservatively with short duration of oral steroid in tapering dose, topical steroid, and cycloplegic agent.

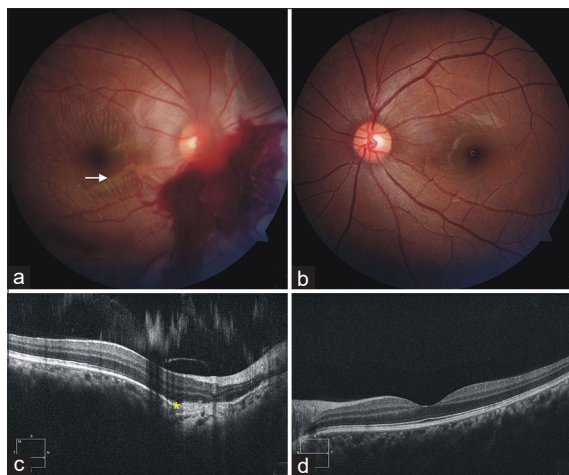


Figure 1: Fundus photograph of the right eye at presentation (a) showing commotio retinae, preretinal hemorrhage in peripapillary area, and choroidal rupture (CR; white arrow). Optical coherence tomography (OCT) of right eye (c) showing disruption of retinal pigment epithelium-Bruch's Membrane layer corresponding to the area of choroidal rupture. FP (b) and OCT (d) of the left eye was unremarkable

Three months from the initial presentation, she returned to us with a complaint of further deterioration of vision in OD. Examination revealed BCVA of 20/80 in OD. Fundus of OD [Figure 2a] showed the resolution of preretinal hemorrhage, well-delineated CR abutting the optic disc, and a subfoveal yellowish lesion at infero-temporal extent of the CR. Repeat SD-OCT scan of OD [Figure 2b] showed disruption of the RPE-BM complex and hyperreflective lesion involving the ellipsoid zone and interdigitation zone. Considering her doubtful drug allergy history, conventional fundus fluorescein angiography (FFA) was not performed. OCT angiography (OCTA) (Angiovue, Optovue, Inc., Fremont, CA, USA) enface scan demonstrated the presence of a well-defined network of anastomotic vessels with hyperintense signal in the outer retina and choriocapillaris layer [Figure 3c and d] with sparing of superficial and deep retinal vasculature [Figure 3a and b]. Corresponding OCT B-scan in Optovue [Figure 3e and f] revealed the presence of intraretinal fluid adjacent to hyperreflective material. A diagnosis of secondary CNVM associated with traumatic CR was made. She was explained about the need of treatment and was administered a single dose of intravitreal injection ranibizumab (0.5 mg/0.05 ml) in OD under aseptic precautions. At 4 weeks follow-up after intravitreal injection, her visual acuity improved to 20/40 in OD. Repeat enface scans using OCTA at 6 weeks [Figure 4a-d] revealed disappearance of hyperintense signal at the level of the outer retina and choriocapillaris, suggesting the regression of CNVM following intravitreal ranibizumab. Corresponding OCT B-scan [Figure 4e and f] demonstrated the reduction of hyperreflective material, disappearance of intraretinal fluid, and partial resolution of the RPE-BM layer disruption. At 6 months of follow-up

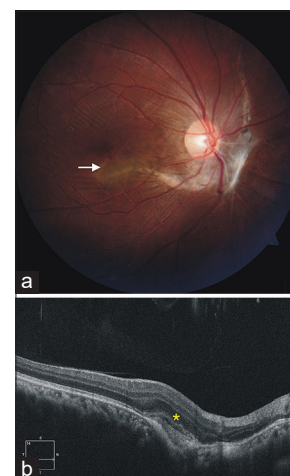


Figure 2: Fundus photograph of right eye after 3 months (a) showing choroidal rupture abutting the optic disc and a juxta-foveal yellowish lesion (white arrow). Optical coherence tomography of right eye (b) showed disruption of retinal pigment epithelium-Bruch's membrane complex and hyperreflective lesion (yellow star) involving the ellipsoid zone and interdigitation zone

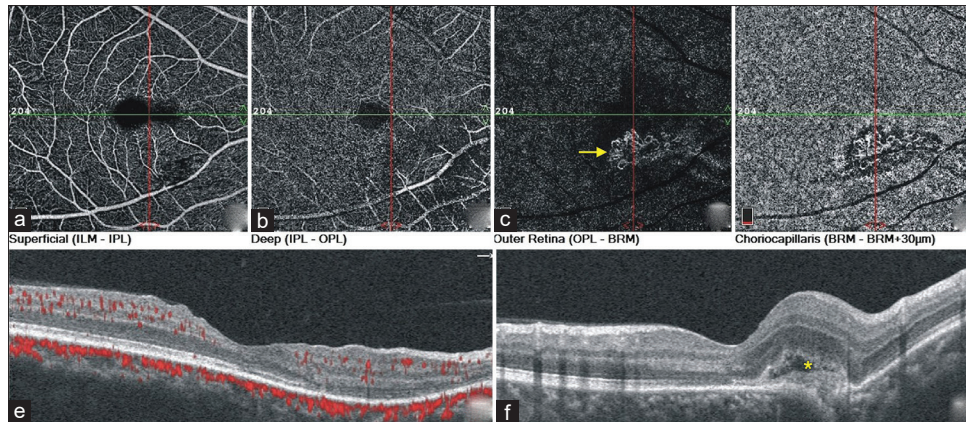


Figure 3: Optical coherence tomography angiography enface scan at 3 months demonstrated a vascular anastomotic network with hyperintense signal (yellow arrow) in the outer retina (c) and choriocapillaris layer (d) with sparing of superficial and deep retinal vasculature (a and b). Corresponding optical coherence tomography B-scan (e and f) revealed the presence of intraretinal fluid adjacent to hyperreflective material (yellow star)

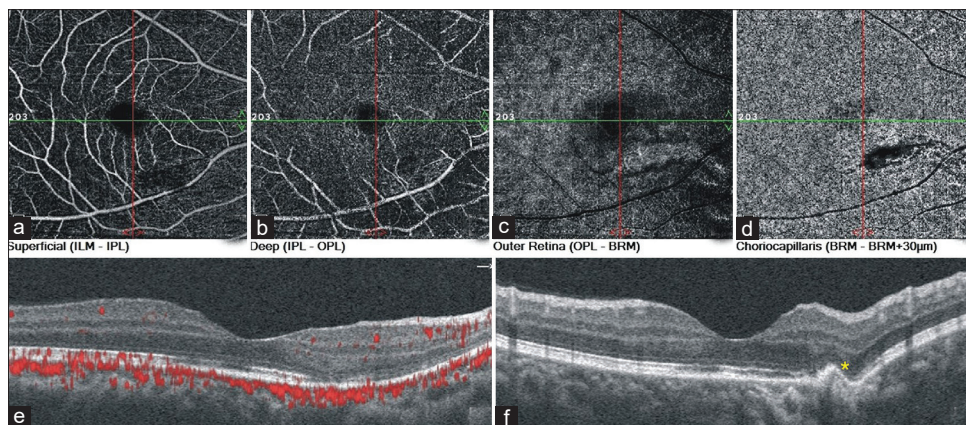


Figure 4: Optical coherence tomography angiography enface scans (a-d) 6-week after intravitreal ranibizumab showing disappearance of hyperintense signal. Corresponding optical coherence tomography B-scan (e and f) showed the resolution of intraretinal fluid and partial healing of wound involving retinal pigment epithelium-Bruch's membrane layer (yellow star)

from initial presentation, her BCVA improved to 20/30 in OD. Fundus showed old CR with peripapillary fibrosis [Figure 5a] and OCT [Figure 5b] revealed healed RPE-BM layer wound without any activity of CNVM. She was kept under monthly follow-up thereafter.

Discussion

CR has been reported in 5%–10% of cases of blunt ocular trauma.^[5] It may occur directly at the site of impact or more commonly at a site distant from the place of injury due to countercoup mechanism.^[5] Various hypotheses have been postulated to explain the genesis of CR. Sudden anteroposterior deformation of the globe following trauma with subsequent equatorial expansion has been incriminated for CR. Temporal half of the retina may be predisposed more in contrast to its nasal counterpart as the temporal ocular coats receive maximum stress due to upward and outward rolling of the globe during protective Bell's phenomenon.^[6] Presenting visual acuity in traumatic CR may vary from 20/20 to as

worse as light perception only, depending upon the site of rupture and other associated ocular injuries.^[2] CR may be complicated by acute subretinal hemorrhage, associated traumatic optic neuropathy, peripapillary pigment epithelial disruption, or development of sub retinal neovascularization;^[7] all of these can have varied impact on the final visual acuity.

The development of secondary CNVM is an infrequent, but well-recognized and visually significant complication of CR. Risk of CNVM has been increasingly associated with length of CR, age, and location within the arcades.^[5] Loss of mechanical and chemical barrier due to interruption in the RPE-BM complex and VEGF upregulation secondary to increased release of elasin-derived peptides from damaged elastic lamina of BM, have been incriminated in pathophysiology of CR associated CNVM.^[8] Abundance of perfusion from the choroidal vasculature and more intense wound-healing process predispose the macular region further to increased propensity to CNVM formation.^[9] Russell *et al.*

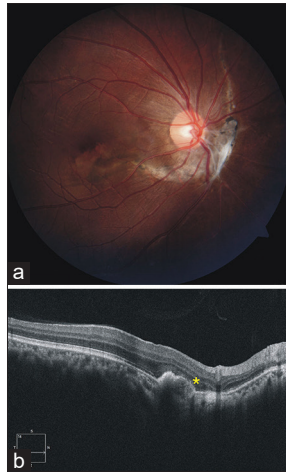


Figure 5: Fundus photograph (a) and optical coherence tomography (b) of right eye 6 months after the initial presentation showing less reflective retinal pigment epithelium and absence of any intraretinal or subretinal fluid (yellow star)

in their retrospective case series including 101 eyes of CR had reported CNVM formation in 8 patients (7.9%) after a mean interval of 2 months following trauma.^[10] In another large retrospective review of 111 patients with traumatic CR, 12 patients were diagnosed with CNVM (10.8%); older age and macular CR were found to have strong association.^[2] However, Barth *et al.* had reported relatively higher incidence of CNVM (20%) out of 54 diagnosed patients with posttraumatic CR.^[3] Thus, although CR itself requires no active intervention, it must be closely monitored for the potential of CNVM formation. In our patient, large crescent-shaped CR was noted; temporal extent of the rupture was close to fovea. Three months following injury she reported sudden decline in visual acuity which raised the suspicion of possible CNVM formation.

Currently, OCTA has evolved as a novel, fast, *in vivo*, noninvasive imaging modality able to provide both structural and functional information of the microvasculature of retina and choroid without any intravascular dye injection. Not only does it provide three-dimensional scans and volumetric angiographic information but also it obtains enface segmentation scans in the different layers of retina and choroid.^[11] OCTA can be used as an early diagnostic tool with good sensitivity and specificity^[12] for the detection of CNVM in traumatic CR. Although classically FFA and Indocyanine Green Angiography have been considered as the primary imaging modalities for the confirmation of diagnosis of CNVM, utility of both of these modalities is particularly limited in the setting of hypersensitivity and allergy. We avoided performing FFA in our patient due to drug allergy being suspected as per patient's history. Although a presumptive diagnosis of CNVM was already made using SD-OCT [Figure 2b], demonstration of the anastomotic vascular network in outer retinal and

choriocapillaris layer by enface scans of OCTA confirmed the presence of active CNVM. The absence of clinically visible retinal hemorrhage and paucity of intraretinal or subretinal fluid associated with CNVM in OCT may raise the dilemma of treatment versus observation. OCTA becomes a very handy noninvasive imaging modality in such equivocal scenario by depicting the hyperintense flow signal pointing active CNVM and need for urgent intervention. We also demonstrated successful regression of CNVM following anti-VEGF injection in subsequent OCTA images.

In a case report by Benillouche *et al.*, the authors demonstrated contraction and remodeling of the neovascular flow in OCTA following treatment with intravitreal ranibizumab in a posttraumatic CR-associated CNVM in a young patient with kidney transplant. They showed that the contraction of CNVM in OCTA was maximum at 1 week after postinjection as compared to at 1 month after injection. However, the authors concluded that other multimodal imaging should also be taken into consideration while planning treatment, as the activity of CNVM lesions may be difficult to assess from OCTA alone.^[13] Preziosa *et al.* and Lorusso *et al.* demonstrated OCTA-guided evolution of posttraumatic CR-associated CNVM managed with single intravitreal bevacizumab in two separate reports.^[14,15] Preziosa *et al.* described a defined tangled network of CNVM within a regular line of severe choriocapillary rarefaction corresponding to the area of CR by OCTA.^[14] In another report by Pierro *et al.*, the authors demonstrated CR by OCTA as breaks in the choriocapillary plexus with a hypointense appearance (due to lack of substance) with projection of underlying hyperintense choroidal vasculature along the break while the retinal vascular plexus was spared. However, there was no CNVM formation in this case.^[16] In a report by Rezaei *et al.*, the authors demonstrated evolution and progression of vascular network patterns in the areas of posttraumatic CR suggestive of possible CNVM by OCT-based microangiography, although the lesion regressed over time without need of any anti-VEGF injection.^[17]

Intravitreal anti-VEGF pharmacotherapy is a well-recognized and effective treatment option for CNVM secondary to traumatic CR. Other treatment modalities include observation, focal laser photocoagulation, submacular surgery, or photodynamic therapy;^[2] all of these modalities were in use prior to anti-VEGF era. Russell *et al.* in their case series of eight patients with CR associated CNVM, had achieved anatomical and visual stability after a median of two intravitreal anti-VEGF agents.^[10] Barth *et al.* have treated five patients of CR associated active CNVM by either bevacizumab or ranibizumab with an average of 4.2 injections per eye for regression over a mean follow-up period of 5 years.^[3]

However, in 40% of eyes single injection of anti-VEGF was sufficient to induce quiescence, and only one eye showed recurrence 6 months post treatment. The remaining three eyes (60%) were treated with upload of three monthly injections initially.^[3] This might explain the higher mean number of injections reported for achieving quiescence by Barth *et al.*^[3] as compared to other reports.^[10] In our case, the patient responded to a single intravitreal anti-VEGF agent and her visual acuity at 6-month follow-up improved to 20/30. As the CR associated CNVM was juxtafoveally located, the CNVM might not be the sole contributing factor for visual decline in this case. It may be additionally attributable to the sequale of other blunt trauma manifestations also like mild disc pallor, localized anatomical or functional photoreceptor degeneration following commotio retinae, and remodelling of subretinal scar tissue.

Although there are a few case series describing the management of posttraumatic CR associated CNVM with anti-VEGF, the literature has very few isolated case reports demonstrating the use of OCTA in depicting the evolution of CR associated CNVM.^[13-17] In the index report, we presented OCTA-guided the diagnosis of secondary CNVM following CR, where conventional FFA could not be performed and also showed its successful management with single injection of intravitreal ranibizumab.

Conclusion

CNVM is a potential sight-threatening complication of CR following blunt trauma. A high index of clinical suspicion, appropriate investigative modalities such as OCTA, and timely intervention with intravitreal anti-VEGF injection may help in restoring visual acuity in such patients. OCTA helps as a novel, noninvasive, fast-imaging modality to diagnose and follow-up such patients with precision and accuracy especially where conventional FFA is contraindicated. However, future prospective studies with OCTA involving more number of patients and longer follow-up periods may provide additional insight regarding posttraumatic CR associated CNVM.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initial will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

The authors declare that there are no conflicts of interests of this paper.

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