Early Onset Choroidal Neovascular Membrane after Choroidal Rupture: Case Report

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Abstract

Introduction: Choroidal rupture is a break in the retinal pigment epithelium, Bruch membrane and the choroid. Case Presentation: A 58-year-old male patient was admitted to the clinic with complaint of vision loss on the left eye. He had blunt trauma to his left eye 3 weeks ago. He had had choroidal neovascularisation (CNV) due to choroidal rupture. Discussion: CNV is usually a late complication after choroidal rupture, but it may also occur at an early stage. *Keywords: Blunt Trauma; Choroid Neovascular Membrane; Choroidal Rupture; Optic Coherence Topography*

Introduction

Choroidal rupture is a break in the retinal pigment epithelium, Bruch membrane and the choroid. It mostly occurs after blunt trauma but it was showed that it could occur after penetrating and perforating traumas [1-3]. One of the most important complication of choroidal rupture is choroid neovascular membrane (CNVM). In this report, we presented a patient with choroidal neovascular membrane due to choroidal rupture 3 weeks after blunt trauma.

Case Presentation

A 58-year-old male patient was admitted to the clinic with complaints of vision loss on the left eye for 3 weeks. The vision at the arrival of the patient is 1.0 (Snellen equivalent (SE)) on the right eye and 0.4 (SE) on the left eye. Anterior segment examination; natural on the both eyes. Intraocular pressure (IOP) was bilaterally 18mHg. Dilated fundus examination showed normal optic nerve, macula and retinal vascularisation on the right eye and there was choroidal rupture which is close to temporal side of the optic nerve on the left eye. And there was choroidal neovascular membrane on the choroidal rupture area. We learnt from the patient that he had blunt trauma 3 weeks ago. Optic coherence topography (OCT), colour fundus photo and fundus auto florescence were taken (Figure 1-3). Pigment epithelial detachment (PED), choroidal neovascularisation were observed in the OCT of the patient. We arranged the indocyanine green fundus fluorescein angiography (ICGA) which is ideal for imaging of choroid circulation [4]. Early phase (30 seconds), ICGA showed crescent shaped, hypercyanescent lesion and hypocyanescent rupture area at the inferonasal of the macula. Late phase (15 minutes); hypercyanescent corresponding to choroidal rupture with CNVM (Figure 4 and 5). ICGA demonstrated sign of active CNV such as dye leakage or staining. Additionally, there was subretinal fluid on OCT. Intravitreal anti-vascular endothelial growth factor (VEGF) injection treatment procedure was started. The patient has been seen by our retina department for follow-up.

Discussion

Retinal detachment, scleral rupture, lens dislocation, intravitreal hemorrhage, traumatic cataract may seen after blunt ocular trauma. Choroidal rupture is one of the rare complication which can occur after blunt trauma. And these ruptures may occur from direct contact at the site of injury or indirect contact at the opposite site of injury which calls as countercoup. The latter is the more common. As our case, ruptures are more commonly located temporal to the disc than nasal and in the macula. This can be explained with the high blood flow and oxygenation [1,5,6]. The mechanism of the choroidal rupture is antero-posterior compression and horizontal expansion of the globe. This causes rupture in the choroid. Because choroid is less flexible than the retina and less stronger than sclera. So choroid layer can be ruptured easily [5,6].

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Figure 1: Colour fundus photo, OS. Pigment epithelial detachment (long arrow) is seen in the infero-temporal to the optic nerve and the second one (short arrow) is smaller and is located in the temporal side of the macula.



Figure 2: Fundus autofluorescence, OS. Choroid rupture is seen as crescent-shaped streak and hyperfluorescent on autofluorescence imaging (arrow).

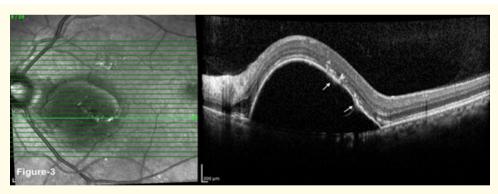


Figure 3: Optic Coherence Topography (OCT), OS revealed a disruption on of the retinal pigment epithelium (RPE)/Bruch membrane (BM) complex (arrows).



Figure 4: ICGA early phase, OS. Crescent shaped, hypocyanescent rupture site (arrow) and there is hypercyanescent area around the rupture.

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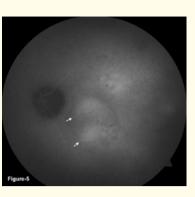


Figure 5: ICGA late phase, OS. After 15 minutes hypercyanescent lesions corresponding to the CNV.

Several factors such as Bruch membrane and retinal pigment epithelial layer disruption and inflammation after trauma which can increases the VEGF level can be the reasons for CNV due to choroidal rupture [3]. CNVs mostly occur within the first year after trauma. Older age, location of rupture within the arcades, longer length of the rupture, multiple choroidal ruptures and submacular haemorrhage increase the risk of CNV. Also pathological myopia is the cause of CNV on its own, so it can be thought that these patients are prone to formation of CNV after choroidal rupture [7-9]. In our study, early onset CNV mechanism can explain with macular localisation of the rupture and the age. Other studies also emphasized that retina pigment epithelium cannot inhibit neovascularization completely around the fovea so CNVs are more in the perifoveal area [9].

It also prefers to create type 2 CNV. Choroidal ruptures without complication do not require treatment. If the patients have foveal CVNM or subretinal fluid; laser photocoagulation, photodynamic treatment, intravitreal anti-VEGF injections or surgery can be perform [7].

There is no enough study for intravitreal anti-VEGF injection treatment after CNV due to choroidal rupture. Some studies stated that CNV may still occur after years. Therefore long time period follow-ups are necessary and important for the patient and the disease [6]. Visual outcomes for these patients are poor and final visual acuity is mostly worse than 20/40. Localisation of the rupture, rupture size and early treatment are important for the final visual acuity [1,10].

In our case, clinical manifestation has been presented with blurred vision complaint. A detailed examination especially dilated fundus examination is really important for these patients who have vision loss after trauma. CNV is mostly delayed complication after choroid rupture but there are some studies reported that it may occurs at early stage, as our study [6]. Wang., *et al.* treated their patient -who had blunt eye injury 3 days ago- with intravitreal injection of expandable gas and additionally intraocular anti-VEGF injection. The patient had early-stage choroidal rupture-induced subretinal hemorrhage and the complications of suspected CNV. In our study CNV has been seen 3 weeks after blunt trauma without subretinal or intravitreal hemorrhage. The case of this early cvvm is rarely found in the literature. We treated our patient with intravitreal aflibercept (Eylea; Bayer AG, Basel, Switzerland) injection. To our opinion, If early-stage CNV is in the foveal or parafoveal region and decreases the visual acuity, the treatment should be started immediately.

As we mentioned before, treatment procedure can be perform according to localization and the additional pathologies.

Conclusion

As a result, for the diagnosis of CNV; detailed examination, OCT and ICGA imagings are crucial. CNV can be seen as an early complication after choroid rupture. Therefore we should remember it and its treatment in every patient who had trauma to the eye. Also these patients can be treated with anti-VEGF injections, laser photocoagulation or surgery. According to us, for CNVs which are located foveal and parafoveal region, anti-VEGF treatment should be the first option. Finally, we should not forget that this disease will require long-term follow-up and treatment.

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