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Case Report

# The Response to Single Dose Intravitreal Ranibizumab Injection in a Child Case with Traumatic Choroidal Neovascularization

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## Abstract

Choroidal neovascularization (CNV) may occur due to traumatic choroidal rupture and cause visual loss because of the macular hemorrhage or edema. Here we describe one case of CNV related to traumatic rupture of Bruch's membrane which was successfully treated with intravitreal injection of ranibizumab. A 10 years old child admitted to our clinic with a complaint of metamorphopsia and the visual loss in the left eye five months after blunt ocular trauma. His best corrected visual acuity (BVCA) in his left eye was 20/200. Optical coherence tomography showed subretinal liquid accumulation next to hyperreflective lesion attributed possible choroidal neovascularization in the choroidal rupture region at 5$^{th}$ month. Single intravitreal injection of ranibizumab 0.5 mg/0.05 ml was performed. His BCVA improved to 20/50; the complaint of metamorphopsia reduced; subretinal liquid totally resolved.

**Conclusion:** A single intravitreal ranibizumab injection may provide visual improvement and the resolution of subretinal liquid due to traumatic CNV in the child cases.

***Keywords:*** *Traumatic Choroidal Rupture; Intravitreal Ranibizumab; Choroidal Neovascularization*

## Introduction

The choroidal neovascularization (CNV) due to Bruch membrane injury might be developed in %20 of the cases with indirect choroidal rupture due to a closed blunt eye trauma [1]. In previous studies, it has been reported that advanced age, the length of the rupture, the level of initial visual acuity and the localization of the lesion regarding the closeness to fovea are prognostic indicators for choroidal rupture [2]. The usage of laser photocoagulation and photodynamic treatment is limited because of the poor visual prognosis. In recent years, anti-VEGF agents have been used for the treatment of various choroidal neovascular diseases. However, the reports regarding the usage of these agents in the child cases with choroidal neovascularization are limited numbers [3-6].

Here, we report the anatomical and visual response to single dose intravitreal ranibizumab injection in a child case with secondary choroidal neovascularization to traumatic choroidal rupture.

## Case Report

Ten years old child admitted to our clinic with a complaint of the visual loss in his left eye following ocular contusion, caused by a stone two weeks ago. His systemic medical history was unremarkable. His best-corrected visual acuities (BCVA) were 10/10 in the right eye and 20/200 in the left eye. His intraocular pressure (IOP) was 14 mmHg in both eyes. The slit-lamp examination did not reveal any pathological sign in both eyes. Fundus examination is normal in the right eye (Figure 1A). Ophthalmoscopy revealed a vertical choroidal rupture crossing the temporal to foveola and mild retinal hemorrhage around the rupture (Figure 1B). Fundus fluorescein angiography (FFA) did not reveal any CNV and dye leaking except for hypo fluorescence due to mild retinal hemorrhage and retina pigment epithelium (RPE) window defect at the area of choroidal rupture in the left eye at the presentation (Figure 2A and 2B).

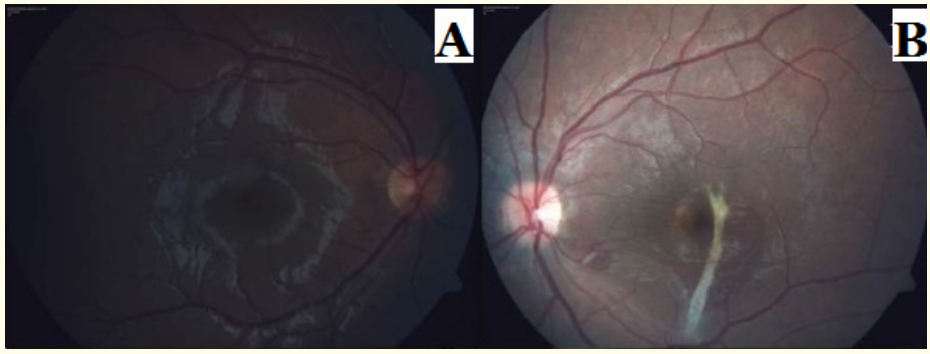


***Figure 1:*** *Color macula images of the right (A) and the left eye with choroidal rupture at temporal to foveola (B) at the presentation.*

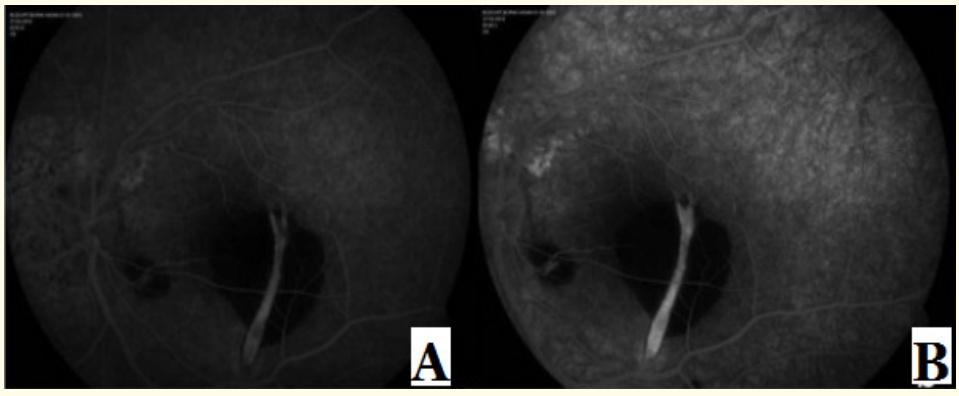


***Figure 2:*** *Early (A) and late (B) stage fundus fluorescein angiographic images of the left eye with choroidal rupture at the presentation. There is no choroidal neovascular membrane and leaking.*

Optical coherence tomography (OCT) scan showed hyperreflective scar formation and the disruption and thickening of RPE-chorio-capillaris (CC)-Bruch membrane complex. However, any subretinal or intraretinal liquid accumulation or edema in the fovea and the area of the choroidal rupture in the left eye was not observed at the first presentation (Figure 3A).

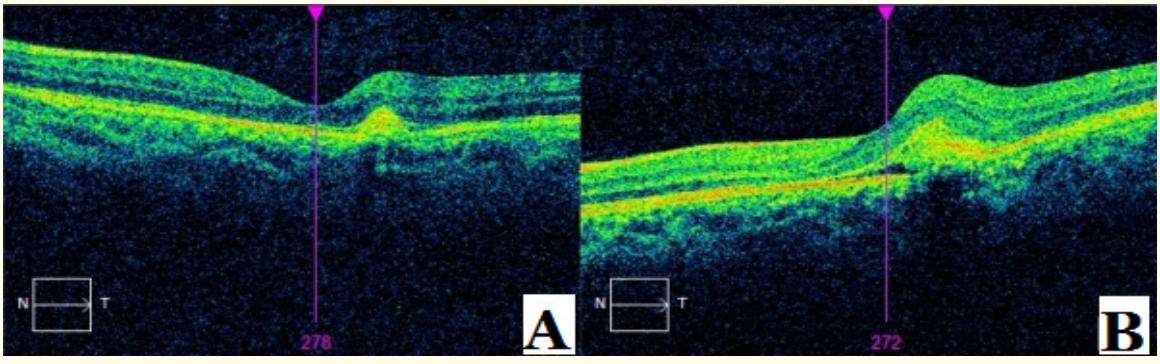


***Figure 3:*** *Optical coherence tomography scans crossing the choroidal rupture in the left eye. It shows hyperreflective scar formation and the disruption and thickening of RPE- CC-Bruch line without foveal edema at the first presentation (A)and subretinal liquid accumulation next to hyperreflective lesion attributed possible choroidal neovascularization at 5 month (B).*

The case admitted to our clinic again with a complaint of metamorphopsia five months after trauma. BVCA in his left eye was 20/200. OCT scans crossing the choroidal rupture in the left eye showed hyperreflective scar formation and the disruption and thickening of RPE-CC-Bruch membrane complex and subretinal liquid accumulation next to hyperreflective lesion attributed possible choroidal neovascularization at 5th month (Figure 3B). Color macula image of the left eye showed the scar formation in the area of choroidal rupture and retinal hemorrhage in foveola next to the choroidal rupture compared to that in the first presentation (Figure 4A and 4B). An afferent pupillary defect in the left eye was also noted in this ophthalmological examination. FFA was recommended again for the detecting the CNV. However, the case and parents did not accept to be performing FFA once again. So, a CNV secondary traumatic choroidal rupture was diagnosed based on OCT scan and color fundus photography and intravitreal injection of ranibizumab was recommended. Written informed consent was obtained from parents in accordance with the Declaration of Helsinki. With intravenous sedation, single intravitreal injection of ranibizumab 0.5 mg/0.05 ml (Lucentis; Novartis Pharma AG, Basel, Switzerland) was performed. Follow-up examinations were performed monthly during the first 3 months, and then quarterly until month 12.

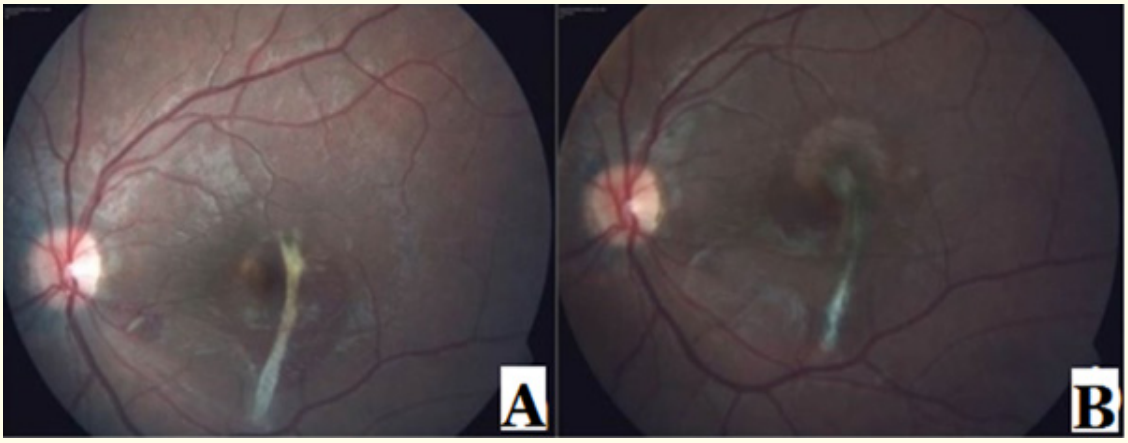


***Figure 4:*** *Color macula images of the left eye at the first presentation (A)and at 5th month (B). It shows the scar formation in the area of choroidal rupture (A) and retinal hemorrhage in foveola next to the choroidal rupture (A, B).*

At the examination of the post-injection 2nd month, BCVA improved to 20/50. The complaint of metamorphopsia reduced. OCT scans crossing the choroidal rupture showed the total resolution of subretinal liquid next to scar formation at the follow-up examinations at the first (Figure 5A) and 6th and 12th (Figure 5B) month after the intravitreal ranibizumab injection. At 12-month follow-up, VA remained stable at the level of 20/50. Any ocular or systemically side effect was not observed along follow-ups. Repetition of injection was not required.

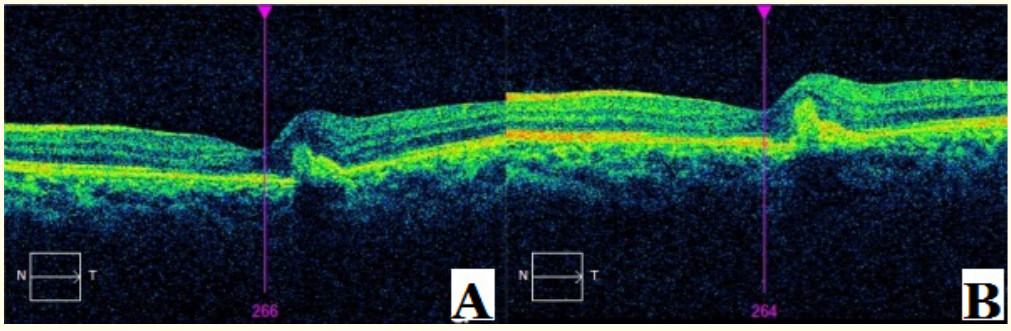


***Figure 5:*** *Optical coherence tomography scans crossing the choroidal rupture in the left eye. It shows the resolution of subretinal liquid next to scar formation at the follow-up examinations at the first (A) and 12th (B) month after the intravitreal ranibizumab injection, respectively.*

## Discussion

Traumatic choroidal rupture occurs due to the breakdown of RPE-CC-Bruch membrane following blunt ocular trauma. Choroidal rupture may be developing direct, occurring at the site of blunt trauma, or indirect, occurring far away from the primary site of blunt trauma (countercoup effect). According to previous reports, %80 of choroidal ruptures is indirect [1-3]. Indirect choroidal rupture is generally associated with non-penetrating closed- blunt ocular trauma. It has been considered that the implicated various mechanisms including breaks in the Bruch's membrane, inflammatory process, and angiogenic stimuli might play role in pathogenesis of CNV [1]. The Bruch's membrane is a physiologic barrier to the development of CNV [2,3]. However, Bruch's membrane has more tendencies to traumatic rupture because it is less elastic than the retina and is less tensile than the sclera [1-3].

The choroidal neovascularization (CNV) due to indirect Bruch membrane rupture might be developed in %20 of the cases with closed-blunt eye trauma [1]. In the injury of Bruch membrane, VEGF levels increase via the release of elastin-derived proteins and the promotion of inflammatory process [7].

In the treatment of CNV secondary traumatic choroidal rupture, multiple therapeutic approaches including the photodynamic therapy, laser photocoagulation and recently anti-vascular endothelial growth factor (VEGF) agents depending on CNV localization in relation to the foveal area have been used to limit or to reverse the visual loss. The reports on the usage of anti-VEGF agents in the children with CNV are limited. However, all of these reports demonstrated single intravitreal anti-VEGF injection (ranibizumab or bevacizumab) provided significant visual and anatomical improvement along at least of 12 months [3-6]. Also in our case, a single intravitreal ranibizumab injection provided vision improvement and total resolution of subretinal liquid due to traumatic CNV at least for 12 months. However, reports having more cases and long-term follow-ups, and studying security and safety of anti-VEGF therapy for traumatic CNV in children are needed.

## Financial Support

None.

## Conflicts of Interest

None.

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