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Case report

# Choroidal rupture after blunt ocular trauma in a 21-year-old patient: Case report

Natalia Kwaśniewska[1], Przemysław Ciszewski[1], Sebastian Sirek[2,3], Dorota Wyględowska-Promieńska[2,3]

[1]Students' Scientific Club, Department of Ophthalmology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
[2]Department of Ophthalmology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
[3]Department of Adult Ophthalmology, Prof. Kornel Gibinski University Clinical Center, Medical University of Silesia, Katowice, Poland

**Address for correspondence:**
Natalia Kwaśniewska
Studenckie Koło Naukowe
Klinika Okulistyki
ul. Ceglana 35, 40-514 Katowice
e-mail: s85936@365.sum.edu.pl

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## ABSTRACT

A choroidal rupture is a disruption of the continuity of the choroid Bruch's membrane, and the retinal pigment epithelium (RPE), most commonly associated with blunt ocular trauma. It occurs in 5–10% of patients after globe injury, more frequently in young men and during work-related activities or physical exercises. A 21-year-old female patient presented to the Emergency Department after sustaining blunt trauma to the left eye caused by being struck with a mobile phone. On admission, best-corrected visual acuity (BCVA) was 0.01, and intraocular pressure was 39 mmHg. Ophthalmic examination revealed subluxation of the crystalline lens, prolapse of the vitreous body into the anterior chamber and a choroidal rupture. Flash visual evoked potentials (VEP) showed normal latencies. However, the amplitude in the left eye was reduced to 50%. Fundus examination of the left eye demonstrated a rupture with displacement of the vascular bundle, signs of fibrosis, retinal hemorrhages, choroidal rupture, post-hemorrhagic vitreous opacities and macular edema. Fluorescein angiography revealed preserved flow in the major vessels, an area of hypo- and hyperfluorescence in the peripapillary region and scattered foci of blocked fluorescence inferiorly corresponding to dispersed hemorrhages. Based on the clinical presentation and ancillary test results, systemic and topical treatment was initiated, resulting in clinical improvement. Visual acuity remained at 0.01, while intraocular pressure decreased to 16 mmHg following pharmacological therapy. Post-traumatic choroidal rupture frequently coexists with other ocular structural injuries. It requires comprehensive diagnostic evaluation and management and the prognosis depends on the location and extent of the lesions as well as appropriately selected pharmacotherapy.

KEYWORDS

ocular trauma, lens subluxation, choroidal rupture

## INTRODUCTION

Choroidal rupture (CR) was first described by von Graefe in 1854 in patients with blunt ocular trauma. It is defined as a disruption of the choroid, Bruch's membrane, and the retinal pigment epithelium (RPE). Ruptures may be classified as direct (coup injury) or indirect (countercoup injury). Direct ruptures occur at the site of impact, typically parallel to the ora serrata and the peripheral retina. In contrast, indirect ruptures occur at a site distant from the impact, usually at the posterior pole, where they assume a crescent shape arranged concentrically around the optic disc. Approximately 80% of choroidal ruptures are indirect [1].

Choroidal rupture occurs in about 5–10% of patients with globe trauma and is three times more common in closed-globe injuries than in open-globe injuries [2]. During trauma, the globe

undergoes rapid mechanical deformation. The sclera, composed mainly of connective tissue, provides structural strength and relative resistance to injury. The retina is elastic and capable of stretching, whereas the RPE–Bruch's membrane–choroid complex has limited elasticity and tensile strength, making it susceptible to rupture. This disruption damages choroidal capillaries and results in bleeding into the subretinal space [3].

Sports injuries, physical assaults, and occupational accidents are the most common causes of CR, which explains its higher prevalence among young men. Patients with angioid streaks are at increased risk due to the fragility of Bruch's membrane [4]. Choroidal rupture may also occur during forceps-assisted delivery or as a complication of intravitreal anti-VEGF injections used in the treatment of retinopathy of prematurity [2].

This report presents a case of a young female patient who developed choroidal rupture with lens subluxation following blunt ocular trauma.

## CASE REPORT

A 21-year-old female presented to the Ophthalmic Emergency Department after sustaining blunt trauma to the left eye when struck with a mobile phone during an altercation with a friend. The patient was otherwise healthy and reported no relevant comorbidities.

On admission, best-corrected visual acuity (BCVA) in the left eye was 0.01, and intraocular pressure (IOP) measured 39 mmHg. Ophthalmic examination revealed subluxation of the crystalline lens, prolapse of the vitreous body through the pupillary margin into the anterior chamber, and choroidal rupture.

Fundus examination demonstrated a rupture with displacement of the vascular bundle, fibrotic changes, retinal hemorrhages, post-hemorrhagic vitreous opacities, macular edema, reduced perfusion in the inferior quadrant, and interrupted retinal vessels.

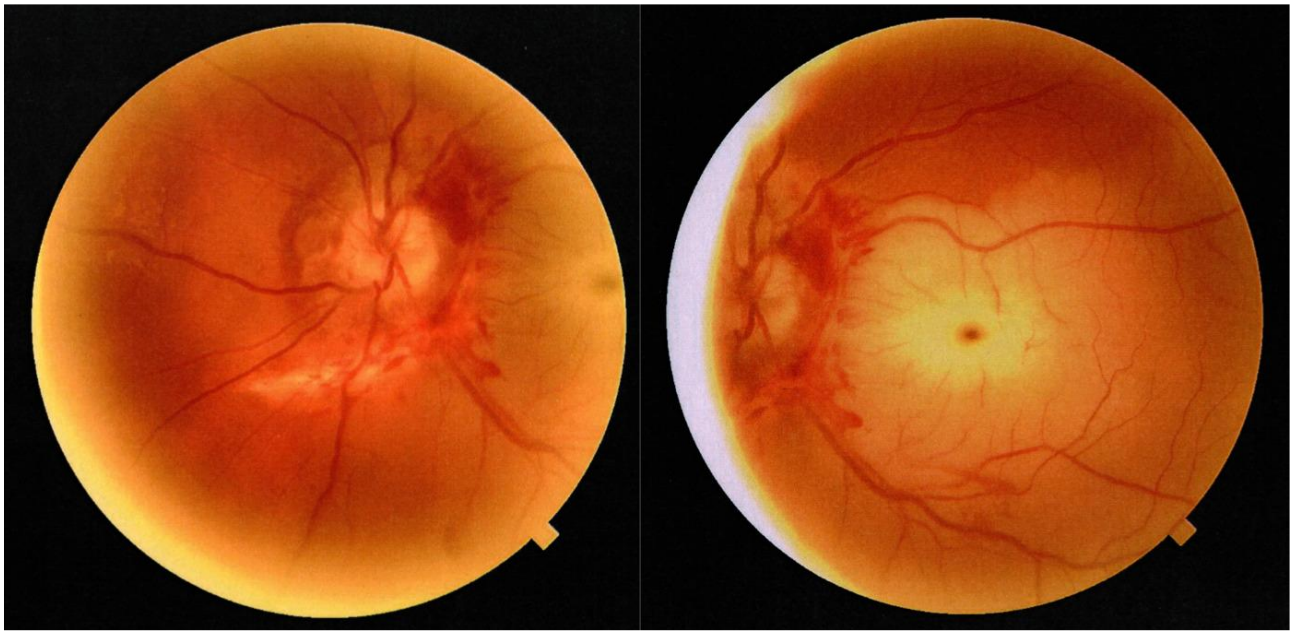

**Fig. 1.** Fundus photograph of the left eye

Flash visual evoked potentials showed normal latency; however, the amplitude in the left eye was reduced to 50% compared with the right eye. Fluorescein angiography demonstrated preserved flow through the major vessels. Visualization of the fundus was slightly hazy. A peripapillary area of hypo- and hyperfluorescence suggested edema, while scattered inferior foci of blocked fluorescence corresponded to dispersed retinal hemorrhages.

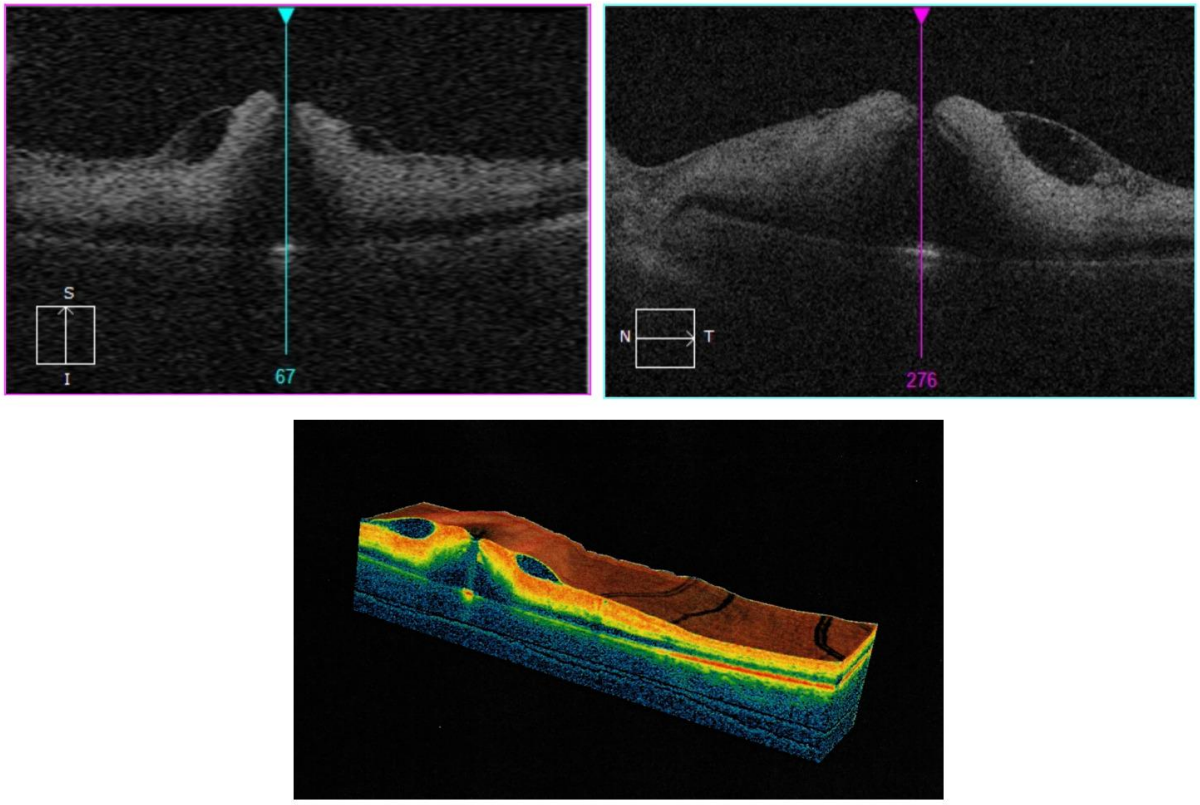


**Fig. 2.** OCT of the left eye

Based on the clinical findings and ancillary test results, combined systemic and topical treatment was initiated, resulting in gradual clinical improvement. Systemic therapy included acetazolamide, methylprednisolone (2 mg), and supplementation with magnesium and potassium. Topical therapy consisted of latanoprost once daily, bromfenac three times daily, a fixed combination of dorzolamide and timolol, tropicamide twice daily, and fluorometholone three times daily.

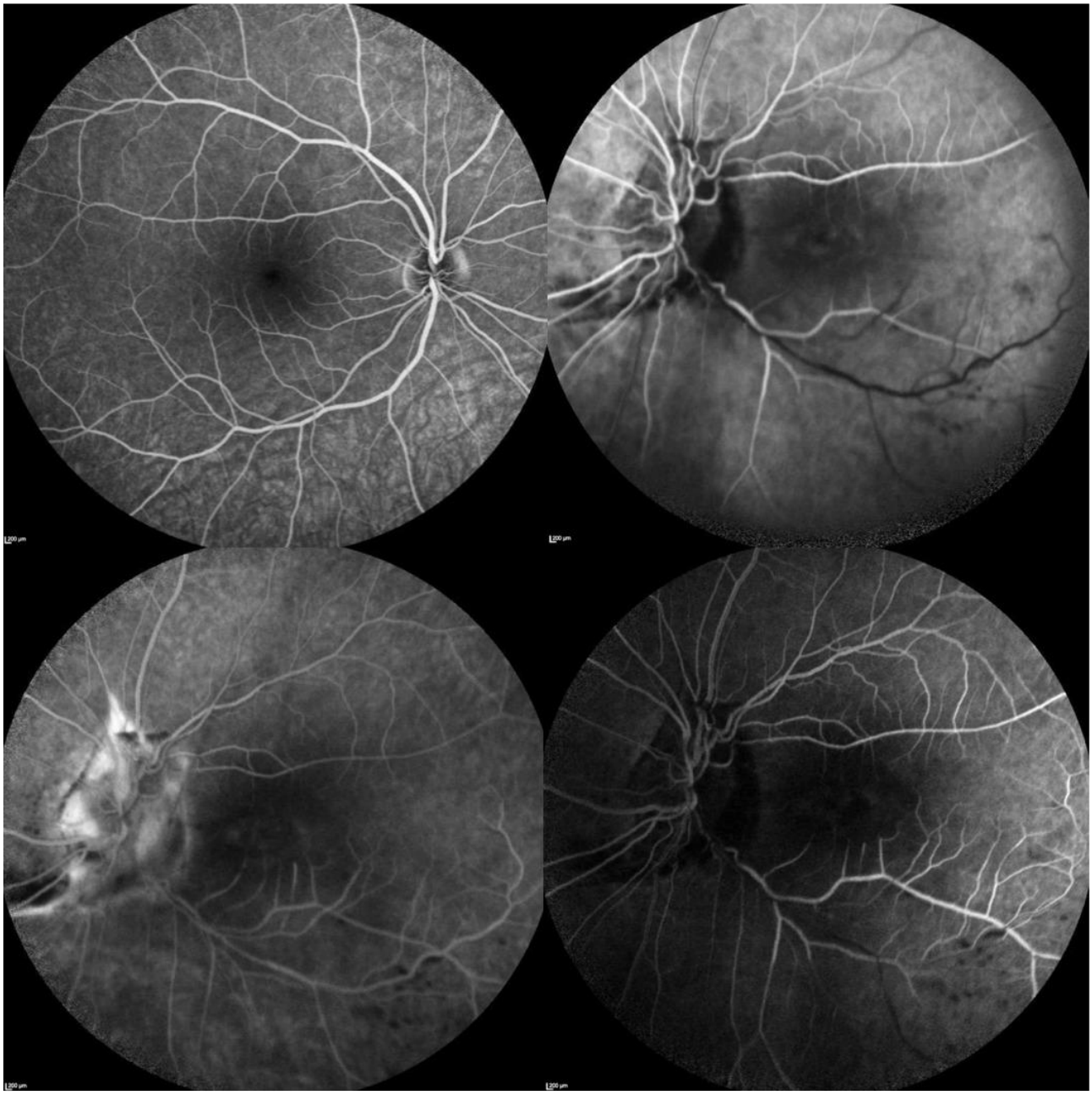

**Fig. 3.** Fluorescein angiography of the left eye

At a follow-up visit three weeks later, visual acuity remained 0.01, while intraocular pressure normalized to 16 mmHg. Ophthalmic examination revealed persistent vitreous prolapse, iridodonesis, inferior pigment dispersion within the vitreous, a pigment granule at the 10-o'clock position on the iris margin, and continued lens subluxation.

Topical treatment with bromfenac, fluorometholone, and tropicamide, as well as systemic acetazolamide and electrolyte supplementation, was continued. Other medications were discontinued, and another follow-up visit was scheduled in one month.

## DISCUSSION

Choroidal rupture is a complex complication of blunt ocular trauma that requires careful evaluation to guide appropriate management. Visual impairment most commonly results from secondary choroidal neovascularization (CNV) [2]. In the present case, the patient developed acute mechanical

complications, including subretinal hemorrhage and macular edema, without evidence of CNV. Therefore, a conservative approach to macular management was adopted, as anti-VEGF therapy is reserved for cases with confirmed neovascular activity [5].

The main clinical challenge in this patient was a significant elevation of intraocular pressure to 39 mmHg. This was most likely caused by mechanical disruption of the lens–iris complex following trauma, resulting in lens subluxation and vitreous prolapse. Such disturbances often lead to iridodonesis and pigment dispersion within the anterior chamber, a well-recognized mechanism contributing to secondary traumatic glaucoma in young adults [6,7].

Despite reduced fundus visibility and the presence of multi-level ocular trauma, visual evoked potential testing provided valuable prognostic information. The observed electrophysiological pattern—reduced amplitude with preserved latency—may indicate partial traumatic optic nerve involvement while suggesting potential for functional recovery [8]. As there was no evidence of optic canal fracture or complete loss of light perception, surgical optic nerve decompression was not indicated [9]. Nevertheless, early pharmacological treatment remains important to limit neurodegenerative changes associated with traumatic optic neuropathy [10].

The topical and systemic treatment regimen was therefore tailored to address the patient's acute clinical presentation. Acetazolamide, beta-blockers, and prostaglandin analogues were administered to rapidly reduce intraocular pressure and prevent glaucomatous damage [7,11]. Corticosteroids and non-steroidal anti-inflammatory drugs were used concurrently to control post-traumatic inflammation and stabilize the disrupted blood–ocular barrier [11].

Significant lens displacement and vitreous prolapse may require surgical intervention, including pars plana vitrectomy and lensectomy [3,12]. In the present case, however, surgery was postponed. The immediate priority was medical stabilization of intraocular pressure and inflammation. Surgical reconstruction may be considered after resolution of the acute hemorrhagic and edematous phase, which reduces the risk of intraoperative complications [12].

Management of choroidal rupture itself typically involves observation. Anti-VEGF therapy is primarily indicated when CNV develops [5]. Visual prognosis largely depends on lesion location. Ruptures involving the fovea are associated with poorer outcomes and a higher risk of CNV. Additional unfavorable prognostic factors include extensive injury involving the optic disc, macular holes, optic atrophy, or pronounced pigmentary changes [2]. CNV develops in approximately 7.9% of choroidal rupture cases, most frequently in lesions involving the fovea [13].

Intravitreal anti-VEGF therapy has proven safe and effective for CNV secondary to choroidal rupture, resulting in lesion regression and visual improvement. In a retrospective study with a mean follow-up of five years, these outcomes were achieved with an average of 4.2 injections per eye, while 40% of patients required only a single injection. All treated patients achieved regression of neovascularization, and most experienced visual improvement. Compared with neovascular age-

related macular degeneration, post-traumatic CNV typically requires fewer injections but still necessitates long-term follow-up because of the risk of late recurrence [5].

Earlier therapeutic approaches included laser photocoagulation and photodynamic therapy (PDT). Argon laser photocoagulation was limited to extrafoveal lesions and did not significantly improve long-term visual outcomes. PDT enabled treatment of subfoveal lesions and provided partial visual stabilization in some cases, although its effectiveness was often limited and temporary [2].

In the present case, fluorescein angiography showed no evidence of active neovascularization, supporting an observational strategy. However, isolated reports have described the use of intravitreal bevacizumab in traumatic choroidal rupture without CNV, resulting in faster resolution of subretinal fluid and improved visual outcomes [14].

## CONCLUSIONS

In this case, early monitoring and prompt control of post-traumatic complications allowed preservation of visual function despite a complex posterior segment injury. Individualized management that considers the location of the choroidal rupture, lens stability, and the risk of neovascularization is essential for optimizing patient outcomes.

This study was conducted in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

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