

Review Article

A practical clinical approach to traumatic choroidal rupture

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ABSTRACT

Background: Traumatic choroidal rupture is a posterior segment manifestation of trauma and is more common in closed-globe injuries. It is defined as a tear of the choroid and Bruch's membrane following blunt trauma. This narrative review summarizes the current literature and provides a practical clinical approach to the diagnosis and management of traumatic choroidal rupture and its complications.

Methods: In this narrative review, we searched the PubMed/MEDLINE database using the search term *choroidal rupture* to provide a practical and updated approach to traumatic choroidal rupture, focusing on its definition, etiology, diagnosis, imaging, management of complications, and prognosis as mentioned in the literature over the last two decades.

Results: Traumatic choroidal rupture occurs due to increased tensile stress on the eye wall and is three-fold more common in closed-globe injuries than in open injuries. Subretinal or sub-retinal pigment epithelial hemorrhage and macular edema are early signs. Macular involvement is associated with poor visual prognosis. Damage to Bruch's membrane increases the risk of subretinal choroidal neovascular membrane (CNVM). Traumatic epiretinal membrane is another complication. Imaging modalities such as spectral-domain optical coherence tomography, indocyanine green angiography, conventional fundus fluorescein angiography, and optical coherence tomography angiography (OCT-A) can be used in diagnosis or monitoring for complications. OCT-A offers unique opportunities for the diagnosis, treatment, and follow-up of both the initial presentation and possible complications. Frequent follow-up has been suggested in the first year after trauma. Intravitreal anti-vascular endothelial growth factor (anti-VEGF) injection is effective and less invasive in managing CNVM. The visual outcome depends on the location of the rupture, baseline visual acuity, and presence of optic atrophy or macular holes. Risk factors for developing CNVM include rupture involving the macula, longer rupture length or rupture closer to the foveal center, and older age.

Conclusions: Choroidal rupture is a posterior segment entity that usually occurs after trauma, compromises choroidal vessels and Bruch's membrane, and can lead to CNVM. The use of novel noninvasive imaging modalities and efficient anti-VEGF therapy to manage this entity or its subsequent complications produces better visual outcomes. Early diagnosis and frequent follow-up of these patients allow treatment of possible complications, thereby improving the visual prognosis.

KEYWORDS

blunt injury, choroid, rupture, choroidal disease, VEGFs, intraocular injection, choroid neovascularization, fluorescence angiography, optical coherence tomography, angiography

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INTRODUCTION

Traumatic choroidal rupture is a posterior segment manifestation of trauma and is more common in closed-globe injuries [1]. It is defined as a tear of the choroid and Bruch's membrane following blunt trauma [2]. The incidence of traumatic choroidal rupture, among other traumatic choroidal injuries, is 4.8%, with 6.8% of eyes having unfavorable outcomes [2]. Post-traumatic choroidal neovascular membrane (CNVM) occurs in approximately 12% of eyes with traumatic choroidal rupture and mostly occurs within one year of blunt ocular trauma [3].

Detailed post-traumatic fundus examination in conjunction with multimodal imaging such as fundus autofluorescence (FAF) [4], spectral-domain optical coherence tomography (SD-OCT) [4], indocyanine green angiography (ICG-A) [5], and optical coherence tomography angiography (OCT-A) [5, 6], along with monitoring by Amsler grid [7], is a practical approach to early detection of choroidal rupture and follow-up for possible complications such as CNVM.

Successful management of CNVM secondary to choroidal rupture has been reported using various treatment modalities such as photodynamic therapy [8] or argon laser photocoagulation [9]. The advent of anti-vascular endothelial growth factor (anti-VEGF) therapy has led to positive visual outcomes in eyes complicated with CNVM [6], even over a long follow-up period [10]. Interestingly, regression of CNVM has occurred in all reported cases of CNVM secondary to choroidal rupture treated using anti-VEGF [10]. Even children with CNVM who were treated with anti-VEGF injections experienced significant vision improvement that was maintained in the long term [11]. However, spontaneous regression of subfoveal CNVM secondary to choroidal rupture has been detected using OCT-A [12].

This narrative review summarizes the current literature and provides a practical clinical approach to the diagnosis and management of traumatic choroidal rupture and its complications. This paper describes the definition, etiology, general pathology, complications, imaging, currently available treatments, and prognosis of this sight-threatening entity.

Definition, etiology, and general pathology

Traumatic choroidal rupture, first described by von Graefe in 1854 as damage to Bruch's membrane and the retinal pigment epithelium (RPE) [4], is a result of traumatic tensile stress on the eye wall. Subtypes include direct (at the site of contusion, located in the periphery) and indirect (located at the posterior pole) [2, 4]. The traumatic force induces a sudden globe deformation at the site of impact and causes stress folding of the peripheral globe wall [2, 4, 13].

Choroidal rupture is usually the result of a traumatic injury [14] and is the most common retinal macular finding in eyes with blunt ocular trauma [3]. The incidence of choroidal rupture ranges from 5% to 10% after non-penetrating ocular trauma and is three-fold more common in closed-globe injuries than in open injuries [14].

Complications

Subretinal or sub-RPE hemorrhage may occur at the time of choroidal rupture [15]. However, choroidal rupture is associated with a low risk of retinal detachment [2]. Damage to Bruch's membrane increases the risk of subretinal neovascularization [16]. Untreated patients with subfoveal neovascularization, with or without fibrosis, have a poor prognosis [14]. Macular involvement is associated with poor visual prognosis [15]. CNVM is a well-known complication of traumatic choroidal rupture that can develop early or late in the disease course. It may threaten vision in undiagnosed or untreated cases [12, 14].

Traumatic epiretinal membrane is another complication of traumatic choroidal rupture. In a published case report, surgical intervention was considered, but was not performed because of the possibility of insufficient improvement in visual acuity [17].

Imaging

Diagnosis and monitoring for possible complications of traumatic choroidal rupture can be accomplished using various imaging modalities. SD-OCT [3, 18], OCT-A, ICG-A [19], and conventional fundus fluorescein angiography (FFA) [3, 18, 19] are beneficial for both diagnosis and follow-up to determine early treatment options, complications, and prognosis [3, 12, 18-20].

We now present the case of a 23-year-old man who visited the ophthalmology clinic in the early post-traumatic period and underwent multimodal imaging. Figure 1A shows a color fundus photograph acquired using a fundus camera (TRC-NW8, Topcon Corporation, Tokyo, Japan). Figure 1B shows an OCT image taken

using the Heidelberg Spectralis (Heidelberg Engineering, Heidelberg, Germany). A vertical choroidal rupture (yellow-white crescent-shaped lesion) (Figure 1A) originated from the upper nasal quadrant, concentric to the optic nerve. This presentation is atypical, as approximately 80% of choroidal ruptures are temporal to the optic disc [21]. The lesion then converted (Figure 1A) to a horizontal choroidal rupture with damage to Bruch's membrane and the RPE involving the subfoveal region of the macula, as evident in the foveal section of the OCT image (Figure 1B). This is consistent with reports that the macula is affected in approximately two-thirds of cases [21].

OCT has been widely used since its introduction in clinical practice [22]. It revolutionized the recognition of traumatic pathology and defined the anatomical localization and possible functional effects of traumatic lesions [15, 23-25]. In a previously reported case, OCT imaging was performed in a patient with multiple traumatic choroidal ruptures. The images revealed a loss of continuity in the RPE-Bruch's membrane complex and hyperplasia of the RPE at the margins of the rupture [26]. These data demonstrate the usefulness of OCT in the diagnosis of choroidal rupture.

Figure 2A-C is an OCT-A image (AngioPlex, Carl Zeiss Meditec, Inc., Dublin, CA, USA) of our patient showing possibly new vessels above the foveal avascular zone and the area of choroidal tear extending both nasally and temporally to the fovea (Figure 2C). Hypointense choroidal tear in the choriocapillaris plate and relative loss of choriocapillaris in the area of the hypointense choroidal tear is evident (Figure 2C). OCT-A, the most recent imaging method to enter clinical ophthalmology practice, has facilitated early detection and follow-up of complications [27]. It has good sensitivity and specificity in diagnosing CNVM [28]. Preziosa et al. [29] used OCT-A, along with other imaging modalities, in the diagnosis and follow-up of CNVM after traumatic choroidal rupture. Their patient received an intravitreal bevacizumab injection for CNVM. This injection

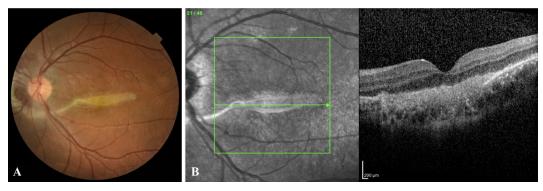


Figure 1. (A) Color fundus photograph acquired using a fundus camera (TRC-NW8, Topcon Corporation, Tokyo, Japan) shows the clinical features of choroidal rupture as a yellow-white crescent-shaped lesion with a vertical course originating from the upper nasal quadrant and extending around the optic nerve to the macula. (B) Foveal section of optical coherence tomography (middle and right image) taken using the Heidelberg Spectralis (Heidelberg Engineering, Heidelberg, Germany) shows a horizontal choroidal rupture damaging Bruch's membrane and the retinal pigment epithelium due to trauma in the subfoveal region.

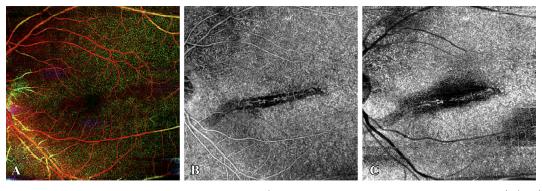


Figure 2. Optical coherence tomography angiography image (AngioPlex, Carl Zeiss Meditec, Inc., Dublin, CA, USA). (A, B) show the presence of possibly new vessels overlying the area of choroidal rupture extending both nasally and temporally to the fovea with the foveal avascular zone. (C) Shows a hypointense choroidal rupture in the choriocapillaris slab with relative loss of choriocapillaris in the hypointense area.

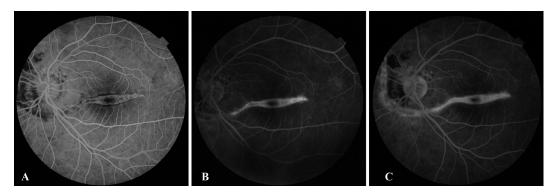


Figure 3. Fundus fluorescence angiography images captured using a retinal camera (TRC-NW8, Topcon Corporation, Tokyo, Japan) in the early stage show (A) relative hypofluorescence in the area of choroidal rupture due to a lack of choriocapillaris in the lesion area and (B) large choroidal vessels below several points in the choroidal rupture area. In the late phase (C), hyperfluorescence and scleral tissue staining are observed in the lesion area due to choroidal rupture along with late staining of fibrous tissue.

reduced the existing neovascular network, and the neovascular network narrowed and the borders of the lesion improved after anti-VEGF treatment as documented by OCT-A [29]. OCT-A allows detection of normal and abnormal retinal and choroidal flow without contrast injection, and OCT-A alone can help manage CNVM without conventional FFA [30, 31].

Saraswat et al. [30] reported a 25-year-old woman with a traumatic choroidal rupture. Conservative management was chosen at the time of diagnosis. At three months post-trauma, the patient demonstrated further deterioration of visual acuity in her right eye. OCT-A confirmed the presence of CNVM at the site of choroidal rupture. The patient was administered a single intravitreal injection of ranibizumab. Six weeks later, a repeat OCT-A showed regression of the CNVM [30].

Conventional FFA has been used along with other imaging modalities at baseline and during follow-up of eyes with CNVM secondary to traumatic choroidal rupture [5, 6, 27]. We performed FFA in our patient (Figure 3A-C). Figure 3A shows early-stage FFA images captured using a retinal camera (TRC-NW8, Topcon Corporation, Tokyo, Japan), with relative hypofluorescence in the lesional area due to deficient choriocapillaris (Figure 3B) and large choroidal vessels under a few spots in the choroidal tear area. FFA image in the late phase (Figure 3C) showed hyperfluorescence and scleral tissue-derived staining in the lesion area due to choroidal tears and late staining of fibrous tissue.

Along with other modalities, OCT-A imaging in traumatic choroidal rupture offers unique opportunities in diagnosis, assessing treatment response, and follow-up of both the initial presentation and the complications that may develop [5, 6, 12, 27, 29-31].

Management

Frequent follow-up is suggested in the first year after trauma [14]. CNVM can develop within or adjacent to the traumatic choroidal rupture during follow-up [3]. Although there is no definitive treatment, reported treatment modalities for CNVM secondary to traumatic choroidal rupture include argon laser photocoagulation, surgery, argon laser photocoagulation followed by surgery, and photodynamic therapy [1, 9]. Intravitreal anti-VEGF injections may be used in the presence of CNVM and are less invasive [18, 20].

Benillouche et al. [6] reported a double vertical macular lesion indicating choroidal rupture in the right eye of a 19-year-old man with a history of blunt trauma. SD-OCT, FFA, and ICG-A revealed an active type 2 CNVM secondary to choroidal rupture. OCT-A images revealed a high-flow neovascular network, consistent with those of conventional multimodal imaging. One month after intravitreal ranibizumab injection, OCT-A showed narrowing and remodeling of the neovascular flow, and exudative findings on multimodal imaging had disappeared [6].

Rezaei et al. [31] demonstrated that OCT-based microangiography detected choroidal rupture and secondary CNVM in a 20-year-old man. The submacular hemorrhage was displaced using outpatient injection of intravitreal expansile perfluoropropane gas with face-down positioning for one week. The patient's visual acuity improved after gradual absorption of the submacular hemorrhage. The final best-corrected vision reached 20 / 50 without further intervention [31].

Preziosa et al. [29] described the case of a 20-year-old woman with choroidal rupture on OCTA images after blunt ocular trauma. Secondary CNVM developed six months later and was treated using a single intravitreal injection of bevacizumab. Initially, OCT-A showed high-flow neovascularization, which appeared contracted after treatment [29].

In a study of five eyes with traumatic CNVM treated using anti-VEGF injections with an average follow-up of five years, Barth et al. [10] reported that intravitreal anti-VEGF therapy was a safe and effective treatment option. These eyes can be managed with fewer anti-VEGF injections than those with exudative age-related macular degeneration [10].

Another viable treatment option is photodynamic therapy followed by ICG-A-guided feeder vessel photocoagulation, which was reported as an effective treatment for subfoveal CNVM secondary to choroidal rupture in a 61-year-old woman after blunt head trauma [19].

Prognosis

Visual acuity at presentation depends on the location of the rupture, the presence and size of retinal hemorrhage, and macular edema. As subretinal edema and hemorrhage heal, vision may improve [32, 33]. The visual outcome depends on the location of the rupture [9, 30], baseline visual acuity [9], and the presence of optic atrophy or macular holes [16]. Choroidal ruptures temporal to the optic disc, especially those involving the subfoveal region, have the poorest visual prognosis [1].

There is a risk of CNVM formation due to a tear in Bruch's membrane [16], and CNVM growth in the subfoveal area causes reduced central vision that may be regained after anti-VEGF injection [3]. Risk factors for developing CNVM include indirect choroidal rupture involving the macula, longer choroidal ruptures or ruptures closer to the foveal center, and older age [15]. CNVM has been reported even six years after blunt trauma in a 17-year-old boy without apparent choroidal rupture [34]. However, spontaneous regression of CNVM is also likely [1].

The likelihood of CNVM development is higher in choroidal ruptures closer to the fovea than in ruptures away from the fovea [3, 9], and also in older patients [9]. In a retrospective review of 111 patients with traumatic choroidal ruptures, Ament et al. found that rupture location and baseline visual acuity were independent factors associated with visual outcomes [9].

In this narrative review, we have summarized the practical aspects of managing traumatic choroidal rupture and its complications, especially the use of multimodal diagnostic imaging, as mentioned in the literature over the last two decades. However, this study did not use other indexing sources or a systematic review approach, which should be addressed in future review studies on this topic. Interesting challenges remain for further research to increase the efficiency of managing this sight-threatening complication of blunt eye injury. These include investigating the value of new noninvasive diagnostic imaging modalities such as OCT-A in the initial evaluation or follow-up of these patients, as well as the efficacy and safety of other new treatments in the management of CNVM following traumatic choroidal rupture.

CONCLUSIONS

Choroidal rupture is a posterior segment entity that usually occurs after trauma, compromises choroidal vessels and Bruch's membrane, and can lead to CNVM. Early diagnosis and frequent follow-up of these patients allow treatment of possible complications, thereby improving the visual prognosis. The use of novel noninvasive imaging modalities and efficient anti-VEGF therapy to manage this entity or its subsequent complications produces better visual outcomes.

ETHICAL DECLARATIONS

Ethical approval: Ethical approval was received on 28/12/2022 at the department level. **Conflict of interests:** None

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