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Full-Thickness Scleral Rupture with Choroidal Prolapse, Grade III *Hyphema*, and Conjunctival Laceration Following Blunt Ocular Trauma: A Case Report

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ABSTRACT
Open-globe injuries caused by blunt ocular trauma remain a major cause of irreversible monocular blindness worldwide. Full-thickness scleral rupture accompanied by choroidal prolapse and high-grade *hyphema* represents one of the most severe forms of ocular trauma, often resulting in poor visual outcomes despite prompt surgical intervention. Early recognition and immediate management are therefore essential to preserve globe integrity and minimize secondary complications. This study aims to describe the clinical presentation, emergency surgical management, and prognostic considerations of a patient with severe blunt ocular trauma presenting with full-thickness scleral rupture, grade III *hyphema*, and choroidal prolapse. A qualitative case report approach was employed. Clinical data were obtained from medical records, comprehensive ophthalmologic examinations, orbital computed tomography findings, intraoperative observations, and postoperative follow-up. The patient's clinical course was descriptively analyzed and interpreted according to current literature and established ocular trauma management guidelines. A 60-year-old male presented with no light perception following blunt trauma to the left eye after falling from stairs. Clinical examination revealed full-thickness scleral rupture, choroidal prolapse, grade III *hyphema*, conjunctival laceration, and canalicular injury. Emergency primary globe repair was successfully performed within six hours of injury, including scleral closure, conjunctival repair, repositioning of viable uveal tissue, and canalicular reconstruction. At one-week follow-up, the globe remained anatomically intact without evidence of endophthalmitis; however, visual acuity remained at no light perception, indicating an extremely poor functional prognosis. This case highlights that prompt surgical intervention is critical for preserving anatomical integrity and preventing postoperative complications in severe blunt open-globe injuries.

INTRODUCTION

Ocular trauma remains one of the leading causes of monocular blindness worldwide, particularly in developing countries, with open globe injuries accounting for a substantial proportion of severe cases (Blanch et al., 2024). The incidence of open globe injury has been reported to range between 3.5–3.8 per 100,000 population annually, with higher rates observed in occupational and domestic accidents.

Blunt trauma can result in rupture of the globe when the force transmitted to the eye causes a rapid and transient increase in intraocular pressure exceeding the tensile strength of the sclera. This often occurs at structurally weaker regions such as the limbus, rectus muscle insertions, or areas of previous surgical scars (Agrawal et al., 2013; Kuhn & Pieramici, 2016). The resulting injury pattern frequently involves multiple concurrent intraocular complications.

Full-thickness scleral rupture represents the most severe form of open globe injury and is frequently associated with intraocular complications including *hyphema*, vitreous hemorrhage, retinal detachment, and choroidal prolapse (Jain & Talreja, 2022). The presence of these findings is strongly correlated with poor visual outcomes, particularly when initial visual acuity is no light perception (NLP) (Chaudhary et al., 2022). The Ocular Trauma Score (OTS) is a validated prognostic tool widely used to stratify visual outcomes in such cases (Borjan et al., 2025; Rezaei et al., 2026).

Beyond the biomechanical mechanisms of trauma, post-traumatic inflammatory responses also play a crucial role in determining the extent of ocular tissue damage and visual prognosis (Toh et al., 2020). Open globe injuries trigger the release of inflammatory mediators such as pro-inflammatory cytokines, interleukins (IL-1β, IL-6), and tumor necrosis factor-alpha (TNF-α), leading to increased vascular permeability, tissue edema, and secondary damage to intraocular structures. Uncontrolled inflammatory processes may aggravate the primary injury and increase the risk of subsequent complications, including proliferative vitreoretinopathy, endophthalmitis, and phthisis bulbi (Yuki et al., 2025). Therefore, early management aims not only to restore the anatomical integrity of the globe but also to limit secondary damage caused by inflammatory responses.

Another relevant theory is related to the concept of ocular compartment syndrome, which refers to an acute increase in intraocular pressure caused by hemorrhage or post-traumatic edema, resulting in impaired retinal and optic nerve perfusion. Severe *hyphema*, intraocular bleeding, and tissue prolapse may accelerate the onset of ocular ischemia (Shukla et al., 2024). A short-term reduction in retinal blood flow can cause irreversible damage to retinal ganglion cells and the optic nerve, thereby worsening visual prognosis, particularly in patients presenting with initial no light perception (NLP) visual acuity. This mechanism explains why some patients continue to experience permanent visual loss despite optimal anatomical reconstruction (Czarnek-Chudzik et al., 2024).

Furthermore, the wound healing theory in ocular tissues explains that post-traumatic healing occurs through inflammatory, proliferative, and remodeling phases. In cases of full-thickness scleral rupture, healing success is strongly influenced by the timeliness of surgical intervention, stability of wound closure, and viability of prolapsed tissues. Delayed wound closure increases the risk of intraocular infection and excessive scar formation, potentially resulting in distortion of globe anatomy and permanent visual impairment. The principle of primary globe repair emphasizes the importance of restoring structural integrity as early as possible to reduce complications and preserve residual visual potential.

Blunt ocular trauma is particularly challenging because the external appearance of the injury may not fully reflect the extent of internal structural disruption. Rapid anteroposterior compression of the globe produces equatorial expansion and an abrupt increase in intraocular pressure, which can rupture the eyewall at anatomically vulnerable areas such as the limbus, rectus muscle insertions, or previous surgical scars. The resulting injury may include scleral

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rupture, *hyphema*, uveal or choroidal prolapse, vitreous hemorrhage, retinal detachment, traumatic cataract, canalicular injury, and optic nerve damage. The manuscript underlying this research presents a 60-year-old man who sustained blunt trauma to the left eye after falling from stairs. Examination revealed no light perception, eyelid edema, periorbital ecchymosis, conjunctival chemosis and full-thickness laceration, corneal injury, grade III *hyphema*, absent direct pupillary response, scleral rupture, choroidal or uveal tissue prolapse, canalicular laceration, and active hemorrhage. Computed tomography confirmed globe disruption without an intraocular foreign body, demonstrating a clinically severe combination of anterior, eyewall, uveal, and adnexal injuries.

Previous research has established several predictors of unfavorable outcomes after open-globe injury. Fujikawa et al. examined visual outcomes and prognostic factors among patients with open-globe injuries and emphasized that the initial clinical condition substantially influences final vision. A subsequent review by Tan et al. identified initial visual acuity and injury zone as the most influential prognostic factors, while retinal involvement, retinal detachment, relative afferent pupillary defect, vitreous hemorrhage, vitreous prolapse, injury mechanism, *hyphema*, lens damage, and treatment timing were also associated with final outcomes. These findings are highly relevant to the present case because the patient exhibited initial no-light-perception visual acuity, extensive scleral disruption, high-grade *hyphema*, and prolapsed uveal tissue. This combination indicates that the prognosis cannot be determined from scleral rupture alone; rather, it reflects the cumulative effects of mechanical destruction, hemorrhage, tissue ischemia, inflammatory injury, and possible retinal or optic nerve involvement.

Research has also focused specifically on eyes presenting with no light perception, a condition traditionally regarded as an indicator of irreversible visual loss. Bhagat et al. reported that initial no-light-perception status does not invariably indicate permanent blindness, because selected patients may recover limited vision after primary globe repair. Across previous series, reported recovery from no light perception to light perception or better ranged from approximately 4% to 33%; in one series of 73 repaired eyes, 17 eyes, or 23%, achieved light perception or better, although most remained profoundly visually impaired. More recent research similarly indicates that a small subgroup of patients with extremely poor presenting visual acuity may regain variable visual function. These findings support a management approach that avoids premature assumptions of complete irreversibility and prioritizes primary anatomical repair when clinically feasible. Nevertheless, the likelihood of meaningful recovery becomes considerably lower when no-light-perception vision coexists with posterior scleral extension, choroidal prolapse, retinal damage, or afferent pupillary abnormalities.

The management of traumatic *hyphema* represents another important component of severe blunt ocular trauma. *Hyphema* results from disruption of vessels in the iris or ciliary body, allowing blood to accumulate within the anterior chamber. High-grade *hyphema* can obstruct visual assessment and increase the risk of recurrent bleeding, elevated intraocular pressure, corneal blood staining, secondary glaucoma, optic nerve injury, and permanent visual deterioration. Chen et al. emphasized that even relatively small anterior-chamber hemorrhages may produce serious complications, making appropriate surveillance and treatment essential. In an eye with an open-globe injury, however, assessment and management are more complicated because conventional intraocular-pressure measurement or forceful manipulation

may further disrupt the wound or promote extrusion of ocular contents. The coexistence of grade III *hyphema* and choroidal prolapse in the present case therefore represents not simply two parallel diagnoses, but an interconnected injury pattern involving extensive vascular disruption, impaired visualization, possible pressure dysregulation, inflammation, and profound risk to both the anterior and posterior ocular segments.

Timely surgical intervention is consistently regarded as a central determinant of anatomical preservation following open-globe injury. A systematic review and meta-analysis involving 15 studies and 8,497 injured eyes found that primary repair performed within 24 hours was associated with significantly lower odds of post-traumatic endophthalmitis than repair performed after 24 hours. Current reviews similarly emphasize early wound closure, systemic and topical antimicrobial therapy, protection of viable intraocular tissues, cautious removal of contaminated or necrotic tissue, and avoidance of unnecessary intraocular manipulation. In the present case, emergency surgery was completed within six hours of presentation. The prolapsed viable tissue was repositioned, contaminated tissue was excised, the scleral wound was closed with interrupted nylon sutures, the conjunctival laceration was repaired, and the injured canaliculus was treated using a monocanalicular silicone stent. At one week, the globe remained anatomically intact and showed no evidence of endophthalmitis, although vision remained at no light perception.

Despite the extensive literature on open-globe injury, a clinically relevant gap remains in the integrated description of full-thickness scleral rupture occurring simultaneously with choroidal prolapse, grade III *hyphema*, conjunctival laceration, and canalicular damage following blunt trauma. Many previous studies have analyzed broad cohorts and quantified prognostic variables, but cohort-based analyses often provide limited detail regarding the interaction between individual anatomical injuries, emergency decision-making, intraoperative tissue management, and early postoperative outcomes. In addition, publications frequently focus on either posterior-segment predictor, traumatic *hyphema*, wound-repair timing, or final visual acuity as separate issues. This fragmentation limits understanding of how multiple severe findings should be interpreted when they occur in the same patient. The present case addresses that gap by connecting the biomechanical mechanism of blunt rupture, the clinical grading of *hyphema*, the significance of prolapsed choroidal tissue, the principles of primary globe closure, concurrent adnexal reconstruction, and the interpretation of persistent no-light-perception vision within one comprehensive clinical account.

The urgency of documenting this case lies in the need to distinguish successful anatomical management from successful functional recovery. In severe open-globe trauma, restoration of vision may be impossible even when surgical treatment is timely and technically adequate. Nevertheless, primary repair remains important for maintaining globe architecture, limiting microbial contamination, reducing the likelihood of endophthalmitis, preserving facial symmetry, supporting ocular comfort, and facilitating subsequent assessment or reconstructive intervention. The novelty of this report is therefore not based solely on the presence of scleral rupture, but on the simultaneous clinical trial of full-thickness scleral rupture, grade III *hyphema*, and choroidal prolapse, accompanied by conjunctival and canalicular injury, in an older patient presenting with no light perception. It further demonstrates the contrast between a favorable early anatomical result an intact globe without infection and an extremely poor early visual result. This contrast offers an important clinical lesson: anatomical salvage and

visual restoration are separate outcome domains that should be clearly communicated during treatment planning and informed consent.

Accordingly, this research aims to describe the clinical presentation, diagnostic evaluation, emergency surgical management, early anatomical outcome, and visual prognosis of severe blunt ocular trauma involving full-thickness scleral rupture, choroidal prolapse, grade III *hyphema*, conjunctival laceration, and canalicular injury. Its objectives are to explain the relationship between the biomechanical force of blunt trauma and the resulting multi-structural damage, evaluate the prognostic significance of initial no-light-perception visual acuity and associated ocular findings, and demonstrate the role of rapid primary repair in preserving globe integrity and preventing early infection. The study contributes an integrated case-based perspective that may complement evidence from larger prognostic and epidemiological studies. Its practical benefits include supporting emergency physicians and ophthalmologists in early recognition, referral, surgical prioritization, prognosis communication, and postoperative monitoring. Academically, the report may provide a reference for future comparative studies examining anatomical survival, visual recovery, infection, phthisis bulbi, sympathetic ophthalmia, and patient-reported outcomes after complex blunt open-globe injuries.

METHOD

This study employed a qualitative method using a case study approach to explore in depth the clinical presentation, diagnostic process, management, and outcomes of a patient with an open globe injury caused by blunt ocular trauma accompanied by full-thickness scleral rupture, choroidal prolapse, and grade III *hyphema*. Data were obtained through medical record reviews, ophthalmologic examination findings, documentation of emergency surgical procedures, and observation of postoperative clinical progression. The analysis was conducted descriptively by interpreting clinical findings based on current literature and established ocular trauma management guidelines, aiming to provide a comprehensive understanding of the relationship between injury severity, immediate surgical intervention, and visual prognosis in severe ocular trauma cases.

CASE PRESENTATION

A 60-year-old male presented to the emergency department with a history of blunt trauma to the left eye following a fall from stairs approximately 30 minutes prior to admission. The patient complained of sudden severe ocular pain with immediate visual loss and active bleeding from the affected eye. There was no prior history of ocular disease, surgery, or systemic comorbidities.

On general examination, the patient was haemodynamically stable, except for mild hypotension with a blood pressure of 80/60 mmHg; neurological status was intact with a Glasgow Coma Scale (GCS) score of 15/15. No other systemic injuries were identified on initial survey.

Ophthalmological examination of the left eye demonstrated: (1) no light perception (NLP); (2) eyelid oedema and periorbital ecchymosis; (3) conjunctival chemosis with full-thickness conjunctival laceration; (4) corneal oedema with associated epithelial and stromal injury; (5) grade III *hyphema* occupying more than half of the anterior chamber; (6) anisocoria with absent direct pupillary light reflex; (7) visible scleral rupture with prolapse of intraocular

tissue consistent with uveal/choroidal tissue; and (8) canalicular laceration with active hemorrhage. The right eye was unremarkable. Axial computed tomography (CT) of the orbits confirmed globe disruption without intraocular foreign body.

Based on these findings, the patient received a diagnosis of full-thickness scleral rupture with choroidal prolapse, grade III *hyphema*, and conjunctival rupture of the left eye (Zone I–II injury per the Birmingham Eye Trauma Terminology [BETT] system). Emergency surgical intervention was performed under general anesthesia within six hours of presentation.

Intraoperatively, the scleral wound was identified extending from the limbus posteriorly. Prolapsed uveal tissue was gently repositioned where viable, and contaminated tissue was excised. Primary watertight closure was achieved using interrupted 8-0 nylon sutures. The conjunctival laceration was repaired with 8-0 vicryl sutures. Canalicular repair was performed with a *monocanalicular* silicone stent. Postoperatively, the patient was commenced on topical and systemic antibiotics, cycloplegic agents, and corticosteroids. At the one-week follow-up, the globe remained intact with no signs of endophthalmitis, though visual acuity remained at NLP.

Clinical Figures

Figure 1. Preoperative external clinical appearance of the left eye following severe blunt ocular trauma. Extensive periorbital ecchymosis, eyelid oedema, and subconjunctival haemorrhage are visible,

Figure 2. Anterior segment documentation showing grade III hyphema filling more than half of the anterior chamber, accompanied by conjunctival laceration and corneal oedema after blunt trauma.

Figure 3. Close-up clinical image demonstrating the rupture site with prolapsed pigmented intraocular tissue consistent with uveal or choroidal prolapse through a full-thickness scleral defect.

RESULT AND DISCUSSION

Blunt ocular trauma generates rapid anteroposterior compression of the globe accompanied by equatorial expansion, producing a sudden rise in intraocular pressure that may transiently exceed 1000 mmHg. This hydraulic mechanism eventually causes rupture at the structurally weakest regions of the sclera most commonly at the limbus, posterior to the rectus muscle insertions, or at sites of previous surgical incisions (Guo & Ni, 2024; Lin et al., 2025). Unlike penetrating injuries, blunt trauma distributes kinetic energy diffusely throughout the globe, explaining the characteristic coexistence of multiple concurrent injuries as observed in this case.

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The Ocular Trauma Score (OTS), a validated prognostic system incorporating initial visual acuity, globe rupture status, endophthalmitis, perforating injury, retinal detachment, and afferent pupillary defect, assigns this patient to the lowest prognostic category (OTS score 1), corresponding to a less than 1% probability of achieving ambulatory vision. Posterior extension of scleral wounds, prolapsed uveal tissue, and initial NLP acuity are independently associated with the poorest visual outcomes (Rao et al., 2010).

Hyphema following blunt trauma results from disruption of iris or ciliary body vasculature, with subsequent accumulation of blood in the anterior chamber. Grade III *hyphema* defined as hemorrhage occupying 50–99% of the anterior chamber is associated with a substantially elevated risk of secondary hemorrhage (occurring within 2–5 days), corneal blood staining, secondary angle-closure glaucoma, and optic nerve damage from chronically elevated intraocular pressure (Feng et al., 2011). Elevated IOP in the context of an open globe demands particular vigilance, as standard pressure-lowering interventions must be applied cautiously to avoid extrusion of intraocular contents.

Choroidal prolapse occurs when uveal tissue herniates through a full-thickness scleral defect, indicating severe disruption of the posterior segment with heightened risk of retinal detachment and proliferative vitreoretinopathy (PVR). Prolapsed tissue should not be aggressively repositioned, as contamination and additional avascular injury may promote infection and worsening ischemia. When tissue viability is uncertain, conservative debridement followed by watertight closure is preferred.

Early surgical repair ideally within 24 hours of injury has been consistently associated with improved anatomical outcomes and a significantly reduced incidence of endophthalmitis (Blanch et al., 2019; Bravetti et al., 2026). Key surgical principles include: (1) meticulous watertight closure of scleral and conjunctival wounds; (2) preservation of all viable intraocular tissue; (3) excision of necrotic or contaminated tissue; (4) avoidance of intraocular manipulation unless absolutely necessary; and (5) concurrent repair of adnexal injuries such as canalicular lacerations to minimize long-term lacrimal morbidity .

Despite optimal surgical management, visual prognosis in patients presenting with NLP acuity, combined scleral rupture, choroidal prolapse, and high-grade *hyphema* remains poor. However, primary repair remains essential to preserve globe architecture, prevent secondary infection, maintain cosmesis, and reduce the risk of sympathetic ophthalmia in the fellow eye a rare but potentially devastating immune-mediated complication following penetrating uveal injury. The long-term risk of phthisis bulbi should be discussed with the patient and family as part of informed consent and longitudinal follow-up planning.

CONCLUSION

This case report demonstrates that full-thickness scleral rupture accompanied by choroidal prolapse, grade III *hyphema*, conjunctival laceration, and canalicular injury represents one of the most devastating forms of blunt ocular trauma, requiring immediate recognition and emergency surgical intervention. The patient's initial presentation of no light perception (NLP), extensive scleral disruption, and prolapsed uveal tissue indicated an extremely poor visual prognosis despite prompt management. Emergency primary globe repair performed within six hours successfully restored the anatomical integrity of the globe, prevented early postoperative complications such as endophthalmitis, and preserved ocular

structure. However, the persistence of NLP vision at the early postoperative follow-up emphasizes that successful anatomical reconstruction does not necessarily translate into functional visual recovery. This case further highlights the importance of comprehensive clinical assessment, prognostic evaluation using established ocular trauma principles, and timely surgical management in optimizing patient outcomes while minimizing the risk of secondary complications following severe open-globe injury. Future studies should involve larger multicentre case series or prospective cohort designs to improve understanding of prognostic factors influencing both anatomical and functional outcomes in patients with severe blunt open-globe injuries. Further investigation is warranted to evaluate the long-term visual prognosis, incidence of complications such as proliferative vitreoretinopathy, phthisis bulbi, and sympathetic ophthalmia, as well as the effectiveness of different surgical techniques and postoperative management strategies. Additionally, integrating advanced ocular imaging, standardized trauma scoring systems, and patient-reported quality-of-life measures would provide a more comprehensive evaluation of treatment outcomes. Such evidence may contribute to the development of evidence-based clinical guidelines for the management of complex ocular trauma and support more accurate prognostic counseling for patients and their families.

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