

Beyond the Broken Orbit: Ocular Complications of Orbital Fractures—A Comprehensive Narrative Review

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ABSTRACT

Background: Orbital fractures represent a frequent form of midfacial trauma and are commonly accompanied by varying degrees of ocular involvement. The clinical spectrum may range from relatively minor findings, such as periorbital swelling and subconjunctival hemorrhage, to serious complications including globe rupture, retrobulbar hematoma, and traumatic optic neuropathy that can jeopardize vision. Owing to the intimate anatomical relationship between the orbit and its contained ocular structures, a thorough ophthalmologic assessment is imperative in all cases of orbital injury. This narrative review discusses the underlying mechanisms of trauma, associated ocular manifestations, diagnostic evaluation, and current management principles. Prompt identification of ocular injuries and coordinated multidisciplinary care are essential for optimizing visual outcomes and achieving successful anatomical reconstruction.

Keywords: Orbital fractures; Ocular trauma; Diplopia; Enophthalmos; Orbital floor fracture; Traumatic optic neuropathy.

INTRODUCTION

Orbital fractures are a common consequence of maxillofacial trauma and account for a substantial proportion of facial skeletal injuries. The orbit houses critical structures, including the globe, optic nerve, extraocular muscles, vascular networks, and supporting connective tissues within a relatively confined bony framework.¹ As a result, disruption of the orbital walls can adversely affect vision, ocular motility, and facial symmetry. Motor vehicle collisions, interpersonal violence, sports-related trauma, and accidental falls remain the leading causes of orbital injuries worldwide. Because ocular damage may not be immediately apparent during the initial assessment, a systematic ophthalmologic evaluation is essential in all patients presenting with orbital trauma.²

Mechanisms of Injury

Several theories have been proposed to explain the pathogenesis of orbital fractures. According to the hydraulic mechanism, blunt trauma to the orbit produces a rapid increase in intraocular pressure, causing fracture of the weakest portions of the orbital walls.² In contrast, the buckling mechanism suggests that impact forces applied to the orbital rim are transmitted posteriorly, resulting in failure of the orbital floor. Contemporary evidence indicates that both mechanisms may operate simultaneously in many cases. The posteromedial segment of the orbital floor is recognized as the region most frequently involved in blowout fractures.³

Ocular Manifestations

Clinical manifestations of orbital fractures range from minor soft-tissue injuries to severe vision-threatening complications (Fig1.). Frequently

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observed findings include periorbital ecchymosis, subconjunctival hemorrhage, facial swelling, diplopia, sensory disturbances in the infraorbital nerve distribution, and limitations in ocular movement. Diplopia is commonly associated with entrapment of extraocular muscles or surrounding orbital soft tissues within the fracture defect. Enophthalmos may develop due to expansion of orbital volume and prolapse of orbital contents into adjacent sinuses.⁴



Fig 1: Clinical presentation of orbital wall fracture.

More serious ocular injuries include hyphema, vitreous hemorrhage, retinal detachment, traumatic optic neuropathy, globe rupture, and retrobulbar hematoma. Among these, traumatic optic neuropathy is particularly concerning because it may lead to irreversible visual impairment. Retrobulbar hemorrhage constitutes an ophthalmic emergency, as rapidly increasing orbital pressure can compromise optic nerve perfusion and threaten vision if prompt decompression is not performed.⁵(Table 1)

Table 1: Immediate, delayed & late complications in orbital wall fractures

IMMEDIATE COMPLICATIONS	DELAYED COMPLICATIONS	LATE COMPLICATIONS
A. Loss of vision Optic nerve injury Ophthalmic vascular injury Ocular globe injury Retrobulbar Hemorrhage and Optic nerve compression Palpebral Edema/Proptosis Orbital and Periorbital Hemorrhage, Epistaxis Orbital	A.Naso-Orbital Skeletal Deformities Nasal deformity Septal or conchal deformity B.Loss or displacement of prosthetic implants Orbital and Periorbital Soft-Tissue changes Fibrosis and scar formation Orbital fat atrophy or volume	A. Extraocular Muscle Imbalance (Persistent Diplopia) B. Enophthalmos C. Malposition of the Orbital Floor or Implant D. Eyelid Malposition - Ectropion - Entropion - Ptosis E. Lacrimal Drainage Disorders - Epiphora - Nasolacrimal duct obstruction F. Reconstruction Failures - Implant migration - Implant infection - Implant exposure - Non-union or malunion G. Pediatric Fractures and Growth Disturbances

Emphysema	changes	• Facial asymmetry
Skeletal and Structural defects	Persistent vertical diplopia	• Orbital growth abnormalities
Displaced orbital wall fractures	secondary to muscle fibrosis or malposition	• Persistent diplopia
Bone defects causing orbital volume changes		
Soft-Tissue and Extraocular Muscle		
Entrapment		
Inferior rectus entrapment		
Medial rectus entrapment		
Orbital fat herniation		

Diagnostic Evaluation

Assessment of patients with orbital fractures should include a comprehensive ophthalmic examination. Essential components include measurement of visual acuity, evaluation of pupillary responses, assessment of color vision, examination of ocular motility, determination of globe position, and fundoscopic evaluation. Forced duction testing may be useful in differentiating mechanical restriction caused by muscle entrapment from neurogenic causes of limited eye movement.⁶ (Fig. 2)



Fig 2: Forced duction test

Computed tomography (CT) remains the gold-standard imaging modality for orbital trauma because it provides excellent visualization of bony defects, soft-tissue herniation, extraocular muscle incarceration, and alterations in orbital volume. Three-dimensional image reconstruction may further assist in surgical planning, particularly in extensive or complex fractures.⁷

Management

Treatment strategies are determined by fracture characteristics, associated ocular injuries, and the

presence of functional or aesthetic deficits. Conservative management with observation is appropriate for selected patients with minimally displaced fractures who exhibit no significant visual or motility disturbances.⁸ Surgical intervention is generally recommended for persistent diplopia, clinically significant enophthalmos, extraocular muscle entrapment, extensive orbital floor defects, progressive ocular dysfunction, or noticeable cosmetic deformity.⁸

Various reconstructive materials are available for orbital wall repair, including titanium mesh, porous polyethylene implants, patient-specific implants, and autogenous bone grafts.⁹ Early surgical correction is especially important in pediatric trapdoor fractures and cases involving muscle incarceration, as delayed treatment may result in permanent ocular motility disorders.¹⁰ (Table 2)

Table 2: Ocular manifestations in orbit fracture with their management

MANIFESTATION	CLINICAL FEATURES	MANAGEMENT
Periorbital ecchymosis	Bruising around the eye ("raccoon eyes") Appears within 1-2 hours after injury	Cold compress for first 24-48 hours Head elevation Analgesics Reassurance (usually resolves within 1-2 weeks)
Subconjunctival Hemorrhage	Red patch over the sclera Usually painless	Reassurance Lubricating eye drops (if symptomatic) Resolves spontaneously within 1-2 weeks
Facial Swelling	Swelling of eyelids and surrounding tissues May be associated with tenderness	Cold compress initially Head elevation Analgesics Treat associated injuries
Diplopia	Double vision Worse in specific gaze directions	Identify and treat underlying cause (muscle entrapment, edema, nerve injury) Advise avoidance of nose blowing Surgical repair if persistent or associated with entrapment
Sensory Disturbances (Infraorbital Nerve Distribution)	Numbness or tingling of the lower eyelid, cheek, lateral nose, upper lip, and maxillary teeth	Reassurance (often improves spontaneously) Avoid further nerve injury Treat fractures when indicated
Limitation of Ocular Movement	Restricted eye movements Due to muscle entrapment, edema, or nerve injury	Evaluate cause (CT scan, forced duction test) Corticosteroids if due to edema Surgical intervention if muscle entrapment is present

HypHEMA	Blood in the anterior chamber Pain, photophobia, blurred vision	Bed rest with head elevation Eye shield Topical corticosteroids and cycloplegics Avoid NSAIDs and anticoagulants Surgical washout in severe cases
Vitreous Hemorrhage	Blood in the vitreous cavity Floaters and decreased vision	Head elevation Restrict physical activity Monitor intraocular pressure (IOP) Vitrectomy if non-clearing or complicated
Retinal Detachment	Flashes, floaters, curtain-like shadow Decreased vision	Urgent referral to a retinal specialist Surgical repair (scleral buckle, pneumatic retinopexy, or pars plana vitrectomy as indicated) Appropriate postoperative positioning
Traumatic Optic Neuropathy	Sudden decrease in vision Relative afferent pupillary defect (RAPD) May present immediately or be delayed	High-dose intravenous corticosteroids (controversial) Observation and supportive care Surgical decompression in selected cases (controversial) Visual prognosis is guarded
Globe Rupture	Full-thickness injury of the globe Severe pain and vision loss	Place a rigid eye shield (avoid pressure on the globe) Broad-spectrum intravenous antibiotics Urgent surgical repair Tetanus prophylaxis
Retrobulbar Hematoma (Orbital Compartment Syndrome)	Proptosis Severe orbital pain Decreased vision Tense orbit	Ophthalmic emergency Immediate lateral canthotomy and cantholysis if indicated Lower intraocular pressure (e.g., mannitol, acetazolamide) Definitive treatment of the underlying cause

DISCUSSION

Orbital fractures remain among the most clinically significant injuries encountered in maxillofacial trauma because of their close association with ocular morbidity. Although the bony orbit provides substantial protection to the globe and its adnexal structures, disruption of the orbital walls may transmit traumatic forces to the optic nerve, extraocular muscles, vascular structures, and surrounding soft tissues. Consequently, visual impairment following orbital trauma is not solely dependent on the severity of the fracture but also on the nature and extent of associated ocular injuries.^{1,2} Successful management therefore extends beyond restoration of facial anatomy and

requires prompt recognition and treatment of concomitant ophthalmic complications.

The incidence of ocular injury associated with orbital fractures varies considerably across published studies, reflecting differences in patient populations, mechanisms of injury, and diagnostic criteria. Previous reports have shown that while many patients present with relatively benign findings such as subconjunctival hemorrhage, eyelid edema, or periorbital ecchymosis, a smaller but clinically significant proportion develop vision-threatening complications including globe rupture, retinal detachment, retrobulbar hemorrhage, or traumatic optic neuropathy.² This variability emphasizes that the external appearance of an orbital injury does not reliably predict the severity of intraorbital damage. Even fractures that appear minimally displaced on imaging may harbor significant ocular pathology, reinforcing the need for comprehensive ophthalmologic evaluation in every patient.^{2,5}

The mechanisms responsible for orbital fracture formation have been debated for several decades. The hydraulic and buckling theories, once considered competing explanations, are now regarded as complementary mechanisms that frequently coexist during blunt facial trauma.^{1,3} The magnitude, direction, and duration of the applied force determine the resulting fracture pattern and the likelihood of ocular injury. Low-energy trauma may produce isolated blowout fractures with limited soft-tissue damage, whereas high-energy injuries frequently involve multiple orbital walls, complex midfacial fractures, and extensive ocular disruption. Understanding these biomechanical principles is clinically relevant because fracture configuration often predicts associated functional deficits, including diplopia, enophthalmos, and extraocular muscle entrapment.¹

Diplopia remains one of the most common functional complaints following orbital fractures and may arise from several mechanisms. Entrapment of the inferior rectus or inferior oblique muscles, edema of the orbital soft tissues, direct muscle contusion, cranial nerve injury, or fibrosis during healing may all contribute to persistent ocular motility disturbances. Forced duction testing, combined with detailed ocular motility assessment and high-resolution CT imaging, remains invaluable

in differentiating mechanical restriction from neurogenic dysfunction and identifying patients who may benefit from early surgical intervention.^{6,7}

Enophthalmos represents another important consequence of orbital fractures and is primarily related to enlargement of orbital volume following disruption of the orbital walls. Herniation of orbital fat and extraocular muscles into adjacent paranasal sinuses further contributes to posterior displacement of the globe. Although mild enophthalmos may initially be masked by post-traumatic edema, delayed presentation is common as swelling subsides. Repeated clinical examinations during follow-up are therefore essential to identify progressive globe displacement before permanent cosmetic deformity develops.⁸

Among vision-threatening complications, traumatic optic neuropathy remains one of the most devastating because of its unpredictable clinical course and controversial management. Direct injury to the optic nerve, vascular compromise, or secondary compression resulting from edema or hemorrhage may all contribute to irreversible axonal damage. Clinical suspicion should be heightened in patients presenting with sudden visual loss, impaired color vision, visual field defects, or a relative afferent pupillary defect despite minimal external signs of injury. Current evidence regarding the benefits of high-dose corticosteroids and optic nerve decompression remains inconclusive; therefore, management should be individualized and guided by multidisciplinary clinical judgment.¹⁰

Retrobulbar hematoma deserves particular attention because it constitutes a true ophthalmic emergency. Progressive accumulation of blood within the confined orbital compartment rapidly elevates intraorbital pressure, resulting in ischemia of the optic nerve and retina. Visual prognosis is directly related to the duration of ischemia, making prompt recognition and immediate decompression critical. Clinicians should therefore remain vigilant for severe orbital pain, rapidly progressive proptosis, tense eyelids, decreased visual acuity, ophthalmoplegia, and elevated intraocular pressure. Early lateral canthotomy and cantholysis, when indicated, can be vision-saving procedures.¹⁰

Computed tomography continues to represent the gold-standard imaging modality in orbital trauma because of its excellent spatial resolution and ability to evaluate both osseous and soft-tissue structures. Multidetector CT with multiplanar and three-dimensional reconstruction facilitates accurate assessment of fracture extent, orbital volume changes, muscle incarceration, and associated facial fractures, thereby improving surgical planning. Nevertheless, imaging findings should always be interpreted in conjunction with clinical examination because radiographic evidence of muscle contact with a fracture does not invariably indicate true entrapment.⁷

The timing of surgical intervention remains an important determinant of outcome. Most uncomplicated orbital floor fractures may initially be managed conservatively while edema subsides, permitting more accurate assessment of persistent diplopia and enophthalmos. In contrast, pediatric trapdoor fractures with muscle entrapment or oculocardiac reflex require urgent surgical release to prevent irreversible muscle fibrosis and long-term ocular motility disorders. Similarly, patients with extensive orbital floor defects, significant enophthalmos, or persistent diplopia generally benefit from early reconstruction.⁸

Advances in orbital reconstruction have significantly improved functional and aesthetic outcomes. Titanium mesh, porous polyethylene implants, autogenous bone grafts, and more recently patient-specific implants have expanded reconstructive options for orbital wall defects. Computer-assisted planning and customized implants enable more precise restoration of orbital volume and contour, particularly in complex fractures. Despite these technological advances, successful outcomes continue to depend on meticulous surgical technique, accurate anatomical reduction, and preservation of viable orbital soft tissues.⁹

An important theme emerging from this review is the indispensable role of multidisciplinary collaboration. Optimal management of orbital fractures frequently requires coordinated care involving oral and maxillofacial surgeons, ophthalmologists, radiologists, otolaryngologists, plastic surgeons, and neurosurgeons. Early ophthalmologic consultation facilitates prompt

diagnosis of occult ocular injuries, while collaborative treatment planning ensures that visual function and facial reconstruction receive equal priority.²

Although substantial progress has been made in understanding orbital trauma, several challenges remain. Much of the current evidence is derived from retrospective studies with heterogeneous patient populations and varying outcome measures. Differences in fracture classification, surgical indications, timing of intervention, and assessment of visual outcomes limit direct comparison among studies. Future prospective multicenter studies using standardized diagnostic criteria and validated outcome measures are required to establish stronger evidence-based guidelines for the management of orbital fractures and associated ocular complications.¹⁰

Overall, the available literature supports a patient-centered approach combining meticulous ophthalmic examination, appropriate radiologic evaluation, timely recognition of vision-threatening complications, and individualized surgical management. Preservation of visual function should remain the foremost priority, while restoration of orbital anatomy and facial aesthetics represents an equally important objective. Continued advances in imaging, biomaterials, computer-assisted surgical planning, and multidisciplinary trauma care are expected to further improve patient outcomes following orbital fractures.⁸

CONCLUSION

Orbital fractures can produce a wide range of ocular manifestations, extending from minor periorbital injuries to devastating complications resulting in permanent vision loss. Accurate diagnosis requires a detailed understanding of orbital anatomy, fracture patterns, mechanisms of injury, and associated ophthalmic findings. Timely clinical assessment, appropriate radiologic evaluation, and coordinated multidisciplinary management are essential for preserving visual function, restoring orbital anatomy, and optimizing overall patient outcomes

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