Management of Orbital Floor Fractures

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Abstract

Fractures of the orbital floor represent a common yet difficult to manage sequelae of craniomaxillofacial trauma. Repair of these injuries should be carried out with the goal of restoring normal orbital volume, facial contour, and ocular motility. Precise surgical repair is imperative to reduce the risk of long-term debilitating morbidity. This article aims to review concepts on the management of orbital floor fractures in the hope of further elucidating perioperative evaluation and decision-making regarding operative intervention.

Keywords
- orbital reconstruction
- orbital blowout
- orbital floor fracture

Orbital floor fractures are common in the setting of craniomaxillofacial trauma. Isolated orbital floor fractures represent approximately 10% of all facial fractures, with nearly 30 to 40% of all facial fractures involving the orbit.[1–4] These injuries are managed in a multidisciplinary setting involving ophthalmic plastic surgeons, oral and maxillofacial surgeons, otolaryngologists, and plastic surgeons. Treatment of these fractures is therefore variable according to both surgeon training background and experience, as well as specific fracture pattern and clinical presentation. A summary of current evidence regarding evaluation and management is presented in this article to further aid surgeons in developing patient-specific treatment plans while mitigating risk of emotional, functional, and occupational patient morbidity.

Anatomy and Pathophysiology of Floor Fractures

The orbit is composed of seven facial bones including the frontal, zygomatic, maxillary, ethmoid, sphenoid, lacrimal, and palatine bones. The frontal, maxillary, and ethmoid bones also delineate the boundaries of their corresponding pneumatized sinuses. The sinuses act as "crumple zones" or areas of force absorption in blunt trauma that protect the globe. The orbital cavity has a pyramidal shape bounded by the roof, floor, medial, and lateral walls. The orbital floor, forming the roof of the maxillary sinus, slopes superiorly toward the apex of this pyramid, approximately 44 to 55 mm posterior to the orbital rim. The orbital cavity measures approximately 30 mL in volume, with the globe itself occupying 7 mL.[1,2]

A detailed understanding of orbital anatomy is of paramount importance in both the evaluation and treatment of the patient presenting with orbital trauma. The thinnest and most susceptible area to fracture along the orbital floor is medial to the infraorbital canal, with an average thickness of 0.4 to 0.5 mm. This area is particularly susceptible to fractures following blunt force trauma as the resultant force is propagated to this segment.[3] Several theories involving the mechanism of orbital floor fractures have been proposed. In 1901, Le Fort proposed the "buckling" theory which states that "blowout" fractures, in which the orbital floor is displaced downward into the maxillary sinus, occur through transmission of force through the more rigid infraorbital rim to the relatively weak orbital floor.[3–7] Pfeifer later postulated that hydraulic pressure propagated from the globe to the surrounding osseous orbit results in fracture of the orbital floor. This became termed the "hydraulic" theory.[3,8] Recent cadaveric experimental studies have shown that a combination of both mechanisms may result in floor fractures.[3,9,10] These injury mechanisms have been proposed to result in the

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production of differing fracture characteristics. Propagation of force through buckling has been shown to result in smaller, more linear fractures along the anterior orbital floor, with limited periorbital soft tissue herniation and lower rates of enophthalmos.[9–12] In contrast, hydraulic injuries correlate with larger, posterior fracture patterns involving both the floor and medial wall.[10–12] However, when both mechanisms occur in conjunction, there appears to be a summative effect resulting in fractures that are significantly more expansive.[12,13]

"Trapdoor" orbital fractures, particularly within the pediatric population, present with distinct clinical and radiographic findings deserving special consideration. Trapdoor fractures represent isolated orbital floor fractures, in which an osseous fragment hinged medially is transiently displaced inferiorly, allowing herniation of orbital contents into the maxillary sinus. These contents may then become entrapped as the bony fragment returns toward its native position. In all patients with suspected trapdoor fracture, extraocular movements should be evaluated to assess for entrapment of extraocular muscles. This typically presents with upward gaze restriction and diplopia secondary to the mechanical restriction from soft tissue entrapment within the orbital floor defect. In uncooperative or neurologically impaired patients, ocular motility may be evaluated through forced duction testing. Fine forceps are used to grasp conjunctiva at the attachment point of the inferior rectus, and movements assessing range of motion of the globe are attempted.[14–16] A more detailed discussion on emergent indications for repair and consideration of orbital fractures in the pediatric population is presented later in this article.

The superior orbital fissure provides passage for neurovascular structures from the middle cranial fossa to the orbital cavity. These include the oculomotor, trochlear, and abducens nerves, as well as the ophthalmic division of the trigeminal nerve (V_1) and both the superior and inferior ophthalmic veins.[17–20] Although rare, fractures extending to the superior orbital fissure may result in injury of these nerves with resultant ptosis secondary to paresis of the Müller muscle or levator palpebrae superioris. Proptosis, ophthalmoplegia, and associated sensory loss in the ophthalmic distribution of the trigeminal nerve are also potential sequelae of fractures through the superior orbital fissure.[19,21] The constellation of these symptoms, first described by Hirschfield in 1858, is termed "superior orbital fissure syndrome." These findings in conjunction with ipsilateral blindness should concern the surgeon for involvement of the optic canal, which carries the optic nerve and ophthalmic artery through the greater wing of the sphenoid. The combination of the above with optic neuropathy has been termed "orbital apex syndrome." The individualized use of high-dose steroids with or without surgical decompression of the optic canal has shown utility in some cases of vision compromise.[22–28]

Patient Evaluation

Clinical History and Physical Examination

Following thorough evaluation of patients with clearance of any potential life-threatening injuries, it is imperative to perform a thorough history including timing and mechanism of the surrounding injury. Consideration of potential non-accidental trauma is important in both children and elderly presenting with concern for abuse. A complete history is important for overall perioperative patient management.

This should be promptly followed by physical examination. Common presenting signs and symptoms in the context of orbital floor fractures include diplopia, periorbital ecchymosis, eyelid edema, subconjunctival hemorrhage, sensory deficits in the distribution of the inferior orbital nerve, and localized pain. Any soft tissue trauma, such as avulsion and laceration injuries, as well as bony step-offs, should be noted. The patient should then be evaluated for enophthalmos, hypoglobus, or general dystopia. Enophthalmos, posterior displacement of the globe within the anteroposterior axis, may be clinically detected at 2 mm or greater. Hypoglobus, inferior displacement of the globe following loss of the structural integrity of the orbital floor, may result in pseudostrabismus, in which there is vertical asymmetry of both globes with otherwise normal alignment of the visual axes of both eyes.[29] Orbital floor blowout results in an increase in overall orbital volume with relaxation of surrounding periorbita. Studies have indicated that as little as a 5% increase in orbital volume may result in clinically significant enophthalmos.[30–34] Therefore, globe position should be carefully evaluated in all orbital floor fractures. Hertel exophthalmometry may be employed to quantitatively evaluate sagittal globe position compared to the unaffected eye. Vertical asymmetry indicative of hypoglobus may be detected through appraisal of the pupillary light reflex. Assessment of visual acuity is critical in evaluating patients with orbital trauma. Color perception may be useful in determining injury to the optic nerve. Loss of color saturation, particularly within the red spectrum, is an early sign of optic neuropathy. Pupillary size and shape should be documented, as well as assessment for relative pupillary defect, which again is suggestive of optic neuropathy. Examination will reveal paradoxical dilation of the unaffected eye during a swinging light test from the unaffected eye to the affected eye.[35,36]

Radiographic Assessment

In cases of significant periorbital craniomaxillofacial trauma, one should have a low threshold for radiographic imaging, namely computed tomography (CT) with coronal and sagittal reconstructions from axial scans.[31,37,38] Thin-cut (1–2 mm) coronal sections are of particular utility in analyzing orbital floor fractures. Sagittal sections may be useful in assessing premorbid orbital contour prior to reconstructive efforts. They are also useful in the postoperative assessment of orbital floor reconstruction and restoration of orbital volume.[39,40] Despite its utility in delineating reliable data regarding the size of orbital defects, and resultant volumetric change within the orbital cavity, the role of CT imaging in predicting the need for operative management has been inconsistent.[41] Studies examining the size of fracture, fracture pattern (anterior/posterior floor, nasoethmoid strut), degree of soft tissue displacement, and rounding of the inferior rectus in predicting degree of enophthalmos or gaze restriction have shown varying results.[41–45] Preliminary studies categorizing fractures based on location and premorbid-to-postinjury orbital volume ratios have shown to be predictive of patients requiring surgical intervention but have not shown to be consistently reliable.[45,46]

Surgical Management

Immediate Intervention

Indications for repair of orbital floor fractures continue to remain controversial, and management has therefore primarily been guided by specialty training and clinical experience.[47–49] Immediate repair is defined as repair occurring within 24 to 48 hours following injury. Indications for immediate repair include early enophthalmos greater than 2 mm, isolated orbital floor or combined orbital floor/medial wall defects measuring greater than 2 cm^2, pediatric trap door fractures, and CT findings concerning entrapment with clinical correlation. Patients presenting with any of the aforementioned indications have been found to have improved outcomes and decreased rates of persistent diplopia or late enophthalmos when surgery is performed within 48 hours.[49–51] The length of muscle entrapment has been shown to correlate with persistent postoperative diplopia.[50–53] In severe cases, corrective motility surgery may be required in order to address persistent diplopia.[53–55]

Oculocardiac reflex, the triad of bradycardia, nausea, and syncope, is an indication for urgent exploration and repair of floor fractures. This reflex is elicited by either increased intraorbital pressure or periorbital soft tissue entrapment with a subsequent increase in vagal tone. The afferent pathway is carried by the ophthalmic division of the trigeminal nerve through the ciliary ganglion with efferent signals transmitted through the vagal nerve to the cardiac and gastric tissue. Persistence or worsening of these symptoms may result in cardiac dysrhythmia and is life-threatening. Therefore, immediate surgical exploration and reduction of any incarcerated tissue is warranted.[49,51,54,56] Urgent surgical intervention is recommended in patients presenting with oculocardiac reflex in order to prevent compartment syndrome of the inferior rectus muscle with resultant Volkmann contracture, an osseofascial compartment syndrome resulting in irreversible muscle ischemia.[49,54,55]

As previously mentioned, an approximate increase in orbital volume of 5% may result in clinically apparent enophthalmos.[30–32] With this in mind, some surgeons implement floor defect size criteria of 1 to 2 cm^2 or defects greater than 50% of the native floor as indications for early intervention.[41,47] A survey study evaluating the surgical management of orbital fractures found that 87% of surgeons employed fracture size as a determinant of intervention. Furthermore, rounding of the inferior rectus on CT has been associated with orbital blowout fractures. The muscle is typically displayed as an oval shape on cross-section on coronal reconstructions. The long axis is oriented within the transverse plane, with a resultant height-to-width ratio of < 1. Orbital floor fractures potentially result in distortion of this intrinsic shape secondary to edema, intramuscular hemorrhage, or loss of structural support. This finding on CT has been associated with enophthalmos, with a preoperative height-to-width ratio of > 1 prognosticating late enophthalmos.[57–59] Therefore, in the aforementioned cases, early surgical repair may be warranted to prevent late complications, resulting in dystopia following resolution of periorbital edema.

Delayed Intervention

Patients who lack the aforementioned findings requiring immediate or early repair may be reevaluated within a 2-week time frame to allow for resolution of edema while evaluating for progressive or persistent symptoms including diplopia, late enophthalmos, and infraorbital nerve hypesthesia.[49,51] Infraorbital hypesthesia is not typically an indication for intervention. However, progressive hypesthesia is indicative of nerve compression, and evidence, primarily limited to case reports, exists that intervention may improve outcomes.[60,61] Early diplopia following orbital fractures is a common finding secondary to muscle contusion and mechanical compression from surrounding edema. Diplopia should resolve within 2 weeks following injury. Persistent symptoms may be because of underlying intramuscular hemorrhage or motor nerve palsy. Persistent diplopia within 30 degrees of primary gaze with associated symptomology, positive forced duction, or radiographic findings suggestive of entrapment should be treated with surgical repair.[49,51] When possible, surgical correction should be delayed in order to facilitate resolution of edema. It is believed that deferment allows for increased mobility of displaced orbital contents that are otherwise restricted to a finite volume. This facilitates visualization of the entire fracture line and retrieval of orbital contents from within the paranasal sinuses while allowing for accurate shaping and placement of the reconstructive implant.[55] In theory, this will result in restoration of a final orbital volume, which is more reflective of the premorbid contour and thus more accurate. However, resolution of edema may unveil persistent enophthalmos or hypoglobus not appreciated on initial evaluation. Current evidence suggests that the presence of these signs 6 weeks following injury onset warrants surgical repair.[40,62]

Surgical Approaches to Repair

A variety of surgical approaches to the orbital floor have previously been described, including subciliary, subtarsal, transantral, transcaruncular, and transconjunctival techniques.[63,64] The subciliary approach has exhibited a high rate of postoperative cicatricial ectropion.[63,64] The transconjunctival approach has become the preferred technique due to low rates of complications, wide surgical exposure, and uncomplicated dissection. It can also be combined with a lateral canthotomy for additional exposure. Transcaruncular extension for medial exposure is also an option, although there is an associated risk of nasolacrimal obstruction secondary to scar formation.[65] Patients with prior procedures necessitating an external lid incision have also been found to be at risk for ectropion when using a transconjunctival approach.[64,66,67] In comparison, the subtarsal incision allows for direct access to the orbital floor and is technically facile but results in poor aesthetic outcomes with significant risk of scarring.[63,64] Studies reviewing surgical approaches have failed to find high-level evidence, implicating the use of one technique over another when evaluating ocular outcomes. However, a consistent trend toward lower complications with the use of tranconjunctival techniques and higher rates of complications requiring revision surgery with subciliary and subtarsal methods have been documented.[64–67]

Implant Materials in Floor Reconstruction

Following reduction of orbital contents, reconstruction of the orbital floor may be performed through the use of one of several implant materials. Allogenic or autogenous materials present the potential benefit of improved biocompatibility, whereas implementation of synthetic substrates has been historically correlated with high rates of both infection and extrusion. Rates of implant-related complications are low and have been attributed to devitalized and potentially contaminated orbital soft tissue at the time of repair.[68] Nevertheless, debate continues regarding the preferred material with which orbital reconstruction is performed. An ideal reconstructive implant is one which is chemically inert, bio-friendly, nonimmunogenic, and noncarcinogenic. This material should preferably be cost-effective, able to withstand decontamination, and easily manipulated yet able to retain its shape once implanted.[68] A brief overview of available materials for reconstruction follows. Interested readers are directed to the references for comprehensive reviews of biomaterials.[68–70]

Biological Materials

Biological implants offer the potential benefit of biocompatibility while being associated with high donor-site morbidity. These materials include autologous cartilage and bone, dural allograft, collagen, or dermal xenograft.[70] However, despite their advantages, biological grafts display variable resorption rates, resulting in a degree of long-term unpredictability with regard to reconstructive orbital volume, enophthalmos, and visual outcomes.[69–71]

Alloplastic Material

Alloplastic implant material may be further differentiated into resorbable and nonresorbable substrates, each with characteristic benefits and disadvantages. Synthetic resorbable alloplasts include poly-L-lactic acid, polydioxanone, polyglycolic acid, and composite polymers. Although these substrates are readily available and malleable and facilitate osseous growth, various studies have indicated an association with inflammation, delayed enophthalmos, and dystopia.[70–72] These materials have shown limited utility in pediatric fractures due to a lack of growth of the implanted material with the patient.

In comparison, permanent alloplastic materials provide longer term rigidity with a higher risk of infection. Porous polyethylene allows for adequate structural support while allowing vascular ingrowth. However, this material has been shown to result in an inflammatory response with the risk of adhesion formation to surrounding extraocular muscles.[69,70,73] In contrast, titanium mesh implants provide biocompatibility and conform easily to orbital contours. The surgeon must, however, consider that titanium has been previously shown to result in significant fibrosis, which may present a challenge in the setting of secondary surgery. Hybrid implants, composed of both the aforementioned materials, have attempted to address individual deficiencies associated with each implant material.[69–71] A systematic review of biomaterials used in orbital floor reconstruction, comparing variable outcome measures, did not demonstrate strong evidence in support of one specific material when compared with another.[69] Therefore, surgeons are encouraged to develop individualized surgical plans that exploit the strengths of each material on a case-by-case basis.

Pediatric Orbital Fractures

Orbital fractures in the pediatric subpopulation represent a distinct subdivision of orbital fractures due to unique differences, particularly with respect to osteology within this patient population. Recent data suggests that regardless of defect size, pediatric orbital blowout fracture may be managed conservatively in the absence of entrapment, dystopia, or enophthalmos.[74,75] In the setting of operative intervention in pediatric patients, consideration must be given to current and future growth patterns when selecting the reconstructive material. Implementation of rigid alloplasts results in growth restriction or implant entrapment within actively remodeling orbital bone. With this in mind, biocompatible resorbable substrates are preferred for reconstruction.[76,77] Due to the greater elasticity of pediatric bone, there is an increased susceptibility to greenstick fractures. Similarly, trapdoor fractures are more common in this population. These fractures may present without subconjunctival hemorrhage and have been therefore referred to as “white-eyed blowout fractures.” Oculocardiac reflex is particularly robust in pediatric patients presenting with this type of injury pattern.[78] Clinicians must therefore maintain a high level of suspicion in pediatric patients presenting with periorbital trauma and symptoms suggestive of entrapment. Early surgical intervention, defined as within 48 hours, has been associated with improved overall visual outcomes in these patients.[79] Nylon foil has shown utility as an inert nonporous alloplastic implant within the pediatric population with low complication rates. It represents a safe and viable option while mitigating risk of tissue integration and orbital adherence due to its nonporous nature.[80]

Complications

Common complications following repair of the orbital floor include infraorbital nerve dysfunction, ectropion, persistent diplopia, and enophthalmos. Postoperative diplopia occurs in approximately 20 to 50% of patients.[81–83] Further studies have revealed upward of 50% incidence of postoperative hypesthesia or dysesthesia attributable to infraorbital nerve dysfunction. However, early identification of these complications followed by repair within 48 hours resulted in significant reduction in persistent symptoms.[84] Conversely, lower lid malposition, either ectropion or entropion, are cited relatively infrequently, with rates less than 5%.[85] Implant-associated complications include extrusion, infection, palpability, and migration. The majority of complications in orbital reconstruction are attributable to inadequate reconstitution of the osseous orbital contours. Overall enlargement of the orbital cavity is seen in approximately 8.5% of orbital reconstructions.[86] This is because of the inability to distinguish landmarks particularly in the setting of severe bony destruction.[86,87] Further potential may therefore lie in use of preoperative stereolithographic modeling, intraoperative navigation systems, and CT imaging. Consideration should therefore be given for use of this technology in complex fracture patterns.

Conclusion

Orbital floor fractures are common in the setting of facial trauma. Despite continuing efforts toward developing uniform, evidence-based management protocols, there continues to be evolving management paradigms across multiple subspecialties. Indications for surgical management should therefore be evaluated on an individual basis. Significant dystopia, enophthalmos, extraocular muscle entrapment with associated motility restriction, and oculocardiac reflex are important distinguishing features that influence the decision to pursue early surgical repair. Timing of intervention otherwise may vary based on the patient's clinical presentation. A high index of suspicion is important in the pediatric population as "white-eyed" blowout fractures may exhibit minimal physical signs of orbital trauma but may have muscle entrapment with a greater associated risk of muscle ischemia.

Conflict of Interest
None.

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