Elderly Man with Orbital Blow-out Fractures After a Motor Vehicle Accident

Case Presentation

A 71-year-old white man presented to his primary care physician complaining of a swollen left eye for 2 days. He had been involved in a major motor vehicle accident and the airbags did not deploy. He hit his head against the steering wheel but only suffered a broken left thumb and never lost consciousness. When he was assessed in the emergency department, he had no complaints of mental status changes, diplopia, enophthalmos, or blurry vision. He did have mild left periocular ecchymosis but no crepitus or irregularity of the orbital rim on palpation. His vital signs were stable. The patient was treated and discharged. During the next 2 days he developed increasing left lower-eyelid swelling.

Examination revealed mild swelling and ecchymosis of the left eye but no visible enophthalmos. Visual acuity was 20/20 in the right eye and 20/15 in the left eye. There was marked subcutaneous emphysema but no orbital rim tenderness or step-off. Extraocular movements were completely intact. He did not have diplopia in any extremity of gaze. Examination of the eye with a direct ophthalmoscope did not reveal any injury.

Given the mechanism of injury and presentation of subcutaneous emphysema, a computed tomography (CT) scan with coronal and axial cuts was obtained to evaluate for orbital blow-out fracture. The scan revealed several fractures of the left orbital floor, the lamina papyracea, and the lateral wall of the left orbit. Marked orbital emphysema was also noted (Figure). He was prescribed a 10-day course of oral amoxicillin/potassium clavulanate (Augmentin) and a decongestant to be used twice daily and was advised not to blow his nose. The patient was referred to an orbital specialist for repair. He had a benign postoperative course without any visual complaints.

Discussion

 Orbital blow-out fractures are often the result of blunt trauma to the ocular region, typically with an object whose diameter is greater than the width of the orbital outlet. By far, the most common cause of blow-out fractures in the adult population is motor vehicle accidents, followed by physical assault and sports-related injuries. Since emergency department and primary care physicians are often the first to encounter patients with these injuries, they must be able to recognize and appropriately manage them.

Clinically, blow-out fractures are characterized (in order of frequency) by periorbital ecchymosis, diplopia, subconjunctival hemorrhage, enophthalmos (sunken globe), inferior orbital nerve paresthesia, and periorbital swelling or edema. Systemic symptoms, as a result of the oculocardiac reflex (typically caused by entrapment of soft tissues), may include nausea, vomiting, and bradycardia. Because very little force is usually required to cause a blow-out fracture (78 mJ), the presentation may be subtle, as in our patient.

Anatomy

The 7 bones of the orbit are the maxilla, zygoma, lacrimal, ethmoid, palatine, sphenoid, and frontal. They form a cone that supports and protects the globe. The rim of the orbit is thick and strong, whereas the orbital walls, especially the floor and medial walls, are thin and easily fractured. The medial wall is composed of the maxilla, lacrimal, ethmoid (lamina papyracea), and sphenoid bones. The orbital floor, which is made up of the maxillary bone, overlies the maxillary sinus. Although the medial wall (specifically the lamina papyracea) is thinner than the orbital floor, it is reinforced by ethmoid air cells that form a “honeycomb” structure, making it stronger than the orbital floor.

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The floor is dissected diagonally by the infraorbital groove. The infraorbital nerve, which provides sensation to the cheek and ipsilateral upper alveolus and teeth, traverses the intraorbital groove and creates a natural “break line” that alleviates any pressure generated within the globe.\(^3\) Orbital fractures, thus, act as a safety valve against any sudden intense rise in intraorbital pressure.

### Mechanism of trauma

There are 2 theories for how orbital floor fractures occur with blunt trauma. The buckling theory contends that increased pressure from trauma causes the weakest part of the orbit—the floor—to snap. The hydraulic theory proposes that blows to the rim create pressure gradients transmitted by bone to the weakest points, thereby shattering them. This effectively dissipates the energy from the trauma. Many studies have supported both mechanisms of action. Regardless of mechanism, the design ultimately preserves vision by protecting the globe.\(^3\)

### Evaluation

Although orbital blow-out fractures usually require urgent care, the initial evaluation of ocular trauma should rule out vision-threatening injuries to the eye. The presence of vision loss or blurring, hyphema, proptosis, afferent papillary defect, and severe eye pain warrant immediate evaluation by an ophthalmologist to rule out ocular emergencies, such as optic nerve compromise, orbital hematoma, globe rupture, orbital foreign bodies, retinal detachment, or optic nerve sheath hematoma. In some cases (eg, retrobulbar hemorrhage), irreversible vision loss resulting from increased ocular pressure can occur within 90 minutes without decompression.\(^4,5\)

Once ocular injury has been safely ruled out, an evaluation for blow-out fractures may continue. The triad of diplopia, enophthalmos, and infraorbital paresthesia is highly suggestive of orbital floor fracture.\(^3\) A retrospective review of 199 patients found that the most common signs and symptoms of orbital blow-out fractures were periorbital ecchymosis (73.5%), diplopia (45.5%), subconjunctival hemorrhage (38.6%), enophthalmos (33.9%), and infraorbital nerve paresthesia (20.6%).\(^1\)

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The most obvious concern in any blow-out fracture is herniation and entrapment of fat, muscle, or the globe. The presence of diplopia and severe pain with any attempted eye movement indicates entrapment of the inferior rectus and oblique muscles because they are herniated through the fracture. Diplopia can also be the result of periorbital edema that prevents the superior oblique muscle from functioning. The degree of enophthalmos is directly proportional to the extent of tissue herniation through the fractured walls or floor. Entrapment of muscle or fat may induce the oculocardiac reflex, causing bradycardia, nausea, and vomiting. Ultimately, a diagnosis of entrapment is made clinically and not by radiographic findings.\(^8\) Infraorbital paresthesia is caused by damage to the infraorbital nerve.
Imaging studies
Suspected orbital blow-out fractures can be evaluated by different imaging modalities. Imaging is especially important when the diagnosis is in question, as is the case in our patient. CT scanning with coronal and axial views in 2-mm cuts remains the radiographic gold standard for evaluation of orbital blow-out fractures. It is readily available in most hospitals and does not require direct contact with the globe. CT imaging is faster and less expensive than magnetic resonance imaging (MRI). One important disadvantage of CT is the inability to visualize intraocular structures and rule out eye injury.

Ultrasound has the capability to take real-time images of the eye and does allow visualization of intraocular structures. However, the ultrasonic probe would have to be in direct contact with the eye, which will increase intraocular pressure. Since this may result in or worsen leakage of ocular contents through a ruptured globe, ultrasound should not be performed without the assistance of an ophthalmologist.

Plain x-rays often produce false-negative results (50%) or are nondiagnostic (30%). As a result, care must be taken when interpreting x-rays. MRI has the advantage of being able to evaluate soft tissue and eye injury but is markedly inferior to CT in the evaluation of orbital fractures and plays a less prominent role in management.

Management
Orbital blow-out fractures without evidence of entrapment and ocular injury are treated with supportive care, including broad-spectrum antibiotics, nasal decongestants, and avoidance of any nose blowing. Any increase in nasal pressure can cause intraorbital emphysema and result in compromised retinal blood flow and optic nerve compression. Some studies support the use of systemic corticosteroids to inhibit fibrosis formation.

Patients with signs of entrapment (evidenced by diplopia or bradycardia), enophthalmos more than 2 mm, or symptoms suggestive of retrobulbar hemorrhage, including severe eye pain, proptosis, or decreased eye movement, should be immediately referred to an ophthalmologist because of the danger of optic-nerve damage and muscle ischemia. In a retrobulbar hemorrhage, an immediate lateral canthotomy is performed to prevent rising intraocular pressure that could cause blindness.

Surgery for eye muscle entrapment is usually postponed for 7 to 10 days to allow the swelling to subside. Diplopia may resolve during this time as the edema diminishes. Previously, it was believed necessary to wait up to 6 months before any surgical intervention, but intervention is now recommended within 7 to 10 days to prevent fibrosis of the muscles.

Conclusion
Orbital blow-out fractures are the result of an extraordinary anatomical design that protects the eye from true ocular emergencies. Physicians must be cognizant of the possibility of these fractures in patients who have sustained any orbital trauma. When suspicion for a blow-out fracture is high, coronal and axial CT scans of the orbit should be obtained. It is vital to recognize a blow-out fracture, but it is even more important to first rule out ocular injuries that can threaten vision and require emergent referral to an ophthalmologist.

References
We applaud the author’s excellent review of the diagnosis and management of orbital fractures and would like to add our own experience. As ophthalmologists, our primary concern is the preservation of vision. Thus, we believe that any case of suspected orbital or ocular trauma warrants consultation with an ophthalmologist, regardless of whether ocular signs or symptoms are present.

The most important condition to rule out is a ruptured globe. The patient should not be allowed to take anything by mouth. A protective eye shield that rests on the bony orbital rim should be placed and adequate pain and nausea control instituted. Pain control is essential to prevent extrusion of the ocular contents from excessive Valsalva pressure. All manipulations of the globe should be avoided until the globe has been deemed intact by ophthalmic examination.

As described in this case, retrobulbar hemorrhage is an ocular emergency that requires immediate attention. Prompt diagnosis and treatment is essential to prevent blindness. An expanding retrobulbar hemorrhage can cause an orbital compartment syndrome, leading to ischemia of the optic nerve. Once this happens, permanent and irreversible blindness can occur in as little as 90 minutes, based on primate studies. Keys to the diagnosis of an orbital hemorrhage include proptosis, decreased vision, increased intraocular pressure, and the presence of a relative afferent papillary defect. Management includes performing a lateral canthotomy of the overlying skin and muscle and, more importantly, lysis of the lateral canthal tendon (cantholysis). Only after the tendon is lysed can the orbital contents prolapse forward, relieving the compartment syndrome. If lateral canthotomy fails to alleviate the compartment syndrome, orbital decompression should be performed in an emergent operative setting.

Pediatric cases of orbital trauma require special considerations. The anatomy of the orbit and adnexal tissues undergo dramatic changes as we age. The frontal sinus is the last of the paranasal sinuses to pneumatize and is not visible radiographically until the sixth year of life. As a result, frontal trauma can result in orbital roof fractures in young children. In adults, the fully developed frontal sinus acts as a crumple zone that absorbs trauma. The presence of pulsatile proptosis, cerebrospinal fluid rhinorrhea, signs of meningeal irritation, or pneumocephalus on brain imaging should alert the clinician to the possibility of orbital roof fractures. Immediate neurosurgical consultation is warranted in such cases.

Orbital floor fractures are also different in children. In adults, the rigid nature of the orbital floor causes most fractures to remain displaced. In children, the orbital floor is more pliable, causing a higher rate of inferior rectus muscle entrapment because of a “trapdoor” effect. Prolonged entrapment can cause ischemic necrosis of the muscle or permanent scarring, leading to impaired ocular motility. In visually immature children, this induced diplopia can result in vision loss secondary to amblyopia. Sometimes, life-threatening bradycardia can result from excessive stimulation of the oculocardiac reflex as a result of an impinged extraocular muscle. Urgent surgical repair is mandatory.

Trauma to the orbit and adnexal tissues requires timely diagnosis and evaluation. Knowledge of the vision-threatening complications of orbital trauma and prompt ophthalmic evaluation will ensure optimal patient outcomes.

References