

ORBITAL TRAUMA- A REVIEW

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ABSTRACT:

Trauma to the mid-facial region commonly causes ocular injuries of varying degrees. Majority of the patients suffer injury to the eye who have sustained trauma to the mid-facial region. Orbital contusion or fractures have been reported in as many as 30% of the cases and approximately 15% have decreased vision. Only 2 ocular emergencies require treatment within minute's I.e.: chemical burns of the eye and central retinal artery occlusion. Apart from these there is adequate of time for thorough history taking and examination. Isolated orbital blow-out fractures will have an associated eye injury in up to one third of patient. Both prospective and retrospective studies of patients who have sustained bifacial fractures indicate that as many as 20% may sustain serious ocular injury that warrants ophthalmological referral.

KEY WORDS: Head & Neck , reconstruction, Mortality

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INTRODUCTION:

In 2001 an estimated 1.9 million reported cases were treated in the USA. Orbital fractures are of varying degree from fractures causing trauma to globe or seemingly minor trauma may cause fracture without the injury to the globe. The supra orbital and lower maxillary regions are more resistant to trauma when compared to the glabella area, the nasal bridge, and the malar eminence⁸⁸. In contrast, the resilient structure of the globe allows it to withstand blows of considerable

force without rupture. Orbital floor fractures have been associated with a 33% incidence of ophthalmic complications^{1,2}. Severity of injuries may range from a mild corneal abrasion and traumatic iritis to optic nerve avulsion and globe rupture. Some ophthalmic injuries are immediately obvious but other less overt complications must be excluded. Examination of the eye is mandatory for every patient who has sustained mid facial trauma severe enough to cause fracture since inadequate care can result in blindness.

Clinical examination:

It should assess the function and anatomy of both eyes. Thorough history taking, a step by step assessment of vision, and examination of the eye and the adnexal tissue. Vision before the injury is important information, and a record of acuity exists if the patient has an ophthalmologist. The following information should be sought but may not be attainable because of the severity of the injury:

1. The time place and circumstances of the injury including any loss of consciousness.
2. Exact nature if injury, and any possible intraocular or intra-orbital foreign body.
3. The type of object that caused the injury [blow out orbital fractures are usually caused by the size of the object great than the orbit.]
4. The amount duration and the direction of the force.
5. Occurrence of the diplopia, epistaxis or rhinorrhoea.
6. Whether eyeglasses were worn at the time of the injury as they may have protected the eyes or caused a glass foreign body.
7. Past ocular history involving visual status, prior trauma, intraocular surgery, strabismus and amblyopia.

Following are 8 parts – eye examinations:

1. Visual acuity
2. External examination
3. Ocular motility

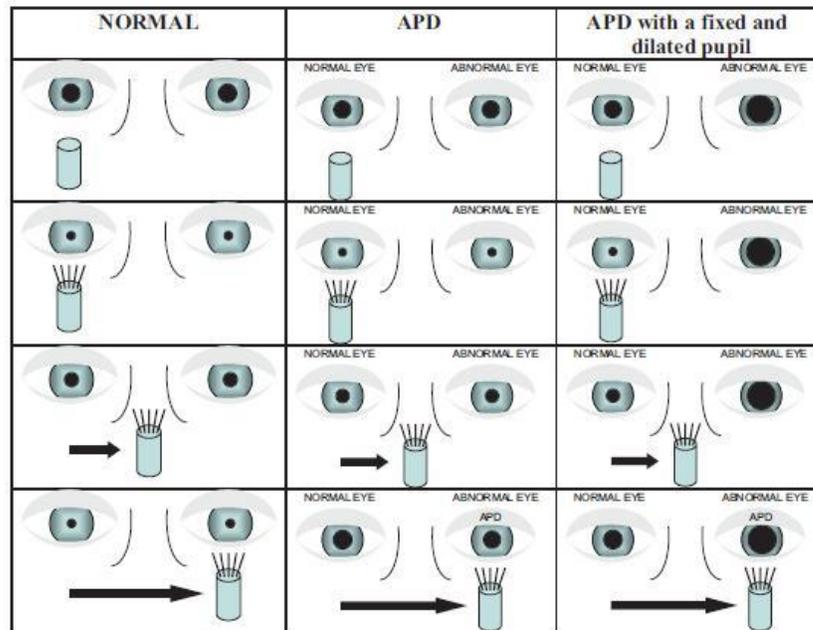
4. Pupils
5. Visual fields
6. Slit-lamp examination
7. Tonometry
8. Fundus examination

The eyelids are inspected for oedema, ecchymosis, burns, foreign bodies, lacerations, ptosis, canalicular damage or avulsion of the canthal tendons. When searching for the foreign body it is important to Evert the upper eyelid with the help of lid retractor. A lid retractor is also valuable in separating the eyelids when examining patients with significant pre-orbital or orbital hematoma. The orbital rim should be palpated for crepitus or step-off deformity. Retraction or proptosis of the globe can be best assessed from below or from the side and can be measured with a hertel exoptalmometer³.

Extra ocular movements are then tested for evidence of restriction or paralysis and for the presence of diplopia in any direction of gaze. To diagnose a restrictive cause a forced duction test can be performed which determines each eyes resistance to passive rotation toward the limited field of gaze. Pupillary size and light reactions should be checked. Optic nerve compromise can be detected by the presence of a Marcus-gunn pupil {apparent pupillary defect} which is elected with the swinging flashlight test⁴.

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Figure 3. Afferent Pupillary Defect (APD) As Detected With The “Swinging Flashlight Test”



In the normal condition, both pupils constrict regardless of which eye is illuminated, due to intact direct and consensual light reflexes. With an APD, the pupil in the normal eye dilates upon illumination of the pathological eye because of a lack of stimulus for the consensual light reflex.

EXAMINATION OF THE ORBIT:

Examination of the orbit should include the bony structures, globe position and the surrounding soft tissues. Oedema, ecchymosis, and crepitus may indicate that the orbit has sustained serious injuries such as fractures or hematomas. Globe position and intercanthal distance should be reported as well because abnormalities with these indicate that severe injury is present. Bony deformation can often be palpated but this can be masked by extensive amounts of soft tissue oedema. Foreign bodies must be sought when there is disruption of the soft tissues and clinical suspicion may be upon the mechanism of trauma⁵.

The position of the globe may indicate where the pathology is present [e.g.; lateral globe displacement from a process in the medial orbit]. Also a widened intercanthal distance may exist in naso-orbital ethmoidal fractures. Soft tissue wounds are potential entry sites for foreign bodies and need thorough examination. Clinical findings assist the evaluation of radiographic imaging such as computed tomography [CT], which can help determine the full extent of the injuries and the location of foreign bodies.

ANTERIO-POSTERIOR GLOBE DISPLACEMENT [ENOPHTHALMOS OR EXOPHTHALMOS]

A number of traumatic mechanisms can cause anterior or posterior positioning of the globe. Immediately following injury, anterior protrusion of the globe, or exophthalmoses, and bony fragments within the orbit. As the soft tissue swelling diminishes, posterior positioning of the globe, or enophthalmos, may develop. Alternatively, enophthalmos may be seen immediately following injury when a large orbital floor defect is present. Inspection may reveal a deepening of the suprasternal sulcus and pseudo ptosis of the upper eyelid secondary to backward disposition of the globe⁶.

The antero-posterior displacement is most accurately measured with an exoptalmometer by comparing the globe position in relation to the globe, and a difference of more than 2 mm is considered abnormal⁷. This instrument, however, uses the lateral orbital margin as the reference point and cannot be used in cases in which the orbital rim has been displaced. Another means of assessment in these cases is to position the back of the patient's chair at a 45-degree angle or to ask the patient to lift his/her chin up and then examine the patient from below, comparing the positions of the corneas with respect to the malar surfaces. A patient with left-sided enophthalmos is shown in the figure.

NONPERFORATING EYE INJURIES

Blunt trauma to the eye can cause both direct and compressive injuries. Direct injury results from concussive forces striking the eye. Compressive injuries occur when a force presses against the globe, temporarily shortening its antero-posterior dimensions and lengthening the vertical dimensions. The internal structures of the eye that are circumferentially attached may tear from their insertions as a result. The various anatomic locations and associated non-perforating injuries are described⁸.

CONJUNCTIVA

Trauma to the surface of the eye often leads to sub-conjunctival haemorrhage. This appears as painless, bright red blood on the surface of the eye beneath the conjunctiva and may be associated with chemosis, or oedema. Bleeding may be localised from a vessel within the

conjunctiva, or in deeper injuries may extend from the orbit and track anteriorly. Orbital injury will have associated signs such as exophthalmoses, limitations of ocular motility, and periocular ecchymosis. Patients with 360 degrees of sub-conjunctival hemorrhage and diminished vision may require surgical exploration to rule out an occult rupture of the eye. Lacerations of the conjunctiva need to be completely explored to ensure that deeper injury does not exist, and larger laceration [$>10\text{mm}$] should be sutured⁹.

CORNEA.

Abrasion of the corneal surface with or without a foreign body causes severe pain, blurry vision, tearing and photophobia. An abrasion results when the epithelial surface of the eye is disrupted for many reasons.



Direct trauma, prolonged exposure to air, or contact with causating agents [e.g.; cleaning agents, alcohol etc.] all can cause an abrasion. The diagnosis is made by fluorescein staining of the denuded epithelium, which will fluoresce with blue light. A topical anesthetic will provide enough relief to permit examination. Slit-lamp bio microscopy is recommended to determine the full extent of an eye surface injury. When the patient has been struck by a piece of metal with high velocity [e.g.; hammering material on metal], foreign bodies and penetration of the globe should be suspected. The depth of the wound needs to be clearly visualized since small penetrating injuries can be self-sealing.

Abrasions leave the corneal stroma vulnerable to infection and are treated with topical antibiotics until the protective epithelium can regenerate. Small abrasions often heal within 24 hours and these patients need daily examinations until the abrasion is gone. Larger abrasions with associated

photophobia can be treated with an additional cycloplegic drop, such as cyclopentolate. This treats the pain of ciliary body spasm by temporarily paralyzing the muscle. A topical non-steroidal anti-inflammatory drug may also be of benefit¹⁰.

If a foreign body is present, it should be removed with great care using binocular magnification if possible. A hypodermic needle held tangential to the corneal surface is used to elevate the foreign body after a topical anaesthetic has been instilled. The patient must not move, and the forehead band on the slit-lamp prevents the patient from moving forward towards the needle. The patient is encouraged to rest with the both eyes closed in order to prevent the continuous rubbing action of the eyelids over the fragile healing epithelium. The patient is examined daily until the epithelium has healed, and topical antibiotics are necessary on a frequent basis.

Blunt injury can also cause oedema of the cornea by affecting the innermost corneal layer composed of endothelial cells. Damage to the endothelium may result from direct contusion, increased intraocular pressure, and/or reactive inflammation^{11,12}. This monolayer of cells continuously pumps water out of the corneal stroma, providing the clarity necessary for vision. Damage to these cells produces an opaque, thickened, oedematous cornea. The oedema usually resolves spontaneously but may require topical hyper-osmotic medication and/or corneal grafting.

ANTERIOR CHAMBER.

The anatomic space between the cornea and the iris is the anterior chamber, which is filled with aqueous fluid secreted from the ciliary processes. Eye trauma can tear iris and ciliary body blood vessels, resulting in bleeding in the anterior chamber and inflammation. Also, the anterior chamber may become deep or shallow depending on the extent and location of the injury¹³.

HYPHEMA.

A hyphema is blood in the anterior chamber and the most often results for tearing of the blood vessels at the root of the iris.



The patient usually represents with pain, photophobia, and blurred vision, and red blood cells can be seen in the anterior chamber. Microscopic evidence of red blood cells in the aqueous without layering inferiorly is called a microhyphema. Larger amounts of blood can be seen without magnification and will collect along the bottom of the chamber when the patient is upright. The prognosis is related to the amount of blood present. The height of the blood needs to be measured daily and this can be followed as a clinical sign of improvement or worsening¹⁴.

Patients are prescribed bed rest to mineralizes re-bleeding, and the head of the bed should be elevated at least 45 degrees at all times. Atropine 1% decreases the pain and helps constrict the blood vessels, and prednisolone 1% decreases intraocular inflammation. Aminocaproic acid may be used in the early course of treatment for larger hyphemas. The intraocular pressure must be evaluated daily because blood and inflammation may impair aqueous outflow. In the event of raised intraocular pressure, corneal blood staining may result, necessitating washout of the anterior chamber on an urgent basis. Other indications for surgery are significant visual deterioration, total filling of blood in the anterior chamber, persistent clot in the angle for 7 days, and increased intraocular pressure despite medical treatment [$>50\text{mm Hg}$ for 5 days or $>35\text{mm Hg}$ for 7 days]¹⁵.

ABNORMAL DEPTH OF THE ANTERIOR CHAMBER.

An abnormal depth of the anterior chamber is a sign of damage to the eye, and either shallow or a deep chamber provides clues to the side of pathology. The distance between the cornea and the iris can be evaluated by observing the eye from an oblique angle, and this is performed most effectively using an external source of light and high magnification. A shallow anterior chamber

can result from low pressure from blood or oedema within the posterior segment of the eye. A deep anterior chamber may indicate that the iris and/or lens are torn from its insertion, or the eye could be ruptured. Intraocular pressure helps differentiate these pathologic processes^{16,17}.

PENETRATING OCULAR INJURIES:

Penetrating injuries are classified into rupture or lacerating injuries. Rupture may occur secondary to a fall or blunt trauma and lacerating injuries are secondary to a glass cut or knife stab wound. Non-penetrating injuries often result from concussive or blunt trauma to the globe and may lead to various types of ocular damage and visual loss as previously described. Patients involved in traffic accidents or those with multiple facial lacerations should be examined carefully for the possibility of a penetrating ocular injury. Sharp objects such as glass or a fast flying missile [e.g.; bullet] can cause laceration of the cornea or sclera, or both. Severe blunt trauma can result in scleral rupture and an open globe. The two most common locations of a rupture are the limbus and posterior to the extra ocular muscle attachment, where the sclera is usually the thinnest. On presentation the visual acuity should be obtained if possible. Visual acuity on presentation is a reliable predictor of the long-term visual prognosis. Ocular examination should be carried out without any pressure on the globe. The pupils should be examined for shape and size and the presence or absence of an afferent pupillary defect. The cornea and sclera are inspected for lacerations and possible prolapsed of iris and uveal tissue through the wounds. Absence of uveal tissue prolapse does not rule out an open globe. An ophthalmologist should examine patients with a suspected open globe injury because delayed diagnosis and repair of an open globe increase the risk of endophthalmitis and permanent visual loss.

OPTIC DISC AVULSION

Optic disc avulsion may result from a penetrating orbital trauma a backward pulling force on the optic nerve. It can also occur from forward displacement of the globe or a severe sudden rise in intraocular pressure with a rupture of the lamina cribrosa. There is a total loss of vision on presentation. Hemorrhage obscuring the optic nerve and vascular occlusion are seen acutely.

INTRAORBITAL FOREIGN BODY.

Intraorbital foreign body may present with pain, decreased vision and double vision or it can be asymptomatic. With a history of trauma, CT of the orbit is necessary to identify an orbital foreign body¹⁸. On examination, patients present with or without a palpable mass, limitation of ocular motility, proptosis, periorbital erythema and swelling. Organic or wooden foreign bodies increase the risk of infection, orbital cellulitis and they must be removed in cases. Posterior inert foreign bodies are usually left alone in the absence of ocular motility disturbances or impingement on the optic nerve because of the possibility of iatrogenic damage to the optic nerve and other orbital structures.

TRAUMATIC RETROBULBAR HAEMORRHAGE.

Retrobulbar hemorrhage occurs in the setting of penetrating or blunt trauma with or without orbital bone fractures.



It is usually a rapidly expanding hematoma; however, some evolve over time. Patients taking anticoagulants who sustain orbital trauma may present with a delayed Retrobulbar hematoma. Early recognition of retrobulbar haemorrhage is critical. Patients typically present with proptosis, ecchymosis and subconjunctival haemorrhage. Visual compromise is secondary to compartment syndrome or compression of the optic nerve, leading to poor perfusion of the optic nerve and retina.

Lagophthalmos in patients with severe proptosis results in damage to the cornea with exposure keratopathy that contributes further to visual compromise. Intraocular pressure may be elevated

secondary to increased orbital pressure. An afferent pupillary defect may be present, reflecting damage to the optic nerve from compression or a stretch effect in cases of extreme proptosis. Immediate intervention includes lateral canthotomy and lateral canthal tendon lysis to relieve orbital pressure. If the vision does not show improvement, orbital decompression and evaluation of the hematoma should be considered. The anticoagulation status of trauma patients secondary to medication or underlying medical condition should be reviewed and reversed as it can exacerbate a traumatic retro bulbar hemorrhage¹⁹.

TRAUMATIC OPTIC NEUROPATHY

The optic nerve may be injured directly or indirectly by craniofacial trauma. The most common sites or injury are the intracanalicular and intracranial portions of the optic nerve²⁰. The impact of forces from a frontal injury tends to be concentrated and transmitted to the optic canal. Direct injury can be caused by transection of nerve fibers, avulsion of the optic nerve, or compression by intrasheath hemorrhage. Indirect injury to the optic nerve may occur with relatively minor or more severe cranio-orbital trauma. It is the most common form of traumatic optic neuropathy and occurs in 0.5% to 5% of patients who suffer closed head trauma. Indirect injury results from avulsion of the vascular supply by shear forces of the intracanalicular segment of the optic nerve. Orbital hemorrhage may cause progressive compression of the optic nerve and secondary vision loss. The intracranial segment of the optic nerve may be injured secondarily by the falciiform dural fold during the impact²¹.

Visual loss may be immediate or delayed. An afferent pupillary defect is invariably present in unilateral injuries. In bilateral injuries, an afferent pupillary defect is absent but the pupillary reaction is sluggish. In anterior optic nerve injuries, infarction or hemorrhage is seen on fundoscopic examination. In more posterior injury to the nerve, an afferent papillary defect and visual loss may be despite a normal fundoscopic examination.

Treatment of traumatic optic neuropathy is controversial. The use of mega-doses of corticosteroids is based on the success observed in spinal cord injuries. High-dose intravenous corticosteroids have an antioxidant and membrane stabilizing effect when administered within 8 hours of injury. Beyond 8 hours, corticosteroids decrease oedema around the nerve but have little neuroprotective effect on the injured axons²². The international optic nerve trauma study, a

multicentre randomized comparative analysis, did not find a clear benefit of either intravenous corticosteroids or surgical optic canal decompression. The general guidelines advocate the use of high-dose corticosteroids as soon as possible if the patient's condition warrants: methylprednisolone 30 mg/kg over 30 minutes followed by methylprednisolone 15 mg/kg 2 hours later; then the treatment is continued at 15 mg/kg every 6 hours for 24 to 48 hours. If visual function improves, corticosteroids are tapered. Patients who show deterioration or no response to corticosteroids may be offered optic canal decompression with discussion of potential risks of surgery against the possible benefits^{23,24}.

DISPLACEMENT OF THE GLOBE FOLLOWING INJURY [GLOBE DYSTOPIA]

The average orbital volume is 30ml. thus a small increase in volume into the orbit from oedema, blood or air can result in significant displacement of the globe. The direction of the displacement indicates the site of the pathology and CT imaging gives an anatomic correlation. Proptosis [exophthalmos] is often present immediately following orbital injury, secondary to bleeding, oedema and/or emphysema. This resolves with time. Persistent proptosis indicates either a large, slowly resorbing hematoma or bony fragments causing anterior displacement of globe²⁵.

Enophthalmos, or posterior placement of the eye, is usually a late complication because it can be masked by the oedema and hemorrhage from trauma. Bony expansion of the orbit, prolapse of orbital contents into the sinuses, and orbital fat atrophy all contribute to enophthalmos⁴³. Vertical displacement of the globe indicates pathology along the roof or the floor of the orbit. Fractures may result in bony fragments that displace the globe. Localized hematomas or air pockets can displace the globe in any direction, depending on their location. CT imaging is always warranted when globe dystopia is present and fractures are suspected.

CAROTID CAVERNOUS FISTULA.

Trauma can cause arterio-venous fistulas, which are abnormal communications between previously normal arteries and veins. The most common trauma responsible for these fistulas is basal skull trauma, leading to a communication between the internal carotid artery and the cavernous sinus. Examination discloses a red eye secondary to tortuous blood vessels on the eye surface and a bruit may be audible to the patient and the examiner. Pulsatile proptosis may also be present.

Diagnosis is made using arteriography of the orbit and cavernous sinus. Interventional radiologists can treat these by obstructing the artery with embolic materials, but this is difficult. Damage to the eye may result from ischemia and increased venous pressures²⁶.

DISCUSSION:

The goals of surgery for orbital floor fractures are to release entrapped tissues and correct enophthalmos and hypo Globus. When the inferior rectus is entrapped within a fracture, surgical release of the muscle is required on an urgent basis. Immediate intervention is recommended and has been shown to prevent muscle fibrosis and paresis-induced diplopia. Also, diplopia resolves more quickly with earlier intervention.

Large fractures involving more than half of the orbital floor are usually repaired as these fractures often lead to noticeable enophthalmos and hypo Globus. Three millimeters or more of enophthalmos is predictable and should be repaired. Severe fractures involving the entire floor may lead to herniation of the globe into the maxillary sinus, but this is a relatively rare complication^{26,27}. Fractures are repaired by releasing all entrapped orbital contents and periorbital and resurfacing the floor with an implant to support the orbital soft tissues. Various materials have been used, including autogenous bone and cartilage grafts and, more recently alloplastic materials²⁸.

Medial orbital wall fractures are associated with floor fractures 7% to 53% of the time. These commonly involved the ethmoid bone and may be a continuous extension of the fractured floor. Isolated medial orbital wall fractures are not common but they can be overlooked because of a lack of symptom²⁹. Signs of these fractures include epistaxis from a severed anterior ethmoidal artery, orbital emphysema, and motility disorders in horizontal gaze³⁰. Medial rectus entrapment and late enophthalmos are rare complications but should be ruled out in each case. Medial canthus displacement, lacrimal apparatus damage and cerebrospinal fluid rhinorrhoea may occur in more extensive fractures³¹. A rare but well organized complication of medial orbital wall fractures is incarceration of the medial rectus. A recent review of medial orbital wall fractures with muscle entrapment shows that both adduction and abduction are usually impaired and the adduction deficit is usually greater.

The majority of these cases had minimal external signs of soft tissue contusion and the motility exam was vital in making the diagnosis. Again, with periorbital trauma, a complete exam including extra-ocular motility is necessary. CT imaging is useful in the diagnosis and also shows the extent and location of the fracture³².

Treatment of medial orbital wall fractures is rarely indicated. Enophthalmos does not usually occur even with larger fractures and conservative management with prophylactic antibiotics and observation is recommended³³. Entrapment of the medial rectus is extremely uncommon but should be ruled out clinically with the motility exam and/or forced duction of the muscle and radio graphically by CT imaging³⁴. Incarcerated medial rectus muscles should be released as soon as possible. These surgical patients have minimal residual enophthalmos even without resurfacing of the medial wall and ocular motility improves greatly.

Orbital roof fractures are present about 5% of the time in patients with facial fractures¹²⁸. They are more common in young children and may involve the brain and cribriform plate because the frontal sinus is not yet pneumatized. In adults, the frontal sinus acts like a crumple zone and absorbs the trauma³⁵. The many complications of orbital roof fractures include intracranial injuries, pneumocephalus, cerebrospinal rhinorrhoea, orbital hematoma, globe displacement and ocular motility disorders. In severely comminuted fractures, displaced bone fragments may impinge upon the globe, superior rectus and levator complex. This can result in globe dystopia, motility restriction and/or ptosis and removal of the fragment may be necessary. However, roof fractures rarely need repair and treatment indications are usually neurosurgical.

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