Ophthalmologic considerations in maxillofacial trauma
Anatomy and diagnostic evaluation

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Abstract

The high incidence of orbital involvement in facial injuries necessitates a thorough ophthalmologic evaluation in virtually all cases. This article will provide the oral and maxillofacial surgeon with a review of the anatomy of the eye and ocular adnexa and present a systematic method of conducting and interpreting an ophthalmologic examination.

Introduction

The anatomical location of the orbits and globes predisposes them to injury in instances of maxillofacial trauma. Therefore, a thorough and systematic evaluation of these structures is mandatory in the assessment of most individuals who sustain midfacial injuries. This paper reviews the anatomy of the orbital region and recommends a systematic ophthalmologic evaluation.

Anatomy

Skeletal[1,2,3]

The anterior dimension of the bony orbit is approximately 35 mm high and 40 mm wide. The four walls of the orbital cavity are much thinner than the rim and they converge towards the optic foramen. The optic canal extends posteriorly from the foramen into the middle cranial fossa and is only 9 mm long.

The inferior orbital fissure communicates with the pterygomaxillary fossa.[2] The infraorbital groove and/or canal courses anteriorly in the maxilla along the center of the orbital floor and connects the infraorbital fissure with the infraorbital foramen. The infraorbital foramen is located approximately 6 mm below the inferior orbital rim. The superior orbital fissure is located in the lateral wall above the inferior orbital fissure and communicates with the middle cranial fossa (Fig. 1).

The medial wall of the bony orbit is thin and fractures easily, especially in its thinnest portion, the lamina papyracea of the ethmoid bone. The lacrimal fossa and the nasolacrimal canal are important landmarks in this area. The anterior and posterior ethmoid foramen are located posteriorly along the frontoethmoidal (Fig. 2).

Neurovascular[2,3]

The optic nerve (II) and ophthalmic artery enter the orbital cavity via the optic foramen. The superior orbital fissure transmits the motor innervation of the extraocular muscles (III, IV, VI), the sensory innervation from the skin and globe (ophthalmic and nasociliary of V) with the sympathetic and parasympathetic nerves to the iris, and the superior ophthalmic veins which drain into the cavernous sinus. The inferior orbital fissure provides access to the orbit from the pterygomaxillary fossa for the infraorbital and zygomatic nerves (V), infraorbital artery (internal maxillary artery), and occasionally a vein from the inferior ophthalmic veins which drain into the pterygoid plexus. The temporal and zygomatic branches of the facial nerve (VII) arise from the deep surface of the parotid gland and course superiorly over the zygomatic body of the lateral rim of the orbit to innervate some of the periorbital muscles. The periorbital lymphatics drain laterally.

Muscles and ligaments[4,5,6,7,8]

Voluntary muscles: The palpebral portion of the orbicularis oculi muscle functions in closure of the eyelids (Fig. 3). A small portion of this muscle is known as the pars lacrimalis (muscle of Horner or tensor tarsi) (Fig. 3). It arises from the posterior lacrimal crest and inserts in the eyelid margin. Innervation is via the temporal and zygomatic branches of the facial nerve (VII). Some authors suggest this muscle may facilitate the function of the lacrimal apparatus.

The levator palpebrae superioris muscle inserts into the upper eyelid between the fibers of the orbicularis oculi (Fig. 3). It produces the characteristic superior palpebral fold and is innervated by the oculomotor nerve (III). The posterior portion of the levator palpebrae constitutes an involuntary superior tarsal muscle (of Muller)(Fig. 3). It receives sympathetic innervation and also elevates the upper eyelid, but does not contribute to the superior palpebral fold. Therefore, the presence or absence of the superior palpebral fold enables one to localize the peripheral nerve injury when ptosis is present.

The four rectus and the superior oblique muscles arise from a common oval tendon, the annulus of Zinn, which surrounds the optic foramen and a portion of the superior orbital fissure (oculomotor foramen) and extends anteriorly to insert onto the globe near the limbus.[7] The superior oblique muscle extends anteriorly to the trochlea, located on the superomedial aspect of the orbital rim, and is redirected posteriorly and laterally to attach on the medial surface of the globe in the posterosuperior quadrant. The inferior oblique muscle does not arise from the apex, but rather has its origin on the orbital floor near the posteroinferior aspect of the nasolacrimal canal. It inserts over the lateral surface of the globe in the posteroinferior quadrant of the globe.

Four of the extraocular muscles are innervated by the oculomotor nerve (III). The lateral rectus muscle abducts the globe and is innervated by the abducens nerve (VI). The superior oblique muscle passes through a trochlea and is innervated by the trochlear nerve (IV).

A fascial ligamentous sling of Lockwood supports the globe and prevents vertical displacement and subsequent diplopia following orbital fractures in many instances.[6] This filamentous network of connective tissue runs from the posterior portion of the orbit within the periorbital fat and forms distinct fascial layers anteriorly. These strands of fascia attach to the cheek ligaments medially and laterally, the muscle sheaths, and Tenon's capsule, and form a hammock which suspends the globe when the ligamentous attachments to the orbital walls are preserved following a blow-out fracture.

Involuntary muscles: The palpebral, ciliary, and two iridial muscles are the primary involuntary muscles of the eye. The superior palpebral muscle (of Muller) has been discussed. The ciliary muscle attaches to the lens, receives its innervation from postganglionic fibers of the ciliary ganglion via parasympathetic outflow along the oculomotor nerve (III), and permits accommodation.

The iris is composed of two muscles, the circular sphincter pupillae and the radial dilator pupillae , which regulate the size of the pupils. Pupillary constriction results when a stimulus is induced along the afferent pathway of the parasympathetic system via the optic nerve (II), progresses through a hemidecussation at the optic chiasma, and enters the Edinger-Westphal nucleus in the midbrain. The efferent response to this stimulus arises in the nucleus and courses along the oculomotor nerve (III) to the ciliary ganglion and the short ciliary nerves to innervate the sphincter pupillae and constrict the pupil.[6]

The enlargement of the pupil results from a sympathetic stimulus that arises in the ciliospinal center of Budge and continues through the white rami at the first and second thoracic vertebrae to the superior cervical sympathetic ganglion in the carotid plexus. From the ganglion it travels with the ophthalmic (V), nasociliary (V), and long ciliary nerves (V), to the posterior surface of the globe where the fibers penetrate the sclera and run forward to the dilator pupillae muscle to dilate the pupil.[6]

Globe: The fibrous tunic covering the globe consists of the white sclera and the transparent cornea. The sclerocorneal junction is called the limbus. There is no blood supply to the cornea and it receives sensory innervation from the long ciliary nerves (V). Intraocularly, the anterior and posterior chambers are separated by the lens and ciliary apparatus. Aqueous humor is supplied to the anterior chamber via the ciliary process and drained by the canal of Schlemm. Interference in the resorption of aqueous humor results in increased intraocular pressure and a painful condition called glaucoma.

Lacrimal apparatus[9]

The lacrimal apparatus consists basically of a gland, canaliculi, sac, and nasolacrimal duct. The lacrimal duct lies in the lacrimal fossa in the superiolateral portion of the orbital cavity. Innervation to the gland is by the lacrimal nerve (V) and the parasympathetic fibers from the sphenopalatine ganglion. The tears accumulate in the medial lacrimal lake and are drained via small puncta on the eyelid margins. The action of the pars lacrimalis during the blink reflex

apparently provides the negative pump pressure which induces drainage of fluid from the lacrimal lake. The fluid passes through the canaliculi and valve of Rosenmuller into the sac, down the nasolacrimal duct, through the valve of Hasner, into the inferior nasal meatus. The valve of Hasner is actually a pseudovalve which prevents retrograde flow of fluid from the nose and subsequent inflammation of the lacrimal sac (dacrocystitis). Interruption in this drainage causes a persistent overflow of tears (epiphora).

This brief anatomical overview of the eyes and their adnexial structures illustrates the complex anatomy in this region. It is easy to realize the potential effects midfacial injuries may have on ocular structures. Clearly, a knowledge of basic anatomy in this area is essential to proper diagnosis and management of maxillofacial trauma-related ophthalmologic pathology.

Evaluation

History: Pertinent information from the patient or a companion regarding the nature of the injury, previous eye disease or corrective (contact) lenses is often valuable. Preexisting ocular pathology must be elicited to properly interpret the findings from the evaluation after injury. If corrective lenses were damaged, critical assessment of the possibility of intraocular glass must be made, since glass fragments in the eye are notoriously difficult to visualize.

Circumorbital: The nasal dorsum and intercanthal disease are inspected to determine the status of the medial canthal tendons. The integrity of the nasal bones and canthal tendons are determined by palpation. The intercanthal distance is measured. Orbital rims are palpated with firm digital pressure to access subcutaneous or bony crepitation. Cotton aplicators may be used to evert the lids and carefully probe lacerations (Fig. 4). Proptosis or enophthalmos are often difficult to access acutely when the patient has sustained periorbital trauma because of the initial edema. Possibly the conditions can be ascertained by noting the width of palpebral fissure, the position of the globe relative to the orbital rims, and the depth of the superior palpebral fold.

Visual acuity: A gross assessment of visual acuity can be made of each eye with newsprint, the examiner's fingers, or a flashlight. Greater accuracy can be achieved with a standard Snellen chart or Rosenbaum pocket card. In a well-lit room, the card is held 14 inches from the eye being tested and read with and without glasses. The reading is expressed as a fraction with the numerator indicating the distance at which the line of letters should be read by a normal eye.[10] Ordinary newsprint is approximately 20/70 and headlines are 20/400 at 14 in. Diplopia and decreased or blurred vision is noted. The presence of diplopia is assessed in all six cardinal positions of gaze similar to the evaluation of the visual fields.

Visual fields: The visual fields are assessed by directing the patient to cover one eye with the heel of his hand and fixate on the examiner's eye with the other. The examiner then directs the patient to count the number of fingers held up in the various quadrants. The procedure is repeated for the oposite eye.

Pupils: A great deal of information can be gained from thorough evaluation of the pupils.[11] The size and shape of the pupils are compared under indirect lighing. The light reflex is tested for both direct and concentric responses. The Marcus Gunn phenomenon will help to demonstrate an afferent pupil defect when the concentric response is questionable.[12] In this test, a weaker light is moved rapidly from the affected eye to the normal eye and back. Initial failure of constriction of the pupil in the affected eye followed by dilatation indicates the presence of a peripheral sensory defect on the affected side. A tremulous iris (iridodonesis) usually indicates lenticular displacement.

Extraocular movements: The movements of the eyes are assessed in the six positions of gaze for dysconjugate movement, ophthalmoplegia, strabismus, or nystagmus. When limited extraocular movement is evident, muscle entrapment can be differentiated from neurologic deficit by performing the forced duction test nder local anesthesia (Fig. 5). The inferior rectus muscle is grasped with a fine forceps and the globe is rotated superiorly and inferiorly. Mechanical entrapment of the inferior rectus or oblique muscles prevents rotation to the same extent as the unaffected globe. Forced duction must be done on both globes for adequate comparison. A test for strabismus is made by directing the patient to fixate on a penlight held 14 in in front of him. Each eye is covered independently and the contralateral eye is observed for compensatory movement.[10]

Conjunctiva: Eyelid retractors or cotton applicators are necessary to thoroughly examine the conjunctiva for lacerations, ecchymosis, or chemosis (Fig. 4). However, if one suspects a perforation of the globe, the maneuver is avoided to prevent extravasation of the intraocular contents. Tonometry is usually not performed in these instances because the intraocular pressure of a perforated globe is often variable and tonometry would not necessarily be diagnostic.

Cornea: The transparent cornea is difficult to evaluate by simple inspection. The integrity of the corneal reflex may be tested with a wisp of cotton. Once the possibility of an ocular perforation has been eliminated, a topical anesthetic (proparacain HCL 0.5%) may be applied to the cornea to facilitate the examination. Fluorescein dye may be helpful to visualize corneal abrasions. When a corneal laceration is suspected the presence of a foreign body on the cornea or within the globe must be strongly considered. Although foreign bodies are seldom removed, it is important that their presence be documented and that an ophthalmologist be consulted.

Fundus: The fundiscopic examination is often difficult in the patient with maxillofacial trauma. Nonetheless, this aspect of the evaluation is most important for both ophthalmic and neurologic reasons. The examination can be facilitated with certain pharmaceutical and manual assistance. Topical anesthesia will permit an assitant to retract swollen and tender eyelids and to wash the cornea with sterile ophthalmic solution. Photophobia can be minimized by utilizing the slit lamp lens on the ophthalmoscope. If the patient's neurologic status is satisfactory, a mydriatic (mydrioly 1%) may be instilled to permit better visualization of the intraocular chambers, lens, and retina for tissue damage or blood.

Radiographs: The routine radiographic evaluation for maxillofacial trauma should include cervical spine, skull, and facial series along with a Rhese (optic foramen view).[13]

Interpretation

Accurate diagnosis of ophthalmologic lesions or deficits is dependent upon a thorough examination and a knowledgeable interpretation of the findings. The clinical manifestations of various pathological ocular disorders which are commonly encountered in the patient who sustains facial trauma will be presented and followed with brief explanations.

Excessive intercanthal distance: An intercanthal distance greater than 35 mm in the adult is abnormal. Increased intercanthal distance with blunting of the medial canthal angles suggests detachment of the medial canthal tendon or a naso-orbito-ethmoid fracture.

Proptosis: Traumatic exophthalmos may follow intraorbital retrobulbar hemorrhage, a compressive wall fracture, cranial floor fractures with brain herniating into the orbit, or air in the orbit. It often manifests itself by a widened palpebral fissure and protrusion of the globe at least 3 mm beyond the intact inferior orbital rim. The intraocular pressure is also elevated in cases of traumatic exophthalmos. Proptosis may occur with superior orbital fissure syndrome due to loss of tone in the extraocular muscles.[14]

Enophthalmos: Recession of the eyeball into the orbit following orbital trauma is usually gradual due to a defect in the orbital floor and atrophy of periorbital fat. However, badly displaced malar bone fractures with a gross defect in the orbital floor may cause enophthalmos acutely. It presents with deepening of the superior palpebral fold, lowering of the pupil on the affected side with diplopia, and limited extraocular movement due to edema or muscle entrapment.

Ptosis: Drooping of the eyelid results from a variety of neurological deficits. A peripheral lesion of the facial nerve causes ipsilateral hemifacial paralysis and slight ptosis due to involvement of the orbicularis oculi muscle. However, unilateral central lesions do not cause this minor degreeof ptosis because there is a semi-decussation of fibers in the temporofacial branch of the facial nerve. Ptosis, miosis, and enophthalmos result from a sympathetic nerve deficit and are termed Horner's syndrome. These symptoms arise due to loss of tone in the superior palpebral msucle (of Muller) and the dilator pupillae muscle. The ptosis associated with the superior orbital fissure syndrome demonstrates no superior palpebral fold due to oculomotor nerve deficit and the loss of tone in the levator palpebrae muscle. Compression of the other structures transmitted through the superior orbital fissure results in ophthalmoplegia, ophthalmic paresthesia, and occasional mydriasis (Fig. 6).

Epiphora: A persistent overflow of tears following trauma to the orbit is generally expected because of eyelid swelling and poor drainage. It persists when there is damage to the lacrimal apparatus or eyelid eversion (ectropion) following a subciliary approach to the orbital floor.

Eyelid lacerations: Lacerations in the periorbital region should alert the clinician to a possible perforation of the globe. Inspection should enable one to determine whether the wound is superficial or deep to the tarsal plate. Injury to the tarsal plate increases the likelihood of injury to the overlying conjunctiva or perforation of the sclera. Special precautions are indicated under these circumstances and an ophthalmologist sgould be consulted. Lacerations on the medial aspect of the eyelid are potentially hazardous for the lacrimal apparatus. Lacerations, primarily vertical, on the lateral aspect of the eyelid will compromise lymphatic drainage and result in significant and prolonged edema of the eyelid.

Globe perforations: Perforations of the globe are often difficult to diagnose and when they are found they are considered an ophthalmologic emergency. The examination should cease when a perforation of the globe is suspected, the affected eye should be protected with a shield and sedation used when necessary to prevent a valsalva maneuver and extravasation of ocular contents. Damage to the choroid layer may initiate an autoimmune response following severe ocular trauma. Antibodies are produced which subsequently attack the unaffected eye (sympathetic) several days to years later. This phenomenon is called sympathetic ophthalmia and can lead to blindness in the unaffected eye if the injured globe is not enucleated.

Crepitus: Crepitus elicited by manual palpation of the orbital rims may be caused by subcutaneous emphysema or comminution of the underlying bone. Subcutaneous emphysema arises in the lower eyelid from the maxillary sinus and the upper eyelid from the ethmoid sinus.

Visual acuity deficits: The most common visual disturbance is blurred vision. It may arise from excessive lacrimation, corneal abrasion, foreign body, or swelling of the eyelids. Diplopia occurs with less frequency, but is of more diagnostic significance. Binocular diplopia commonly occurs when the globe is enophthalmic and Lockwood's suspensory ligaments are damaged, when cranial nerves III, IV, VI are injured, and when one or more extraocular muscles are entrapped within a fracture of an orbital wall. Monocular diplopia occurs rarely and only when there is subluxation of the lens or retinal detachment.

Sudden onset of blindness results from compression or complete transection of the optic nerve somewhere along the afferent pathway. Retrobulbar hemorrhage, severely perforated globe, or severe orbital and base of the skull fractures may cause blindness.

Visual field defect: Uniform narrowing of the visual fields is associated with degenerative neurological or psychoneurotic disorders. Specific areas of anopia (absence of sight) following trauma are often due to edematous eyelids or interruption in the sensory pathway.[15] Hemianopia is a bilateral defect in the visual field of each eye caused by an intracranial lesion (optic chiasm, tract, or brain). The hemidecussation of fibers in the chiasm results in the right brain serving the right halves of each retina and the left brain serving the left halves. Homonymous hemianopia occurs when the lesion is in the optic tract or brain and the same side of each visual field is affected. The pupillary reflex is also absent when the lesion is in the optic tract, but is intact when the brain is affected. Bitemporal hemianopia occurs with damage to the optic chiasma only, and the lateral (temporal) fields are intact.[10]

Afferent pupil defect: The appearance of direct and consensual pupil responses bilaterally indicate an intact neuropathway. Loss of the direct and consensual responses in one eye indicate that there is an ipsilateral lesion proximal to the optic chiasma (intracranial). Loss of the direct response in one eye and loss of the consensual response in the opposite eye indicates there is an ipsilateral lesion distal to the optic chiasma (extracranial).

Anisocoria: Normal pupillary diameter is approximately 2 mm under indirect lighting. When the pupils differ in size by 0.3 mm it is termed anisocoria.[16]Approximately 30% of normal subjects demonstrate a small degree of this phenomenon without underlying pathology. When the difference becomes 2 mm or greater, there is indication of a unilateral sympathetic (miosis) or parasympathetic (mydriasis) lesion and a neurosurgeon should be consulted.

Limited extraocular movements: Diplopia and strabismus are common complaints arising from limitations in extraocular movement. These limitations may result from contused or entrapped extraocular muscles or from compressed or transected extraocular nerves (III, IV, VI). Paralysis of two or more ocular muscles is termed ophthalmoplegia. Partial nilateral ophthalmoplegia occurs with a lesion of the oculomotor nerve (III) alone and total unilateral ophthalmoplegia results when all three nerves (III, IV, VI) are involved; i.e. superior orbital fissure syndrome, orbital apex syndrome, or cavernous sinus thrombosis.

The orbital apex syndrome appears like the superior orbital fissure syndrome with the addition of blindness. Bilateral ophthalmoplegia occurs only when a lesion is focused at the base of the brain. Conjugate movement with failure of lateral or vertical gaze occurs with a disturbance in the frontopontine pathway. There is no diplopia associated with these lesions.

Bell's phenomenon: (reflex exaluation of the globes with the lids closed) distinguishes a frontopontine lesion from the combined bilateral paralyses of the superior recti and inferior oblique muscles (III).[10] Persistence of the reflex is indicative of a supranuclear lesion.

Nystagmus: Irritation or destruction of the vestibular nerve (VIII) commonly produces nystagmus during fixation of the eyes. The nystagmus is described by the direction of the fast component. Horizontal nystagmus is the most common type, though vertical and rotatory forms exist. Nystagmus may be produced by injuries to the cerebral cortex, optic reflex pathways, or the cerebellum.[10] Slow nystagmus is less than 40 jerks per minute and fast is greater than 100 per minute. Jerks less than 1 mm are fine and greater than 3 mm are coarse.[10]

Subconjunctival hemorrhage: Scleral ecchymosis occurs after direct orbital trauma or orbital rim fracture. Blunt trauma to the globe produces a diffuse or blotchy hematoma. Orbital rim fractures often produce a triangular or flameshaped hematoma with the apex near the fracture site (Fig. 7).

Corneal defects: Corneal lacerations present with a flattened anterior chamber and decreased intraocular pressure, a foreign body, or perforated iris. Both lacerations and abrasions cause pain and blurred vision. Corneal abrasions are best visualized under fluorescin dye and

black light.

Hyphema: Damage to the ciliary body may cause hemorrhage into the anterior chamber. A hyphema may block drainage of aqeous humor and lead to galucoma. Reflex hyperemia may contribute to secondary hemorrhage and recurrent hyphema on the third to fifth day following the initial injury (Fig. 8).

Lenticular displacement: Slight displacement of the lens from its usual place behind the pupil is termed subluxation and iridodonesis occurs with horizontal movement. Fundiscopic examination reveals a dark curved line across the pupillary aperture (lens equator) and a double image on the retina.[10] Complete displacement is called dislocation.

Papilledema: A choked optic disc presents with swelling of the nerve head and obliteration of the margins as a result of increased intracranial pressure.[10] Symptoms vary from minor visual disturbances to total blindness.

Optic atrophy: Compression of the optic nerve presents with pallor of the nerve head due to decreased vascularity.[10] The symptoms present as loss of visual acuity and changes in the visual field.

Commotia: Periorbital trauma and contusion of the globe occasionally results in generalized edema. This edema causes a uniform pallor of the retina and decreased visual acuity, but does not lead to blindness.

Retinal detachment: The accumulation of fluid between the neural layer and the pigmented epithelial layer of the retina seldom follows trauma. However, once it does occur, progressive blindness will result unless treated promptly.

Radiographs: Skull and facial radiographs should be viewed in a systematic manner. Identification of each structure best visualized on that particular radiograph will prevent oversight. The lateral view demonstrates fractures of the frontal bone and orbital roof. The posteroanterior view demonstrates the orbital rims and naso-orbito-ethmoid complex. The Water's view demonstrates fractures of the orbital floor resulting from zygomatic complex fractures. Blow-out fractures of the orbital floor are best visualized on Caldwell or Rhese tomography (Fig. 9). Remember, identify each normal landmark on every radiograph and the abnormalities will most often be obvious.

Summary

The ophthalmologic considerations in maxillofacial trauma are numerous. Therefore it is imperative that patients with midfacial injuries are approached in a knowledgeable and systematic manner. This article approaches the topic with a review of orbital anatomy and presents a diagnostic evaluation in preparation for the proper management of orbital injuries. Whenever the examination reveals a significant alteration in visual acuity, penetrating wounds to the eyes or

eyelids, corneal lacerations and/or intraocular foreign bodies, hyphema, optic atrophymor retinalk detachment, an ophthalmologist should be consulted. A neurological surgeon should be consulted whenever significant anisocoria, papilledema, or an unreactive pupil are discovered. The diagnosis of these important and other relevant pathologic ocular disorders discussed herein are necessary before the surgical repair of facial lacerations or fractures.

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