Ocular Injuries Associated with Midface Fractures: A 5 Year Survey

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Abstract

Aim The aim of this study is retrospective analysis of ocular injuries after maxillofacial trauma reporting to the Department of Oral and Maxillofacial Surgery at V.S. Dental College and Kempegowda Institute of Medical Sciences and Hospital, Bangalore.

Study design A retrospective analysis of ocular injuries following facial trauma from 2008 to 2013.

Results Road traffic incident was the most common etiology (55.46 %). Zygomaticomaxillary complex fracture was the most common fracture associated with ocular injuries (67.22 %). Out of 119 patients, 5 had severe visual impairment and (0.84 %) had blindness.

Conclusion Maxillofacial trauma particularly that associated with Lefort II, Lefort III and Zygomaticomaxillary complex fracture may lead to ophthalmic injuries and even blindness. A thorough ophthalmic examination should be carried out for every patient with maxillofacial trauma and suspected cases should be placed under closed observation so that immediate and active treatment can be taken if necessary.

Keywords Zygomaticomaxillary complex fracture · Lefort II · Lefort III · Ocular injuries · Blindness

Introduction

Worldwide an estimated 1.6 million people are blind as a result of eye injuries and further 19 million have monocular blindness or low vision due to eye trauma [1]. Eye injuries in association with maxillofacial trauma are particularly important as these injuries have high risk of threatening vision [2]. Blindness in patients with maxillofacial trauma is usually caused by optic nerve or optic canal injuries. It is, however an uncommon complication of facial trauma, with a reported incidence of 0.32–9 % [3, 4]. Hippocrates was the first person to record the association between facial trauma and blindness [5]. Midface fractures like Zygomaticomaxillary complex, Lefort II, Lefort III and Naso orbital ethmoid are mainly associated with ocular injuries. By definition, Zygomaticomaxillary complex fractures are the most common fractures associated with orbital involvement [6].

According to Lefort, the face resists the force mainly because of the face's elasticity, its periosteum, and its soft tissues [7–9]. Direct injury to the globe of the eye is relatively rare as compared to the frequency of orbital trauma [10]. This is explained by the fact that a number of factors protect the globe from injury, including the prominence of the bones of the orbit and the natural reflexes of self-protection such as blinking, protecting the eye with the hand, and averting the head [11]. In addition, the resilient structure of the globe allows it to withstand blows of considerable force without rupture. Damage to the optic nerve is also uncommon because of the presence of a dense bony ring that protects the nerve as it enters the orbit [12, 13].

Blindness may also occur after surgical repair of midface fracture. A thorough ophthalmic examination should be done before reduction and fixation of midface fractures.

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Materials and Methods

A 5 year retrospective study from 2008 to 2013 was conducted on patients with maxillofacial injuries admitted under oral and maxillofacial surgery unit. The study involved a total of 119 patients with 103 males and 16 females who had sustained fractures of the middle third and the upper third of the face with orbital involvement.

In all these cases, ophthalmic examination was done at the Department of Ophthalmology, Kempegowda Institute of Medical Sciences and Hospital. Ophthalmologic review chart and information was recorded and analyzed for the relationship between maxillofacial fracture, ophthalmologic injury and blindness.

Analysis

See Tables 1, 2 and 3.

Table 1 Age distribution of patients

Age (years)	No. of patients
11–20	4
21–30	37
31–40	28
41–50	26
51–60	18
>60	6

Table 2 Etiology of maxillofacial fractures

Etiology	No. of patients	Percentage
Road traffic accident	66	55.46
Self fall	28	23.52
Assault	17	14.28
Work related injuries	6	5.04
Sports injury	2	1.68

Table 3 Fractures associated with ocular injuries

Fracture site	No. of patients	Percentage
Zygomatic complex fracture	80	67.22
Lefort III	8	6.72
Lefort II	10	8.40
Naso orbital ethmoid fracture	2	1.68
Panfacial trauma	19	15.96

Result

The average age group of the patients was between second to fourth decades of life. Thirty-seven patients were between 21 and 30 years while 54 patients were between 31 and 50 years. The most common cause of injury was road traffic accident (67.22 %), followed by self fall (23.52 %). Fracture sites associated with ocular injuries mainly involved Zygomaticomaxillary complex fracture (67.22 %), panfacial trauma (15.96 %), Lefort II fracture (8.40 %), Lefort III fracture (6.72 %) and naso orbital ethmoid fracture (1.68 %). Computerized tomography, paranasal sinus view and submentovertex view were used to confirm the clinical diagnosis. Most of the patients were treated with open reduction and internal fixation. There were 107 patients who had periorbital edema, 89 patients had subconjunctival ecchymosis, chemosis was seen in 27 patients, 11 patients had diplopia, retrobulbar hemorrhage was detected in 2 patients leading to optic nerve compression, 2 patients had traumatic mydriasis while retinal detachment leading to blindness was seen in 1 patient (0.84 %). Rest all distribution of the clinical findings are mentioned in (Table 4). All the recorded data including both extraocular and intraocular injuries was based on preoperative and postoperative clinical examination done under the supervision of staff of oral and maxillofacial surgery and ophthalmology.

Discussion

There are several differences among international series regarding the epidemiology and presentation of maxillofacial fractures. Selection of patients and treatment often differ between studies; therefore, any comparison should be made with caution. Classically, the main causes of facial fractures are road traffic accidents, followed by assault, self fall, and sports injury [1]. However, more recent studies have identified assaults as the main cause [2, 5]. According to our study, the most frequent etiologic factor was road traffic accident (66.72 %) followed by accidental fall and assault.

As it is evident from our data that 0.84 % of trauma cases lost vision; this is quite high when the importance of an organ is taken into consideration. Careful ocular examination should be performed in all the patients with midfacial trauma. Examination should include a record of perception to light, color, form, visual acuity, pupillary reactions, eye movements, fundus examination to detect intrabulbar hemorrhage, retinal edema, detachment and optic nerve compression.

Direct trauma may result in bruising of the eyelids leading to the classical “black eye”. Tissue laxity permits free tracking of the blood through the loose subcutaneous tissue which commonly remains extravasated for several weeks. No treatment is necessary for the same. However,

Table 4 Associated ocular injuries

Type of lesion	Clinical presentations (signs and symptoms)	No. of patients
Extraocular	Periorbital edema	107
	Subconjunctival ecchymosis	89
	Chemosis	27
	Ptosis	11
	Restriction of extra ocular movements	17
	Extraocular muscle paresis	12
	Telecanthus	6
	Enophthalmos	11
	Exophthalmos	6
	Infra orbital nerve paresthesia	66
	Transient diplopia	11
	Optic nerve compression	2
Intraocular	Retrobulbar hemorrhage	2
	Retinal detachment	1
	Traumatic mydriasis	2
	Blindness	1

further inspection is necessary to exclude a concomitant injury to the globe. Subconjunctival ecchymosis frequently described as pathognomonic of fracture involving one or more orbital walls, with a resultant hemorrhage which tracks forward external to muscle cone and under the conjunctiva as far as the margin of avascular cornea. This ecchymosis remains bright red in color due to ability of the atmospheric oxygen to diffuse through the conjunctiva and prevent reduction of haemoglobin in effused blood. Non-penetrating injury to the conjunctiva may lead to conjunctival edema (chemosis) which usually resolves spontaneously.

Ptosis following trauma may be mechanical or neural in origin. Edema of the upper eyelid produces a temporary mechanical ptosis. Damage to the muscular (levator palpebrae superioris) and facial surface of upper eyelid should be identified and repaired primarily. Ptosis secondary to traumatic nerve paresis (oculomotor) usually resolves slowly.

Extraocular movements are evaluated to rule out any mechanical entrapment or paresis of extraocular muscle leading to restricted movement of eye ball. Computed tomography scan finding should be correlated with any clinically noted entrapment. If mechanical entrapment is suspected, then eye should be topically anesthetized and forced duction test should be performed. The point of doing forced duction test is to determine whether diplopia is due to restricted motion from entrapment, scarring or fibrotic contracture or a neurogenic motility disorder (Cranial nerve III, IV, VI).

Diplopia is a disabling problem, especially when it occurs close to primary position or downward gaze. Different authors have reported an overall frequency between 5 and 37 % [14], but diplopia as a persisting symptom has been

reported in 5–7 % of the patients [15, 16]. It can be either monocular or binocular. Monocular diplopia is usually due to lens opacification or displacement or other disturbances in clear media along visual axis. Acute binocular diplopia, secondary to trauma, derives from one of these basic mechanisms; edema or hematoma followed by restricted mobility due to muscle entrapment, neurogenic injury, loss of orbital contents (fat), and displacement of attachment of suspensory ligament of globe.

Restricted mobility or entrapment is commonly found with orbital floor and medial wall fracture, less frequently with roof fracture and rarely with lateral wall fractures. Diplopia in the primary and downward gaze usually resolves along with the edema in 7–10 days. Slight diplopia in extreme peripheral fields of gaze persisting for a month is rarely problematic. Persistent post traumatic diplopia is best evaluated by an ophthalmologist.

Every effort should be made to record the size and reaction of pupil to illumination using direct and consensual light reflex. Ipsilateral oculomotor nerve lesion will reveal fixed dilated pupil on affected side under direct light source while consensual light reflex is preserved. On contralateral stimulation, in case of optic nerve lesion, reveals fixed dilated pupil under direct reflex while consensual reflex is preserved because parasympathetic pathway is intact. This is important for the maxillofacial surgeons because fractures of facial skeleton may occasionally affect the optic foramen and damage the optic nerve.

The eyes should be assessed for the presence of position of eye ball post trauma. Incidence of enophthalmos and exophthalmos is mainly attributed to the displacement of the zygoma. Other authors have reported an incidence of either enophthalmos or exophthalmos depending on the pattern of

displacement of fractured zygomatic bone in 2–7 % of the patients [17]. The post traumatic enophthalmos is due to orbital volume expansion which has been repeatedly confirmed using CT imaging [18, 19]. Inferior and posterior displacement of zygoma produces varying degree of disorganization of the soft tissue of orbital cavity with bony expansion causing enophthalmos. Whitehouse et al. emphasized on the enophthalmos, which was less marked than would be predicted from orbital expansion when measured within first 20 days after injury (due to hemorrhage and edema) but after this time, resolution of edema and hemorrhage permitted a good correlation between orbital volume expansion and the degree of enophthalmos, each 1 cm [5] of increase in orbital volume corresponding to 0.8 mm enophthalmos. On other side, medial dislocation of zygoma may compress the volume of the orbit and produce exophthalmos [19]. Thus a wide variety of ocular displacement and visual symptoms accompany zygomatic fracture.

Traumatic telecanthus is seen when there is increase in the inter-canthal distance. The normal range for inter-canthal distance is 25.5–37.5 mm in women and 26.5–38.7 mm in men. Disruption to the attachment of medial canthal ligament is the most common etiology following naso orbital ethmoid fractures. The medial canthal ligament has an anterior limb that attaches to anterior lacrimal crest and a posterior limb lying posterior to lacrimal sac. Disruption of anterior limb alone does not produce telecanthus. Telecanthus is a sign of disruption of both the anterior and posterior limbs of the ligament.

Blindness following maxillofacial trauma has less incidence. Nevertheless, it cannot be neglected. As it is evident from our studies that 0.84 % of trauma cases lost vision; this is quite high when the importance of an organ is taken into consideration. Blindness is mostly due to optic nerve compression, retrobulbar hemorrhage, increased intraocular pressure which leads to optic nerve atrophy. Immediate, complete and permanent blindness suggests compression from displaced bone whereas delayed progressive blindness suggests compression of the nerve by hemorrhage or interstitial edema.

Retrobulbar hemorrhage may rarely (<1 %) occur as a consequence of midfacial trauma. It is characterized by an injury within the confines of the extraocular muscle (intraconal space) that ruptures one or more of the posterior ciliary arteries. Although the intraconal space communicates with extraconal space through a small poster lateral channel, the former essentially acts as a closed compartment. This eventually leads to increased venous congestion and edema around optic nerve and finally obstruction of central retinal artery. Treatment approaches include:

1. Surgical decompression through lateral canthotomy to release the intra ocular pressure.
2. Reduction of intraocular pressure using combination of intravenous mannitol (200 ml, 20 %) and acetazolamide 500 mg. Intraorbital edema and circulatory spasm can also be minimized by using intravenous steroid (hydrocortisone, 100 mg) or dexamethasone sodium phosphate 3–4 mg/kg as a bolus and then gradually tapering the dose.
3. Since it is impossible to assess the pupillary response accurately during general anesthesia, some experts advocate that decompression of the orbit be supplemented by the retrograde injection of spasmolytic agent such as papaverine (40 mg bolus) through a cannula introduced into the supraorbital artery [14]. Patients who initially have vision after an injury but subsequently lose it should be given active treatment because they have a greater chance of regaining vision [20]. Patients who lose their sight immediately have some degree of permanent visual impairment regardless of treatment, but treatment should be instigated immediately [20].

Traumatic mydriasis after blunt trauma may persist, often affecting young adults causing glare, blurred vision, and poor cosmesis. In case of failure of recovery, treatment with a muscarinic agent such as pilocarpine drops (1 % four times daily), is available. However it causes brow ache, miosis, and decreased accommodative amplitude. Thymoxamine drops (0.5 %), a competitive α antagonist, causes pupillary constriction. It affects accommodation less in comparison to 1 % pilocarpine and needs to be administered twice daily.

Conclusion

Maxillofacial trauma particularly those associated with Lefort II, Lefort III and Zygomaticomaxillary complex fracture may lead to ophthalmic injuries and even blindness. A thorough ophthalmic examination should be carried out for every patient with maxillofacial trauma and suspected cases should be placed under close observation so that immediate and active treatment can be taken if necessary. Internal fixation after a correct reduction is the preferred treatment option, but any attempt to reduce the fracture associated with possibility of vision loss should not be performed.

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