Blindness After Facial Fractures: A 19-Year Retrospective Study

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Purpose: We conducted a 19-year review of patients with facial fractures who were treated in the Iranian Maxillofacial Unit at the Mobasher Emergency Hospital, Hamedan Province, Iran, to specifically consider those fractures that resulted in blindness or severe visual impairment.

Materials and Methods: During the period of February 16, 1984, to March 20, 2003, a total of 2,503 patients with facial fractures were operatively treated. Of these, 550 (22%) patients had orbital region fractures and were specifically studied.

Results: From our facial fractures database, 83 (3.31%) patients were identified as having ocular or extraocular injuries. Of these, 39 patients (1.56%) had severe visual impairment or blindness.

Conclusions: Laterally directed forces are implied as major causative factors in blindness or visual impairment. Males (83.3%), left eye (63.3%), third and fourth age decades (53.3%), and motor vehicle accidents (63.3%) were the most commonly involved gender, site, age, and cause of monocular blindness, respectively.

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Hippocrates was the first person to record the association between facial trauma and blindness (Latinate from Greek: *amaurosis*, or "darkening," and *amblyopia*, of "sluggish eye, blindness").[1] However, it was nearly 2,300 years before anyone scientifically examined the relationship between trauma and the optic nerve. In 1879, Berlin[2] noted that trauma could lead to fracture of the optic canal. 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% to 9%.[3-5] Blindness may also follow surgical repair of facial fractures. Many mechanisms, such as intraoperative direct nerve injury, retinal arteriolar occlusion associated with orbital edema, or delayed presentation of indirect optic nerve injury sustained at the time of the initial trauma, have been implicated in causing this blindness. Postoperative ophthalmic complications seem to be primarily mediated by indirect injury to the optic nerve and its surrounding structures. The most frequent cause of postoperative visual loss is an increase in intraorbital pressure in the optic canal. In addition, blindness may be attributable to intraorbital hemorrhage or unspecified mechanisms of increased intraorbital pressure within the restricted confines of the optic canal. Even small changes in pressure potentially may cause ischemic optic nerve injury.[3]

According to Le Fort,[6-8] the face resists the force mainly because of the face's elasticity, its periosteum, and its soft tissues. Direct injury to the globe of the eye is relatively rare compared with the frequency of orbital trauma.[9] 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.[10] 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.[11,12] There is no such protection for the long and short posterior ciliary arteries that lie within the muscle cone and enter the eye around the optic nerve. Therefore, a bleed into the cone could compress these vessels and produce anterior ischemic neuropathy, which is the most likely mechanism of visual loss in these cases.[7]

Several authors have reported sporadic cases of blindness in association with facial trauma.[3,11,13-18] The incidence of loss of vision in at least one eye varies depending on the method of initial examination, the fractures considered in each study, the extent of investigation (retrospective, prospective) and

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the reporting institution.[4,19] Using objective examination of the pupil, some have found visual impairment to be as high as 15%.[20]

Fujino and Makino[21] and Smith and Regan[22] reported extensively on the mechanism of blowout fractures. The soft tissues are compressed by a force into the orbital cavity and rupture the thin (0.5- to 1-mm thickness) orbital floor,[22] where the ensuing posttraumatic edema leads to an increased hydraulic pressure that further increases the prolapse and entrapment of the orbital contents into the sinus.[21]

Pure blowout fracture of the orbit complicated by retrobulbar hemorrhage or emerging proptosis and pain is an emergency occurrence and warrants special attention.[23-25]

Many reports concern the diagnosis and treatment of facial fractures, but they seldom describe the simultaneous ocular injuries in a systematic fashion, although the latter constitutes the area of greatest functional loss and greatest concern to the patient.[9,23-26] In the past decade, only one report appeared in the literature regarding ocular injuries in Iran. Taher[27] reviewed retrospectively the cases of 367 patients from a 7-year period (1984 to 1990) and found that blindness or visual impairment was most frequently caused by gunshot injuries (54%) as a consequence of Iran-Iraq conflict, followed by road traffic accidents (40%). More than 95% of severe ocular injuries were associated with fractures of the facial middle third, and the remaining were complications of upper third injuries. The purposes of this retrospective analysis were to assess visual impairment (9 cases) and/or blindness (30 cases) and to disclose the relationship between the type of ocular injury and type of facial fracture. A discussion of the pathophysiology of this problem and management precautions are included.

Materials and Methods

This retrospective chart review was performed at the Mobasher Emergency Hospital, an urban trauma center, in Hamedan Province, Iran, which treats a population of approximately 1.925 million. During the 19-year period between February 16, 1984, and March 20, 2003, 3,180 patients with maxillofacial injuries were admitted to the Oral and Maxillofacial Surgery Service. From this group, 2,503 patients had sustained maxillofacial fractures. The staff of our unit examined these patients. In cases of zygomaxillary complex (ZMC) and Le Fort II and Le Fort III fractures and in other cases as necessary, an ophthalmologic examination was requested. Charts were reviewed for age and gender of the patient, cause of injury, type of facial fracture, type of ocular trauma, and cause of blindness. The anatomic location of mandible fractures was classified according to the system described by Ivy and Curtis.[28] The anatomic location of zygoma fractures was classified according to the system described by Knight and North.[29] The anatomic location of maxillary fractures was classified according to the system described by Le Fort.[6-8] The ophthalmologic injuries were classified according to the system described by Dutton and Al-Qurainy.[10] The charts were reviewed, information was recorded on a flow sheet, and the information was analyzed for the relationships between facial fractures, ophthalmologic injury, and blindness. There were 1,987 men and 516 women, and injuries included 1,298 mandibular fractures, 75 fractures of the ZMC, and 260 Le Fort–type fractures.

Table 1. ASSOCIATED INJURIES OF EYE AND/OR ADNEXAE

Types of Lesions	Type of Injury	No.
Extraocular lesions sustained		
Muscle/periorbital soft tissues	Entrapment	6
Position of eyeball	Enophthalmos	8
	Exophthalmos	1
	Medial displacement	1
Cranial nerve lesions	Oculomotor nerve	3
	Abducent nerve	3
Medial canthal ligament	Telecanthus	14
Lacrimal apparatus	Damage	4
Intraocular lesions sustained		
Loss of vision	Optic nerve lesion	19
	Eyeball destruction	11
Anterior structures	Corneal laceration	2
	Perforation of eyeball	2
Posterior structures	Severe retinal edema	7
	Macular hematoma	1
	Retinal detachment	1
Total		83

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Results

Thirty cases of blindness were recorded, and 9 patients were diagnosed with severe visual impairment (Table 1). The majority (19 cases) had been involved in road traffic accidents. Sixteen patients were between 20 and 39 years old (third and fourth age decades); 25 were men and 5 were women (Table 2). There were 9 fractures of the zygo-orbital complex, 5 Le Fort III fractures, and 1 Le Fort II fracture. Other fractures were zygoma (n = 14) and nasoethmoidofrontal (n = 1) fractures (Table 3). Other associated injuries were 5 mandibular and 1 skull fracture. The floor of the orbit was fractured in 8 cases in association with other fractures. In all cases,