Blindness Following Facial Fracture: Treatment Modalities and Outcomes

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ABSTRACT

Blindness is an uncommon, yet documented complication of facial trauma. Numerous case studies, series, and retrospective analyses have been published, with a reported incidence around 3%. Hippocrates first noted the association between maxillofacial trauma and blindness; millennia later, this was expounded upon by Berlin, who discovered such trauma may directly lead to fracturing of the optic canal. As diagnostic modalities such as computed tomographic scanning evolved, particularly over the past few decades, more specific, in-depth reports analyzing maxillofacial trauma and subsequent sequelae have emerged. It is the goal of this article to examine the current literature for those publications that have addressed the issue of blindness following facial trauma (including operative interventions) and create a concise review for maxillofacial surgeons.

KEYWORDS: Maxillofacial trauma, facial fracture, blindness, traumatic optic neuropathy, optic nerve injury

Blindness is an uncommon, yet documented complication of facial trauma. Numerous case studies, series, and retrospective analyses have been published, with a reported incidence around 3%. Hippocrates first noted the association between maxillofacial trauma and blindness; millennia later, this was expounded upon by Berlin, who discovered such trauma may directly lead to fracturing of the optic canal.[1] As diagnostic modalities such as computed tomographic scanning evolved, particularly over the past few decades, more specific, in-depth reports analyzing maxillofacial trauma and subsequent sequelae have emerged. It is the goal of this article to examine the current literature for those publications that have addressed the issue of blindness following facial trauma (including operative interventions) and create a concise review for maxillofacial surgeons.

MATERIALS AND METHODS

The current published literature was extensively reviewed for works analyzing blindness subsequent to maxillofacial trauma or surgery. A PubMed search was conducted covering the years 1966 to 2008. Search terms included "maxillofacial trauma," "facial fracture," "blindness," "traumatic optic neuropathy," "steroids," "optic canal decompression," and "observation." Twenty-seven articles were identified that met our selection criteria. The bibliography of each article was cross-referenced and unique articles also pulled for review. These articles were collected and key points analyzed, including the fractures described, the mechanisms for blindness in such cases, operative and nonoperative interventions, and ultimate outcomes. Because a large proportion of the current literature regarding this subject was in the form of isolated case reports and case series, specific

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percentages for particular components of fracture patterns and associated blindness were not enumerated. There are a handful of thorough, eloquent, retrospective analyses of ocular complications of maxillofacial trauma, from which specific quantitative figures have been assembled. All research in this area can be categorized as level III using the U.S. Preventive Services Task Force system for ranking evidence about the effectiveness of treatments. Table 1 represents a comprehensive review of case reports, series, and studies that have addressed blindness secondary to maxillofacial trauma or maxillofacial surgery. Table 2 outlines the various treatment modalities and the actual visual acuity. Blanks in each table indicate where data were absent in each series report.

INCIDENCE OF BLINDNESS

One of the earliest analysis of maxillofacial trauma outcomes was published in the *Journal of Trauma* by Cruse et al in 1980.[2] In this article, the authors reported on 33 patients noted to have naso-orbito-ethmoid fractures, 10 of whom were found to suffer severe ocular injury with initial or subsequent loss of sight. Three years later, Holt et al published one of the largest such series to date, which analyzed 727 facial fractures with a blindness incidence of ~3%, laying the groundwork for today's incidence reporting.[3]

Many groups have confirmed these numbers. Al-Qurainy et al describe 363 patients with a total of 438 midfacial fractures with loss of vision of 2.2% when analyzed per patient.[4] Similar figures were described by Kallela et al in 1994. Here, retrospective analysis of 614 patients presenting with midfacial fractures (four as isolated zygomatic fractures and six with LeFort fractures) revealed 10 patients with a total of 14 blind eyes: a blindness rate of 2.3%.[5] Higher rates of blindness secondary to traumatic midfacial fractures have been published: Ashar et al noted 22% of midfacial fractures involving the orbit resulted in permanent blindness.[6] This high incidence seems to be isolated. Even when focused attention is paid to orbital fractures, blindness rates of ~2.9% are reoprted.[7]

In more recent years, several very large retrospective analyses have been completed. MacKinnon et al described in eloquent detail a retrospective review of a total of 2516 patients with facial fractures that required operative intervention. Of these patients, 19 were noted to have severe visual impairment or blindness, largely secondary to laterally directed forces of impact.[8] Interestingly, MacKinnon et al demonstrated that the lateral orbital wall along with the zygomaticomaxillary buttress were the most commonly fractured areas in the patients with significant ocular sequelae. These substantiate similar reports in the literature.[9,10] It should be noted that MacKinnon et al's blindness rate of 0.8% of patients sustaining facial fractures is significantly lower than previously published reports.

This reduced rate of blindness has also been documented in other very large series, including Zachariades et al in their review of 5936 facial trauma patients, in which 19 cases of blindness occurred, yielding a rate of 0.32%.[11] The authors found vision loss was most commonly seen in Le Fort III level fractures.

A retrospective chart review undertaken by Ansari in 2005 revealed 30 cases of blindness following facial trauma and subsequent facial fractures requiring operative intervention in 2503 patients.[12] Their analysis revealed the vast majority of cases were secondary to zygoma or zygomaxillary complex fractures with retrobulbar hemorrhage or severe damage to the eye as a whole being present in most.

MECHANISM OF INJURY LEADING TO BLINDNESS

Numerous case reports and case series have added to our understanding of this phenomenon in the setting of maxillofacial trauma. These span a range of mechanisms, clinical courses, and ultimate outcomes ranging from traumatic blindness following a displaced lateral orbital wall fracture to traumatic retrobulbar hemorrhages and malar fractures.[13–16] Other interesting yet rare complications have been published in the form of transient complete blindness following nose blowing after an orbital floor fracture and orbital cellulitis secondary to fracture with subsequent subperiosteal abscess formation and permanent blindness.[17–19]

The facial skeleton is designed to withstand large force loads, with directed energy resistance in the form of elasticity, surrounding periosteum, and affect of soft tissues. Rene LeFort[20] originally described the common fracture patterns associated with maxillofacial trauma and laid the groundwork for our understanding of facial trauma patterns. When one directs attention specifically to the globe and ocular periorbit, other factors protecting the globe include the prominence of the orbital bones themselves, as well as natural reflexes such as blinking and head aversion.[21] Cushioning of the contents of the orbit in the form of orbital fat and the extraocular muscles also protect the ocular mechanism from injury secondary to blunt external forces.

Injury to the optic nerve itself is the most common cause of blindness following traumatic fracture. It should be noted, however, that, secondary to a bony ring and relative laxity of the optic nerve within the optic canal, the optic nerve itself is not commonly injured during traumatic facial fracture. The transmitted force from frontal impact through the orbital wall and apex deforms the optic canal, leading to secondary ischemic necrosis from damage to the vasonervorum.[22] In addition to the mechanism of compression, shearing forces

Table 1 Fracture Patterns and Their Associated Ocular Injuries

Reference	Fracture Pattern	Ocular Injury
2	Naso-orbita-ethmoid fracture (10)	Optic nerve trauma (5) Globe perforation (5)
3	Midface fractures (12) Frontal fracture (3)	Optic nerve injury Retinal detachment Corneal-scleral rupture (no numbers given)
4	''Severe malar comminution'' (number not supplied)	Traumatic optic neuropathy (8)
5	LeFort II/III level fracture (6) Zygoma fracture (10 patients, 14 blind eyes)	Optic nerve defect (9) Unknown (2) Hyphema (1) Vitreous hemorrhage (1) Perforation (1)
7	Zygoma fracture (10) Nasoethmoidofrontal fracture (8) LeFort III level fracture (4) Orbital blowout (2) LeFort II level fracture (2) (25 fractures for 15 patients)	
8	Orbitozygoma fracture (4) Naso-orbital-ethmoid fracture (2) LeFort III level fracture (2) Zygoma fracture (1) Bilateral condyle/mandible fracture (2) Frontal fracture (2) Supraorbital bar fracture (1) Fronto-orbital-ethmoid fracture (1) Nasomaxillary fracture (1) Orbital rim fracture (1) LeFort level II fracture (1) Orbital floor fracture (1)	Traumatic optic neuropathy (7) Ruptured globe (5) Enucleation (2) Macular hole (1) No diagnosis (1) Penetrating eye injury (1) Retro-orbital abscess (1) Optic nerve infarct (1) Vitreoretinal hemorrhage (1) Optic nerve contusion (1)
11	ZMC fracture (8) LeFort III level fracture (8) LeFort II level fracture (2) Bilateral ZMC fracture (1)	Retrobulbar hemorrhage (10) Complete eye damage (5) Optic nerve laceration (2) Optic nerve compression (1) Postoperative (1)
12	Zygoma fracture (14) ZMC fracture (9) LeFort III level fracture (5) LeFort II level fracture (1) Nasoethmoid-frontal fracture (1)	Retrobulbar hemorrhage (14) Complete eye damage (11) Laceration of optic nerve (3) Optic nerve compression (2)
13	Displaced lateral wall fracture	Lateral rectus transaction Direct optic nerve compression
14	Orbital floor fracture	Retrobulbar hemorrhage
17	Zygoma/orbital floor fracture	Retrobulbar emphysema
18	Naso-orbital with subperiosteal	Ischemic optic neuropathy
24	LeFort I/BSSO/genioplasty LeFort I with advancement	Unknown Bony fragment displacement into optic canal with pterygoid comminution
25	LeFort I with advancement Abscess	Sphenoid discontinuity

Table 1 (*Continued*)

Reference	Fracture Pattern	Ocular Injury
26	LeFort I osteotomy	Maxillary sinus roof bony impingement on optic nerve
	LeFort I osteotomy with genioplasty	
		Retrobulbar optic nerve lesion
27	Displaced ZMC fracture s/p Gilles	Anterior ethmoidal artery hemorrhage/ hematoma
15	Lateral orbital wall, malar fracture, medial displacement of greater sphenoid wing	Optic nerve compression
40	Nondisplaced zygomatic, lateral orbital wall, frontal sinus wall fracture	Bony impingement on optic nerve
19	Minimally displaced ZMC fracture	Subperiosteal abscess
6	NOE/frontal/zygoma fracture (3)	Traumatic optic nerve injury (7)
	Zygoma fracture (2)	Ruptured globe (3)
	NOE/zygoma/LeFort II/III (2)	
	LeFort II/III/zygoma fracture (1)	
	NOE/LeFort II/III (1)	
	LeFort II/III (1)	
28	Blunt facial trauma (40)	Orbital fracture (53)
	Penetrating facial trauma (21)	No fracture (8)
29	Frontal head trauma with monocular blindness (7)	Neurogenic vision loss
30		Traumatic optic neuropathy (22)
33	Facial trauma (7)	Optic nerve compression
34	Blunt head trauma (14)	Acute unilateral optic nerve injury
35	Closed head trauma (31)	Neurogenic vision loss
37	Blunt head trauma (4)	Neurogenic vision loss
38	Facial trauma (33)	Traumatic optic neuropathy

BSSO, bilateral saggital split osteotomy; NOE, naso-orbito-ethmoid; ZMC, zygomaticomaxillary complex.

on the optic nerve itself may damage the nerve's intimate blood supply, which is not as resilient as the nerve, resulting in ischemic neuropathy. Furthermore, the long and short posterior ciliary arteries lie unprotected in the muscle cone and may be more susceptible to injury following blunt trauma than the optic nerve itself.[12]

Ophthalmologic examination of patients with blindness or severe visual impairment secondary to ischemic neuropathy reveals a pupil that constricts with accommodation, but not to light. Notably, these patients, if not suffering other ocular injuries, will have a normal fundoscopic exam.

Patients with ocular abnormalities following maxillofacial trauma most commonly present with immediate deficits; however, delayed presentations have also been described. Ansari outlined in detail those pathogeneses associated with immediate versus delayed visual loss and surmised the causes of immediate loss of vision in the posttraumatic blind patient, which include indirect optic nerve contusion, necrosis, concussion, laceration, vaso nervorum disruption, or intraneural/intrasheath hemorrhage, as well as intracerebral bleeding, vascular insufficiency, or compressive local edema.[12] These are contrasted to those factors associated with delayed posttraumatic blindness: optic nerve edema, optic nerve necrosis, infarction, intraneural hemorrhage, visual tract injuries, optic chiasm hemorrhages, and callus formation into the optic canal/foramen.

Other than direct injury to the optic nerve itself, blindness secondary to maxillofacial fractures in the trauma patient may also be seen secondary to retrobulbar hemorrhage.[23] These hemorrhages are commonly arterial in nature, often arising from the infraorbital artery or the anterior/posterior ethmoidal arteries. Brisk, high-pressure bleeding into the closed confines of the orbit results in ischemic injury, compartment syndrome, and subsequent atrophy of the optic nerve.[14]

It is also important to emphasize that iatrogenic fractures in the form of operative osteotomy are not without risk and complication. Naturally, in the controlled atmosphere of the operative theater, these rates are substantially lower than scar in traumatic facial fracture, but the risk exists nonetheless. Such factors were investigated by Girotto et al, who documented three cases of ophthalmic complications secondary to LeFort I osteotomies ranging from diplopia to permanent blindness.[24]

Table 2 Treatment Strategies and Outcomes Following Traumatic Optic Injury

Reference	Treatment	Outcomes
2		Severe ocular injury with initial/ subsequent loss of vision
3		Blindness
4		Blindness
5	Optic nerve decompression (4)	Blindness
7		Blindness
8	None (11)	Severe visual impairment (11)
	Scleral repair (3)	Blindness (8)
	Dexamethasone (1)	
	Abscess drainage (1)	
	Evisceration (11)	
11		Blindness
12		Blindness
13	Open reduction/internal fixation	Blindness
14	Transantral decompression	Blindness
17	Acetazolamide/dexamethasone	Transient blindness
18	Orbital decompression	Blindness
24	Steroids	Severe visual impairment
	Lumbar drain/antibiotics/steroids	Diplopia
	Steroids	Blindness
25	Steroids	Blindness
26	Dexamethasone	Blindness
	Steroids	Loss of superior visual field
27	Acetazolamide/dexamethasone/ lateral canthotomy/cantholysis/ hematoma evacuation	''Slight visual acuity''
15	Dexamathasone/open reduction/internal fixation	Blindness
40	Bone fragment resection	Transient blindness with return of vision
19	Incision and drainage	Blindness
6		Blindness
28	Steroids (25)	Visual improvement (22)
	Optic canal decompression (7)	No visual improvement (39)
	Open reduction/internal fixation (21)	
	Observation (13)	
29	Transethmoid-sphenoid optic canal decompression (4)	Return of vision (4)
		Blindness (3)
	Megadose steroids (3)	
30	High-dose methylprednisolone (13)	Visual improvement (19)
	High-dose dexamethasone (18)	No visual improvement (3)
33	Transethmoidal optic canal decompression (7)	Visual recovery (7)
34	External ethmoidectomy with perioperative dexamethasone	Visual improvement (11)
		No visual improvement (3)
35	Perioperative steroids with Transethmoidal optic canal decompression	Improvement in visual acuity (22)
		No improvement in visual acuity (9)
37	Steroids (2)	Return of vision (4)
	Observation (2)	
38	Observation (25)	Visual improvement (9)
	High-dose steroids (4)	No visual improvement (24)
	High-dose steroids and transethmoidal optic canal decompression (4)	

They hypothesized that the uncontrolled nature of pterygomaxillary disjunction may result in the extension of this fracture to the skull base or optic canal, resulting in optic nerve compromise. Their subsequent investigation utilizing a cadaveric model examining pressure transduction through the optic canal during maxillary down fracture revealed both increases of pressure and propagation of fracture lines through the pterygoid bones. Such reports of blindness or severe visual impairment following maxillary down fracture have also previously been noted in the literature.[25,26]

Iatrogenic causes of severe ocular impairment secondary to maxillofacial surgery are not limited to LeFort I osteotomies, as this has also been published in the literature. Pigadas and Lloyd reported such a case following Gilles repair of a displaced zygomaticomaxillary complex fracture in 2005.[27]

TREATMENTS AND OUTCOMES

For decades, debate has surrounded the treatment modality of choice for patients sustaining traumatic optic neuropathy. Although the management of traumatic optic neuropathy and posttraumatic blindness has remained controversial, observation, megadose corticosteroid therapy, and surgical decompression of the optic nerve canal have all been investigated. The results of these studies are varied and often contradictory. Table 2 summarizes the reported treatment modalities and outcomes.

Recently, Wang et al conducted a retrospective review of 61 consecutive patients presenting with a visual acuity deficit following facial trauma to their institution in Baltimore over a 12-year time period.[28] These patients were managed both operatively and nonoperatively, with 41% receiving steroids alone, 11% undergoing optic nerve decompression, 34% undergoing open reduction and internal fixation of facial fractures, and observation in the remaining 21%. The authors found no significant difference in posttreatment visual acuities across the varying treatment modalities. However, it was noted that patients who had suffered blunt trauma demonstrated a significantly higher rate of improvement in visual acuity when compared with those who had received penetrating periocular trauma. Posttreatment improvement was also significantly higher in those patients who had some degree of light perception at their time of presentation versus those who had presented with total blindness. In all, 45% of patients in this series demonstrated some improvement in visual acuity following blunt facial trauma after observation or medical or surgical intervention.

Anderson et al first advocated for the use of megadose steroid therapy for treatment of blindness following facial trauma in 1982.[29] The authors published a case series of seven patients who presented with abrupt-onset monocular blindness following frontal head trauma. Four of these patients underwent surgical decompression of the optic canal, with only one patient achieving minor return of vision. Three of the seven patients demonstrated return of vision after receiving a course of megadose steroid therapy. The authors recommended that a 12-hour trial of megadose steroid treatment be employed prior to consideration of surgical optic nerve canal decompression.

Steroid therapy was further investigated by Spoor et al.[30] These authors investigated 21 patients who had some visual impairment in 22 eyes following facial trauma and who were treated with megadose methylprednisolone (13 patients) or dexamethasone (18 patients). Notably, there was a wide range of time to presentation, from 4 hours to 15 days. No significant difference in outcome was noted between these two treatment groups, with seven of nine eyes and 12 of 13 eyes demonstrating visual improvement in the dexamethasone and methylprednisolone groups, respectively. However, those patients treated with methylprednisolone were noted to demonstrate improvement in visual acuity significantly faster than those who had received dexamethasone.

If one considers that indirect optic trauma is a focal central nervous system insult, the treatment of such may be supported by evidence for appropriate treatment modalities for other traumatic central nervous system events. In 1995, the Brain Trauma Foundation published their "Guidelines for the Management of Severe Head Injury," which recommended against glucocorticoid therapy in the severely head injured patient.[31] These recommendations were contradictory to those published by the National Spinal Cord Injury Study (NASCIS) group, which advocated for utilization of glucocorticoid therapy in the setting of acute spinal cord injury.[32] However, following the NASCIS 2 and NASCIS 3 investigations, only modest benefit was noted in post hoc analyses. The findings of these two investigations have yet to be independently confirmed, thus confounding treatment recommendations for the patient sustaining acute traumatic central nervous system injury. The most current recommendations for the management of acute cervical spine and spinal cord injuries are guided by the American Association of Neurological Surgeons, Spine Section and the Congress of Neurological Surgeons. With respect to acute spinal cord trauma, the available medical evidence does not support a significant clinical benefit from the administration of methylprednisolone in the treatment of patients for either 24 or 48 hours' duration. The neurological recovery benefit of methylprednisolone when administered within 8 hours has been suggested but not convincingly proven. However, administration of methylprednisolone for 24 hours has been associated with a significant increase in severe medical complications. This is even more significant when given for 48 hours. Because

clinical trials failed to convincingly demonstrate a significant clinical benefit of high-dose steroids when given in the face of acute spinal cord trauma, and with the increased risks of medical complications associated with its use, high-dose methylprednisolone in the treatment of acute traumatic optic neuropathy should only be undertaken with the knowledge that the evidence suggesting harmful side effects is more consistent than the evidence suggesting clinical benefit. This cannot be overemphasized.

Other treatment modalities have been described for the management of posttraumatic optic neuropathy. The transethmoidal approach to surgically decompressing the optic canal was first described by Niho et al in 1961.[33] Prior to this publication, surgical decompression of the optic canal was undertaken via a frontal approach, which unroofed the canal but failed to excise the medial wall. Niho and colleagues' description of the procedure noted it to be undertaken under local anesthesia, as subjective responses in terms of return of vision guide subsequent steps of the operation. Their first case series was comprised of seven patients, all of whom were reported to enjoy visual recovery. Similar outcomes have been described by Joseph et al following ipsilateral external ethmoidectomy. They noted visual improvement in 11 of 14 patients following blunt head trauma.[34] However, these authors utilized perioperative dexamethasone as part of their treatment regimen. Levin et al in 1994 performed a retrospective analysis of 31 cases of neurogenic vision loss following closed head trauma for which transethmoidal decompression of the optic canal was undertaken.[35] Again, all patients had received a perioperative course of steroid therapy. The authors noted that 71% of these patients had improvement in visual acuity, with 19% achieving an acuity of 20/40 or better. They also found vision was significantly improved in those patients less than 40 years of age.

These case numbers, however, pale in comparison to those published by Fukado in 1975.[36] The author presented 400 cases of surgical decompression of the optic canal with excellent results. However, these numbers have been called into question due to a lack of clear patient selection criteria.

Observation of such injuries has been described but is not strongly supported in the current literature. Wolin and Lavin described four cases of spontaneous return of vision after blunt head trauma that had initially caused blindness.[37] They noted that two of these patients did indeed receive steroids, but the authors stated that visual improvement had begun prior to their institution. They went on to advocate for surgical decompression of the optic canal when visual loss initially improves with corticosteroid therapy but repeatedly deteriorates with tapered doses.

Lessell investigated 33 cases of posttraumatic optic neuropathy in which 25 patients were observed and only five of whom had improvement in visual acuity.[38] Four patients received high-dose corticosteroids with one patient improving; the remaining four patients received both steroid therapy and transethmoidal decompression, and three improved. This failure of observation alone was substantiated by Cook et al, who published their results of a meta-analysis of 45 articles involving 244 cases of traumatic optic neuropathy.[39] In it, they found no significant difference in visual outcomes following treatment with steroids, surgical decompression, or a combination of these two modalities. Significance was demonstrated, however, in those patients treated with any of the above-mentioned modalities who improved more than those observed.

The appropriate treatment modality for patients suffering blindness or severe optic neuropathy secondary to blunt head trauma continues to be debated. Support exists for both megadose corticosteroid therapy as well as surgical decompression of the optic canal. The relatively small number of such cases precludes a large, prospective randomized trial to aid in the elucidation of the appropriate management plan. It must also be emphasized that the administration of high-dose steroids has been associated with a significant increase in severe medical complications. Therefore, its use in the treatment of acute traumatic optic neuropathy should only be undertaken with the knowledge that the evidence suggesting harmful side effects is more consistent than the suggestion of clinical benefit.

CONCLUSION

Blindness following facial fracture, either traumatic or iatrogenic, is a rare yet documented complication of injury or surgery in the orbital region. Although there are multiple mechanisms, the common pathway of direct or indirect optic nerve compromise is most common. It should be noted many fracture patterns may result in such a devastating complication, although high lateral loads seem to carry the highest correlation. Intraoperatively, care should be taken during osteotomies, particularly those of the LeFort I variety, to avoid uncontrolled fracturing and pressure transduction as these have been documented to lead to severe ocular complications. Management of the patient suffering blindness or severe visual impairment secondary to traumatic optic neuropathy is complex and disputed. Megadose corticosteroid therapy, as well as surgical decompression of the optic canal via a transethmoidal approach, is supported in the published literature, whereas observation alone is not. However, steroid use in the treatment of acute traumatic optic neuropathy should only be undertaken with the knowledge that the evidence suggesting harmful side effects is more consistent than the suggestion of clinical benefit. The debate over the proper management of

these patients will likely continue until a large, prospective randomized trial can be undertaken.

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