Chapter 22

TRAUMATIC OPTIC NEUROPATHY

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INTRODUCTION

Approximately 1.5% to 5% of patients with closed head injuries have damage to the visual pathways (4–6/100,000 general population/y). These injuries can be divided into anterior and posterior lesions. Anterior lesions show ophthalmoscopic abnormalities (eg, central retinal artery occlusion) and are usually associated with a variety of easily recognized injuries to the globe. Anterior lesions may include optic nerve avulsion, traumatic anterior ischemic optic neuropathy, anterior optic nerve sheath hematoma, and optic nerve compression from an anterior orbital hematoma. Posterior lesions, on the other hand, are often free of ophthalmoscopic findings, but disc edema (acutely) and optic nerve pallor (eventually) do occur. Posterior traumatic optic neuropathy is characterized by visual loss that occurs in the presence of an afferent pupillary defect (APD) but without evidence of injury to the eye or optic nerve.[1–3] This chapter focuses primarily on the diagnosis and management of posterior traumatic optic neuropathy.

Etiology

Blunt trauma, penetrating injuries, and self-mutilations are the most common causes of optic nerve injury. Blunt trauma classically occurs following rapid deceleration injuries to the anterofrontal regions of the head. Trauma to the outer third of the superior orbital rim is transmitted directly to the optic canal, where the optic nerve is tethered at both ends by dura. Conversely, the optic nerve is redundant in the orbit and protected by orbital fat and resistant to injury at this site.[1,4]

The severity of the trauma does not always correlate with the degree of visual loss. Incidents such as minor falls after tripping, or hitting the side of the head against a solid object resulting in a frontal blow are adequate to produce a posterior traumatic optic neuropathy. The most common mechanism of injury is bicycle accidents, followed by motor vehicle accidents. Most bicycle accidents (90%) are solo spills; the protective impact of bicycle helmets has not been studied with respect to incidence of optic neuropathy.[1]

Also, the presence or severity of orbital fractures neither directly predicts the severity of visual loss nor determines prognosis. Fractures of the medial orbital wall, optic canal, zygoma, or floor may be present.[1] One patient with an optic canal fracture may regain normal vision without intervention, but another with no fractures may present with no light perception (NLP) vision that persists despite all interventions.

Traumatic optic neuropathy is most often seen in boys in their first or second decade of life, but case series have included a wide age range and both genders. In one series,[5] patients older than 40 years of age were found to have a worse visual outcome independent of mechanism of injury, severity of visual loss, or intervention utilized.

Pathology

The optic nerve axons lie in two compartments: the intradural and the intrafascicular. Closed-space edema, contusion necrosis, nerve fiber tears, and infarction due to thrombosis or spasm have all been implicated as potential mechanisms of optic nerve injury. Shearing, stretching, compression, and contusion at the level of the intracanalicular optic nerve are probably all important in creating the dysfunction. Surgery can only open the dura and cannot relieve intrafascicular pressure elevations. Interruption of venous flow may also play an important role. Myelin is more sensitive to edema and acidosis than axonal structures. Typical findings found at autopsy include hemorrhage, demyelination, focal necrosis, and axonal changes.[4,6]

NEUROOPHTHALMIC EVALUATION

The complete ophthalmic examination has been detailed in this textbook in Chapter 3, Ocular Trauma: History and Examination. Identification of optic nerve injury can be challenging, especially in uncooperative, inebriated, or unresponsive patients. Best-corrected visual acuity is usually quantitated at the bedside with a near card. Patients who are 45 years old or older should be offered plus correction if vision is less than 20/20 on initial testing. Formal color testing is not essential, but comparing red saturation is very helpful and can easily be performed at the bedside. Even a patient who has been mistakenly or traumatically dilated can see a red-topped eyedrop container. Red desaturation, a characteristic of optic nerve dysfunction, causes the cherry-red color to appear orange or brown. The gold standard for identifying a unilateral or asymmetrical optic neuropathy is, of course, the swing-

ing flashlight test. This test is performed using a penlight, muscle light, or indirect ophthalmoscope. The light is moved from the left to the right pupil and back again. The amount and timing of constriction and redilation are then computed.

Asymmetry of constriction (1+ to 3+) or redilation (trace) is consistent with an APD. Even if the pupil on the side of interest is unreactive, the status of the optic nerve pathways can be evaluated by looking for a reverse APD. The optic nerve head is usually normal in appearance even when vision is NLP. Other associated abnormalities (eg, peripapillary choroidal rupture, hemorrhage, edema) should be sought as well. If sectoral swelling of the optic nerve head is present, especially in the presence of a small cup-to-disc ratio and altitudinal field defect, then posttraumatic anterior ischemic optic neuropathy should be suspected.

Clinical Features

Posterior traumatic optic neuropathy is often difficult to recognize in patients with multiple injuries, especially in unconscious patients. Care must be taken to examine the patient as early as possible in the evaluation process, as APD may be the only sign of injury. Typically, the optic nerve head is initially normal. Visual deficits range from normal vision with subtle visual field defects (inferior altitudinal is most common) to complete loss of light perception. In most cases, the visual loss is severe and instantaneous.[1,6–8]

Even seemingly trivial trauma may result in dramatic optic nerve impairment. The severity of visual loss does not necessarily correlate with the degree of overall trauma, but total loss of vision has been associated with the presence of fractures. Nau and colleagues[4] found that 90% of patients with total visual loss had evidence of bony fracture by computed tomography (CT); 33% presented with Le Fort III fractures, 33% had orbital fractures, and 33% had frontobasal fractures. This same study also revealed that all the patients had a laceration in the region of the eyebrow on the affected side.[4] On the other hand, Lessel,[1] reporting 33 cases in which the most common cause of injury was a bicycle accident, found no correlation between fractures and degree of visual loss. Visual evoked potentials (flash) have limited utility and may give false-negative results prior to the onset of optic atrophy.

Imaging

Although most patients with posterior traumatic optic neuropathy have normal imaging studies, CT without contrast should be performed in all cases. Spiral CT allows rapid data acquisition in uncooperative adults and children. Imaging will allow identification of associated fractures (Figure 22-1), optic nerve avulsion, optic nerve sheath hematoma (Figure 22-2), and optic nerve compression due to an orbital hematoma (Figure 22-3). The optic nerve injury may not be isolated (Figure 22-4); associated

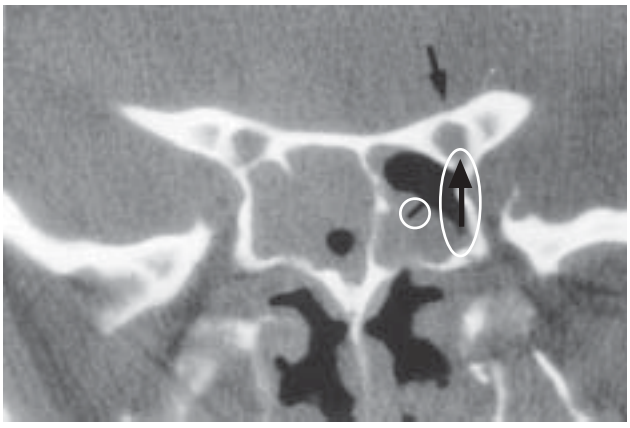


Fig. 22-1. A computed tomography (CT) scan demonstrating a fracture of the optic canal (arrows). CT scan: Courtesy of Department of Ophthalmology, Allegheny General Hospital, Pittsburgh, Pa.

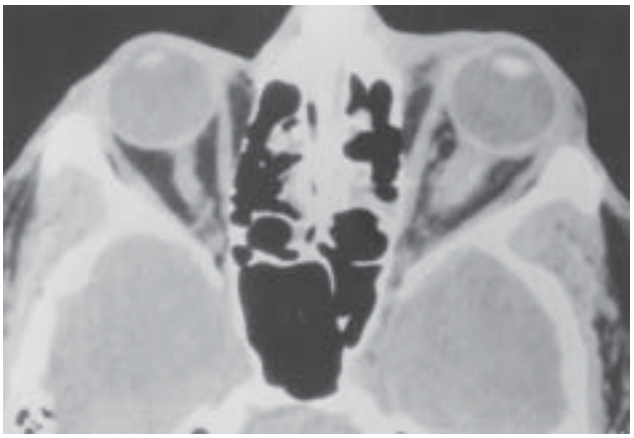


Fig. 22-2. A computed tomography (CT) scan demonstrating a left optic nerve sheath hematoma. Preseptal swelling is also present. CT scan: Courtesy of Department of Ophthalmology, Allegheny General Hospital, Pittsburgh, Pa.

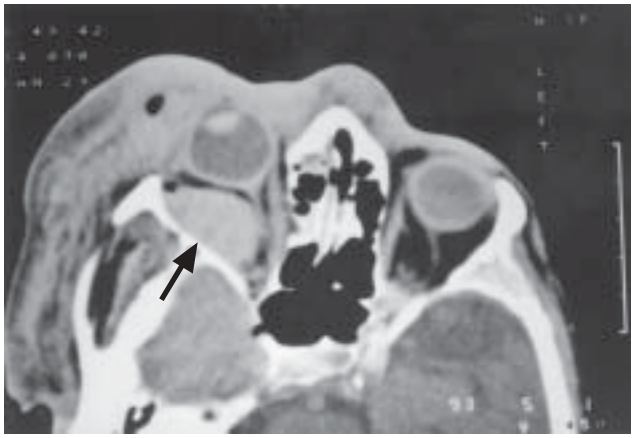


Fig. 22-3. Massive blunt trauma has resulted in preseptal swelling and a lateral orbital hematoma (arrow) that is compressing the right optic nerve, as seen in this axial computed tomography (CT) scan. CT scan: Courtesy of Department of Ophthalmology, Allegheny General Hospital, Pittsburgh, Pa.

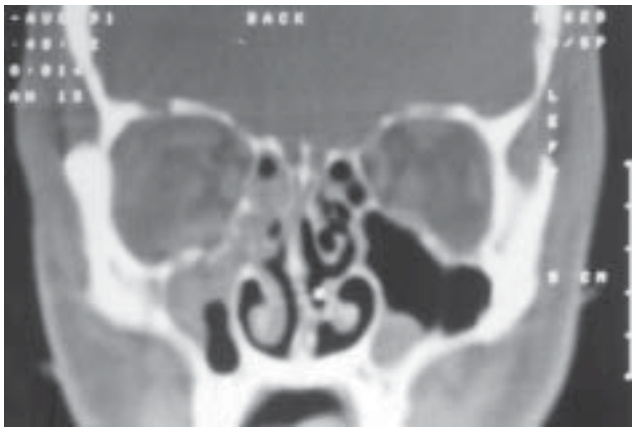


Fig. 22-4. Medial wall and blowout fractures are present in a patient with optic nerve dysfunction due to indirect blunt trauma (posterior posttraumatic optic neuropathy). As seen in this computed tomography (CT) scan, optic nerve avulsion is not present and the optic canal is intact. CT scan: Courtesy of Department of Ophthalmology, Allegheny General Hospital, Pittsburgh, Pa.

fractures and injuries can be identified when the scope of evaluation is expanded to include otolaryngology, oral, maxillofacial, and neurosurgery colleagues. Magnetic resonance imaging is only indicated if intracranial injuries are present that are inadequately detailed with CT imaging.[1,6,9]

NATURAL HISTORY

TABLE 22-1

NATURAL HISTORY OF TRAUMATIC OPTIC NEUROPATHY

Author (Year of Study)	Number of Patients	Spontaneous Improvement (%)
Tang (1986)[1]	13	38
Millesi (1988)[2]	7	57
Lessell (1989)[3]	25	20
Seiff (1990)[4]	15	33
Levin (1999)[5]	9	57

Data sources: (1) Tang R, Li H, Regner V, Bridges MB, Prager TC. Traumatic optic neuropathy: Analysis of 37 cases. *Invest Ophthalmol Vis Sci*. 1986;27(suppl):102. (2) Millesi W, Hollmann K, Funder J. Traumatic lesions of the optic nerve. *Acta Neurochir.* 1988;93:50–54. (3) Lessell S. Indirect optic nerve trauma. *Arch Opthalmol*. 1989;107:382–386. (4) Seiff SR. High dose corticosteroids for treatment of vision loss due to indirect injury to the optic nerve. *Ophthalmic Surg*. 1990;21:389–395. (5) Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: The International Optic Nerve Trauma Study. *Ophthalmology.* 1999;106:1268–1277.

The natural history of traumatic optic neuropathy is difficult to characterize because each patient is different. Attempts to study patient outcomes have also been hindered by the assumption that corticosteroids are helpful and that not offering them would be unethical. Visual prognosis and likelihood of spontaneous improvement are independent of the initial visual acuity.[10,11]

The natural history of indirect optic nerve injuries has been described in several clinical series. The rate of spontaneous visual improvement ranges from 20% to 57% (Table 22-1). Even patients who had a return of normal central visual acuity did not regain an entirely normal afferent examination. Persistence of visual field and color defects, and APD are typical. Optic nerve pallor or nerve fiber layer changes also develop over the months following the injury. Patients with NLP on presentation can sometimes recover-useful vision without intervention.[1,11]

The prognosis tends to be better for patients who have a lucid interval or an enlarged nerve sheath[12] but poorer for patients older than 40 years.[5] The following factors do not appear to correlate with visual outcome[5]:

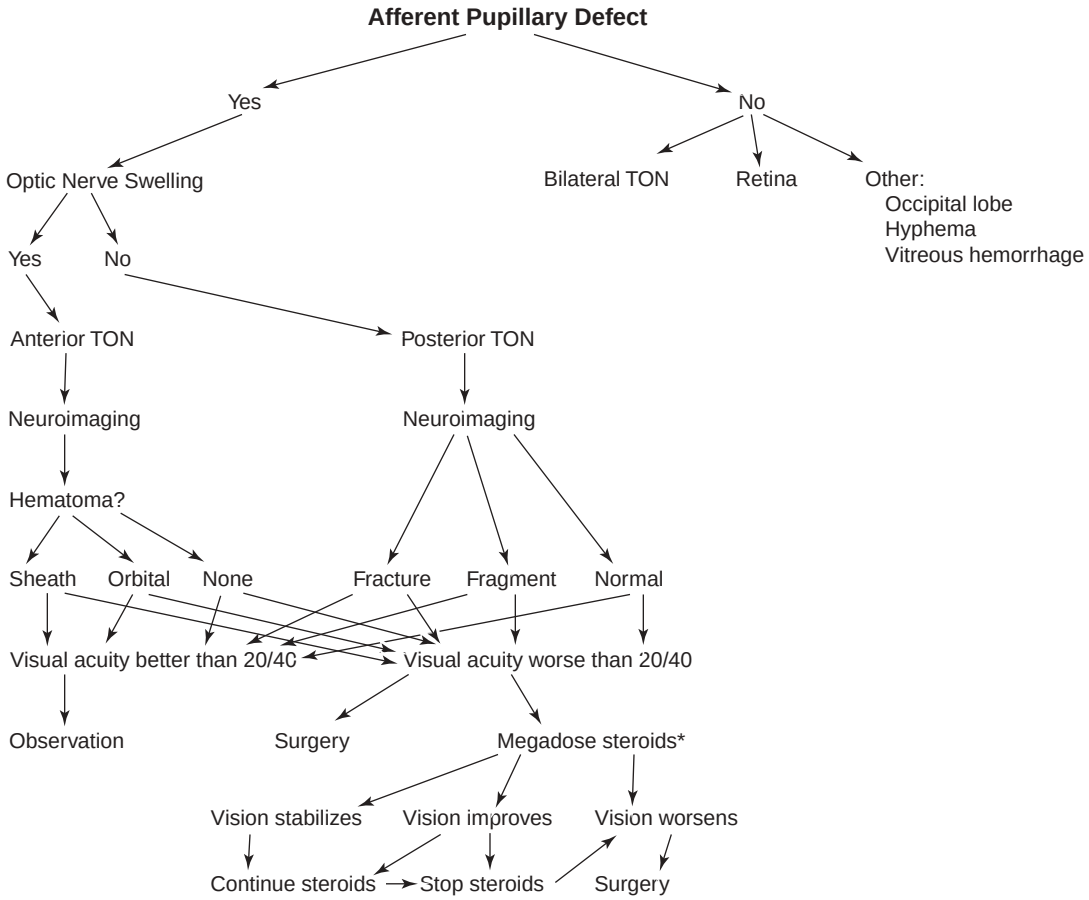
- gender,
- level of consciousness,
- mechanism of injury,
- initial visual acuity (including NLP),
- presence of fractures (including optic canal fracture), and
- time from injury to intervention (within the first 7 d).

TREATMENT

During the mid 1970s, observation was replaced by steroid therapy and surgery as a variety of case series promoted the success rates of these interventions, and since then, significant improvement without treatment has been well documented (see Table 22-1). Despite the rate of spontaneous improvement, however, the standard of care has been to offer high-dose corticosteroids. The role of surgical intervention is still debated. The International Optic Nerve Trauma Study[13] (published in 1999) demonstrated no clear benefit from either corticosteroid therapy or optic canal decompression surgery. Observation has again become an acceptable option when there is no evidence of intrasheath hematoma, orbital hematoma, or optic canal fracture fragments (Figure 22-5).

Corticosteroid Therapy

Widely used for several decades, corticosteroids are thought to stabilize lipid membranes, reduce spasm, increase blood supply, and reduce neural



*Loading dose: 30 mg/kg followed by 15 mg/kg 2 h later. The maintenance dose could then be 15 mg/kg at 6-h intervals or 5.4 mg/kg/h for at least 48 h before deciding whether response has occurred.
TON: traumatic optic neuropathy

Fig. 22-5. Algorithm for medical and surgical management of traumatic optic neuropathy

tissue edema and necrosis. Megadose steroids were first studied in animal models with brain edema and then in humans with spinal cord injuries.[14] Spoor and colleagues[15] were the first to use spinal cord doses for patients with traumatic optic neuropathy. They divided their patients into two groups, and treated

- one group (13 patients) with methylprednisolone (30 mg/kg initially, then 15 mg/kg given 2 h later, and then in divided doses every 6 h), and
- the second group (8 patients) with high-dose dexamethasone (20 mg every 6 h).

Both therapies were continued for 48 hours, then followed by a rapid taper. Of the treated eyes (one per patient), 17 of 21 (81%) improved in visual function during these protocols. The relative dose of steroid was much higher for the methylprednisolone group, but the overall outcome was no different. The methylprednisolone group appeared to make a more-rapid recovery, however. A delay in treatment did not appear to alter outcome in this study. The mean onset of therapy was 4.2 days. The authors of the study believed that neither the initial severity nor the type of nerve injury allowed the response to steroid therapy to be predicted, as five of eight eyes in this study initially had NLP but regained significant function.

Another series by Bendel and colleagues[16] described 17 patients treated with 2 g methylprednisolone initially, then 1 g, divided, every 6 hours for 48 hours. Vision improved in all patients, with an average pretreatment vision of 20/100 and average posttreatment vision of 20/25. Best vision occurred between 1 and 6 days after treatment (mean = 2.9 d). Three patients demonstrated deteriorating visual function during steroid taper and were treated with extracranial optic nerve decompression; all three had eventual improvement.

Mauriello and colleagues[12] reported a series in which 9 of 16 patients (56%) treated with a more-conventional steroid dose had significant improvement (1 g loading dose, then 250 mg in divided doses every 6 h for 48 h). Almost all patients who did not improve had NLP vision initially. They also noted that all 5 of their patients with a lucid interval following injury had eventual improvement.

In summary, a short course of high-dose steroid may be considered unless there is clear evidence of optic nerve transection or avulsion by clinical or radiographic criteria. A delay in onset of therapy and the degree of visual loss have not been clearly shown to alter prognosis (Table 22-2).[6–8,12,15,17,18]

Surgical Intervention

Optic canal decompression was first described in 1916, wherein a transcranial unroofing was performed in patients with afferent dysfunction who required craniotomies for other reasons.[19] Extracranial techniques were later described to minimize the possible complications associated with craniotomy. A transethmoidal approach was investigated as early as 1926 but not popularized until the 1960s.[6] External, transantral, Caldwell-Luc, and transnasal approaches have all been tried.[20] Comparison of different clinical series is difficult because of differences in techniques, selection criteria, and quantification of visual improvement. Many patients also

TABLE 22-2

REPORTED OUTCOMES USING CORTICOSTEROIDS

Author (Year of Study)	Number of Patients and (Treatment Received)*	Visual Improvement (%)
Tang (1986)[1]	5 (C)	20
	11 (M)	36
Millesi (1988)[2]	2 (U)	50
Lessel (1990)[3]	4 (U)	25
Seiff (1990)[4]	21 (M)	62
Spoor (1990)[5]	22 (M)	86
Bendel (1993)[6]	17 (M)	100
Levin (1999)[7]	85 (M)	52

*C: conventional steroid dose (1 g methylprednisolone loading dose followed by 250 mg methylprednisolone qid)
M: megadose steroid doses (30mg/kg methylprednisolone loading dose followed by 15 mg/kg every 6 h or 5.4 mg/h for 24–48 h)
U: unspecified dose

Data sources: (1) Tang R, Li H, Regner V, Bridges MB, Prager TC. Traumatic optic neuropathy: Analysis of 37 cases. *Invest Ophthalmol Vis Sci*. 1986;27(suppl):102. (2) Millesi W, Hollmann K, Funder J. Traumatic lesions of the optic nerve. *Acta Neurochir.* 1988;93:50–54. (3) Lessell S. Indirect optic nerve trauma. *Arch Opthalmol.* 1989;107:382–386. (4) Seiff SR. High dose corticosteroids for treatment of vision loss due to indirect injury to the optic nerve. *Ophthalmic Surg.* 1990;21:389–395. (5) Spoor TC, Hartel WC, Lensink DB, Wilkinson MJ. Treatment of traumatic optic neuropathy with corticosteroids. *Am J Ophthalmol.* 1990;110: 665–669. (6) Bendel RE, McHenry JG, Ramocki JM, Spoor TC. Traumatic optic neuropathy and intravenous megadose corticosteroids. *Invest Ophthalmol Vis Sci.* 1993;34(suppl):1215. (7) Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy. The International Optic Nerve Trauma Study. *Ophthalmology.* 1999;106:1268–1277.

receive at least conventional doses of corticosteroids in the perioperative period, further confusing the ability to attribute improvement to the surgical intervention. The frequency of reported visual improvement ranges from 12% to 79%.[5,6,13,17–19,21–26] In many medical centers, the decision to operate is governed by the criteria established in 1966 by Walsh (Exhibit 22-1).

Mauriello and colleagues[12] treated 23 patient with steroids and operated on 7 nonresponders based on CT evidence of surgical pathology (optic nerve sheath enlargement or narrowing of the optic canal by bone spicules). Of the 3 patients treated with optic nerve sheath fenestration alone, only 1 had significant improvement. The remaining 4 patients were treated with both fenestration and optic canal decompression. Only 1 patient had significant improvement (NLP to 20/200).

Joseph and colleagues[26] reported visual improvement in 11 of 14 patients treated with steroids and transethmoidal decompression of the optic canal. This report was a retrospective review and not compared to any large, steroid-only treatment group, but the results are similar to those in the study of Spoor and colleagues,[15] who used megadose corticosteroids (described above).

Levin and colleagues[13] reported the outcome of the International Optic Nerve Trauma study in 1999. The goal of this endeavor was to compare the visual outcome of traumatic optic neuropathy treated with corticosteroids, treated with optic canal decompression surgery, or observed without treatment. Patients who were randomized to the surgical group did not receive steroids in the perioperative period. Intervention occurred within the first 7 days following injury. The main outcome measure was defined as visual acuity improvement of three or more lines of Snellen acuity. The Inter-

EXHIBIT 22-1

CRITERIA GOVERNING SURGICAL INTERVENTION IN PATIENTS WITH TRAUMATIC OPTIC NEUROPATHY

Absolute Surgical Contraindication:

- Optic nerve avulsion is present on CT imaging

Relative Surgical Contraindications:

- Patient is unconscious
- Total loss of vision and pupillary response

Relative Surgical Indications:

- If visual loss develops despite steroid treatment
- If visual decline occurs during the steroid taper
- If an optic canal fracture is accompanied by potentially compressive bone fragment
- If an optic nerve sheath hematoma is present
- If the visual evoked potential (VEP) response deteriorates over time

Sources: (1) Levin LA, Joseph MP, Rizzo JF. Optic canal decompression in indirect optic nerve trauma. *Ophthalmology.* 1994:101:566–569. (2) Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy. The International Optic Nerve Trauma Study. *Ophthalmology.* 1999;106:1268–1277. (3) Mine S, Yamakami I, Yamaura A, et al. Outcome of traumatic optic neuropathy: Comparison between surgical and nonsurgical treatment. *Acta Neurochir.* 1999;141:27–30. (4) Pomeranz HD, Rizzo JF, Lessell S. Treatment of traumatic optic neuropathy. *Int Ophthalmol Clin*. 1999;39(1):185–194. (5) Li KK, Teknos TN, Lai A, Laurentano AM, Joseph MP. Traumatic optic neuropathy: Result in 45 consecutive surgically treated patients. *Otolaryngol Head Neck Surg.* 1999;120:5–11. (6) Li KK, Teknos TN, Lauretano A, Joseph MP. Traumatic optic neuropathy complicating facial fracture repair. *J Craniofacial Surg*. 1997;8:352–355. (7) Kuppersmith RB, Alford EK, Patrinely JR, Lee AG, Parke RB, Holds JB. Combined transconjunctival/intranasal endoscopic approach to the optic canal in traumatic optic neuropathy. *Laryngoscope.* 1997;107:311–315. (8) Cook MW, Levin LA, Joseph MP, Pinczower EF. Traumatic optic neuropathy: A meta-analysis. *Arch Otolaryngol Head Neck Surg.* 1996;122:389–392. (9) Girard BC, Bouzas EA, Lamas G, Soudant J. Visual improvement after transethmoid-sphenoid decompression in optic nerve injuries. *J Clin Neuro Ophthalmol.* 1992;12:142–148. (10) Joseph MP, Lessell S, Rizzo J, Momose KJ. Extracranial optic nerve decompression for traumatic optic neuropathy. *Arch Ophthalmol.* 1990;108:1091–1093.

national Optic Nerve Trauma study found that only 32% in the surgery group (n = 33) improved significantly. This was in contrast to the visual improvement observed in the untreated group (57%, n = 9) and steroid group (52%, n = 85). The study concluded that there was no clear benefit for intervention. In addition, they confirmed the findings of others that the timing of corticosteroids or surgery within the 7-day window did not affect outcome.

SUMMARY

Traumatic optic neuropathy is a rare but significant cause of posttraumatic visual loss. The responsible blunt trauma to the frontal region may be minor or severe and accompanied by multiple adjacent fractures. Careful documentation of visual acuity, pupillary function, and red desaturation is essential to guide management. CT imaging should be performed to document such structural abnormalities as optic nerve avulsion, optic nerve sheath hematoma, orbital hematoma, or optic canal fracture with fragments.

Based on the data from the International Optic Nerve Trauma Study, observation without intervention is a viable option. Patients and their families should be made aware of the information regarding megadose corticosteroid therapy and participate in an informed decision. In particular, if visual acuity begins to deteriorate, then corticosteroid therapy should be considered. If a structural abnormality is present that may be contributing to optic nerve dysfunction (hematoma or fragment) or if the patient's visual acuity deteriorates on corticosteroids, optic canal decompression should be offered. Management of this disorder remains very controversial; involvement of other appropriate subspecialists and careful discussions with the patient and family are essential to maximize visual outcome.

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