Original Investigation

Traumatic Optic Neuropathy and Second Optic Nerve Injuries

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IMPORTANCE Current controversy about the primary treatment of traumatic optic neuropathy (TON) has anchored on final vision following injury, but, to our knowledge, no study has examined the effect of different treatments on regaining and protecting optic nerve reserve or on the outcome of second optic nerve injuries.

OBJECTIVE To assess vision improvement in patients treated by various methods who have a second incidence of TON.

DESIGN, SETTING, AND PARTICIPANTS Retrospective medical record review of 12 patients with a second TON seen in an 18-year period (mean follow-up, 11.3 months) at a single tertiary care oculoplastic practice.

INTERVENTIONS Observation, high-dose corticosteroids, optic nerve decompression, or high-dose corticosteroids plus optic nerve decompression.

MAIN OUTCOMES AND MEASURES Change in vision on the Snellen eye chart.

RESULTS All second TON events involved the same-side optic nerve as initially injured, and with observation alone, corticosteroids, or corticosteroids and partial optic canal decompression, all patients had vision improvement after their initial injury ($P = .004$). However, following the second optic nerve injury, most patients' vision fell to the pretreatment level of the first injury, and subsequent management of the second injury with corticosteroids and/or optic canal decompression provided little or no vision return ($P = .05$). In contrast, optic canal decompressions performed for 91 primary TON injuries resulted in 82.4% having some degree of vision improvement.

CONCLUSIONS AND RELEVANCE Patients with TON may have a second optic nerve insult, and vision recovery from the second event may be limited regardless of primary treatment choice.

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The reported incidence of traumatic optic neuropathy (TON) ranges from 1.5% to 4% following head trauma,[1,2] and the management of such injuries remains controversial. Original treatment options included observation or optic canal decompression through a frontal craniotomy.[3] Later surgical approaches involved external or transantral ethmoidectomy and, more recently, a combined transconjunctival and intranasal endoscopic approach.[4-8]

In 1982, Anderson et al[9] proposed the use of high-dose corticosteroids for TON based on the observation that such treatment was effective in reducing traumatic central nervous system edema.[10] Although the spinal cord and optic nerve are very different structures, many believed that the Second National Acute Spinal Cord Injury Study in 1990 provided further support for corticosteroid intervention within the first 8 hours of optic nerve injury,[11] spurring the advocacy of rapid intervention in the form of a specific dose of methylprednisolone sodium succinate alone or in combination with optic nerve decompression surgery.[12,13]

The Corticosteroid Randomization After Significant Head Injury trial in 2004, however, unveiled an increased mortality rate among patients with head trauma treated with high-dose corticosteroids. Given the frequency of head trauma in patients with TON, the use of corticosteroids for TON was once again questioned, especially with a reported spontaneous vision improvement in as many as 57% of patients with TON who underwent neither corticosteroid nor surgical treatment.[14-16] Thus, although some remain committed to surgical decompression, as the enthusiasm for corticosteroid treatment has waned, the number of those favoring a laissez-faire approach seems to be growing.[15-18]

Table 1. Patients With Second Traumatic Optic Neuropathy Injuries[a]

Patient No.	Mechanism of Trauma	Visual Acuity				Original Treatment	Time to Second Injury, y
	T1/T2	T1i	T1f	T2i	T2f		
1	MVC/MVC	20/200	20/25	20/400	20/200	?	7
2	Assault/assault	CF	20/20	NLP	NLP	C_O	6
3	Trauma/assault	20/100	20/50	20/70	20/70	C_O	7
4	MVC/other	CF	20/40	CF	CF	C_M	4
5	MVC/assault	20/400	20/40	LP	20/400	C_M	13
6	Assault/MVC	20/100	20/50	20/200	20/200	C_O	8
7	Assault/MVC	20/70	20/30	NLP	NLP	?	2
8	Assault/MVC	20/200	20/30	20/400	20/200	C_M	8
9	Assault/other	CF	20/200	CF	CF	C_O	2
10	Trauma/MVC	20/70	20/40	NLP	CF	C_M	4
11	Trauma/MVC	20/200	20/50	20/400	20/400	C_M, S_P	15
12	MVC/assault	20/100	20/50	20/200	20/200	?	8

Abbreviations: CF, counting fingers; LP, light perception; MVC, motor vehicle crash; NLP, no light perception.

[a] Characteristics of the patients with a second episode of traumatic optic neuropathy showing the vision at presentation following their initial insult (T1i), initial treatment (T1f), the second insult (T2i), and the second treatment (T2f), as well as their mechanism of injury. The visions reported at T1f and T2f represent the vision recorded as of the last follow-up visit. T1 indicates the first injury; T2, the second injury; and ?, unknown treatment. C_M is a corticosteroid regimen per the Second National Acute Spinal Cord Injury Study trial.[11] C_O is some other corticosteroid regimen. S_P is a partial optic nerve decompression less than 90°.

Under the assumption that the type of intervention may not make much difference in ultimate vision recovery, recent TON management debate has focused on the safety of treatment options. That is, if all treatments provide roughly the same outcome, should we not choose the safest treatment? This thinking and our efficacy analysis, however, may be shortsighted, lacking consideration of optic nerve resilience against further insult. Herein, we present a case series of catastrophic, unrecoverable vision loss from second optic nerve insults following initial vision recovery from TON, suggesting increased optic nerve vulnerability and raising further questions of the stability of regained vision following TON and which treatment(s) might provide the most optic nerve protection against future injury.

Methods

A retrospective medical record review was performed on patients referred to a single tertiary care oculoplastic practice and treated from 1994 through 2011 for second TON injuries. Patients in the same practice treated surgically for primary TON were also identified for outcome comparison. Institutional review board approval and informed consent from the patients were not required for a retrospective medical record review at the private tertiary referral practice.

All patients with primary or secondary injuries were provided a detailed review of the practice's experience, as well as a summary of the literature to date, and then offered observation alone, high-dose corticosteroids, or high-dose corticosteroids and optic canal decompression. High-dose corticosteroid treatment consisted of one 1.5-mg/kg intravenous dose of methylprednisolone followed by 5.4 mg/kg/h for 72 hours if there was no improvement in vision, until vision improvement stabilized, or until surgery.

Snellen chart visual acuities were measured at presentation, twice daily while taking intravenous corticosteroids, on the day following surgery (if performed), and following completion of corticosteroids or surgery after 1 day, 1 week, 1 month, and 3 months. For the purposes of analysis, changes in visual acuities were assigned line improvement scores as a difference in the number of lines shown on a Snellen eye chart between events. Beyond 20/400, counting fingers, light perception, and no light perception were each counted as additional "lines" of vision.

Using paired 2-tailed *t* tests, Snellen chart visual acuities, and assigned line improvement scores, pretreatment and posttreatment vision change following the first injury, pretreatment and posttreatment vision change following the second injury, posttreatment vision change after the first and second injuries, and posttreatment and pretreatment vision change after the first and second injuries were compared.

Optic canal decompressions were performed using a combined endonasal endoscopic and transconjunctival approach to achieve 120° to 180° of optic nerve exposure.

Results

A total of 131 patients were identified who were seen in the 18-year period from 1994 through 2011 for treatment of TON. Twelve patients had a previous incidence of TON and sought treatment for a second optic nerve insult. Among the 12 patients with secondary TON, 10 (83.3%) were men, the mean age was 42 years (range, 26-51 years), mean visual acuity at presentation was between 20/400 and counting fingers, mean length of time between TON events was 7 years (range, 2-13 years) (**Table 1**), mean loss in vision following the second insult was 5.6 lines (range, 1-11), and mean follow-up was 11.3 months (range, 1-26 months). Of these patients, 10 (83.3%) re-

Table 2. Vision Change Analysis[a]

Interval	Analysis of Mean Lines of Vision	*P* Value	Interpretation of Vision Change
I	First injury pretreatment vs first injury posttreatment	.004	Improved
II	First injury posttreatment vs second injury pretreatment	<.001	Worsened
III	Second injury pretreatment vs second injury posttreatment	.05	Unimproved
IV	First injury pretreatment vs second injury pretreatment	.01	Worsened
V	First injury pretreatment vs second injury posttreatment	.05	Unimproved

[a] Demonstrates the change in vision at the indicated time intervals. Taken across all patients with 2 injuries, vision following the second injury is worse than that seen following the first injury, and vision after treatment of the second injury is no better than the vision seen before treatment of the first injury.

ceived optic canal decompression, with 2 (16.7%) having a minimal vision improvement of 1 line and 2 (16.7%) having improvement of 2 lines. Two patients (16.7%) were treated with high-dose corticosteroids alone, neither of whom had any vision improvement.

One of the 12 patients (8.3%) with secondary TON had previously undergone attempted optic canal decompression after the first injury, but the decompression involved only a small anterior portion of the optic canal with roughly only 30° of radial decompression. Other treatments following the initial TON included methylprednisolone per the Second National Acute Spinal Cord Injury Study (5 [41.7%]), some other corticosteroid regimen (4 [33.3%]), and unknown treatment (3 [25.0%]).

Among the 12 patients with secondary TON, the most common mechanism of injury for their first TON event was aggravated assault (5 [41.7%]), motor vehicle crash (4 [33.3%]), and trauma (3 [25.0%]), whereas second injuries were ascribed to motor vehicle crash (6 [50.0%]), assault (4 [33.3%]), and minor trauma (2 [16.7%]) from a tether ball to the mid-glabella or banging the head against an open kitchen cupboard door.

Of the 115 patients with primary TON, 91 (79.1%) underwent surgical decompression, with 75 (82.4%) achieving improved vision with a mean of 5 lines of improvement and 74 (98.7%) of those with vision improvement after surgery having some improvement within the first 24 hours. In contrast, 24 patients (20.9%) with primary TON declined surgery and received corticosteroids alone. Only 10 (41.6%) had vision improvement with a mean of greater than 5 lines of improvement.

In the patients with secondary TON, there was a significant improvement in lines of vision following the initial treatment ($P = .004$); however, following the second insult, vision typically fell to or below that seen after the first injury, and no substantive improvement was obtained following treatment for the second insult ($P = .05$).

Discussion

The management of TON, a potentially devastating injury with risk for irreversible vision loss, remains controversial, with some recommending no intervention, others strongly advocating high-dose corticosteroids with or without optic canal decompression, and still others emphasizing a necessity of rapid, early treatment.[13-16,19] Perhaps the wide disparity in vision recovery success reported with various interventions reflects in part not only the mechanism of the TON but also patient characteristics not yet elucidated. One such characteristic may be the "resilience" of the optic nerve, which could depend on multiple factors, such as overall microvascular health, a tendency toward regional edema, general nutritional status of the patient, or previous optic nerve injury. A related characteristic might be the amount of anatomic redundancy of undamaged optic nerve fibers that provide no perceptible increase in optic nerve function, which we term *optic nerve reserve*. Optic nerve reserve, or the ability to lose a degree of neuronal anatomy without losing nerve function, is well recognized throughout the body.[20,21]

Frisén and Quigley[20] explored the idea of ophthalmic neuronal reserve and showed that a patient may have 20/20 visual acuity despite losing more than 40% of retinal or optic nerve neural channels, whereas a visual acuity of 20/50 on the Snellen eye chart represented a 90% loss of neural channels. They suggested that below a certain threshold of neuronal channel loss, linear neural loss creates a nonlinear vision loss.[22]

Herein, we reviewed all medical records of patients with TON treated by a tertiary care oculoplastic surgical practice in an 18-year period, and we were surprised to find that 12 of 131 patients (9.2%) had had documented ipsilateral optic nerve injury with vision loss. Following their first injury, all 12 patients had some degree of vision improvement, but 2 to 15 years later (mean, 7 years), each had a presumed second optic nerve insult, and vision fell to or below the loss incurred from the initial injury (**Table 2**). Furthermore, only 4 of 12 patients (33.3%) recovered any vision after treatment of their second injury, and in all patients, the returned vision did not exceed the vision measured directly after the first injury.

In comparing the outcome of these 12 patients with secondary TON with that of 115 patients with primary TON treated in the same practice, 10 of 12 patients (83.3%) with secondary TON elected optic canal decompression, whereas only 91 (79.1%) patients with primary TON underwent surgery. Nevertheless, among the patients with secondary TON, only 3 (25.0%) showed marginal vision improvement, whereas among those with primary TON, 82.4% demonstrated better vision postoperatively.

This finding raises several questions. First, did the patients with a second injury have optic nerve reserve loss during their first injury, making them more susceptible to further unrecoverable vision loss from a second injury, many of which appeared to be quite minor? Second, since none of the 12 patients with secondary TON received meaningful optic canal decompressions (1 patient received 30° decompression) in

the treatment of their first injuries, might a more complete canal decompression (120°-180°) be more protective against further vision loss? Third, should we be recommending constant safety eyeglass use with polycarbonate lenses and titanium frames for all patients who have had TON and regained vision to protect their uninjured eye since the eye with TON may be at higher risk for further vision loss? In several studies, including that by Frisén and Quigley,[20] an instability of regained vision in patients with TON treated with corticosteroids alone has been reported. Fourth, in practices where optic canal decompressions are offered as a treatment option, should patients with known previous optic nerve injuries be excluded? Fifth, if we dig deeply enough into patients' histories, can we better identify and stratify those who have had a previous optic nerve injury and make further studies on TON management outcome analysis more meaningful?

Although very intriguing, this study should be used primarily as a springboard for further thought and prospective investigation since it has many potential flaws. First, the data may be confounded by the retrospective analysis, study size, and probable referral bias. Although 9.2% of our patients with TON had a previous optic nerve injury, 12 patients is nonetheless a small cohort, and it remains unclear whether this high percentage is typical of a national average or reflects local referral patterns of patients with profound vision loss to a tertiary care practice. In fact, given the retrospective character of this study, the true percentage of second injuries may be even higher if more in-depth medical history questioning had been done. This possibility is further highlighted by the fact that all previous TON events identified in our medical records involved the same eye injured a second time, raising the question of whether histories of contralateral optic nerve injury might have been neglected and missed. Furthermore, all patients in this study with secondary TON had significant vision return (mean, 4 lines; range, 2-9 lines) after their initial injuries. We suspect that this also indicates referral bias since patients who had previous TON and continuing severe vision loss may not have been referred after a second injury with relatively minor additional loss. Second, patients with primary TON and second-insult TON are not well-matched cohorts, with mean ages being 37 and 42 years and lines of vision return after initial injury being almost 6 and 4, respectively. Third, only 1 patient with secondary TON had a return to 20/20 visual acuity after the initial injury. Possibly patients with 20/20 visual acuity may have greater preservation of their nerve reserve. Fourth, patients with primary or secondary TON were not stratified in our medical records according to suspected or known mechanism of TON, of which several have been proposed.[3,9,16,23,24] Fifth, few if any of the patients in this study had rigorous color vision analysis, contrast sensitivity study, optical coherence tomography, electrophysiology, or formal visual fields beyond 30° from fixation to best characterize optic function. Finally, our follow-up for patients with primary TON was only 7 months on average, so our hypothesis that 120° to 180° optic canal decompression may be protective against further optic nerve TON is conjecture since none of the 12 patients had received such surgery.

Conclusions

To our knowledge, this is the first case series of patients with second-event TON injuries to the same eye. Despite the fact that a patient's vision might return after an episode of TON, following a second optic nerve insult, high-dose corticosteroids and surgical decompression may not be beneficial. Regardless of vision recovery, the question is whether patients with primary TON should be offered substantial optic canal decompression since it might be partially protective against a second nerve injury. Nevertheless, given the possible lifelong vulnerability of their injured eye, we strongly encourage all patients with past or current TON to always wear eyeglasses with polycarbonate lenses mounted in a titanium frame to protect their other eye.

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Study concept and design: Soparkar.
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