



# Role of delayed wider endoscopic optic decompression for traumatic optic neuropathy: a single-center surgical experience

Shang-Feng Zhao<sup>1</sup>, Li Yong<sup>1</sup>, Jia-Liang Zhang<sup>1</sup>, Jiang-Ping Wu<sup>1</sup>, Hao-Cheng Liu<sup>1</sup>, Si Sun<sup>1</sup>, Gui-Dong Song<sup>1</sup>, Jian-Min Ma<sup>2</sup>, Jun Kang<sup>1</sup>

<sup>1</sup>Department of Neurosurgery, Beijing Tongren Hospital, Capital Medical University, Beijing, China; <sup>2</sup>Beijing Ophthalmology & Vision Science Key Lab, Beijing Tongren Eye Center, Beijing Tongren Hospital, Capital Medical University, Beijing, China

**Contributions:** (I) Conception and design: J Kang; (II) Administrative support: JM Ma; (III) Provision of study materials or patients: J Kang; (IV) Collection and assembly of data: L Yong, JL Zhang, JP Wu; (V) Data analysis and interpretation: HC Liu, S Sun, GD Song; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

**Correspondence to:** Jun Kang. Department of Neurosurgery, Beijing Tongren Hospital, Capital Medical University, No1, Dongjiaomin Xiang, Dongcheng District, Beijing 100730, China. Email: junkang2015@163.com.

**Background:** The aim of the present study was to discuss the efficacy of delayed wider endoscopic optic decompression in traumatic optic neuropathy (TON).

**Methods:** A total of 479 patients were treated with corticosteroids and delayed wider endoscopic optic decompression, including the injury-to-surgery interval, within 2 weeks in patients with no light perception (NLP), and within 1 month in patients with residual eyesight. Based on the traditional decompression range, the superior wall of the optic canal was further decompressed. The preoperative and postoperative visual acuities (VAs) were reviewed, and the therapeutic efficacy was analyzed.

**Results:** The final VA was 0.1 or better in 29 cases, finger count in 79 cases, hand motion in 99 cases, light perception (LP) in 25 cases, and NLP in 247 cases. A total of 136 patients (136/383, 35.5%) recovered after NLP treatment, and 78 patients (69/96, 71.9%) had improved residual eyesight. The improvement rate in patients with residual eyesight was significantly higher than that of patients with NLP ( $P < 0.01$ ). Moreover, the total VA after treatment was better than that before surgery ( $P < 0.01$ ).

**Conclusions:** Delayed wider optic nerve decompression plus corticosteroids remains an effective and safe therapeutic strategy for patients with delayed treatment intervals of more than 1 week, especially for those with residual eyesight within 1 month.

**Keywords:** Traumatic optic neuropathy (TON); corticosteroids; delayed wider decompression; follow-up

Submitted Nov 05, 2020. Accepted for publication Jan 06, 2021.

doi: 10.21037/atm-20-7810

**View this article at:** <http://dx.doi.org/10.21037/atm-20-7810>

## Introduction

Traumatic optic neuropathy (TON), including direct and indirect damage, remains an important cause of traumatic visual loss following closed head injury. It has an incidence of 0.5–5%, and is more common in cases of craniofacial fracture (1–4). Direct injury is caused by direct penetration of the nerve by a foreign body and a displaced optic canal fracture. It is also the result of the impact of external force transmitted into the optic canal, followed by injury to

optic nerve axons, increased intracanalicular pressure, and vascular ischemia, resulting in secondary retinal ganglion cell (RGC) loss (5,6).

To date, there is little consensus on the optimal treatments for TON. A variety of treatment strategies have been studied, including observation, corticosteroid therapy, and surgical decompression of the optical nerve canal with or without corticosteroid therapy (7–10).

Theoretically, endoscopic optic nerve decompression (EOND) and corticosteroids are useful for decreasing

**Table 1** Clinical manifestations and examination results of patients with traumatic optic neuropathy

Characteristics	Number (%)
<b>Sex</b>	
Male	432 (90.2)
Female	47 (9.8)
Age (years)	28.5±12.92
<b>Cause</b>	
Motor accident	338 (70.6)
Fall	87 (18.2)
Crushing blow	38 (7.9)
Other	16 (3.3)
<b>Eyesight</b>	
No light perception	383 (80.0)
Light perception	29 (6.1)
Hand motion	36 (7.5)
Finger count	31 (6.4)
<b>Fundus examination</b>	
Pale and edematous optic papilla	45 (9.4)
Normal optic papilla	434 (90.6)
<b>High-resolution computed tomography</b>	
Fracture of optic canal	403 (84.1)
No fracture of optic canal	76 (15.9)
<b>Magnetic resonance imagine</b>	
Hyperintensity of injured optic nerve	9 (39.1)
Normal optic nerve	14 (60.9)
<b>Interval between trauma and surgery</b>	
1–2 weeks	426 (88.9)
2 weeks–1 month	53 (11.1)

intracanalicular pressure and improving the delivery of nutrients to the optic nerve, which is critical for optic nerve function. However, many issues exist, such as surgical efficacy, operative indications, and surgical timing. Although the optimal timing of the operation is not clear, it is generally believed that the sooner the operation is performed, the better (11,12).

However, early surgery, especially within 3 days, is impractical for almost all patients in our hospital. Most patients in our department are transferred from other

hospitals for surgical treatment after diagnosis, which results in surgery delays >1 week after the injury. A few studies have reported the effectiveness of delayed decompression within several weeks after injury, but these studies have small sample sizes and the results are questionable (13-15).

The aim of the present study was to discuss the efficacy and safety of delayed optic decompression with corticosteroids 1 week after the onset of injury in 479 TON patients. We present the following article in accordance with the STROBE reporting checklist (available at <http://dx.doi.org/10.21037/atm-20-7810>).

## Methods

### Patients

A total of 479 patients with TON who underwent EOND with corticosteroids more than 1 week after injury at the Department of Neurosurgery, Tongren Hospital, from September 2008 to August 2017, were retrospectively analyzed. All patients were followed-up from 1 month to 46 months (average 10.8 months). Patients with bilateral TON or visual acuity (VA) better than 6/60 were excluded from the study. Additionally, blind patients who had been injured for more than 2 weeks or patients with residual vision for >1 month were not included.

All consecutive patients admitted to our hospital were diagnosed by impaired VA with an afferent pupillary defect in the involved eye after head injury. Full ophthalmologic examinations were undertaken to exclude post-traumatic globe disorders, such as open globe injury, traumatic cataract retinal detachment, and vitreous hemorrhage. High-resolution computed tomography (HRCT) scan was then performed with 1-mm sections through the optic canal to evaluate the orbit and optic canal. Optic nerve magnetic resonance imaging (MRI) scans were performed in 24 patients. The flash visual evoked potential was examined in 58 patients upon admission. The characteristics of patients with TON in the present study are shown in *Table 1*.

### Treatment protocol

Upon admission to our department, all patients were treated with high-dose methylprednisolone (1,000 mg/day) intravenously for 3 days. No serious side effects occurred in our series. If the VA did not improve to 6/60 after steroid treatment, EOND was then suggested. EOND was

**Table 2** VA of pretreatment and post-treatment among the different groups

VA pretreatment	VA post-treatment				
	NLP (n)	LP (n)	HM (n)	FC (n)	≥0.1 (n)
NLP [383]	247	19	78	38	1
LP [29]	0	6	13	7	3
HM [36]	0	0	8	21	7
FC [31]	0	0	0	13	18
Total [479]	247	25	99	70	29

FC, finger count; HM, hand motion; NLP, no light perception; VA, visual acuity.

performed on patients who agreed to undergo surgery. Oral vasodilators, intramuscularly injected with mecobalamin and hyperbaric oxygen, were administered for 2 weeks to improve VA following surgery. The present study was approved by the ethical committee of our hospital, and informed consent was obtained from each participant. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013).

### *Surgical technique*

EOND surgery was performed under general anesthesia, with controlled hypotension after routine preparation. Soaked gauze with 0.1% adrenaline and 1% dicitaine was used to constrict the blood vessels in the nasal cavity. Routine endoscopic ethmoidectomy and sphenoidotomy were performed first. The medial wall of the orbit and the orbital apex were confirmed and exposed. The orbital fasciae was protected from damage to prevent the herniation of intra-orbital fat blocking the operative cavity during surgery. The anterior wall of the sphenoid was then opened and enlarged, and the optic canal and carotid artery canal were identified. Under endoscopic observation, the medial, inferior, and superior walls of the bony optic canal were gently removed by a long hand microdrill with a diamond burr. Finally, the optic nerve sheath and the annulus of the Zinn ring were incised with a sharp sickle knife to expose the fasciculus opticus if necessary. In this case series, the incision was performed in only 38 patients who had intraoperative findings indicating obvious optic nerve swelling.

### *Visual assessments*

In the present study, VA was the main assessment criterion to evaluate the efficacy of EOND during the course of treatment. VA was considered to have an increase of 1 line or more on the Snellen visual chart, or an improvement from no light perception (NLP) to light perception (LP) or better, from LP to hand motion (HM) or better, from HM to finger count (FC) or better, or from FC to 6/60 or better.

Follow-up visits were scheduled 1 week, 1 month, and 3 months after discharge, and submitted to examine VA. The evaluation of VA was performed by ophthalmologist.

The VA of 49 patients (10.2%) at discharge, and the VA of 64 patients (13.4%) at 1 month was chosen as the final result because of loss to follow-up.

### *Statistical analysis*

The  $\chi^2$ -test was used to compare the difference in surgical efficacy between patients with residual vision versus those with NLP prior to surgery. Wilcoxon rank and tests of 2 groups were used to compare preoperative and postoperative VAs.

All statistical analyses were performed using SPSS version 22.0 (IBM, Armonk, NY, USA), and  $P < 0.05$  indicated statistical significance.

## **Results**

Clinical manifestations and examination data of patients are described in *Table 1*. A total of 479 patients with a diagnosis of TON underwent EOND in the present study (432 men and 47 women), with an age range from 6 to 61 years (average age  $28.5 \pm 12.92$  years). The main causes of TON were traffic accidents (338 cases), falls (87 cases), and crushing blows (38 cases). Transient loss of consciousness occurred in 91 cases. Of the 479 patients, 383 had NLP, 29 had LP, 36 had hand motion, and 31 had FC upon admission. The surgical timing from injury to surgery ranged from 7 to 31 days (average interval  $11.8 \pm 4.92$  days). A total of 426 patients underwent surgery within 2 weeks, and 53 patients underwent surgery within 1 month.

Of the 479 patients with TON, the final VA reached 6/60 or better in 29 patients, FC in 79 patients, HM in 99 patients, LP in 25 patients, and NLP in 247 patients. The postsurgical VA of different group was described in *Table 2*. The

**Table 3** Comparison of presurgical eyesight and postsurgical eyesight in patients with TON

Group	NLP (n)	LP (n)	HM (n)	FC (n)	≥0.1	Total (n)
Preoperative	383	29	36	31	0	479
Postoperative	247	25	99	79	29	479

Z=-12.636, P=0.000. Significant difference between presurgical eyesight and postsurgical eyesight in patients with traumatic optic neuropathy (TON) (P<0.01). Postoperative visual acuity (VA) improved significantly compared with preoperative VA. Wilcoxon rank and tests of 2 groups were applied for the comparison of preoperative and postoperative values. FC, finger count; HM, hand motion; NLP, no light perception.

**Table 4** Comparison of the postsurgical VA improvement rate in groups of traumatic optic neuropathy patients with different postoperative VAs

Group	Pretreatment	Improvement, post-treatment	VA improvement rate	$\chi^2$	P value
NLP	383	136	35.5	41.466	0.000
No NLP	96	69	71.9		

Comparison of the postoperative visual acuity (VA) improvement rate between the preoperative VA with the no light perception (NLP) group, and the preoperative VA with the no NLP group. VA improvement rate was much better in the preoperative VA with residual VA group than that in the preoperative VA with NLP group;  $\chi^2=41.466$ , P=0.00 (<0.01). The  $\chi^2$ -test was applied to compare VA improvement between the 2 groups.

postsurgical eyesight was significant better than presurgical eyesight (P<0.01) (Table 3). Of the 383 patients with NLP preoperatively, VA recovered in 136 patients (35.5%); of these 136 patients, 19 patients improved to LP, 78 to HM, 38 to FC, and 1 to a VA of 6/60.

Of the 96 patients who retained residual eyesight from LP to residual eyesight <0.1 preoperatively, 69 (71.9%) improved after EOND. There was a significant difference of improvement rate between the group with NLP before surgery and the group with residual eyesight (P<0.01) (Table 4).

Four cases of cerebrospinal fluid rhinorrhea (CSFR) occurred during surgery. All cases of CSFR were repaired by mucosal flap transplantation (the mucosa from excised middle turbinate), and the patients were confined to bed for 7–10 days; all of the patients recovered without complications.

Eight patients developed massive hemorrhage during surgery. Tela iodoformum was immediately inserted into the sphenoid sinus to stop the bleeding, and patients were immediately followed by digital subtraction angiography (DSA) examination.

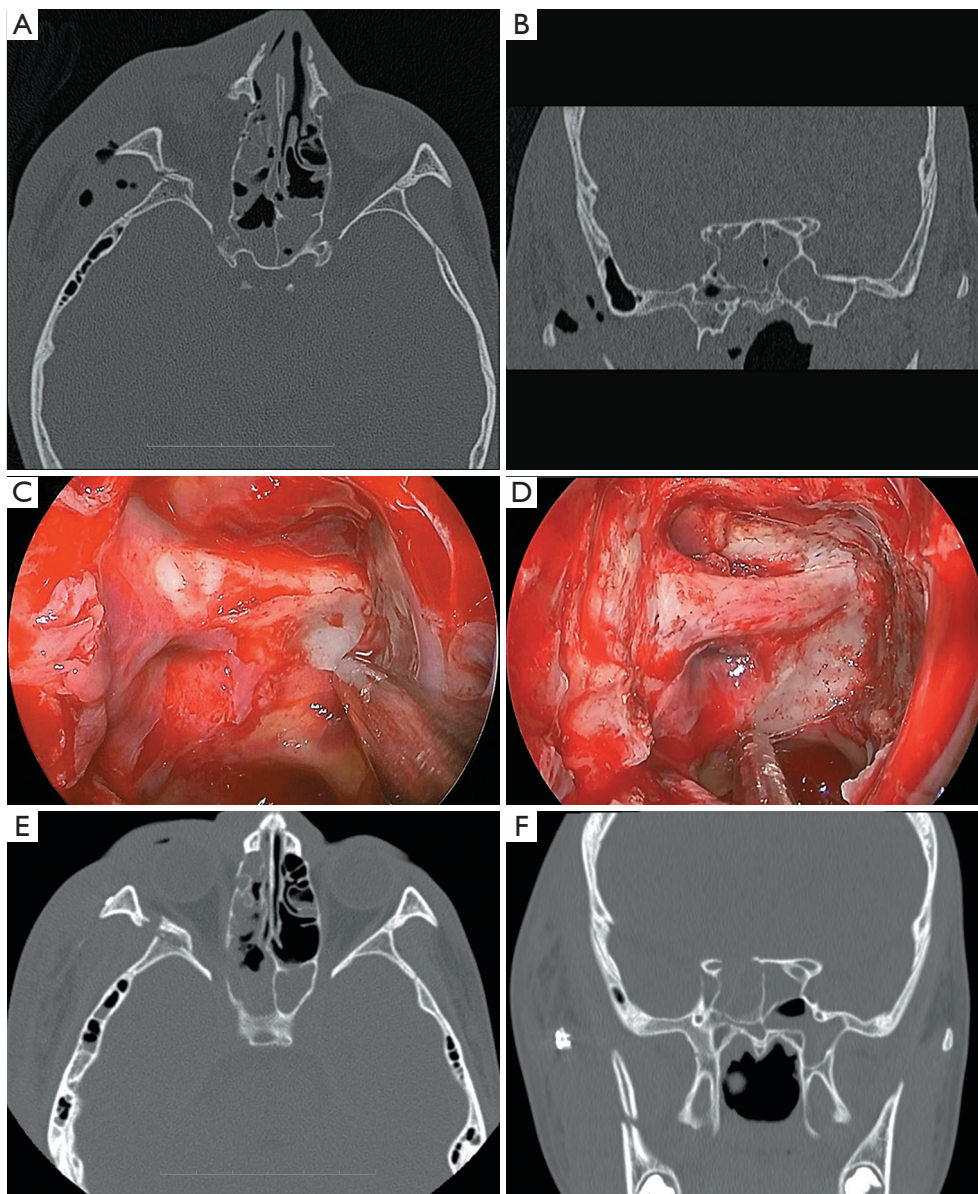
Of the 8 patients, 6 were diagnosed with carotid-cavernous fistula (CCF), and the other 2 patients were diagnosed with traumatic internal carotid artery aneurysm. Balloon embolization was used to treat CCF, and the Willis intracranial stent graft system (Microport, China) was used

for traumatic internal carotid artery aneurysms. All patients were discharged with no deficit in neurological function.

## Discussion

In this case series, 479 patients (432 men and 47 women) with a diagnosis of TON received corticosteroids and underwent EOND. The age range of the patients was 6–61 years (average age 28.5±12.92 years). The main causes of TON were traffic accidents (338 cases, 70.6%), falls (87 cases, 18.2%), and crushing blows (38 cases, 7.9%). Our data were consistent with those of previous studies (1,16–18). However, the main characteristic of the cases in the present study was the long-time interval; 1 week to 1 month (11.8±4.92 days) from injury to surgery. The results revealed that decompression plus corticosteroid therapy was beneficial for improving the VA of TON with total visual loss within 7 days and partial visual loss within 1 month, especially for the latter.

EOND could be undertaken as a major treatment for TON due to its advantages, such as minimal invasiveness and excellent visibility. Based on our experience with EOND since 2006, in the present study, we proposed for the first time, further decompressing the superior wall of the optic canal on the basis of the traditional decompression range to achieve more adequate decompression, especially for patients with bone fractures or impingement of the



**Figure 1** High-resolution horizontal (A) and coronal (B) scanning of the optic canal in a 11-year-old male with left traumatic optic neuropathy. Displacement of fractures from the planum sphenoidale and corresponding sphenoid sinus are evident. Intraoperative endoscopic image of the same patient (C) showed the fracture of the optic canal with no displacement. Wider optic decompression was performed, including the medial, inferior, and superior wall (D). Axial and coronal computed tomography scans (E,F) after surgery indicating decompression of the left optic nerve.

superior wall.

The optic nerve is easily damaged by blunt head trauma, especially in the intracanalicular segment, which is vulnerable to direct contusive forces on the optic canal and nerve or elastic deformations from surrounding bones. The bony fragments or hematomas can lead to secondary

swelling of nerves in the bony canal, a decrease in blood supply, and finally, axon loss (19,20). The force-bearing points are commonly located at the regions anterior and lateral to the eyebrow.

Horizontal HRCT and coronal optic canal HRCT are essential for detecting fractures of the canal and paranasal

sinuses, with reported rates ranging from 6% to 92% (21). CT scans provide a road map for surgery, and revealing anatomic variations in the sphenoid sinus, optic canal, and ethmoid sinus, especially the carotid artery canal and optic canal. Furthermore, a CT-based scoring system or risk-factor analysis can be used to predict the severity of VA and the prognosis of treatment (22-25).

Fractures of the optic canal and hemorrhage in the posterior group of the ethmoid sinuses are regarded as radiological signs of TON. The sensitivity of CT scans is influenced by several factors, such as the scanner machine, the thickness of scanning, the selection of a baseline scan, and the experience of the physician. Although no fractures of optic canal in HRCT scans, fractures could be found and confirmed during surgery, with the rate for missed fractures ranging from 16.1% to 75% (26-28). Preoperative HRCT images showed that 398 patients (83.1%) had fractures of the optic canal, but another 38 patients (7.9%) were confirmed to have fractures of the optic canal during surgery. Therefore, linear fractures, fractures with slight displacement and abnormal anatomy of the optic canal, and adjacent structures are more likely to be misdiagnosed or missed. By now, 1-mm sections through the optic canal and indirect signs on CT scans such as hemorrhage in the posterior ethmoid sinuses or sphenoid sinus may help to reduce the misdiagnosis of mission, but there is no effective to avoid.

Conventional MRI often shows normal optic nerves in patients with TON, which was unable to exhibit consistency with the clinical symptoms. In our study, only 5 patients (5/24) showed hyperintensity in injured optic nerves. MRI with diffusion weighted imaging and diffusion tensor imaging partly reflected the microstructural changes in optic nerves of TON patients. The mean apparent diffusion coefficient (ADC) was significantly decreased and was correlated with poor VA (29,30).

However, a higher ADC and lower fractional anisotropy were obtained in another report, which might be due to the period from injury to examination (31).

Because of the low incidence and variable therapeutic strategies of TON, the current relevant literature mainly comprises retrospective cohort studies, and none of the treatments has been accepted as universal management (7,8,32,33). Although spontaneous recovery of VA was observed in some cases, treatments consisting of endoscopic transethmoidal optic canal decompression (ETOCD) with a combination of steroids were considered more effective (33,34).

Adequate depression of the optic canal is the key point

of the surgical procedure, especially in areas where the optic nerve is compressed by bone fragments. Commonly, the medial and inferior walls of the optic canal are easily decompressed after exposure of the sphenoid sinus and orbital apex. However, the superior wall was difficult to remove because of the tight adherence to the dura of the anterior skull base. We began by gently grinding away the anterior cranial base, exposing the anterior cranial base dura, and then further grinding away the superior wall of the optic canal (*Figure 1*). Unlike the manipulation involved in removing the inferior and medial wall, the narrow space increased the occurrence of injuries to the dura and optic nerve. Although decompression of the superior wall might lead to a high risk of cerebrospinal fluid (CSF) leakage and injury to the brain tissues and arteries of the frontal lobe, it was avoidable by delicate actions using diamond drills by experienced surgeons. No CSF leakage or injury to the artery and subdural tissues occurred in our study during the decompression of the superior wall. Over the decompressive length of optic canal, the cranial and orbital foramina had to be fully decompressed, especially at the cranial foramen, which is prone to insufficient decompression.

Regarding the incision of the dural sheath in the surgical procedure, we do not advocate incising the dura sheath in all cases, only for patients with obvious hemorrhage and edema of the optic nerve. Indeed, such patients may be at risk of damage to the optic nerve or ophthalmic artery, CSF leakage, and secondary meningitis. Only 42 patients underwent incision of the sheath, even though no abovementioned complications occurred.

The time interval from injury to surgery remains controversial. Timely intervention could block the pathophysiological process and relieve nerve swelling and ischemia, possibly recovering the VA. Early intervention (within 3 days or even 7 days) with ETOCD or/and corticosteroid therapy has been proposed, and is regarded to yield a good prognosis (16,35-38). The efficiency rate in patients treated within 3 days was significantly higher than those in patients treated later than 7 days (63.6% *vs.* 35.7%) (36). However, if surgery was delayed, the operation could be completed for patients with NLP within 2 weeks and for patients with residual eyesight within 1 month, based on our experiences. Our study found that 35.5% patients (136/383) with blindness and 71.9% patients (69/96) with residual eyesight improved following EOND plus corticosteroids. According to the current literature, delayed operation time will affect the surgical treatment effect of ton. However, due to the unknown

pathological and physiological mechanism of TON and many influencing factors, it is difficult to confirm this conclusion by randomized controlled trials.

Although the percentage of improvement was not high relative to that of other studies, the majority of patients underwent surgery and corticosteroid therapy within 7 days or less. In addition to the surgical timing, the degree of optic nerve damage may vary, even if the patients all present with the same degree of blindness (8,9).

Compared with surgical timing, the baseline VA is a more important factor affecting prognosis. Patients with residual eyesight have more surviving RGCs and less functional loss of the optic nerve, which indicates greater potential for recovery after medical intervention (39,40).

Satisfactory postoperative results could be obtained in patients with residual eyesight before surgery (41). In our study, the improvement rate of patients with residual eyesight was significantly better than that of patients with blindness, even though the surgery on the former could be delayed until 1 month from onset. Only 136 patients (35.5%) with blindness improved after EOND, which is similar to the results of other investigations reporting improvement rates around 10–30%, but not as high as the 46.9% reported in another study (34,42–44).

Intraoperative CSF leakage is common EOND surgery, especially in patients with anterior skull fracture. Fully exposing the fistula, covering the mucosa, and filling the area with a collagen sponge, followed by patient confinement to bed for 7–10 days after surgery, can successfully repair CSF leakage. Except for the skull fractures, iatrogenic insult was another reason for CSF leakage, with a reported prevalence of 1.2%, which is higher than the 0.13% generally reported for endoscopic sinus surgery (45,46). For the patients with history of cerebrospinal fluid rhinorrhea before operation and serious fracture of anterior skull base on CT scans, it is necessary to operate carefully and avoid removing the displaced fracture bones.

Another rare but most serious complication is hemorrhage caused by CCF and traumatic aneurysm. Bleeding often occurs when the bone fragments are removed from the medial wall of the sphenoid sinus. In the present study, the only measure taken immediately was to inject the sinus with tela iodoformum, followed by DSA examination and interventional therapy. All patients recovered after embolization and were discharged without complications. In some cases, in the early stage after injury, no classic symptoms were evident, but if severe fractures or displacement of the medial wall of the

sphenoid sinus, expanded superior ophthalmic vein, or broadened cavernous sinus existed, computed tomography angiography (CTA) or DSA examination was required to rule out CCF (47). If there were severe fractures in the area around the optic canal that were associated with traumatic subarachnoid hemorrhage, CTA was required to exclude traumatic aneurysm (48). Serious sphenoid sinus fractures and subarachnoid hemorrhage on CT scans must be paid enough attentions and CTA or DSA need to be adopted to exclude the possibility of vascular injury.

Based on the findings of the present study, once diagnosed with TON, even with no definite fracture of the optic canal on HRCT scan, EOND surgery should be performed as soon as possible. However, if surgery cannot be undertaken within 7 days, EOND could still be used as a safe and effective therapeutic approach to improve VA in patients with NLP within 2 weeks and those with residual eyesight within 1 month. Wider decompression, including the superior wall of the optic canal, should be achieved whenever possible, but surgeons must be skilled in endoscopic surgery techniques to avoid complications. The superiority of wider decompression needs to be further demonstrated in large-sample, controlled, prospective studies.

## Conclusions

Preoperative early surgical decompression plus steroid therapy for TON is regarded as an effective protocol, in which a surgical interval time of 7 days and basal VA are key factors influencing prognosis. However, delayed operation plus a megadose of steroid could still be regarded as an effective remedial measure within 2 weeks for patients with NLP and within 1 month for patients with residual eyesight.

## Acknowledgments

*Funding:* The present study was supported by the Beijing Hospitals Authority's Ascent Plan (No. DFL20190201).

## Footnote

*Reporting Checklist:* The authors have completed the STROBE reporting checklist. Available at <http://dx.doi.org/10.21037/atm-20-7810>

*Data Sharing Statement:* Available at <http://dx.doi.org/10.21037/atm-20-7810>

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <http://dx.doi.org/10.21037/atm-20-7810>). The authors have no conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The present study was approved by the ethical committee of our hospital, and informed consent was obtained from each participant. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013).

*Open Access Statement:* This is an Open Access article distributed in accordance with the Creative Commons Attribution-NonCommercial-NoDerivs 4.0 International License (CC BY-NC-ND 4.0), which permits the non-commercial replication and distribution of the article with the strict proviso that no changes or edits are made and the original work is properly cited (including links to both the formal publication through the relevant DOI and the license). See: <https://creativecommons.org/licenses/by-nc-nd/4.0/>.

## References

1. Wang BH, Robertson BC, Giroto JA, et al. Traumatic optic neuropathy: a review of 61 patients. *Plast Reconstr Surg* 2001;107:1655-64.
2. al-Qurainy IA, Stassen LF, Dutton GN, et al. The characteristics of midfacial fractures and the association with ocular injury: a prospective study. *Br J Oral Maxillofac Surg* 1991;29:291-301.
3. Holt GR, Holt JE. Incidence of eye injuries in facial fractures: an analysis of 727 cases. *Otolaryngol Head Neck Surg* 1983;91:276-9.
4. Zachariades N, Papavassiliou D, Christopoulos P. Blindness after facial trauma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;81:34-7.
5. Sarkies N. Traumatic optic neuropathy. *Eye (Lond)* 2004;18:1122-5.
6. Chen YR, Breidahl A, Chang CN. Optic nerve decompression in fibrous dysplasia: indications, efficacy and safety. *Plast Reconstr Surg* 1997;99:22-30.
7. Chaon BC, Lee MS. Is there treatment for traumatic optic neuropathy? *Curr Opin Ophthalmol* 2015;26:445-9.
8. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy: an evolving understanding. *Am J Ophthalmol* 2011;151:928-33.e2.
9. Ropposch T, Steger B, Meco C, et al. The effect of steroids in combination with optic nerve decompression surgery in traumatic optic neuropathy. *Laryngoscope* 2013;123:1082-6.
10. Yang WG, Chen CT, Tsay PK, et al. Outcome for traumatic optic neuropathy-surgical versus nonsurgical treatment. *Ann Plast Surg* 2004;52:36-42.
11. Joseph MP, Lessell S, Rizzo J, et al. Extracranial optic nerve decompression for traumatic optic neuropathy. *Arch Ophthalmol* 1990;108:1091-3.
12. Li KK, Teknos TN, Lai A, et al. Traumatic optic neuropathy: results in 45 consecutive surgically treated patients. *Otolaryngol Head Neck Surg* 1999;120:5-11.
13. Kountakis SE, Maillard AA, Urso R, et al. Endoscopic approach to traumatic visual loss. *Otolaryngol Head Neck Surg* 1997;116:652-5.
14. Girard BC, Bouzas EA, Lamas G, et al. Visual improvement after transethmoid-sphenoid decompression in optic nerve injuries. *J Clin Neuroophthalmol* 1992;12:142-8.
15. Thakar A, Mahapatra AK, Tandon DA. Delayed optic nerve decompression for indirect optic nerve injury. *Laryngoscope* 2003;113:112-9.
16. Song Y, Li H, Ma Y, et al. Analysis of prognostic factors of endoscopic optic nerve decompression in traumatic blindness. *Acta Otolaryngol* 2013;133:1196-200.
17. Li H, Zhou B, Shi J, et al. Treatment of traumatic optic neuropathy: our experience of endoscopic optic nerve decompression. *J Laryngol Otol* 2008;122:1325-9.
18. Lee V, Ford RL, Xing W, et al. Surveillance of traumatic optic neuropathy in the UK. *Eye (Lond)* 2010;24:240-50.
19. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. *Surv Ophthalmol* 1994;38:487-518.
20. Sofferan RA. The recovery potential of the optic nerve. *Laryngoscope* 1995;105:1-38.
21. Cook MW, Levin LA, Joseph MP, et al. Traumatic optic neuropathy. a meta-analysis. *Arch Otolaryngol Head Neck Surg* 1996;122:389-92.
22. Bodanapally UK, Van der Byl G, Shanmuganathan K, et al. Traumatic optic neuropathy prediction after blunt facial trauma: derivation of a risk score based on facial CT findings at admission. *Radiology* 2014;272:824-31.
23. Reddy RP, Bodanapally UK, Shanmuganathan K, et al. Traumatic optic neuropathy: facial CT findings affecting visual acuity. *Emerg Radiol* 2015;22:351-6.
24. Ong HS, Qatarneh D, Ford RL, et al. Classification of



- orbital fractures using the AO/ASIF system in a population surveillance cohort of traumatic optic neuropathy. *Orbit* 2014;33:256-62.
25. Zimmerman R, Rana M, Schumann P, et al. Diagnosis and treatment of optic nerve trauma. *Facial Plast Surg* 2014;30:518-27.
  26. Imachi Y. Clinical and patho-histological investigations on optic nerve lesions caused by head injuries. *Nippon Ganka Gakkai Zasshi* 1967;71:1874-908.
  27. Gupta AK, Gupta AK, Gupta A, et al. Traumatic optic neuropathy in pediatric population: early intervention or delayed intervention? *Int J Pediatr Otorhinolaryngol* 2007;71:559-62.
  28. Wohlrab TM, Maas S, de Carpentier JP. Surgical decompression in traumatic optic neuropathy. *Acta Ophthalmol Scand* 2002;80:287-93.
  29. Bodanapally UK, Shanmuganathan K, Shin RK, et al. Hyperintense optic nerve due to Diffusion Restriction: Diffusion-Weighted Imaging in Traumatic optic neuropathy. *ANJR Am J Neuroradiol* 2015;36:1536-41.
  30. Bodanapally UK, Kathirkamanathan S, Geraymovych E, et al. Diagnosis of traumatic optic neuropathy: application of diffusion tensor magnetic resonance imaging. *J Neuroophthalmol* 2013;33:128.
  31. Yang QT, Fan YP, Zou Y, et al. Evaluation of traumatic optic neuropathy in patients with optic canal fracture using diffusion tensor magnetic resonance imaging: a preliminary report. *ORL J Otorhinolaryngol Relat Spec* 2011;73:301-7.
  32. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. *Cochrane Database Syst Rev* 2013;6:CD006032.
  33. Volpe NJ, Nicholas LL. How should patients with indirect traumatic optic neuropathy be treated. *J Neuroophthalmol* 2011;31:169-74.
  34. Chou PI, Chen YC, Sadun AA, et al. Clinical experiences in the management of traumatic optic neuropathy. *Neuro-Ophthalmology* 1996;16:325-36.
  35. Chen M, Jiang Y, Zhang J, et al. Clinical treatment of traumatic optic neuropathy in children: summary of 29 cases. *Exp Ther Med* 2018;16:3562-6.
  36. Yu B, Ma Y, Tu Y, et al. The outcome of endoscopic transtethmosphenoid optic canal decompression for indirect traumatic optic neuropathy with no-light-perception. *J Ophthalmol* 2016;2016:6492858.
  37. Gupta D, Gadodia M. Transnasal endoscopic optic nerve decompression in posttraumatic neuropathy. *Indian J Otolaryngol Head Neck Surg* 2018;70:49-52.
  38. Emanuelli E, Bignami M, Digilio E, et al. Post-traumatic optic neuropathy: our surgical medical protocol. *Eur Arch Otorhinolaryngol* 2015;272:3301-9.
  39. Peng A, Li Y, Hu P, Wang Q. Endoscopic optic nerve decompression for traumatic optic neuropathy in children. *Int J Pediatr Otorhinolaryngol* 2011;75:992-8.
  40. Yu-Wai-Man P, Griffiths PG. Surgery for traumatic optic neuropathy. *Cochrane Database Syst Rev* 2013;6:CD005024.
  41. Ma YJ, Yu B, Tu YH, et al. Prognostic factors of thans-ethmosphenoid optic canal decompression for indirect traumatic optic neuropathy. *Int J Ophthalmol* 2018;11:1222-6.
  42. Kountakis SE, Maillard AA, EI-Harazi SM, et al. Endoscopic optic nerve decompression for traumatic blindness. *Otolaryngol Head Neck Surg* 2000;123:34-7.
  43. Li HB, Shi JB, Cheng L, et al. Salvage optic nerve decompression for traumatic blindness under nasal endoscopy: risk and benefit analysis. *Clin Otolaryngol* 2007;32:447-51.
  44. Jiang RS, Hsu CY, Shen BH. Endoscopic optic nerve decompression for the treatment of traumatic optic neuropathy. *Rhinology* 2001;39:71-4.
  45. Krings JG, Kallogjeri D, Wineland A, et al. Complications of primary and revision functional endoscopic sinus surgery for chronic rhinosinusitis. *Laryngoscope* 2014;124:838-45.
  46. Dhaliwal SS, Sowerby LJ, Rotenberg BW. Timing of endoscopic surgical decompression in traumatic optic neuropathy: a systematic review of the literature. *Int Forum Allergy Rhinol* 2016;6:661-7.
  47. Tjoumakaris SI, Jabbour PM, Rosenwasser RH. Neuroendovascular management of carotid cavernous fistulae. *Neurosurg Clin N Am* 2009;20:447-52.
  48. Kang Z, Li J, Zou Y, et al. Diagnosis and treatment of traumatic optic neuropathy with carotid artery cavernous segment pseudoaneurysm. *Laryngoscope* 2013;123:2591-7.
- (English Language Editor: R. Scott)

**Cite this article as:** Zhao SF, Yong L, Zhang JL, Wu JP, Liu HC, Sun S, Song GD, Ma JM, Kang J. Role of delayed wider endoscopic optic decompression for traumatic optic neuropathy: a single-center surgical experience. *Ann Transl Med* 2021;9(2):136. doi: 10.21037/atm-20-7810