# Traumatic Optic Neuropathy

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Yun Zhang and Meixia Zhang

**Abstract**

Traumatic optic neuropathy (TON) refers to any injury to the optic nerve secondary to trauma. Injury may be direct or indirect and visual loss may be partial or complete. Direct TON results from an anatomical nerve disruption by penetrating orbital trauma. Indirect injury results from transmission of forces to the nerve. The prognostic value in knowing that an injury was direct or indirect is unclear, and the mechanism of injury is not understood. Also, there are no confirmed protocols for prevention, mitigation, or treatment. Current therapeutic modalities include observation alone, systemic high-dose corticosteroid administration, and/or surgical optic nerve decompression. This chapter includes four cases with brief descriptions, illustrating figures and personal tips and pearls, aiming to provide a guide of diagnosis and management of traumatic optic neuropathy.

**Keyword**

Traumatic optic neuropathy

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## 11.1 Introduction

Traumatic optic neuropathy (TON) is a clinical diagnosis when there is evidence of optic neuropathy following a history of a blunt or a penetrating trauma. This condition may be associated with multisystem trauma especially head injury which needs attention first. The location of trauma-induced injury to the optic nerve can occur anywhere along the nerve's intraorbital to intracranial length. Approximately 0.5–5% of patients with closed head injuries have damage to the visual pathways [1, 2] and 2.5% of patients with midfacial fracture. Eighty-five percent of patients with TON are seen in middle-aged males. The majority of TON causes are motor vehicles and bicycle accidents followed by falls and assaults. TON also can be diagnosed with penetrating trauma (stab wounds, gunshot wounds, foreign bodies) and recreational sports (e.g., paintball injury).

Optic nerve injuries are classically divided into two categories [3]: direct and indirect trauma. Direct trauma results from a tear of the optic nerve itself or a tear caused by a fracture fragment or other foreign body, as well as a compression injury caused by a fracture of the optic canal, intraorbital or intrathecal hemorrhage. Forehead trauma is the most common, especially the contusion of the lateral brow arch. It is supposed to be related to the damage caused by shear force acting on the optic nerve

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or the attachment point of the trophoblastic vessels in the optic nerve canal. Based upon fundus features, TON can be divided into anterior optic neuropathy, posterior optic neuropathy, and optic disc avulsion. Optic disc change that can be found in fundus examination is called anterior indirect trauma, while the most common is posterior indirect trauma in clinic, which is traumatic loss of vision which occurs without external or initial ophthalmoscopic evidence of injury to the eye or its nerve. The avulsion of the optic nerve is caused by the extreme rotation and forward displacement of the eyeball or extrusion that causes sudden increase in intraocular pressure resulting in rupture of the sieve plate; or the optic nerve is pulled backward by an orbital perforation injury, which causes the optic nerve to be strongly pulled back from the scleral canal and a backward dislocation.

During craniocerebral trauma, force applied to the superior orbital rim can be transferred and concentrated on the orbital roof and optic canal. The resultant percussive forces can damage the nerve at transitions between mobile and fixed segments. This commonly occurs at the junction of the intraorbital and intracanalicular segments and results in compression and disruption of pial vessels within the canal, affecting vascular supply of the optic nerve.

Due to an immediate disruption (direct trauma) or mechanical shearing (indirect trauma) to the optic nerve, the primary damage then occurs. Following inflammation and vascular dysfunction gives rise to the secondary damage. Though the primary and secondary pathophysiologic mechanisms of injury differ greatly, patients often suffered damages of both.

The management of TON should be a multidisciplinary approach involving the ophthalmologist, physician, neurosurgeon, and an otorhinolaryngologist. The treatment of TON is somewhat controversial. The optimum management protocol is yet to be elucidated as there is paucity of prospective large-scale clinical trials. The International Optic Nerve Trauma Study was organized to help clarify the value of different treatments of TON, since the natural prognosis of traumatic optic neuropathy is generally poor. Different TON should be given different treatment, but for optic nerve laceration and optic nerve head avulsion, there is no effective treatment. For optic nerve sheath hematoma, optic nerve sheath fenestration may be helpful in the acute stage if optic neuropathy is progressing and no other cause is evident. Effective treatment of posterior indirect TON is, at best, extremely limited. In the vast majority of cases, observation alone is recommended. High-dose corticosteroids should never be offered by ophthalmologists to patients with concomitant traumatic brain injury (TBI) or if the TON is older than 8 h. If steroids are considered (no evidence of TBI, injury within 8-h window, no medical comorbidities), the lack of definitive therapeutic evidence and significant side effects must be discussed with the patient and/or family and the primary team. And for the bone impingement of the optic canal, endoscopic optic canal and orbital apex decompression may be offered in select cases, especially if the optic neuropathy is progressive. However, this option should be approached with extreme caution because of the proximity to the cavernous sinus and carotid siphon and possible bony instability of the skull base. The procedure should only be performed by an otolaryngologist experienced in stereotactic endoscopic sinus and skull base surgery. The patient and/or family should also be informed that there is no definitive data that proves efficacy of this procedure in TON and that optic canal decompression may result in additional damage to the intracanalicular optic nerve.

## 11.2 Case #1: CRAO and CRVO in a Case of TON

### 11.2.1 Case Description

A 17-year-old man was struck in the left eye while playing football, and he immediately experienced severe loss of vision in the eye. On examination of the left eye, vision was no

light perception (NLP). The intraocular pressure was 16.5 mmHg. The pupil was round and reacted consensually but not directly to light. Relative afferent papillary dilatation (RAPD) was positive. Fundus examination showed white-out retina and a cherry-red spot at the macula (Fig. 11.1). Computed tomography (CT) scan of orbit showed that the optic nerve was bulky and no evidence of bony fracture (Fig. 11.2). All systemic investigations including cardiology workup were normal. The patient received intravenous bolus therapy with methylprednisolone 1 g intravenously (IV) for 3 days followed by oral prednisolone. The patient subsequently failed to regain any vision in the left eye. On follow-up after 1 month, there was no improvement of vision in the left eye. But during that time, the white-out retina regained its normal color except in the macular area and the area between the optic disc and macula (Fig. 11.3).

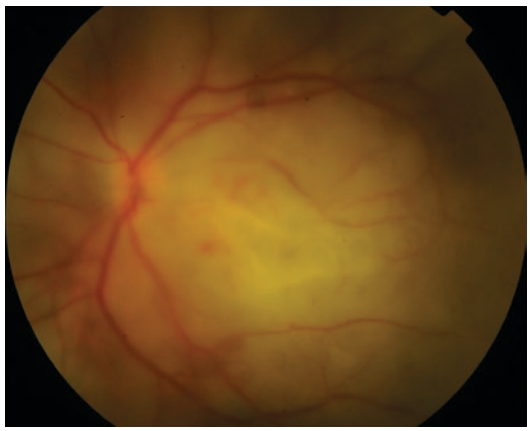

**Fig. 11.1** Fundus photograph after 1 day of trauma shows cherry red spot and retinal edema

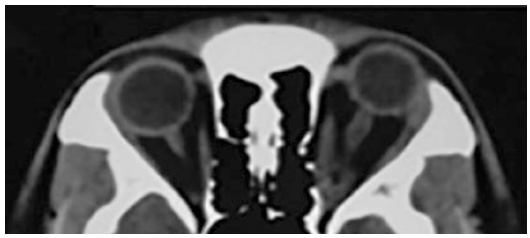

**Fig. 11.2** CT scan of orbit shows bulky optic nerve

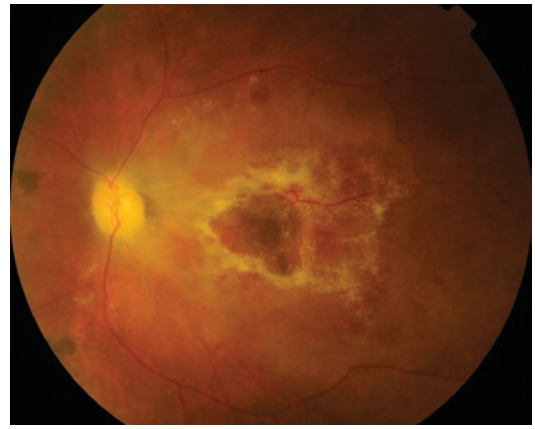

**Fig. 11.3** Fundus photograph after 1 month of trauma shows white macula and pale optic disc

### 11.2.2 Tips and Pearls

Coincident occlusion of CRAO and CRVO is a rare event in ocular trauma. The pathophysiology of the occlusion might be endothelial destruction because of acute extension of retinal vessels caused by sudden trauma and deformations of the eyes. Endovascular rupture is a clear cause of vascular occlusion caused by thrombosis in the human body. Local vasospasm also might be the reason of arterial occlusion and thrombosis, which is a natural steady-state response to trauma [4–6].

It is possible that in some of these cases, the arterial and venous occlusions may not have occurred simultaneously. This case highlights the need for clinicians to be aware of the potential for blunt ocular trauma to cause optic nerve damage and retinal vessel occlusion. And patient with the combination of CRAO and CRVO should be different from the following diseases with systemic disorders, particularly leukemia, hemoglobinopathies, septic cavernous sinus thrombosis, subacute bacterial endocarditis, systemic lupus erythematosus, syphilis, and temporal arteritis, Wegener's granulomatosis, homocystinuria, mitral valve prolapse, atherosclerosis, migraine, sickle cell diseases, Henoch-Schonlein purpura, etc.

## 11.3 Case #2: A Case with Indirect Traumatic Optic Neuropathy

### 11.3.1 Case Description

A 71-year-old man accidentally hurt his right eye with a spade while he was working, and immediately right eye bleeding, vision loss, and headache occurred. He was sent to the hospital for a series of eye examinations. On examination of the right eye, visual acuity was 0.02. The intraocular pressure was 36.1 mmHg. The swelling of the right eyelid is obvious, and the upper eyelid is covered by subcutaneous congestion. About 2 mm skin laceration was seen at the proximal canthus, which had been scabbed. Conjunctiva was hyperemia and edema, irregularly laceration of the conjunctiva from the limbus of cornea at 4 o'clock on the nasal side, and suspected scleral laceration at the corresponding location. The nasal corneal epithelium has flap defect and mild corneal edema. The depth of the anterior chamber is different. Partial lens was adjacent to the corneal endothelium. The pupil is not round, obliquely ellipse, and about the size of 4 × 5 mm, and light reflection disappears. The upper equatorial part of the lens is visible in the pupillary area, the lens is opaque, and the remaining structure is not clear. After admission, anterior segment photography showed conjunctival hemorrhage and edema, the pupil not round, and diameter about 8 mm (Fig. 11.4). P-VEP showed P100 latency was delayed at 60′ (60′ was 117 ms) (Fig. 11.5). Type-B ultrasonic showed the choroidal thickening of the right eye (Fig. 11.6). Orbital CT found the right eyelid swelling, eye ring rough and blurred, right anterior chamber shallowed, lens shifted outward and

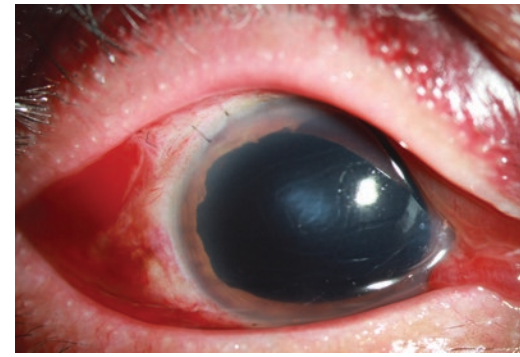

**Fig. 11.4** Conjunctival hemorrhage and edema, pupil not round, and diameter about 8 mm

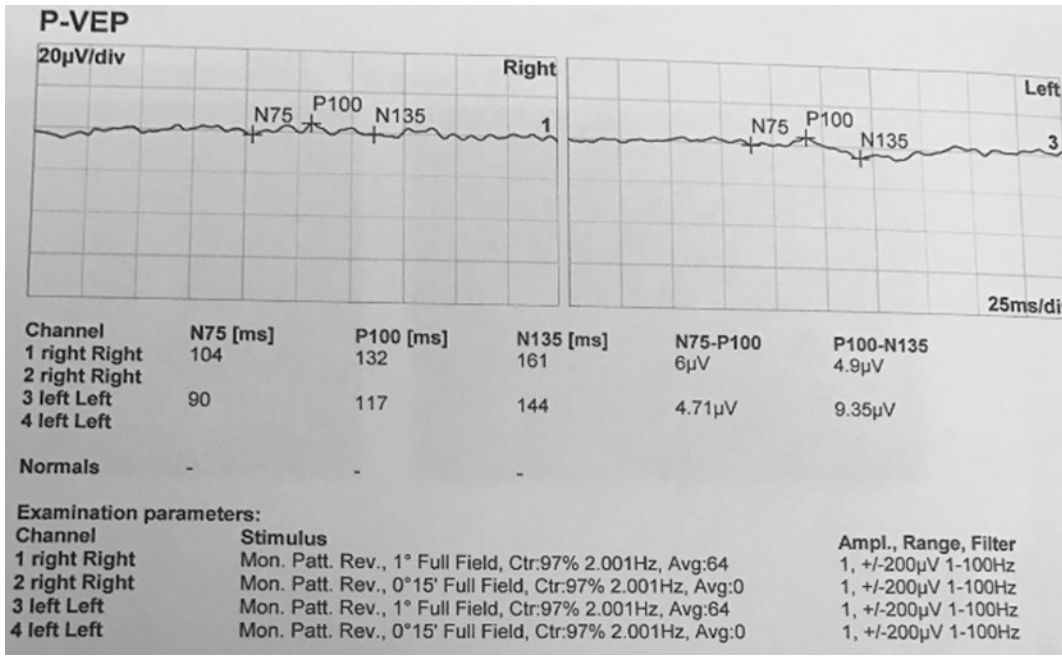


**Fig. 11.5** P-VEP showed P100 latency was delayed at 60′ (60′ was 117 ms)

slightly rotated, and no obvious abnormality in the vitreous body. Local suspicious discontinuity of the medial wall of the right orbit, fracture needed to exclude. The right optic nerve is rough, and the medial rectus muscle is slightly rough (Fig. 11.7). The patient was diagnosed with blunt contusion, subluxation of lens, traumatic cataract, secondary glaucoma, and traumatic optic neuropathy in the right eye. The patient received therapy with surgical operation of subluxation of the lens and traumatic cataract in the right eye. Then oral medicines of methycobal and citicoline sodium tablets were given in treatment of TON. Now, the patient is still being followed up.

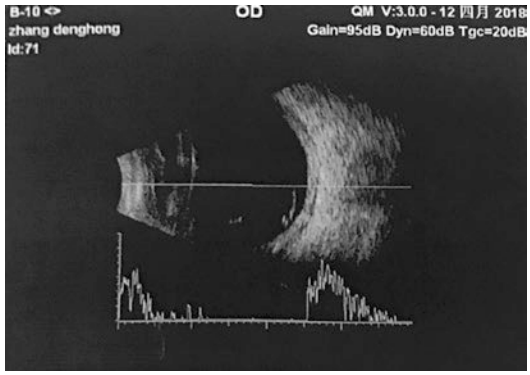


**Fig. 11.6** Type-B ultrasonic showed the choroidal thickening of the right eye

## 11.3.2 Tips and Pearls

According to the definition, indirect traumatic optic neuropathy is a condition that can be clinically diagnosed as optic nerve injury. The patient had a typical history of trauma and clinical feature of visual impairment, dyschromatopsia, visual field impairment, visual evoked potential abnormality, and relative afferent pupillary defect (RAPD).

It is worth noting that RAPD (+) may not exist in patients with binocular optic nerve damage. The optic nerve may perform normally under direct ophthalmoscope while also may gradually develop to paleness or atrophy. Automated computer visual field should also be tested clinically; however, it is possible that the patient's vision is too poor to get valuable results.

In most patients, visual evoked potential (VEP) detection is not required to establish a diagnosis. Especially, VEP may provide confirmatory data and predictive significance where there is an intractable case. In patients with better optic nerve function as reflected in VEP examination, partial or complete recovery of visual acuity is slightly more likely. In addition, VEP may also help in determining treatment options in emergency situations.

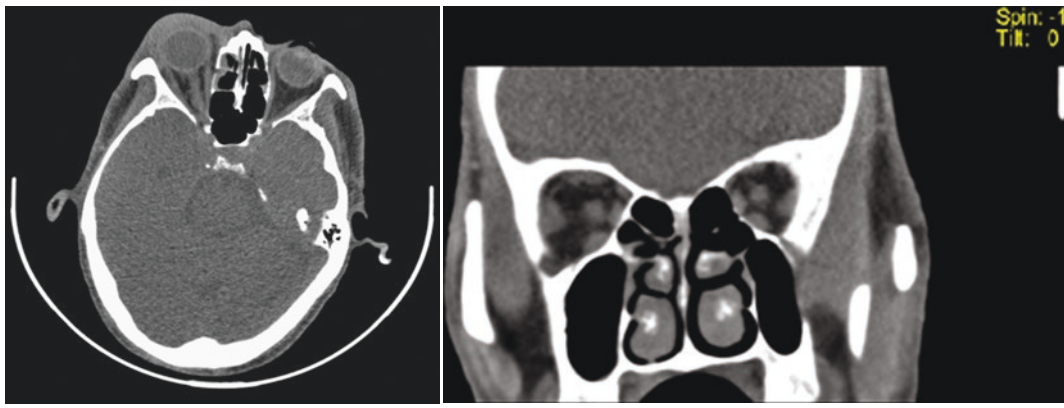


**Fig. 11.7** Orbital CT found the right eye eyelid swelling, eye ring rough and blurred, right anterior chamber shallowed, lens shifted outward and slightly rotated, and no obvious abnormality in the vitreous body. Local suspicious discontinuity of the medial wall of the right orbit, fracture needed to exclude. The right optic nerve is rough, and the medial rectus muscle is slightly rough

Several imaging examinations such as CT, MRI, intraorbital ultrasonography, and craniocerebral imaging can help to judge the degree of trauma and can detect other facial or intracranial injuries, hematoma, and orbital bone fragments related to the injury. The ophthalmologic examination should be carried out as early as possible for the patients with coma and craniocerebral injury to discover and treat the optic nerve injury in time.

## 11.4 Case #3: A Case with Indirect Traumatic Optic Neuropathy

### 11.4.1 Case Description

An 8-year-old girl was struck in her left eye because of motorcycle injury, and she immediately experienced severe loss of vision in the left eye. She was sent to the emergency ophthalmology for a series of eye examinations. On examination of the left eye, visual acuity was 0.06. Binocular corneal transparency, KP(−), normal anterior chamber depth, AR(−), round pupil and diameter approximately 3 mm, direct light reflection sensitivity. Relative afferent papillary dilatation (RAPD) was present. Binocular lens was transparent and no visible bleeding and exudation. Anterior segment photography showed the left eyelid slightly scraped, the pupil dilated medically, and diameter about 7 mm (Fig. 11.8). Perimetry was generally normal (Fig. 11.9). Type-B ultrasonic, OCT and SLO showed no obvious abnormality (Figs. 11.10, 11.11 and 11.12). But P-VEP showed that P100 latency was obviously delayed at 60′ and 15′ (60′ was 129 ms, 15′ was 155 ms) (Fig. 11.13). The amplitude was reduced compared with the right eye. We gave orbital MRI to the patient, and the result showed the anteroposterior diameter of the left eye is shorter, while the intraocular structure was not abnormal, and the bilateral optic nerve was not thickened or thinned (Fig. 11.14). The diagnosis of indirect traumatic optic neuropathy was made. The patient received intravenous bolus therapy with dexamethasone 15 mg intravenously (IV) for 5 days and afterward gradually reduced to dexamethasone 10 mg intravenously (IV) for 5 days and 5 mg for 5 days, followed by oral methycobal and citicoline sodium tablets for 30 days. One week later, left eye visual acuity was 0.08. Now, the patient is still being followed up.

### 11.4.2 Tips and Pearls

This case with traumatic optic neuropathy has no direct imaging evidence of optic nerve injury. It has been reported that in 1/3 of the patients with

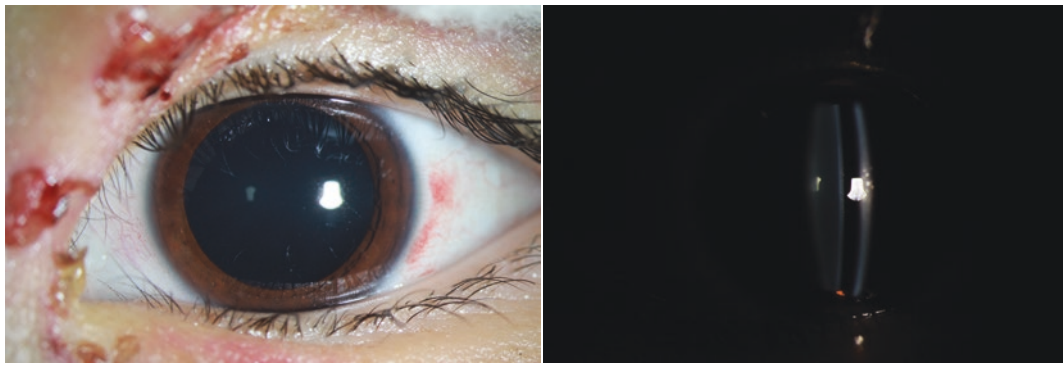

**Fig. 11.8** The left eyelid slightly scraped, the pupil dilated medically, and diameter about 7 mm lens was transparent

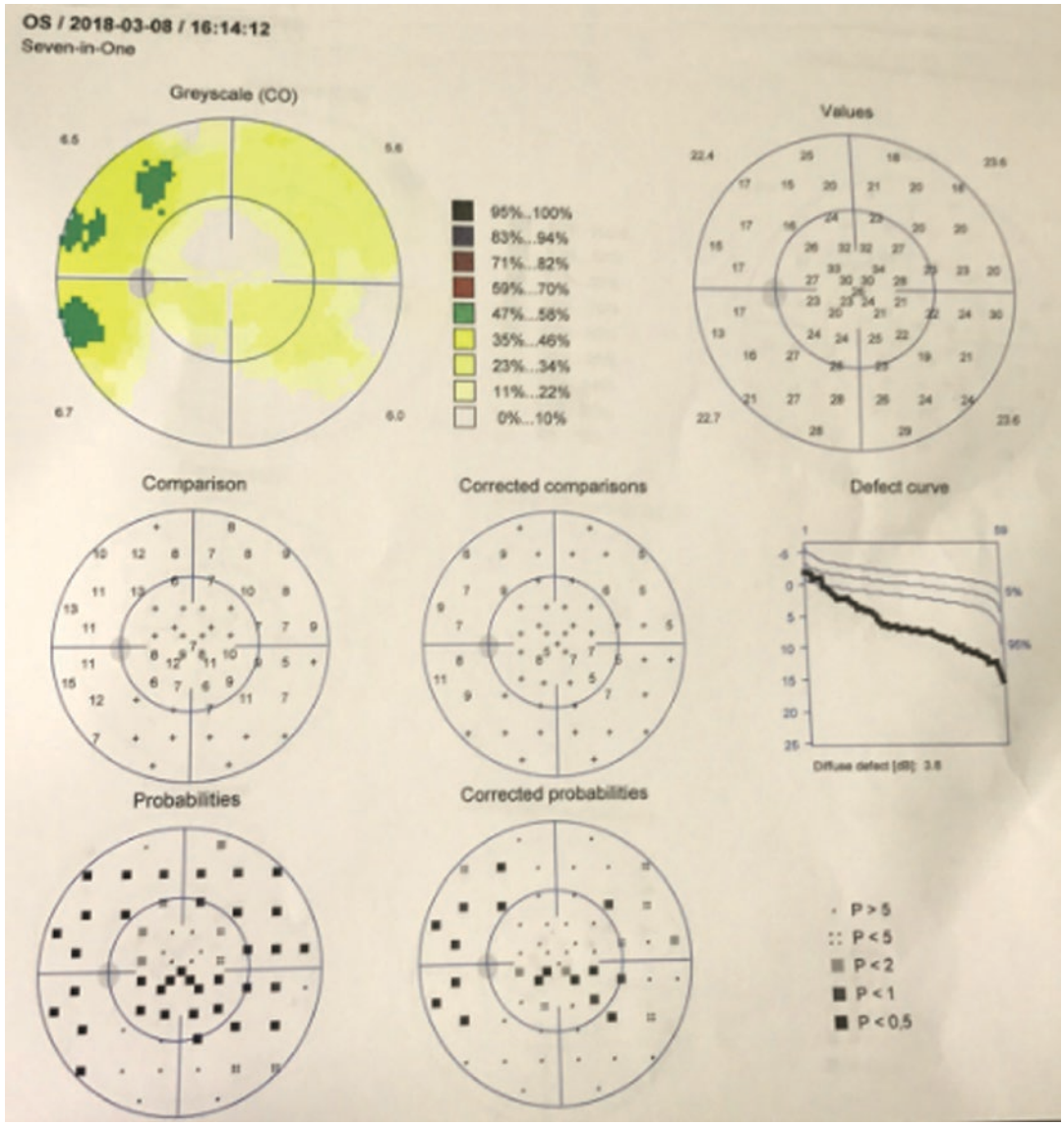


**Fig. 11.9** Perimetry was general normal

optic nerve injury, the neuroimaging manifestations were normal, but the visual improvement was slight after treatment.

The optical coherence tomography (OCT) of optic disc is an important examination to evaluate the structure of retinal nerve fiber layer (RNFL). It can evaluate whether the retina around the optic disc of the left eye is thinner than that of the right eye and whether there is a permanent loss of retinal nerve fiber layer. Therefore, we need to follow up regularly for the next 6 months to assess the changes in the visual acuity and disc color of the patient's left eye.

The loss of RNFL, the final result of traumatic optic neuropathy, reveals a common pathological feature: neuronal atrophy and apoptosis.

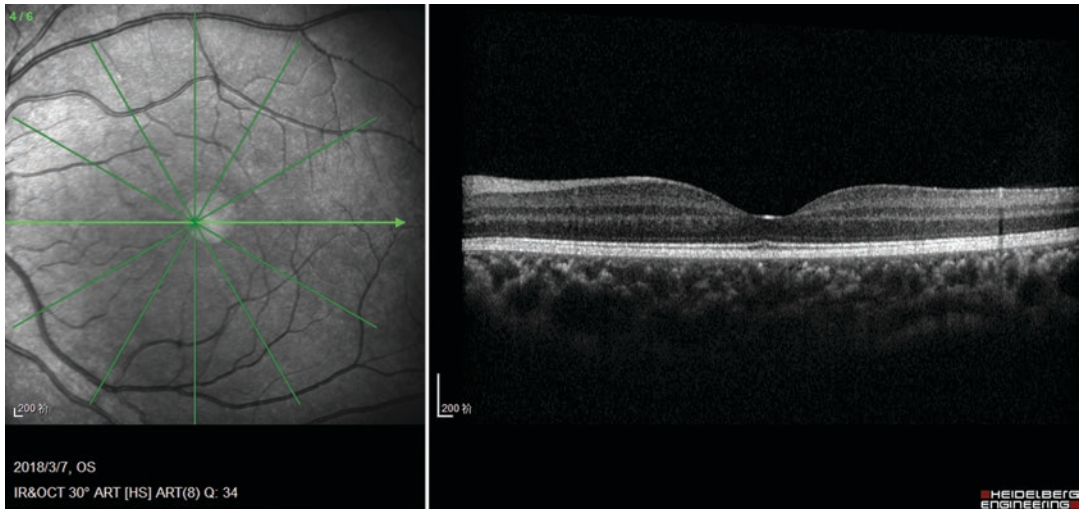


**Fig. 11.11** OCT: normal

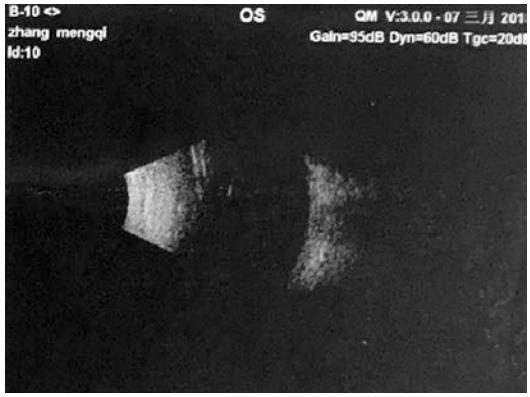


**Fig. 11.10** Type-B ultrasonic: normal

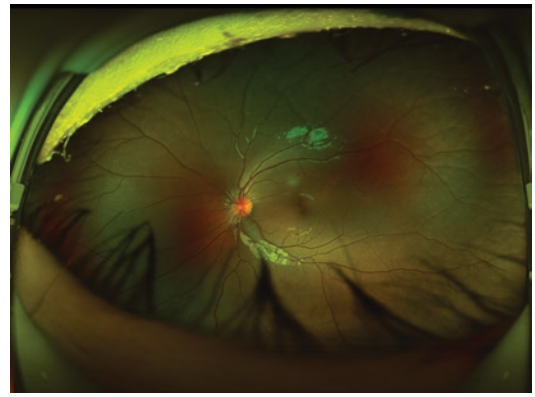

**Fig. 11.12** SLO: normal

Traumatic optic neuropathy is a clinical diagnosis; even if only slight head trauma, its symptoms and signs guide doctors to doubt and diagnose traumatic optic neuropathy, with the symbol of visual function loss. Currently, the therapeutic options include temporary observation with nutritional nerve drugs, corticosteroid therapy, surgery of optic nerve decompression, or combination of corticosteroid and optic nerve decompression.

At present, there is no established treatment standard based on the pathophysiological mechanism, but the ischemia and edema in the early stage of the theoretical injury can be prevented in theory. The large dose of the corticosteroid may limit secondary nerve cell injury caused by the generation of the oxygen free radicals, swelling, blood vessel spasm ischemia, and so on.

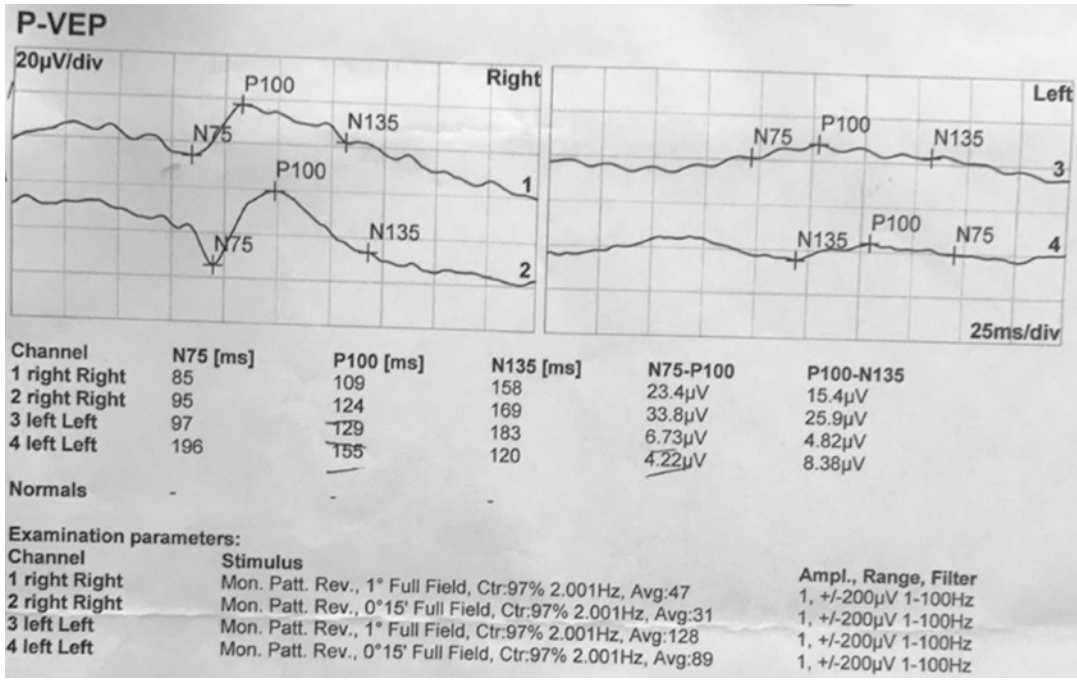


**Fig. 11.13** P-VEP showed that P100 latency was obviously delayed at 60′ and 15′ (60′ was 129 ms; 15′ was 155 ms)

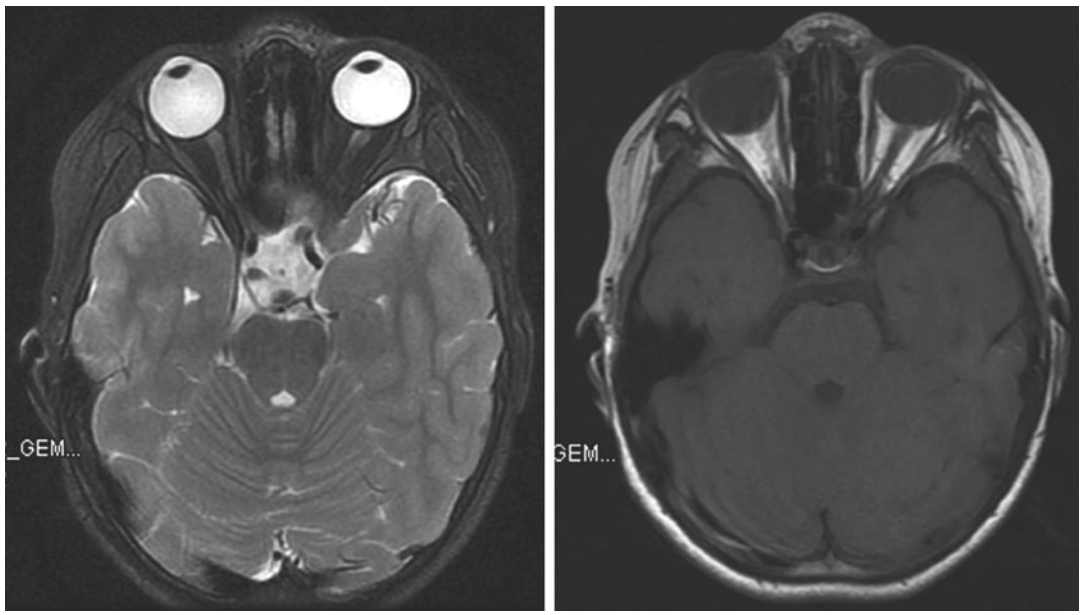


**Fig. 11.14** Anteroposterior diameter of the left eye is shorter, while the intraocular structure was not abnormal, and the bilateral optic nerve was not thickened or thinned

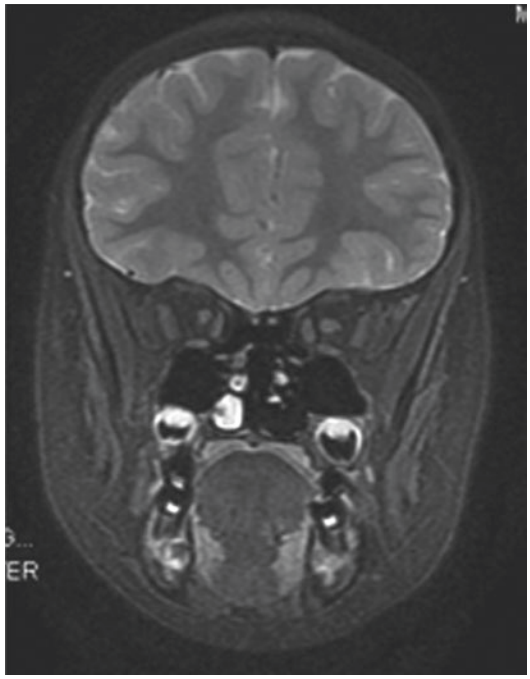

**Fig. 11.14** (continued)

## 11.5 Case #4: A Case with Indirect Traumatic Optic Neuropathy

### 11.5.1 Case Description

A 26-year-old man was struck in his right eye by a bouncing carrot; his left eye is amblyopic since he was a child. He went to the hospital for a series of eye examinations. Corrected visual acuity of the right eye was 0.8 and 0.2 on the left eye. His right pupil was dilated with traumatic dilation (Fig. 11.15). P-VEP showed that bilateral P100 latency was obviously delayed at 60′ (right is 115 ms, left is 112 ms) (Fig. 11.16). OCT is normal (Fig. 11.17). SLO of the right eye found that there was preretinal hemorrhage on the macular area (Fig. 11.18). The diagnosis of indirect traumatic optic neuropathy and traumatic mydriasis was made. The patient received oral drug therapy of methycobal and citicoline sodium tablets and was recommended to wear sunglasses outside or after surgical treatment.

### 11.5.2 Tips and Pearls

Traumatic optic neuropathy is a clinical diagnosis, even if there is only a slight history of trauma, but its history combined with symptoms and signs suggest to suspect and diagnose the traumatic optic neuropathy. The main clinical manifestations are as follows: (1) visual acuity decreased, a few patients could maintain their vision, but the severely damaged patients could be reduced to no light perception; (2) visual field defect; (3) abnormal color vision and asymmetry of binocular color perception; (4) most of the patients have the relative afferent pupillary defect; (5) the fundus manifested variously according to the location of injury which can present normal fundus manifestations, optic disc edema, retinal hemorrhage, and so on; and (6) the optic nerve atrophy occurs 3–6 weeks after optic nerve injury, and the loss of visual acuity may be temporary or permanent and partial or complete. For this patient, we need to follow up his visual acuity and retinal nerve fiber layer thickness with optic disc OCT for the next 6 months.

## 11.6 Treatment

Important signs, examinations, diagnosis, surgical procedures, or postoperative treatment for complications.

Hence, when we diagnose a patient with TON, we should pay attention to the following:

1. History—Mechanism of injury loss of consciousness, nausea and/or vomiting, headache, and clear nasal discharge.
2. Neuroimaging examination—CT and MRI examinations are routine methods for the evaluation of orbital and intracranial injuries.

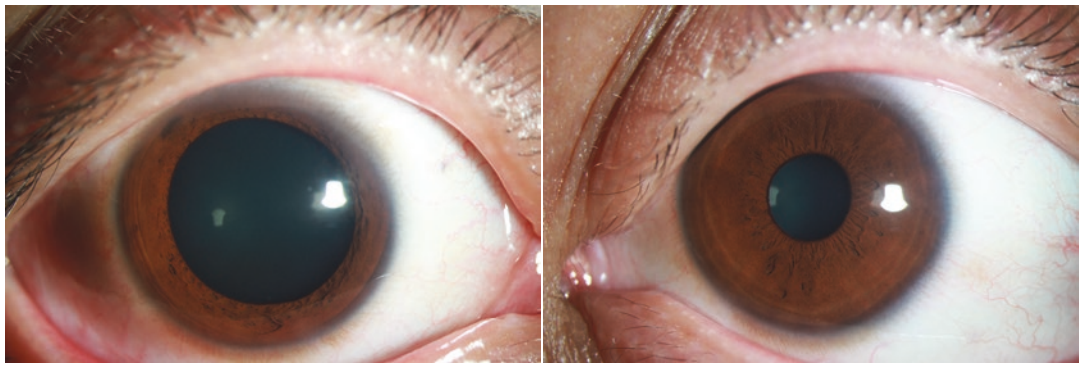

**Fig. 11.15** The right pupil was dilated with traumatic dilation

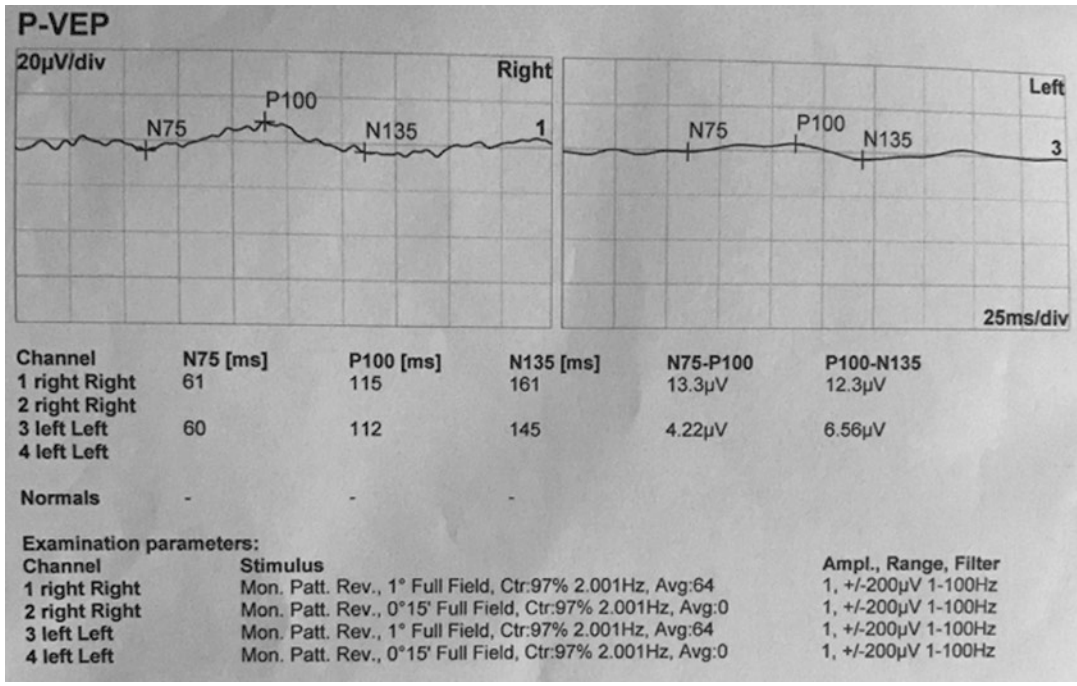


**Fig. 11.16** P-VEP showed that bilateral P100 latency was obviously delayed at 60′ (right is 115 ms; left is 112 ms)

CT should be taken first when a metal foreign body is suspected, because the displacement of the foreign body due to the effect of the magnetic field at the time of MRI examination leads to further damage.

3. Visual acuity testing may be difficult depending on the patient's mental status and use of sedatives and narcotics.
4. Pupillary evaluation—Relative afferent papillary defect (RAPD) is the sine qua non in cases of unilateral TON. In the absence of RAPD, either there is no TON or it is bilateral.
5. Color vision—Checking red desaturation is a useful alternative if color plates are not available.
6. Visual fields—Any type of field defects may be seen in optic nerve trauma.
7. Visual electrophysiological examination—VEP, ERG.
8. Fundus examination—Result from objects that penetrate the orbit and impinge on the

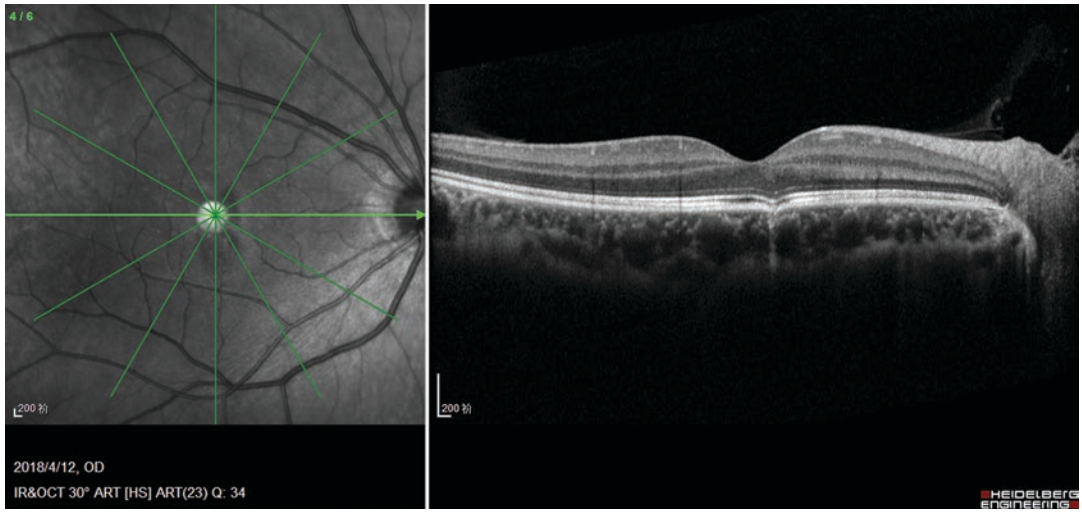


**Fig. 11.17** OCT is normal

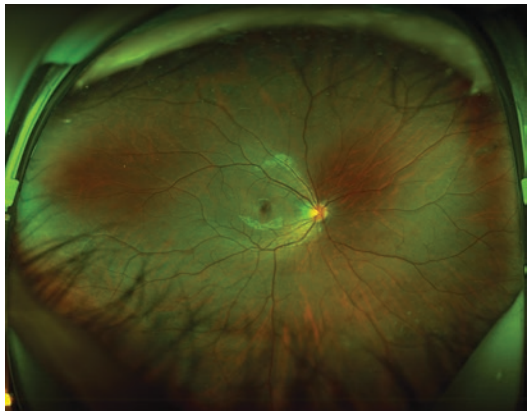

**Fig. 11.18** SLO of the right eye found that there was preretinal hemorrhage on the macular area

optic nerve causing optic neuropathy by partial or complete transection of the optic nerve sheath. Hemorrhages within and around the nerve may also occur which lead to immediate changes in the fundus that can be detected on ophthalmoscopic examination.

9. The methods reported in the literature are different, and the clinical results vary greatly, but the consensus is that the earlier the treatment begins, the better the effect. The principles are generally followed: High dose methylprednisolone infusion can be initiated intravenously guttae as soon as possible in acute cases. It is recommended that 500 mg of methylprednisolone per dose be given twice a day. However, some clinical trials compared the effect of intravenous high-dose corticosteroid therapy to placebo for the treatment of recent traumatic optic neuropathy. It was concluded that there was no difference in improvement with the corticosteroid. A study even indicated that high-dose corticosteroids should not be routinely offered to patients suffering a head injury due to an elevated risk of death. After treatment, if visual function is improved, 48 h after intravenous administration can be changed to oral administration, gradually reduced until 2 weeks.
10. If the medication is ineffective after 12–48 h, or the vision is impaired during the reduction process, it is recommended to consider decompression of the optic canal. However, some scholars believe that if glucocorticoid shock therapy is not effective in the early stage of indirect traumatic optic neuropathy, the surgical effect is also very limited. For patients with progressive loss of visual acuity accompanied with fracture of the optic canal and stenosis or fracture into direct optic nerve injury, decompression of the optic canal to relieve compression and injury

should be performed. Opinions for surgical management of traumatic optic neuropathy include partial removal of the bony optic canal (in cases of optic canal fracture fragments impinging the optic nerve), optic nerve fenestration, and opening up the annulus of Zinn. These procedures principally serve to decompress the optic canal and optic nerve swelling, and the resultant vascular compromise can cause secondary damage.

11. The glucocorticoid can be used to treat optic nerve injury, combined with a dehydrating agent, improved microcirculation drugs, neurotrophic drugs, and so on. Except those, erythropoietin may provide neuroprotection and support axonal growth. Glutamate is the major excitatory neurotransmitter in the eye that induces retinal ganglion cells apoptosis via binding to *N*-methyl-diacetylaspartate (NMDA) and kainate receptors. Glutamate inhibitors and NMDA receptor antagonists have been shown to promote retinal ganglion cell survival in rat models of optic nerve injury. Neurotrophic factors such as brain-derived neurotrophic factor are essential to retinal ganglion cell survival, and their absence is noted in optic nerve damage, for example, nonencephalitogenic myelin peptides.

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