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Open Access Case Report

Post-traumatic Unilateral Vision Loss in a Patient With Severe Traumatic Brain Injury: A Case Report

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Review began 09/03/2026
Review ended 11/15/2026
Published 11/20/2026

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DOI: 10.7759/cureus.120012

Abstract

Traumatic optic neuropathy (TON) is an uncommon but often irreversible cause of vision loss after craniofacial trauma. In indirect TON, forces transmitted through the skull base injure the optic nerve at the orbital apex and optic canal through axonal shearing, ischemia, and secondary compression. Management is controversial: neither high-dose corticosteroids nor optic canal decompression has been shown to be better than observation, and high-dose corticosteroids increase mortality in traumatic brain injury (TBI), which frequently accompanies TON. A 21-year-old male presented after a 20-foot fall with brief loss of consciousness, a Glasgow Coma Scale score of 15, and left eye vision loss. Examination showed no light perception in the left eye with a relative afferent pupillary defect, full ocular motility, and a normal anterior segment and fundus; the right eye was normal. CT showed extensive facial and skull base fractures with pneumocephalus but no optic canal fracture line, and MRI showed compression of the left optic nerve at the orbital apex/optic canal with loss of the perineural cerebrospinal fluid signal. After weighing the risks, megadose intravenous methylprednisolone was started and was stopped at 36 hours because vision had not improved. Decompression was not performed, and the facial fractures were repaired once the edema had resolved. At 11 months, the left eye remained without light perception and had developed a 20-prism-diopter sensory exotropia. This case shows that permanent unilateral vision loss can result indirectly from facial fractures without a visible optic canal fracture. It emphasizes the need for a systematic eye examination, including pupillary testing, in every patient with complex facial fractures, and the importance of individualized, multidisciplinary decisions about corticosteroid use when TBI is present.

Categories: Trauma, Ophthalmology
Keywords: facial fracture, fall injury, traumatic brain injury(tbi), traumatic optic neuropathy (ton), traumatic vision loss

Introduction

Traumatic optic neuropathy (TON) refers to partial or complete loss of visual function caused by injury to the optic nerve. It complicates approximately 0.5%-5% of closed head injuries and is most often associated with high-energy blunt trauma, such as falls, motor vehicle collisions, and assaults [1]. In the only national population-based surveillance study, conducted in the United Kingdom, the minimum estimated incidence of TON was approximately 1 per million population per year; affected patients were predominantly young adult men, and 36% had no light perception (NLP) in the affected eye at presentation [2,3]. Its consequences include permanent monocular or binocular blindness, impaired depth perception, reduced quality of life, and a substantial socioeconomic burden [4]. Globally, eye injuries are estimated to cause blindness in approximately 1.6 million people, making trauma one of the leading causes of preventable monocular vision loss [5].

TON is classified by mechanism as direct or indirect. Direct TON results from physical disruption of the nerve, for example, by a penetrating object or a displaced bone fragment in the orbit or optic canal that lacerates or impinges on it. Indirect TON, the more common form, occurs without penetration of the nerve. Instead, forces from blunt impact to the forehead or midface are transmitted through the skull base and concentrated at the orbital apex and optic canal [2,6,7]. In this region, the intracanalicular optic nerve is tethered to the surrounding periosteum and enclosed in a rigid bony canal. Shearing forces can disrupt its axons and microvasculature, and the edema or hemorrhage that follows has no room to expand. The result is secondary compression and ischemia, which can worsen the initial injury. In patients with extensive craniofacial fractures, this mass effect at the orbital apex can arise from edema, hematoma, or fracture displacement, and it is often a major contributor to vision loss even when the nerve itself is not transected [8]. Orbital, midfacial, and skull base fractures are therefore common in patients with indirect TON, and they deserve particular attention as a cause of vision loss in patients with facial trauma [2].

TON is primarily a clinical diagnosis. Typical findings are reduced visual acuity, impaired color vision, and visual field defects. In unilateral or asymmetric injury, a relative afferent pupillary defect is present. Because the injury is usually posterior to the globe, the optic disc often looks normal at first, and optic atrophy appears only weeks later. Thin-section computed tomography (CT) of the orbits and optic canal is used to

look for canal fractures, bone fragments, and orbital or retrobulbar hemorrhage. TON must be distinguished from retrobulbar hemorrhage with orbital compartment syndrome, which requires emergent canthotomy, and from orbital apex syndrome, in which optic neuropathy is accompanied by ophthalmoplegia from involvement of cranial nerves III, IV, and VI and by reduced sensation in the distribution of the ophthalmic division of the trigeminal nerve [9]. Diagnosis is often delayed in polytrauma patients because they may be sedated, intubated, or have periorbital swelling that prevents a reliable examination.

TON frequently coexists with traumatic brain injury (TBI), because both arise from the same high-energy mechanisms. TBI affects an estimated 69 million people worldwide each year, with road traffic collisions and falls among its leading causes [10,11]. Its management centers on preventing secondary brain injury by avoiding hypoxia and hypotension, monitoring and controlling intracranial pressure, maintaining cerebral perfusion, and performing surgical evacuation or decompression when indicated [12]. Tranexamic acid given within 3 hours of injury reduced head injury-related death in patients with mild-to-moderate TBI in the CRASH-3 trial [13]. Corticosteroids, in contrast, are not recommended in TBI: in the CRASH trial, methylprednisolone given within 8 hours of significant head injury increased mortality compared with placebo [14,15].

There is no established standard of care for TON. The three main approaches are observation, corticosteroids, and surgical optic canal decompression [16]. The megadose corticosteroid regimen used in TON (intravenous methylprednisolone 30 mg/kg bolus followed by 5.4 mg/kg/h), and the convention of starting treatment within 8 hours of injury, were extrapolated from the Second National Acute Spinal Cord Injury Study (NASCIS II) rather than from trials in TON [17]. The International Optic Nerve Trauma Study, a comparative non-randomized study of 133 patients, found no clear benefit of corticosteroids or optic canal decompression over observation [3]. A randomized, double-masked, placebo-controlled trial of high-dose intravenous methylprednisolone likewise found no significant difference in final visual acuity [18], and a Cochrane review concluded that spontaneous recovery is relatively common and that there is no convincing evidence that steroids add benefit over observation [7]. In the Traumatic Optic Neuropathy Treatment Trial, intravenous erythropoietin, intravenous methylprednisolone, and observation produced similar visual improvement, and NLP vision at presentation was associated with poor recovery [19]. Because many patients with indirect TON also have TBI, the increased mortality seen with corticosteroids in the CRASH trial [14,15] makes high-dose steroid therapy particularly controversial and potentially harmful. Management therefore depends on coordinated evaluation by ophthalmology, neurosurgery, and maxillofacial surgery, so that decisions about the optic nerve are balanced against overall trauma stabilization and fracture repair [20,21].

We present a 21-year-old man who developed left-eye NLP vision with a relative afferent pupillary defect and a normal-appearing globe and fundus after sustaining extensive facial and skull base fractures in a 20-foot fall. Imaging showed compression of the optic nerve at the orbital apex and optic canal from the adjacent fractures and edema, without a fracture line through the canal. Vision loss caused indirectly by facial fractures in this way, in a patient with a Glasgow Coma Scale (GCS) score of 15, is uncommon and can be overlooked when attention is focused on other injuries. The case illustrates the ophthalmic findings that distinguish TON from other causes of post-traumatic vision loss, the difficulty of deciding on corticosteroid therapy when evidence is limited and head injury is present, and the long-term consequences of monocular blindness, including sensory exotropia.

Case Presentation

A 21-year-old male with no past medical or ocular history was brought to the Emergency Department by emergency medical services (EMS) after a traumatic fall. He was power washing a roof when he fell about 20 feet onto a porch, then rolled and fell another 2-3 feet onto concrete. Witnesses reported a head strike and brief loss of consciousness. On arrival, he was hemodynamically stable, and his neurological examination was intact, with a GCS of 15 [22]. With brief loss of consciousness, a GCS of 15, and skull base fractures with pneumocephalus on CT, his head injury was classified as a complicated mild TBI. He was bleeding heavily from the oropharynx. The secondary survey showed bilateral orbital ecchymosis, scattered facial abrasions, and vision loss in the left eye, which was noticed upon regaining consciousness. The focused assessment with sonography for trauma (FAST) examination and chest X-ray were negative. The white blood cell count was notably elevated at 17.83 × 10^3/μL, attributable to a trauma response. CT of the head and maxillofacial region showed an extensive central skull base fracture through the sphenoid body, the left greater sphenoid wing, the planum sphenoidale, and the sphenoid sinus. It also showed fractures of the left orbital roof with adjacent pneumocephalus, the left lateral orbital rim at the frontal process, the left frontal bone, the left zygomatic arch, the comminuted left nasal bone, the anterior and posterolateral walls of the left maxillary sinus with herniation of buccal fat into the sinus (Figures *1*, *2*), the medial wall and floor of the left orbit (nondisplaced), the right parasymphyseal mandible, and the left mandibular ramus. Blood in the sphenoid sinus, adjacent to the canalicular optic nerve, raised concern for posterior optic nerve injury, but no fracture line through the optic canal could be seen on CT.

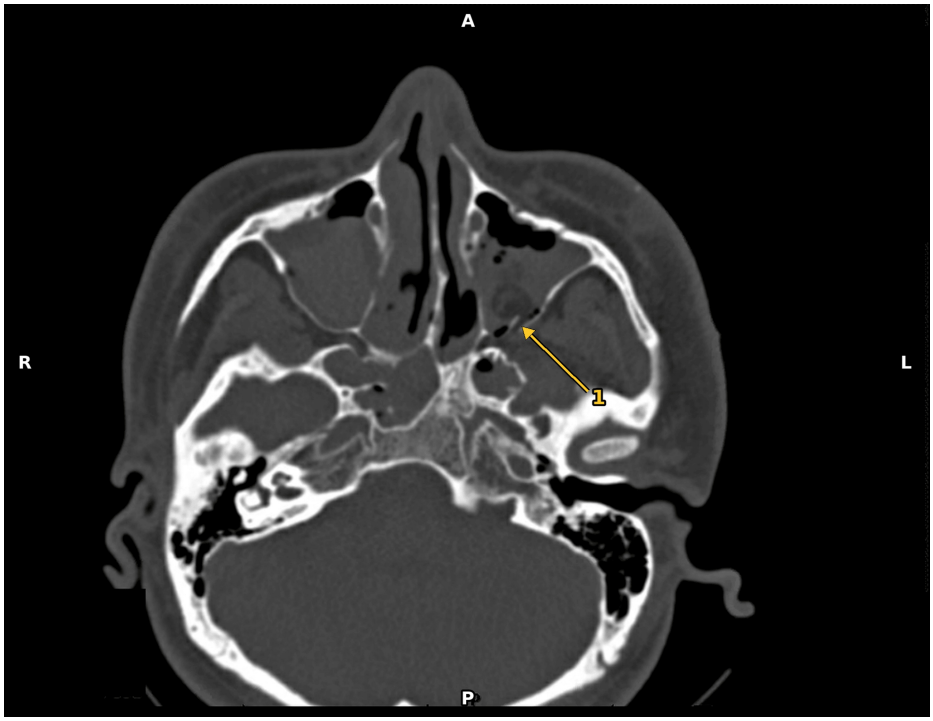


FIGURE 1: Maxillofacial computed tomography (CT), axial section.

Arrow 1 marks the fracture of the posterolateral wall of the left maxillary sinus, with adjacent air. This slice is at the level of the maxillary sinuses, below the orbital apex; therefore, it does not show the optic nerve.

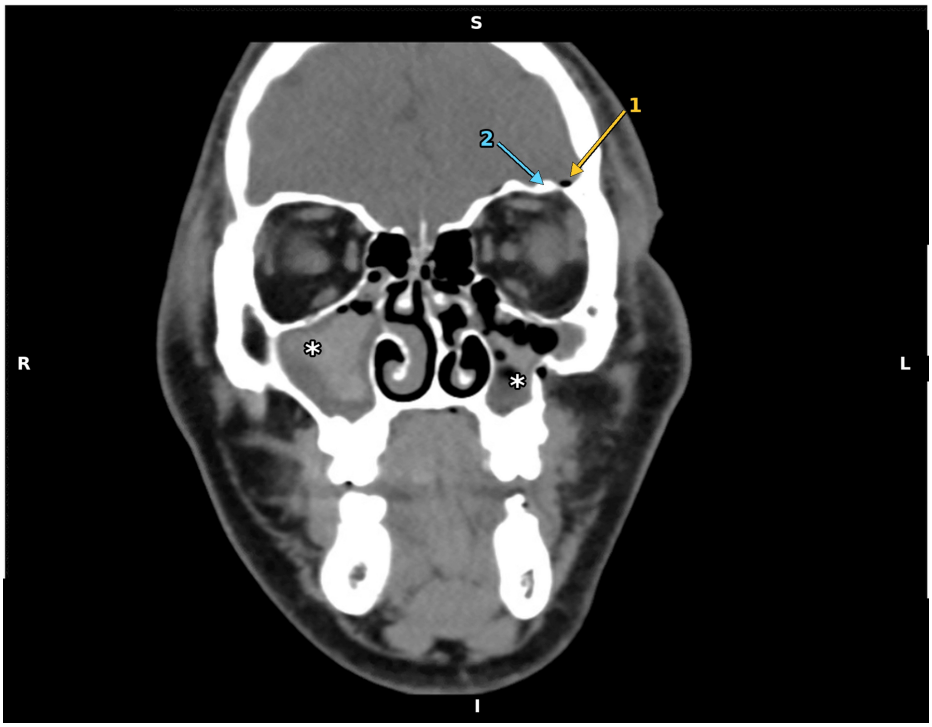


FIGURE 2: Coronal maxillofacial computed tomography (CT).

Arrow 1 indicates a small focus of intracranial air (pneumocephalus) above the left orbital roof. Arrow 2 indicates the left orbital roof at the fracture site adjacent to the pneumocephalus, where the left frontal bone fracture extends into the orbital roof. Asterisks indicate hemorrhage in the maxillary sinuses; blood was also present in the ethmoid air cells.

Ocular examination at presentation

Visual Function

Uncorrected near visual acuity was 20/20 in the right eye (OD) and NLP in the left eye (OS). NLP was confirmed with light testing in all cardinal directions. On optokinetic (OKN) drum testing, the right eye tracked normally, whereas the left eye did not track, supporting the conclusion that the left-eye vision loss was organic. Color vision was 8/8 Ishihara plates OD; the left eye could not be tested. Confrontation visual fields were full to counting fingers OD; the left eye could not be tested.

Pupils

Both pupils were round and reactive. There was a relative afferent pupillary defect (RAPD) in the left eye and none in the right.

Intraocular Pressure and Motility

Intraocular pressure (IOP), measured by Tono-Pen (Reichert Technologies, Depew, NY), was 22 mmHg in both eyes. Extraocular movements were full in both eyes, with no ophthalmoplegia or restriction suggesting muscle entrapment, and the oculocardiac reflex was negative. Facial sensation in the V1 distribution was intact.

Anterior Segment

Externally, the right periorbital area had small abrasions and ecchymosis, whereas the left had edema and ecchymosis. The left eyelids were swollen but soft and opened easily, with no signs of a tense orbit. In both eyes, the conjunctiva and sclera were white and quiet, the corneas were clear, there was no hyphema, the anterior chambers were deep and formed, the irides were round and regular, and the lenses were clear.

Posterior Segment

The eyes were dilated with phenylephrine 2.5% and tropicamide 0.5%, and dilated fundus examination was performed with indirect ophthalmoscopy using a 20 D lens. In both eyes, the vitreous was clear, and the optic discs were flat, sharp, and pink, with a cup-to-disc ratio of 0.3. There was no disc edema or pallor. The maculae were flat with a normal foveal reflex, and the vessels and retinal periphery were normal.

Initial management and MRI

The findings were highly suggestive of posterior (canalicular) TON in the left eye. There was no active intracranial hemorrhage, and the neurological examination remained normal. After the limited evidence and potential risks, including increased mortality with steroids in head injury [14,15], were weighed, the trauma and ophthalmology teams elected a trial of megadose intravenous methylprednisolone: a 30 mg/kg bolus about 7.5 hours after arrival, followed by a continuous infusion of 5.4 mg/kg/h (body weight: 90.3 kg; 5,500 mg overall; no adverse events reported) [17]. Steroids were chosen because suspicion for TON was high, there was no intracranial hemorrhage, and treatment within eight hours has been associated with a somewhat better prognosis [23]; this time window is derived from the NASCIS II spinal cord injury protocol [17]. Therapy was to be stopped if visual function had not improved at reassessment. He was also started on IV ampicillin-sulbactam to cover nasal flora given the sinus-involving fractures and given orbital fracture precautions (no straining or nose blowing, and cool compresses).

MRI of the brain and orbits was performed to assess whether the fracture involved the optic canal. It showed mass effect on the orbital segment of the left optic nerve at the orbital apex/optic canal, with loss of the normal surrounding CSF signal and no intrinsic signal abnormality in the nerve (Figure 3). This was attributed to localized edema and bony deformity from the adjacent fractures. MRI also showed dural thickening over the left frontal and temporal lobes, presumed to be reactive to the fractures, although superimposed infection could not be excluded.

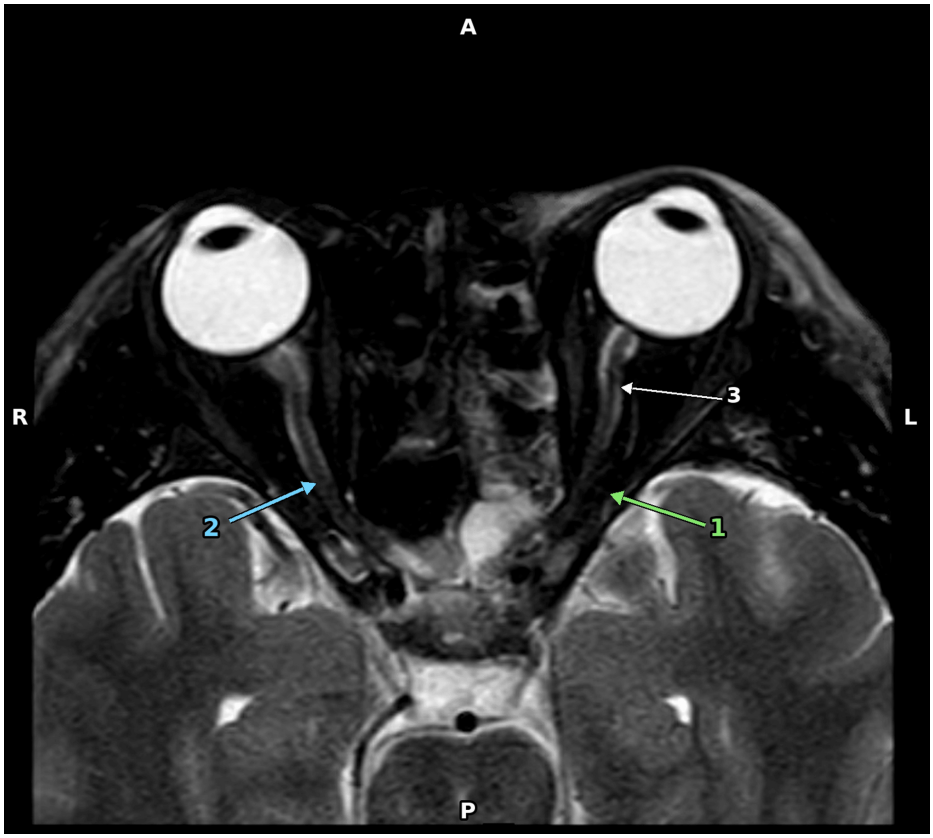


FIGURE 3: Axial T2-weighted 3D DRIVE MRI of the orbits.

Arrow 1: Posterior orbital segment of the left optic nerve at the orbital apex/optic canal, showing loss of the normal bright perineural cerebrospinal fluid (CSF) signal, consistent with compression (mass effect) from the adjacent fractures and edema. Arrow 2: Right optic nerve with normal perineural CSF signal, for comparison. No gross signal abnormality of the right optic nerve is seen. Arrow 3: More anterior segment of the left optic nerve, where the perineural CSF signal is preserved.

DRIVE, driven equilibrium radiofrequency reset pulse

Follow-up ophthalmologic examination

At about 36 hours of IV methylprednisolone, the patient still had no vision in the left eye. Visual acuity was 20/20 OD and NLP OS, with no light perception in any of the nine cardinal directions. The left RAPD persisted. IOP was 20 mmHg in both eyes on Tono-Pen and 19 mmHg OS on repeat measurement. Extraocular movements remained full in both eyes, and color vision and confrontation fields were unchanged. The external and anterior segment findings were stable. The right eye remained normal. Because vision had not improved and to limit steroid exposure in a patient with head injury, ophthalmology recommended stopping the steroids, and the infusion was discontinued at about 36 hours. The patient was counseled on the guarded visual prognosis and given monocular precautions. Ophthalmology and neurosurgery did not recommend surgical intervention on the brain or optic nerve because his vital signs were stable, there was no evidence of further neurological or functional deterioration, and optic canal decompression has not been shown to improve outcomes and carries additional risk in the presence of skull base fractures [3,16].

On hospital day 9, once the facial edema had resolved, the mandibular, maxillary, and nasal fractures were repaired with closed reduction and maxillomandibular fixation, plate fixation of the left lateral maxillary buttress, and closed nasal reduction. On hospital day 10, he was tolerating a liquid diet and was discharged.

Follow-up

At six months after discharge, the patient had returned to baseline function and work without restrictions. He had not needed rehabilitation. Left-eye vision loss was his main limitation. He reported impaired spatial perception and compensatory head-turning, which made walking and night driving difficult. He also reported intermittent discomfort and fatigue in the right eye. These symptoms were attributed to asthenopia from the increased visual demand placed on the only seeing eye.

At 11 months after the injury, visual acuity remained 20/20 OD and NLP OS, with a persistent left RAPD. IOP was 10 mmHg OD and 18 mmHg OS. Ductions were full, but Krimsky testing showed a 20-prism-diopter left exotropia, consistent with sensory exotropia. The slit-lamp examination was normal in both eyes. The patient was bothered by the drift. He was counseled that strabismus surgery carries a risk of recurrence, and he chose to defer surgery for personal reasons. He continues to follow monocular precautions, with strabismus follow-up planned in six months. The clinical course is summarized in Table *1*.

Time point	Findings, investigations, and management
Day 0: injury	Fall of about 20 feet onto a porch, then 2–3 feet onto concrete; head strike and brief loss of consciousness
Hospital day 0: Emergency Department	GCS 15; hemodynamically stable; left-eye vision loss found on the secondary survey; CT: central skull base, orbital roof, left frontal, zygomatic, nasal, maxillary, and bilateral mandibular fractures; pneumocephalus; blood in the sphenoid sinus; no optic canal fracture line
Hospital day 0: ophthalmology examination	VA 20/20 OD (near), NLP OS; left RAPD; color 8/8 OD; IOP 22/22 mmHg; full motility; soft orbit; normal anterior segment and fundus OU
About 7.5 h after arrival	IV methylprednisolone 30 mg/kg bolus, then 5.4 mg/kg/h infusion; IV ampicillin-sulbactam; orbital fracture precautions
Hospital day 1: MRI	Mass effect on the left optic nerve at the orbital apex/optic canal; loss of perineural CSF signal; no intrinsic nerve signal abnormality
About 36 h of steroids	VA 20/20 OD, NLP OS; left RAPD; IOP 20/20 mmHg; no improvement; steroids were stopped, and decompression was not recommended.
Hospital day 9	Repair of mandibular, maxillary, and nasal fractures
Hospital day 10	Discharged
6 months	Returned to work; impaired spatial perception, compensatory head turning, and difficulty with night driving; intermittent right-eye discomfort and fatigue
11 months	VA 20/20 OD, NLP OS; left RAPD; IOP 10/18 mmHg; 20 PD left sensory exotropia; strabismus surgery deferred

TABLE 1: Timeline of diagnosis and management.

CSF, cerebrospinal fluid; CT, computed tomography; GCS, Glasgow Coma Scale; IOP, intraocular pressure; IV, intravenous; MRI, magnetic resonance imaging; NLP, no light perception; OD, right eye; OS, left eye; OU, both eyes; PD, prism diopters; RAPD, relative afferent pupillary defect; VA, visual acuity

Discussion

Traumatic craniofacial injuries can lead to a wide spectrum of visual disturbances, ranging from blurred vision and diplopia to complete vision loss, with TON representing one of the most severe outcomes. Although TON is rare [1,2], its consequences are often irreversible due to axonal disruption, ischemia, or compression of the optic nerve [6]. Fracture-related swelling makes the picture more complicated: localized edema and bone deformity at the orbital apex can compromise the optic nerve even when the globe itself is uninjured [9]. When TON occurs alongside other traumatic injuries, management becomes challenging because interventions aimed at preserving vision may conflict with broader trauma priorities. For example, optic canal decompression may be risky in patients with skull base fractures or intracranial concerns [16]. Systematic reviews have not shown that any single approach (corticosteroids, surgery, or observation) is consistently better than the others [4,7].

In this case report, the left eye had sudden NLP vision with an RAPD, while the globe and fundus were normal. This combination is the typical presentation of posterior TON. The right eye was normal throughout, with 20/20 acuity, full color vision and confrontation fields, and a normal fundus, confirming that the injury was unilateral. The examination ruled out other treatable causes of acute vision loss. A soft orbit with eyelids that opened easily argued against retrobulbar hemorrhage or orbital compartment syndrome. There was no hyphema, no sign of an open globe, and no vitreoretinal pathology, and the normal fundus excluded optic nerve avulsion. Full motility and a negative oculocardiac reflex argued against muscle entrapment. Full motility also excluded orbital apex syndrome, in which optic neuropathy is accompanied by ophthalmoplegia from involvement of cranial nerves III, IV, and VI [9]; the vision loss was therefore attributed to isolated TON rather than orbital apex syndrome. Optokinetic drum testing showed that the left eye did not track, which supported an organic cause for the vision loss. CT did not show a definite fracture plane through the optic canal. However, there was an extensive sphenoid body and planum sphenoidale fracture with blood in the sphenoid sinus. MRI then showed mass effect on the left optic nerve at the orbital

apex and optic canal, with loss of the normal surrounding CSF signal and no evidence of transection. This was attributed to localized edema and bony deformity. Together, these findings place the injury at the canalicular segment. The intracanalicular optic nerve is the only fixed part of the nerve, and it is especially vulnerable to shearing and to secondary swelling inside the rigid canal.

The association between high-energy trauma and TON is well established, as transmitted forces from falls, motor vehicle collisions, or blunt craniofacial impacts can injure the optic nerve directly or indirectly [10]. Here, the mechanism was indirect. A 20-foot fall caused extensive midfacial and skull base fractures, and the resulting compression at the orbital apex and canal injured the nerve. Injuries like this carry a risk of permanent unilateral vision loss, and recognition can be delayed when attention is on competing injuries [24]. In this patient, the left-eye vision loss was found on the secondary survey, and ophthalmology evaluated him the same day. This highlights the value of a systematic visual assessment in any patient with complex facial fractures [8].

Management of indirect TON remains controversial. The main options are high-dose corticosteroids, optic canal decompression, and observation, and the evidence for each is limited [3,4,7,16,18,19,25]. The American Academy of Ophthalmology found no level I evidence and concluded that no therapy represents a standard of care. It advised that patients receiving corticosteroids be told that the supporting evidence is weak at best [25]. Corticosteroids may reduce edema, but they carry risks that include hyperglycemia, immunosuppression, infection, and elevated IOP. In the CRASH trial, high-dose methylprednisolone increased mortality after significant head injury [14,15]. This makes megadose steroids a particularly risky choice in patients with concurrent traumatic brain injury. Surgical decompression may relieve mechanical compression, but it has risks of its own, including CSF leak, infection, and hemorrhage. These risks are a particular concern with skull base involvement [6,25]. Recent meta-analyses disagree. One found better visual outcomes with early decompression than with corticosteroids, especially in indirect TON [26]. Another found no significant benefit of surgery over systemic therapy [27]. Treatment decisions therefore still depend on clinical judgment, timing, and patient-specific factors rather than standardized protocols [4].

In this patient, management was guided by his stable neurologic status, the absence of intracranial hemorrhage, and imaging consistent with compressive canalicular TON. After weighing risks and benefits, the trauma and ophthalmology teams started megadose methylprednisolone: a 30 mg/kg bolus about 7.5 hours after arrival, then 5.4 mg/kg/h. This decision drew on studies suggesting a better prognosis when treatment begins within 8 hours [16,23,28], although this time window derives from spinal cord injury research [17] and has not been validated in TON [3,7]. Vision in the left eye remained NLP in all quadrants, and steroids were stopped at about 36 hours. Ophthalmology and neurosurgery did not recommend optic canal decompression. Their reasons were the skull base fractures, the absence of clinical deterioration, and the limited expected benefit in an eye that had been NLP from the outset. This conservative approach allowed the facial injuries to stabilize, and staged maxillofacial repair followed once the edema had resolved.

The lack of visual recovery is consistent with the poor prognosis of NLP TON. Initial visual acuity is the strongest predictor of outcome, and NLP vision with a dense RAPD predicts limited improvement [29]. Pooled data show that NLP eyes improve much less often than eyes with residual vision, whether they are treated with steroid pulse therapy or with decompression [1]. Optic canal fracture or compression may further reduce the response to corticosteroids alone [1].

Several factors probably explain why vision did not improve with corticosteroids. First, NLP vision at presentation is the strongest predictor of poor outcome, and in the Traumatic Optic Neuropathy Treatment Trial, initial NLP vision was associated with failure to improve regardless of treatment [19]. Second, much of the axonal damage in indirect TON is thought to occur at the moment of impact, through shearing of axons and their blood supply within the canal; this primary injury is not reversed by anti-edema therapy, which targets only the secondary component of the injury [6]. Third, the bony deformity at the orbital apex represented a fixed mechanical compression that corticosteroids could not relieve. In retrospect, no available intervention was likely to have restored vision. Optic canal decompression was the only option directed at mechanical compression, but its benefit is unproven, particularly in eyes that are NLP from the outset, and it carries substantial risk in the presence of skull base fractures and pneumocephalus [3,16]. Observation alone would have been an equally defensible choice [7].

On long-term follow-up, the patient regained baseline function and returned to work without restrictions. However, the monocular blindness continued to affect him. At 6 months, he reported impaired spatial perception and compensatory head-turning, which made walking and night driving difficult. By 11 months, he had developed a 20-prism-diopter left sensory exotropia, a recognized consequence of acquired structural vision loss in one eye [30]. Although cosmesis was considered acceptable, the patient was very bothered by the drift. Strabismus surgery can restore alignment, but because a blind eye has no fusional input, it is prone to progressive drift and recurrence [31]. He was counseled accordingly and chose to defer surgery. The late development of sensory exotropia shows that the effects of TON continue beyond the acute phase. Patients need ongoing ophthalmic follow-up, counseling on monocular precautions, and attention to the psychosocial burden of ocular misalignment.

This case describes unilateral NLP vision loss from indirect, compressive canalicular TON following craniofacial and skull base fractures from a mechanical fall in a young patient with a GCS of 15. Its main educational points are that complete, permanent unilateral vision loss can result indirectly from facial fractures without a visible optic canal fracture, that a systematic eye examination including pupillary testing for an RAPD should be part of the secondary survey in every patient with complex facial fractures, and that full ocular motility helps distinguish isolated TON from orbital apex syndrome. It also underscores the importance of individualized care: the patient made an excellent recovery from his fractures and overall function despite irreversible vision loss. The case has limitations. It does not show that corticosteroids are beneficial, and it is not possible to determine whether corticosteroid therapy affected the outcome, given the lack of definitive evidence for its use [4,7]. The final NLP outcome may also reflect the severity of the initial injury more than the choice of treatment. In addition, objective structural documentation of optic atrophy, such as disc pallor or OCT retinal nerve fiber layer thinning, was not available at follow-up. Further research is needed to define the best management of indirect TON, including the roles and timing of pharmacologic therapy and surgical decompression, and long-term rehabilitation of monocular patients, including strabismus management [20]. By presenting this case, we aim to add to the limited literature on post-traumatic unilateral vision loss resulting indirectly from facial fractures.

Conclusions

Indirect TON can cause complete and permanent unilateral vision loss after craniofacial trauma, even when the globe is intact and no fracture line crosses the optic canal. In this patient, extensive facial and skull base fractures were associated with compression of the left optic nerve at the orbital apex, and vision did not recover despite a trial of high-dose corticosteroids. Three lessons follow. First, every patient with complex facial fractures should have a systematic eye examination as part of the secondary survey, including visual acuity and pupillary testing for a relative afferent pupillary defect, because these may be the only signs of optic nerve injury. Second, the examination should distinguish isolated TON from conditions that require different management, such as orbital compartment syndrome and orbital apex syndrome. Third, no treatment for indirect TON has proven benefit, and high-dose corticosteroids carry particular risks when traumatic brain injury is present; therefore, treatment decisions should be individualized and made jointly by ophthalmology, neurosurgery, and the trauma team. Long-term follow-up remains important because monocular blindness can lead to sensory exotropia and lasting effects on daily function.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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