Content available at: https://www.ipinnovative.com/open-access-journals

Indian Journal of Clinical and Experimental Ophthalmology

Journal homepage: www.ijceo.org

Review Article

Traumatic optic neuropathy: A review

Saravana Bhava[1], Sakthi Gnana Sundaram[1], Suma Kamareddy[1], Adwitya Mohapatra[1]*

[1]Dept. of Ophthalmology, Vinayaka Missions Kirupananda Variyar Medical College & Hospital, Salem, Tamil Nadu, India

Abstract

Traumatic optic neuropathy (TON) is a rare entity resulting in vision loss after a head trauma, predominantly affecting men due to accidents, falls, violence, or sports injuries. The chances of recovery from an insult to the optic nerve depend on whether it is a direct or an indirect injury. Injuries to the optic nerve of direct nature often lead to immediate and severe vision loss that may not improve over time. On the other hand, injuries to the optic nerve which are indirect may cause delayed vision loss, sometimes even after the initial injury. Fractures in the optic canal are effectively detected using thin-section CT scans. MRI is recommended only when intracranial injuries cannot be fully assessed using CT scans. The management of TON includes observation, corticosteroids, and surgery. It is typically recommended to observe the condition, if there are no signs of blood clots or fractures in the optic canal. The treatment for TON involves giving very high doses of intravenous methylprednisolone, followed by slowly tapering with oral prednisolone. Surgical management should be done only when there are compression signs.

Keywords: Traumatic optic neuropathy, Optic nerve, Orbital fracture, Corticosteroids, Ischemic optic neuropathy.

Received: 17-12-2024; **Accepted:** 18-08-2025; **Available Online**: 16-12-2025

1. Introduction

Traumatic optic neuropathy (TON) is the consequence of closed cranial trauma which is of disastrous potential.[1] The cardinal feature is the loss of visual function, manifesting as diminished visual acuity, visual field defects and colour vision abnormalities.[2] A pupillary reaction defect positively corresponds with a pre chiasmal trauma and points towards traumatic optic neuropathy.[3] The incidence of TON is approximated between 0.7% & 2.5% of all the cranial injury cases. Indirect TON has a higher prevalence than direct TON. It occurs in 0.5% to 5% of all patients with closed head injury and 2.5% of patients with midfacial fractures. Intracanalicular part is the most common site of indirect TON (71.4%), followed by the orbital apex (16.7%), combined intracanalicular segment and orbital apex (11.9%).[4-8] Men between ages 20 years and 40 years account for 60–95 percent of total cases, making up the most involved group in cases of TON.[7,9] These injuries causing TON can be of anterior and posterior lesions. Anterior lesions like avulsion of optic nerve, anterior ischemic optic neuropathy, hematoma of anterior optic nerve sheath show fundus abnormalities like Central Retinal Artery Occlusion and usually linked with other traumatic injuries to the eyeball. Posterior lesions are at the intracanalicular part of the optic cranial nerve and is usually free of any fundus findings, but disc oedema and disc pallor may happen. They are defined by loss of vision that occurs in the presence of a pupillary reaction defect but without any signs of globe injury or any signs of injury to the optic nerve.[10,11]

2. Etiological Classification of TON (Figure 1)

Traumatic optic neuropathy (TON) can result from motorcycle and car accidents (almost 15-75%), accidental falls (15-50%), physical violence and sports injuries. There is a significant link between TON and trauma to the cranium, wherein all patients with TON are having coincidental cranial injuries (two-thirds significantly). However, only 2% of the

*Corresponding author: Adwitya Mohapatra
Email: adwityamohapatra@gmail.com

https://doi.org/10.18231/j.ijceo.9460.1761817498

patients with cranial trauma have an attack of TON.[9] Direct traumatic optic neuropathy are usually the result of direct hit to craniofacial structures like penetrating or exhaustive crush injury with displaced fractures of cranio-orbit. Indirect traumatic optic neuropathy is far prevalent than direct variety.[12] On quite the contradictory, indirect TON usually is a result of a blunt trauma or stress transmitted through adnexal tissues and bones of the orbit to the optic nerve progressing from low to high level sight threatening problems.[13,14] Some examples such as fall by tripping or hit to the back of the head may result in a frontal blow leading onto a posterior traumatic optic neuropathy.[15,16] TON may be linked with the fracture of the medial orbital wall, optic canal, zygoma or floor of orbit.[17] The prognosis varies based on whether it is direct or indirect injury. Direct trauma can lead to devastating and fast vision deterioration with minimal recovery. Indirect trauma are seldom linked with sight recovery and has produced gradual diminution of vision that occurs multiple days after the primary injury.[18]

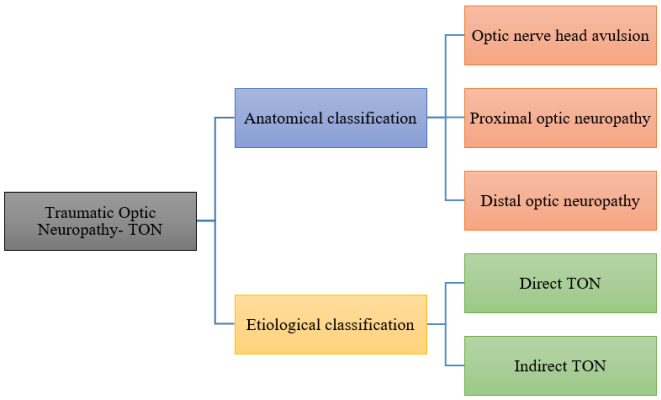

Figure 1: Classification of TON

3. Anatomical Classification of TON: (Figure 1)

1. Optic nerve head avulsion
2. Proximal optic neuropathy
3. Distal optic neuropathy.

3.1. Optic nerve head avulsion[19]

This injury occurs at the origin of the optic nerve where it exits the globe. It is sometimes identified by a characteristic circular hemorrhage encircling the optic disc, visible during fundus examination. The separation of the nerve from the globe may result from a sudden, forceful trauma.

3.2. Proximal optic neuropathy[20]

This refers to trauma involving the initial 10 millimeters of the optic nerve, just before the entry point of the central retinal artery and vein. Damage in this area may lead to vascular complications such as central or branch retinal artery occlusion or anterior ischemic optic neuropathy, often presenting with retinal or optic disc abnormalities.

3.3. Distal optic neuropathy[20]

In this form, trauma affects the optic nerve behind the entry of the central retinal vessels, usually within the optic canal or intracranial portion. These injuries typically do not manifest with immediate optic disc changes during fundus examination. However, visual field deficits, including hemianopia, compartment syndrome may develop depending on the extent and location of the damage.

4. Pathophysiology

The axons of the optic nerve are arranged in double compartments -namely, the intra dural and the intra fascicular compartments. Closed space edema, contusion necrosis, tears in the nerve fibres, and ischemia due to spasm or thrombosis have all been associated as important mechanism for insult to the optic nerve function.[22] Shear, stretch, compression & contusion at intracanalicular portion of optic nerve can lead to its improper function. Surgical intervention can give access to the duramater and can decrease internal fascicular pressure elevation.[23] Disruption of venous blood flow also plays an important role in pathogenesis. Myelin is more sensitive to oedema and acidic reaction than the other axonal constitiuents. An important component of the pathogenesis of indirect TON is the consequence of stress /force on the intracranial and intraorbital contents. Laser interferometry studies on actual skulls showed that injury to the facial region damages the roof of the orbit with optic nerve and the adnexal vessels damage, especially at the entry of the nerve into the optic canal. The power hit to the frontal bone is maximised and sent to the apical part of the bony orbit and the canal of the optic nerve.[24] Bruises of the axons of optic nerve with piamater vessels causes ischemia and oedema which result in further compression of nerve inside the closed compartment of the bony part of the canal of optic nerve and causes a positive loop of feedback which in turn initiate the mechanism of orbital compartment syndrome. After infarction, the cellular events which are the mechanisms behind the nerve injury are being analysed to be the additional events that may lead to optic neuropathy. The roles of oxygen free radical scavengers, enzymatic degradation, cytokinal effects, calcium molecular actions, and other types of reperfusion injury are now discovered via genomic level studies.[25,26]

5. Neuro-Ophthalmic Evaluation (Figure 2)

The diagnosis of TON is mostly by examination at clinical level. Patients with injury to the cranium and facial bones are the most vulnerable. They may have complicated craniofacial trauma, neurosurgical deficits, and other co-morbidities, with no visible signs of injury externally.[27] Optic nerve malfunction is assumed when a reduction of BCVA or field is added by same side relative afferent pupillary reaction defect – 'RAPD' –the 'Marcus Gunn pupil'[28] at the time of initial ophthalmological exam. Comprehensive and exhaustive ophthalmic examination is warranted on all

patients in highly suspicious cases of traumatic optic neuropathy and includes these assessments:[18, 29-33]

1. Visual acuity: Assessment of vision is the initial step which is to be done as soon as the patient is presented and a repeat assessment is done within 24 hours of presentation. It is recommended to assess vigorously the cases of subacute optic neuropathy (almost 10% of TON cases). Impaired color vision is one of the significant findings to be noted.
2. Ocular adnexa: It may showcase fractures of orbital surroundings, oedema, proptosis, enophthalmos, multiple extraocular muscle dysfunction, protrusion of foreign bodies, prolapsed orbital volume, or conjunctival lacerations.
3. Pupil reaction: A relative afferent pupillary defect is important for the diagnosis of TON. Marcus Gunn pupil is examined with the swinging flashlight test (when alternating a light source from one eye to the next -causing the affected pupil's initial constriction followed by a stall or dilatation).
4. Intra ocular pressure: Increased IOP accompanies an orbit haematoma, diffuse orbit haemorrhage, orbit emphysema, or a soft tissue oedema.
5. Fundus examination: Performed with help of cycloplegic drugs on all stabilised individuals. Examination of the retina and choroid vessels and its circulation, head of the optic nerve and its morphology, the visualisation of a ring haemorrhage nearby to the head of the optic nerve and the status of the macula is required.
6. Laboratory studies: Haemostasis is important during the procedure of decompression of optic canal. These lab tests aid in the selection of patients for surgery.

    a. Haemoglobin/ haematocrit
    b. Platelet cell count
    c. Pro- thrombin Time (PT) / activated Partial Thromboplastin Time (aPTT)
    d. Bleeding time (BT)

7. Radiology:[34-41]

    CT scanning is preferred for imaging orbital soft tissue and bony defects over MRI and is recommended without contrast to rule out associated pathologies in posterior traumatic optic neuropathy cases. It can identify fractures, optic nerve avulsion, and hematomas. Thin-sliced CT scans of the nose, sinuses, orbital, and the canal of optic nerve should be used for assessment and as a surgical guide if decompression is needed, although surgical decisions should primarily rely on clinical findings. Optic canal fractures are best shown by thin-sections of CT scans with 1.5 mm cuts and 1mm intervals. Spiral CT is useful for rapid data acquisition in non-cooperative patients.

    MRI is used to assess intracranial injuries when CT imaging is insufficient. It evaluates integrity of optic nerve integrity and detects the optic nerve intra- sheath haematomas. However, MRI is not indicated in suspected metal foreign bodies. Diffusion-weighted imaging (DWI) may reveal optic nerve hyper intensity signals, aiding in the diagnosis of indirect TON.

8. Additional tests

    a. Visual field analysis by Perimetry: TON suspects are subjected to visual field analysis. Quantification of field defects is useful to estimate the prognosis of visual outcome. Simple screening by confrontation method is advised at bedside for unstable patients.
    b. Visual-evoked potential (VEP) and Electro Retino Graphy (ERG) are promising investigations in the modern times for diagnosis of TON. Flash visual evoked potential (fVEP) with a value of more than fifty percentage normal wave pattern is considered a good visual prognosis.[43]
    c. Optical coherence tomography (OCT) records the retinal nerve fibre layer thinning in TON. It is usually not detected in the initial stages. Thus, OCT is not the investigation of choice in TON. OCT might be good in the future to measure nerve reperfusion.[44]

6. Treatment (Figure 3)

Besides from timely monitoring, the other two management modalities that are used include high dose of steroids or surgical decompression of optic nerve. The management of TON is quite cumbersome because monitoring is found to be superior to the other two modalities and there is less data in the literature as to prove this statement as false. The International Optic Nerve Trauma Study (IONTS)—showed that there is no proven success from either steroid treatment or optic canal decompression surgery. When there is no signs of hematoma or fractures of the optic canal, then a mere simple observation is the mainstay of management.[45-47]

6.1. Corticosteroid therapy[48-54]

Corticosteroids are commonly used in the management of traumatic optic neuropathy due to their potential to stabilize cell membranes, reduce vascular permeability, and suppress inflammation. These effects may help limit further damage to the optic nerve by decreasing edema and oxidative stress in neural tissues. The typical treatment protocol involves administering high-dose intravenous methylprednisolone, which is then transitioned to oral prednisolone with a gradual taper. Despite their widespread use, the effectiveness of corticosteroids remains debated. Findings from the International Optic Nerve Trauma Study (IONTS)—a major prospective investigation—indicate no significant difference in visual outcomes among patients treated with corticosteroids, those who underwent surgery, and those who

were simply observed. The study also found that neither the dosage nor the timing of steroid administration influenced the prognosis in a statistically meaningful way. However, other clinical reports have shown varying outcomes. For instance, research by Sitaula et al. compared observation, oral corticosteroid therapy (1 mg/kg of prednisolone for one week followed by a six-week taper), and high-dose intravenous methylprednisolone (1 g/day for three days, then tapered oral prednisolone). Their findings suggested that patients receiving high-dose steroids had a more rapid and favourable visual recovery than the other groups.

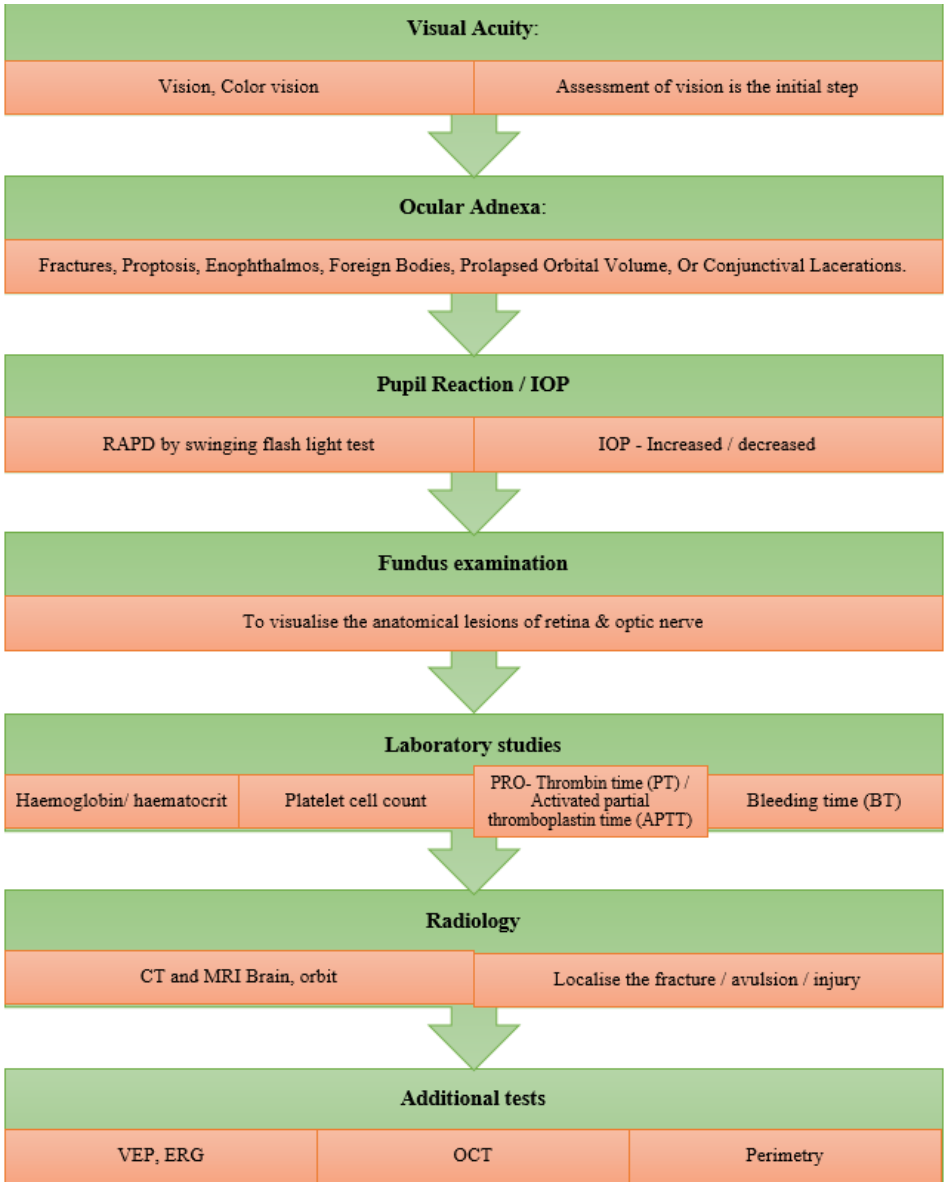

Figure 2: Neuro-ophthalmic evaluation of TON

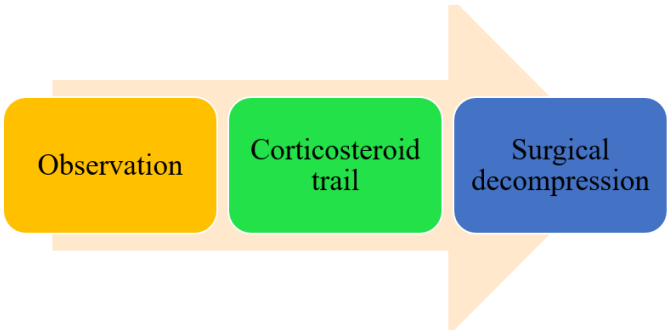

Figure 3: Treatment of TON

6.2. Surgery:[55-62]

Surgical decompression of the optic nerve is one of the treatment modalities considered in cases of traumatic optic neuropathy, particularly when there is evidence of optic canal compression or when vision deteriorates despite medical management. There are three main surgical approaches to achieve decompression: the transcranial route, the transorbital approach, and the endoscopic trans nasal technique. Among these, the transorbital and transcranial methods are gradually falling out of favor due to cosmetic concerns, although they offer a wider surgical field and potentially more complete nerve decompression. The endoscopic trans nasal approach, which does not leave visible scars, is becoming more popular despite some limitations in surgical access and visualization, which may affect outcomes in certain cases. The current literature lacks robust, large-scale studies that clearly support the superiority of any surgical technique over conservative or medical management. As such, the benefit of surgery—either alone or combined with corticosteroids—remains inconclusive. Reported complications from surgical decompression include cerebrospinal fluid leaks, hemorrhage, and postoperative infections. Given the uncertain efficacy and potential risks, the decision to pursue surgical intervention should be made collaboratively, with a clear explanation to the patient and family about the potential outcomes and complications. A case-by-case assessment, guided by clinical judgment and radiologic findings, remains essential.

7. Newer Modalities in Management

7.1. Diagnostic innovations: Doppler sonography[63]

Ultrasound Doppler measurements can be used in TON to measure central retinal artery haemodynamics. There is a reduction in the flowrates of the systole & end-diastole in the patients with decreased peak systolic velocity (PSV) and end-diastolic velocity (EDV) in the central retinal artery (CRA) of the damaged eyes. Measuring the optic nerve sheath diameter (ONSD) can show optic nerve changes using arteriovenous color Doppler.

7.2. Experimental treatments

7.2.1.. Erythropoietin[64]

Erythropoietin, a hormone primarily known for regulating red blood cell production, has demonstrated neuroprotective properties due to its anti-apoptotic and anti-inflammatory effects. Preliminary studies have suggested that EPO administration may contribute to visual recovery in TON by mitigating secondary neuronal injury.

7.2.2. Glutamate antagonists[65-67]

Glutamate plays a major role in excitotoxicity, which can damage retinal ganglion cells following optic nerve trauma. Agents like memantine, phenytoin, and dizocilpine (MK-801), which block NMDA receptors, have been shown in animal models to protect against such excitotoxic injury and support neuronal survival.

7.2.3. Crystallin & citrus naringenin[68]

Crystallins, which belong to the small heat-shock protein family, have demonstrated potential anti-inflammatory and axon-regenerating effects in nerve injury models. Similarly, naringenin, a natural flavonoid found in citrus fruits, has been associated with improved survival of retinal ganglion cells in experimental TON settings.

7.2.4. Nerve growth factors[69]

Growth factors such as fibroblast growth factor-2 (FGF-2), brain-derived neurotrophic factor (BDNF), and ciliary neurotrophic factor (CNTF) are being explored for their capacity to promote regeneration of damaged optic nerve fibers. These molecules may enhance the survival and functional recovery of injured neurons.

7.2.5. Mesenchymal stem cells[70]

Mesenchymal stem cells (MSCs) are gaining attention in neuro-regenerative research. In models of optic nerve trauma, MSCs have demonstrated the ability to promote axonal repair, modulate inflammatory responses, and facilitate the survival of retinal ganglion cells, potentially offering a future therapeutic avenue for TON.

8. Conclusion

Traumatic optic neuropathy (TON) is a notable cause of visual loss following head trauma and often occurs in association with orbital fractures and soft tissue injuries. Early and thorough assessment—including visual acuity, pupillary response, and color vision—is essential for timely diagnosis and management. High-resolution CT imaging is crucial for identifying structural damage such as optic nerve avulsion, hematomas, or fractures involving the optic canal. According to findings from the International Optic Nerve Trauma Study, observation remains a reasonable approach in cases without radiological signs of compression. However, when there is evidence of progressive vision deterioration or structural abnormalities affecting the optic nerve, high-dose corticosteroid therapy should be initiated promptly. If vision continues to deteriorate despite medical treatment, or if imaging reveals compressive lesions, surgical decompression of the optic canal may be considered. Due to the complex and variable nature of TON, a multidisciplinary approach involving ophthalmology, neurology, and radiology is essential. Shared decision-making with the patient and their family is also critical in managing expectations and improving the likelihood of visual recovery.

9. Source of Funding

None.

10. Conflict of Interest

None.

11. Acknowledgement

Nil.

References

1. Evanson NK, Guilhaume-Correa F, Herman JP, Goodman MD. Optic tract injury after closed head traumatic brain injury in mice: a model of indirect traumatic optic neuropathy. *PLoS One*. 2018;13(5):e0197346. https://doi.org/10.1371/journal.pone.0197346
2. Nugent RM, editor. Optic Neuropathy: from Diagnosis to Treatment. New York: Nova Science Publishers, Incorporated; 2020.
3. Pula JH, Yuen CA. Eyes and stroke: the visual aspects of cerebrovascular disease. *Stroke Vasc Neurol*. 2017;2(4):210–20. https://doi.org/10.1136/svn-2017-000079
4. Cockerham GC, Goodrich GL, Weichel ED, Orcutt JC, Rizzo JF, Bower KS, et al. Eye and visual function in traumatic brain injury. *J Rehabil Res Dev*. 2009;46(6):811–8. https://doi.org/10.1682/jrrd.2008.08.0109
5. Edmund J, Godtfredsen E. Unilateral optic atrophy following head injury. *Acta Ophthalmol.* 1963;41(6):693–7. doi: 10.1111/j.1755-3768.1963.tb03588.x.
6. Nau HE, Gerhard L, Foerster M, Nahser HC, Reinhardt V, Joka T. Optic nerve trauma: clinical, electrophysiological and histological remarks. *Acta Neurochir (Wien)*. 1987;89(1-2):16–27. https://doi.org/10.1007/BF01406662
7. Pirouzmand F. Epidemiological trends of traumatic optic nerve injuries in the largest Canadian adult trauma center. *J Craniofac Surg*. 2012;23(2):516–20. https://doi.org/10.1097/SCS.0b013e31824cd4a7
8. Karimi S, Arabi A, Ansari I, Shahraki T, Safi S. A systematic literature review on traumatic optic neuropathy. *J Ophthalmol*. 2021;2021:5553885. https://doi.org/10.1155/2021/5553885
9. Lee V, Ford RL, Xing W, Bunce C, Foot B. Surveillance of traumatic optic neuropathy in the UK. *Eye (Lond)*. 2010;24(2):240–50. https://doi.org/10.1038/eye.2009.79
10. Wyllie AM, McLeod D, Cullen JF. Traumatic ischaemic optic neuropathy. *Br J Ophthalmol*. 1972;56(11):851–3. https://doi.org/10.1136/bjo.56.11.851
11. Miller NR. Traumatic Optic Neuropathy. *J Neurol Surg B Skull Base*. 2021;82(1):107–15. https://doi.org/10.1055/s-0040-1722632
12. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy: an evolving understanding. *Am J Ophthalmol*. 2011;151(6):928–33.e2. https://doi.org/10.1016/j.ajo.2011.02.007
13. Atkins EJ, Newman NJ, Biousse V. Post-traumatic visual loss. *Rev Neurol Dis*. 2008;5(2):73–81.
14. Cirovic S, Bhola RM, Hose DR, Howard IC, Lawford PV, Marr JE, et al. Computer modelling study of the mechanism of optic nerve injury in blunt trauma. *Br J Ophthalmol*. 2006;90(6):778–83. https://doi.org/10.1136/bjo.2005.086538
15. Sarkies N. Traumatic optic neuropathy. *Eye* 18, 1122–1125 (2004). https://doi.org/10.1038/sj.eye.6701571
16. Carta A, Ferrigno L, Salvo M, Bianchi-Marzoli S, Boschi A, Carta F. Visual prognosis after indirect traumatic optic neuropathy. *J Neurol Neurosurg Psychiatry*. 2003;74(2):246–8.
17. Kumaran AM, Sundar G, Chye LT. Traumatic optic neuropathy: a review. *Craniomaxillofac Trauma Reconstr*. 2015;8(1):31–41. https://doi.org/10.1055/s-0034-1393734
18. Yu-Wai-Man P. Traumatic optic neuropathy—clinical features and management issues. *Taiwan J Ophthalmol*. 2015;5(1):3–8. https://doi.org/10.1016/j.tjo.2015.01.003
19. Pujari A, Saxena R, Phuljhele S, Bhaskaran K, Basheer S, Sharma P. Pathomechanism of optic nerve avulsion. *Med Hypotheses*. 2019;125:28–30. https://doi.org/10.1016/j.mehy.2019.02.031
20. Hayreh SS. Management of ischemic optic neuropathies. *Indian J Ophthalmol*. 2011;59(2):123–36. https://doi.org/10.4103/0301-4738.77024
21. Thale A, Jungmann K, Paulsen F. Morphological studies of the optic canal. *Orbit*. 2002;21(2):131–7. https://doi.org/10.1076/orbi.21.2.131.7187
22. Chaon BC, Lee MS. Is there treatment for traumatic optic neuropathy? *Curr Opin Ophthalmol*. 2015;26(6):445–9. https://doi.org/10.1097/ICU.0000000000000198
23. He ZH, Lan ZB, Xiong A, Hou GK, Pan YW, Li Q, et al. Endoscopic decompression of the optic canal for traumatic optic neuropathy. *Chin J Traumatol*. 2016;19(6):330–2. https://doi.org/10.1016/j.cjtee.2016.03.004
24. Mahler S, Huang YX, Liang M, Avalos A, Tyszka JM, Mertz J, et al. Assessing depth sensitivity in laser interferometry speckle visibility spectroscopy through source-to-detector distance variation and cerebral blood flow monitoring in humans and rabbits. *Biomed Opt Express*. 2023;14(9):4964–78. https://doi.org/10.1364/BOE.498815
25. Ahmad S, Fatteh N, El-Sherbiny NM, Naime M, Ibrahim AS, El-Sherbini AM, et al. Potential role of A2A adenosine receptor in traumatic optic neuropathy. *J Neuroimmunol*. 2013;264(1-2):54–64. https://doi.org/10.1016/j.jneuroim.2013.09.015
26. Cansler SM, Evanson NK. Connecting endoplasmic reticulum and oxidative stress to retinal degeneration, TBI, and traumatic optic neuropathy. *J Neurosci Res*. 2020;98(3):571–4. https://doi.org/10.1002/jnr.24543
27. Guy WM, Soparkar CNS, Alford EL, Patrinely JR, Sami MS, Parke RB. Traumatic optic neuropathy and second optic nerve injuries. *JAMA Ophthalmol*. 2014;132(5):567–71. https://doi.org/10.1001/jamaophthalmol.2014.82
28. McGee S. The pupils. In: McGee S, editor. Evidence-Based Physical Diagnosis (Fourth Edition). Elsevier; 2018. p. 161–80.e3. https://doi.org/10.1016/B978-0-323-39276-1.00021-4
29. Tabatabaei SA, Soleimani M, Alizadeh M, Movasat M, Mansoori MR, Alami Z, et al. Predictive value of visual evoked potentials, relative afferent pupillary defect, and orbital fractures in patients with traumatic optic neuropathy. *Clin Ophthalmol*. 2011;5:1021–6. https://doi.org/10.2147/OPTH.S21409
30. Ford RL, Lee V, Xing W, Bunce C. A 2-year prospective surveillance of pediatric traumatic optic neuropathy in the United Kingdom. *J AAPOS*. 2012;16(5):413–7. https://doi.org/10.1016/j.jaapos.2012.04.009
31. Jacobs SM, Van Stavern GP. Neuro-ophthalmic deficits after head trauma. *Curr Neurol Neurosci Rep*. 2013;13(11):389. https://doi.org/10.1007/s11910-013-0389-5
32. Chen B, Zhang H, Zhai Q, Li H, Wang C, Wang Y. Traumatic optic neuropathy: a review of current studies. *Neurosurg Rev*. 2022;45(3):1895–913. https://doi.org/10.1007/s10143-021-01717-9
33. Klein A, Wahbi W. Traumatic Optic Neuropathy Internet. Frontiers In Traumatic Brain Injury. IntechOpen; 2022. http://oi.org/10.5772/intechopen.104731
34. Yang QT, Fan YP, Zou Y, Kang Z, Hu B, Liu X, 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(6):301–7. https://doi.org/10.1159/000330723
35. Li J, Shi W, Li M, Wang Z, He H, Xian J, et al. Time-dependent diffusion tensor changes of optic nerve in patients with indirect traumatic optic neuropathy. *Acta Radiol*. 2014;55(7):855–63. https://doi.org/10.1177/0284185113506900
36. Bodanapally UK, Shanmuganathan K, Shin RK, Dreizin D, Katzman L, Reddy RP, et al. Hyperintense optic nerve due to diffusion restriction: diffusion-weighted imaging in traumatic optic neuropathy. *AJNR Am J Neuroradiol*. 2015;36(8):1536–41. https://doi.org/10.3174/ajnr.A4290
37. Reddy RP, Bodanapally UK, Shanmuganathan K, Van der Byl G, Dreizin D, Katzman L, et al. Traumatic optic neuropathy: facial computed tomography findings affecting visual acuity. *Emerg Radiol*. 2015;22(4):351–6. https://doi.org/10.1007/s10140-014-1292-3

38. Folkestad L, Jönsson L, Karlsson T. Orbital floor fractures—a comparison between computed tomography images and findings at surgery. *Eur Arch Otorhinolaryngol*. 2023;280(6):2795–803. https://doi.org/10.1007/s00405-022-07801-0
39. Hathiram BT, Khattar VS, Sonawane HP, Watve PJ. Traumatic optic neuropathy—our experience. *Indian J Otolaryngol Head Neck Surg*. 2010;62(3):229–35. https://doi.org/10.1007/s12070-010-0072-y
40. Ibanez L, Navallas M, de Caceres IA, Martinez-Chamorro E, Borruel S. CT features of posttraumatic vision loss. AJR. American Journal of Roentgenology. 2021;217(2):469-479
41. Allison JR, Kearns A, Banks RJ. Predicting orbital fractures in head injury: a preliminary study of clinical findings. *Emerg Radiol*. 2019;27(1):31–6. https://doi.org/10.1007/s10140-019-01720-0
42. Sarkies N. Traumatic optic neuropathy. *Eye (Lond)*. 2004;18(11):1122–5. https://doi.org/10.1038/sj.eye.6701571
43. Holmes MD, Sires BS. Flash visual evoked potentials predict visual outcome in traumatic optic neuropathy. *Ophthalmic Plast Reconstr Surg*. 2004;20(5):342–6. https://doi.org/10.1097/01.iop.0000134272.55294.4c
44. Cunha LP, Costa-Cunha LVF, Malta RFS, Monteiro MLR. Comparison between retinal nerve fiber layer and macular thickness measured with optical coherence tomography detecting progressive axonal loss following traumatic optic neuropathy. *Arq Bras Oftalmol*. 2009;72(5):622–5. https://doi.org/10.1590/s0004-27492009000500004
45. He Z, Li Q, Yuan J, Zhang X, Gao R, Han Y, et al. Evaluation of transcranial surgical decompression of the optic canal as a treatment option for traumatic optic neuropathy. *Clin Neurol Neurosurg*. 2015;134:130–5. https://doi.org/10.1016/j.clineuro.2015.04.023
46. Wladis EJ, Aakalu VK, Sobel RK, McCulley TJ, Foster JA, Tao JP, et al. Interventions for indirect traumatic optic neuropathy: a report by the American Academy of Ophthalmology. *Ophthalmology*. 2021;128(6):928–37. https://doi.org/10.1016/j.ophtha.2020.10.038
47. Wolin MJ, Lavin PJ. Spontaneous visual recovery from traumatic optic neuropathy after blunt head injury. *Am J Ophthalmol*. 1990;109(4):430–5. https://doi.org/10.1016/s0002-9394(14)74609-4
48. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. *Ophthalmology*. 1999;106(7):1268–77. https://doi.org/10.1016/s0161-6420(99)00707-1
49. Sitaula S, Dahal HN, Sharma AK. Clinical evaluation and treatment outcome of traumatic optic neuropathy in Nepal: a retrospective case series. *Neuroophthalmology*. 2017;42(1):17–24. https://doi.org/10.1080/01658107.2017.1331362
50. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. *Cochrane Database Syst Rev*. 2007;(4):CD006032. https://doi.org/10.1002/14651858.CD006032.pub2
51. Steinsapir KD. Treatment of traumatic optic neuropathy with high-dose corticosteroid. *J Neuroophthalmol*. 2006;26(1):65–7. https://doi.org/10.1097/01.wno.0000204646.94991.68
52. Pandey S, Reddy GS, Chug A, Dixit A, Raja BS. Comparison of surgical decompression and steroid therapy for the management of traumatic optic neuropathy: a systematic review and meta-analysis. *Craniomaxillofac Trauma Reconstr*. 2023;16(4):306–16. https://doi.org/10.1177/19433875221142682
53. Edwards P, Arango M, Balica L, Cottingham R, El-Sayed H, Farrell B, et al. Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury—outcomes at 6 months. *Lancet*. 2005;365(9475):1957–9. https://doi.org/10.1016/S0140-6736(05)66552-X
54. Lee KF, Nor NIM, Yaakub A, Hitam WWH. Traumatic optic neuropathy: a review of 24 patients. *Int J Ophthalmol*. 2010;3(2):175–8. https://doi.org/10.3980/j.issn.2222-3959.2010.02.20
55. Yu-Wai-Man P, Griffiths PG. Surgery for traumatic optic neuropathy. *Cochrane Database Syst Rev*. 2013;6(6):CD005024. https://doi.org/10.1002/14651858.CD005024.pub3
56. Oh HJ, Yeo DG, Hwang SC. Surgical treatment for traumatic optic neuropathy. *Korean J Neurotrauma*. 2018;14(2):55–60. https://doi.org/10.13004/kjnt.2018.14.2.55
57. Martinez-Perez R, Albonette-Felicio T, Hardesty DA, Carrau RL, Prevedello DM. Outcome of the surgical decompression for traumatic optic neuropathy: a systematic review and meta-analysis. *Neurosurg Rev*. 2021;44(2):633–41. https://doi.org/10.1007/s10143-020-01260-z
58. Chen HH, Lee MC, Tsai CH, Pan CH, Lin YT, Chen CT. Surgical decompression or corticosteroid treatment of indirect traumatic optic neuropathy: a randomized controlled trial. *Ann Plast Surg*. 2020;84(1 Suppl 1):S80–3. https://doi.org/10.1097/SAP.0000000000002186
59. Chen F, Zuo K, Feng S, Guo J, Fan Y, Shi J, et al. A modified surgical procedure for endoscopic optic nerve decompression for the treatment of traumatic optic neuropathy. *N Am J Med Sci*. 2014;6(6):270–3. https://doi.org/10.4103/1947-2714.134372
60. Gupta D, Gadodia M. Transnasal endoscopic optic nerve decompression in post traumatic optic neuropathy. *Indian J Otolaryngol Head Neck Surg*. 2018;70(1):49–52. https://doi.org/10.1007/s12070-017-1211-5
61. Xie D, Yu H, Ju J, Zhang L. The outcome of endoscopic optic nerve decompression for bilateral traumatic optic neuropathy. *J Craniofac Surg*. 2017;28(4):1024–6. https://doi.org/10.1097/SCS.0000000000003743
62. Yu B, Ma Y, Tu Y, Wu W. The outcome of endoscopic transethmosphenoid optic canal decompression for indirect traumatic optic neuropathy with no-light-perception. *J Ophthalmol*. 2016;2016:6492858. https://doi.org/10.1155/2016/6492858
63. Shankar H, Pagel PS. Potential adverse ultrasound-related biological effects: a critical review. *Anesthesiology*. 2011;115(5):1109–24. https://doi.org/10.1097/ALN.0b013e31822fd1f1
64. Ghezzi P, Brines M. Erythropoietin as an antiapoptotic, tissue-protective cytokine. *Cell Death Differ*. 2004;11 Suppl 1:S37–44. https://doi.org/10.1038/sj.cdd.4401450
65. Buonfiglio F, Böhm EW, Pfeiffer N, Gericke A. Oxidative stress: a suitable therapeutic target for optic nerve diseases? *Antioxidants (Basel)*. 2023;12(7):1465. https://doi.org/10.3390/antiox12071465
66. WoldeMussie E, Yoles E, Schwartz M, Ruiz G, Wheeler LA. Neuroprotective effect of memantine in different retinal injury models in rats. *J Glaucoma*. 2002;11(6):474–80. https://doi.org/10.1097/00061198-200212000-00003
67. Schuettauf F, Naskar R, Vorwerk CK, Zurakowski D, Dreyer EB. Ganglion cell loss after optic nerve crush mediated through AMPA-kainate and NMDA receptors. *Invest Ophthalmol Vis Sci*. 2000;41(13):4313–6.
68. Fischer D, Hauk TG, Müller A, Thanos S. Crystallins of the beta/gamma-superfamily mimic the effects of lens injury and promote axon regeneration. *Mol Cell Neurosci*. 2008;37(3):471–9. https://doi.org/10.1016/j.mcn.2007.11.002
69. Tsai NY, Gallo RA, Pelaez D, Tse DT, Kinde B. Traumatic optic neuropathy: challenges and opportunities in developing neuroprotective and neuroregenerative therapies. *Curr Ophthalmol Rep*. 2024;12:23–9. https://doi.org/10.1007/s40135-024-00323-4
70. Cen LP, Ng TK, Liang JJ, Zhuang X, Yao X, Yam GHF, et al. Human periodontal ligament-derived stem cells promote retinal ganglion cell survival and axon regeneration after optic nerve injury. *Stem Cells*. 2018;36(6):844–55. https://doi.org/10.1002/stem.2812

Cite this article: Bhava SG, Sundaram SG, Kamareddy S, Mohapatra A. Traumatic optic neuropathy: A review. *Indian J Clin Exp Ophthalmol*. 2025;11(4):584–590.