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# Review

# **Direct vs. Indirect Etiology of Traumatic Optic Neuropathy**

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# Abstract

When it comes to vision, there is a rare condition which has more potential to impair the vision, and this condition is called Traumatic optic neuropathy. Direct traumatic optic neuropathy occurs when there is direct physical trauma to the optic nerve, often due to penetrating injuries or fractures involving the bones around it. On the other hand, indirect (TON) is caused by blunt force trauma that is transmitted through the tissues or bones around the eye, leading to stretching, tearing, or damage to the blood supply of the optic nerve. Common causes of indirect (TON) include motor vehicle accidents, falls, assaults, and sports-related injuries. The mechanics, blood flow, and inflammation are all sequentially organized in Traumatic optic neuropathy. Mechanical factors disrupt the integrity of the nerve fibers and blood supply, vascular factors reduce the blood flow, and inflammatory factors activate processes that cause cell death within the optic nerve. The signs of (TON) can vary in presentation. Commonly involve a decrease in visual clarity abnormalities in the pupil's reaction to light visual field issues, changes in color perception, and alterations in the appearance of the optic disc. Diagnosing and managing (TON) can be quite challenging since there are no markers or reliable predictors of prognosis. In some cases, observation may be appropriate, while corticosteroids can be employed for their inflammatory properties and potential benefits to optic nerve blood supply. Surgical decompression focuses on relieving pressure or removing obstructions that are affecting the nerve.

Keywords: Direct Traumatic Optic Neuropathy, Etiology, Indirect Traumatic Optic Neuropathy, Trauma, Optic Neuropathy

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#### Introduction

Traumatic optic neuropathy (TON) is also a rare condition, can result in vision impairment due to damage to the nerve. Based on injury, traumatic optic neuropathy can be classified into two categories, which are Direct and Indirect (1, 2). In cases of (TON), the optic nerve is harmed by a penetrating wound or fracture in the surrounding bone, leading to complete severance, tearing, or compression of the nerve. Indirect (TON) occurs when blunt force is transmitted to the nerve through tissues or bony structures of the eye socket, causing shearing stretching or ischemic injury to the nerve. Motor vehicle accidents are a cause of traumatic optic neuropathy (TON), with falls, assaults, and sports injuries also being frequent triggers. In several head injuries, the prevalence of Traumatic optic neuropathy is approximately 0.7-2.5% (3). The main factors that may cause (TON) conditions include mechanical, vascular, and inflammatory factors (4). Mechanical factors include the disruption of the axonal integrity and the blood supply of the optic nerve by the traumatic force. Vascular factors encompass the decrease in blood circulation and oxygen supply to the nerve caused by conditions like narrowed blood vessels, blood clot formation, or swelling. In cases of (TON), it tends to impact the nerve within the orbit or along its canal. Whereas indirect (TON) generally affects the segment of the nerve where it meets the orbital segment within its canal. However, diagnosing and treating (TON) is very difficult to conduct (5). The reason for this is that there are not any reliable indicators that we can trust currently, which may produce more false positive results. Typically, in order to determine the cause, we need to gather information about the patient's background, conduct an examination, and review any relevant imaging tests. There are currently no identified factors that increase the risk of (TON). In the International Optic Nerve Trauma Study, it was observed that 85% of patients diagnosed with (TON) were male, and their average age was 34. The frequent causes of injury leading to (TON) were motor vehicle accidents, bicycle accidents, falls, and assaults. Although abusive injuries are uncommon in causing

(TON), they do raise concerns when it comes to infants. (6). Currently, there are many possible rests available to assess the visual conditions and the function of the eye. Some such tests for measuring acuity, examining the pupillary light reflex, checking the visual field, evaluating color vision, and employing optical coherence tomography (OCT) (7). OCT is an invasive imaging technique that allows us to analyze the thickness and structure of the retinal nerve fiber layer. It gives us valuable information about the individual optic nerve fibers. OCT can help detect subtle changes in the RNFL that may not be visible on fundus examination or other imaging modalities. OCT can also help differentiate between direct and indirect (TON), as direct (TON) tends to cause more severe RNFL thinning than indirect (TON). In some cases, the frequent indicator is a reduction in visual clarity, which can vary from slight impairment to the complete inability to perceive light. Other signs include relative afferent pupillary defects, visual field defects, color vision defects, and optic disc swelling or pallor. Imaging techniques, like CT or MRI scans, can detect fractures, bleeding, or swelling affecting the nerve or canal (8). The treatment options for (TON) are a subject of debate. Include observation, corticosteroids, surgical decompression, or a combination of these approaches. However, there is no evidence that any specific treatment is better than just observation in terms of improving vision. So, this review study aims to gather information and present the current Direct vs. Indirect Etiology of Traumatic Optic Neuropathy.

# Methodology

This study is built upon an examination of the literature conducted on September 11, 2023, utilizing the Medline and PubMed databases. To identify pertinent studies, we employed a search strategy incorporating medical subject headings and relevant keywords tailored to the database requirements. The search terms employed in this investigation encompassed "Direct Traumatic Optic Neuropathy", "Indirect Traumatic Optic Neuropathy", and "Etiology". We exclusively focused on studies involving human subjects and

published in the English language. Additionally, we did not include articles published before 2008 in our search. Following the initial search, meticulously reviewed multiple articles to ensure they met our specific inclusion criteria. Only those studies that were directly relevant to our research objectives were retained for further examination. Afterward, we carefully analyzed the chosen articles, thoroughly examining their content for information. Moreover, we manually reviewed the reference lists of these articles to find any studies that could be valuable for our research.

### Discussion

The optic nerve, which is made up of the axons of retinal ganglion cells and support cells has four segments: 1 mm) 24 mm) intra-canalicular (9 mm) and intracranial (16 mm). Traumatic optic neuropathy (TON) refers to loss caused by injury to the nerve (9). Safety of medical and surgical treatments, for indirect traumatic optic neuropathy (TON) which refers to damage to the nerve that happens after the optic nerve head (10). Motor vehicle accidents are the leading cause of indirect (TON), with falls, assaults, and sports injuries following suit. The precise pathophysiology of both direct and indirect (TON) remains incompletely likely involves mechanical, but understood vascular, and inflammatory factors. Mechanical factors encompass the disruption of axonal integrity and blood supply to the optic nerve caused by traumatic force. Vascular factors also refer to decreased blood flow and oxygen supply to the nerve caused by issues like vasospasm, thrombosis, or edema (11). On the other hand, inflammatory factors involve the activation of cells and cytokines, which result in the apoptosis and necrosis of cells in the optic nerve. The type and extent of injury may vary, with direct (TON) primarily affecting the intra-orbital or intra-canalicular optic nerve segments and indirect (TON) impacting the intracanalicular segment at its junction with the intraorbital segment (12, 13).

Diagnosing and managing (TON) poses significant challenges due to the absence of specific clinical signs and reliable prognostic indicators. Diagnosing a condition often involves taking into consideration the patient's history, conducting an examination, and reviewing imaging studies. A typical indication of the condition is a decrease in clarity ranging from slight impairment to total loss of vision. Additional signs may encompass afferent pupillary defects, visual field abnormalities, color vision deficiencies, and optic disc swelling or pallor. Different types of imaging methods, example as computed tomography (CT) or magnetic resonance imaging (MRI), are used to identify fractures, hemorrhages, or edema that impact the nerves or canal. The treatment choices for (TON) are still a topic of debate, including observing the condition using corticosteroids, opting for decompression, or these different combining approaches. Nevertheless, no definitive evidence suggests any treatment surpasses observation alone concerning visual outcomes.

# Clinical Manifestations

The clinical manifestation of neuropathy can be different in different conditions, and they may contain several factors which includes severity, timing, and location of the injury. Additionally, the presence or absence of associated injuries and individual responses to trauma play a role. The common and significant symptom is vision loss. This can occur in one eye or both eyes. It may be partial or complete (14-16). Visual loss can happen immediately after the injury. Be delayed. It can also be either temporary or permanent. Remain stable or worsen over time. Interestingly, the extent of nerve damage doesn't always correlate with the degree of loss experienced by patients. Some individuals may experience recovery within days to weeks following the injury; others may see no improvement or even experience deteriorating vision. There are factors which influence outcomes. These factors include age, gender, acuity before the injury occurred, whether the trauma was direct or indirect in nature, where precisely in the orbit or optic canal it occurred (intra-orbital vs intra-canalicular), whether there's an optic canal fracture or hematoma present duration of ischemia (lack of blood flow) level of inflammation experienced by the optic nerve tissue

as a response to trauma treatment efficacy as well as genetic factors.

It's worth noting that traumatic optic neuropathy is often associated with injuries in about 72% of cases, followed by frontotemporal injuries at around 12%. The most common causes are traffic accidents and falls. An abnormal direct light reflex provides the most reliable diagnosis in patients with impaired consciousness. Most patients undergo a fundus examination, initially appearing normal if the lesion does not involve the retina and/or anterior optic nerve. However, the patient's optic disc will later become pale and atrophied. Several combined craniofacial or other systemic injuries may delay the diagnosis of (TON). The diagnosis may also be complicated by non-ON damage (damage to the eye or intracerebral visual circuits, etc.). In this case, the diagnosis of (TON) requires a comprehensive clinical assessment (ranging from ophthalmic to radiographic assessments). The majority of patients with (TON) present to the clinic with vision loss. The patient needs to have an eye examination, which includes tests for field (VF) visual acuity (VA) and capturing images of the back of the eye (fundus photography). If performing an eye examination pattern reversal is not feasible, visual evoked potentials (P VEPs) can be employed as an alternative. The P-VEP is helpful in assessing visual function in children and infants, as well as detecting nonorganic vision loss, nonorganic vision loss in complex patients, and optic neuropathy.

Several complications can occur during the onset of this rare disease. Some other clinical complications that may accompany visual loss in (TON) include pupillary abnormalities, ocular motility disorders, eyelid injuries, orbital fractures, facial fractures, intracranial hemorrhage, or cerebral contusion (17, 18). A severe condition such as pupillary abnormalities includes relative afferent pupillary defect, which is a sign of asymmetric optic nerve dysfunction, and fixed or dilated pupils, which may indicate severe optic nerve compression or transection. In the case of orbital fractures, this may include blowout fractures, Zygomaticomaxillary complex fractures, or naso-orbital-ethmoid fractures that may cause orbital emphysema, enophthalmos,

or telecanthus. These fractures can potentially affect functions like breathing, jaw alignment, and fluid drainage. If any bleeding takes place inside the skull, it will be considered as hemorrhage. Such bleeding can lead to increased pressure inside the skull, herniation, or stroke. Cerebral contusion includes coup or contrecoup injury, which may cause cognitive impairment, seizures, or coma.

# Management

The management of traumatic optic neuropathy is a subject that often sparks debates and involves considerations. The primary objectives of managing (TON) are to preserve or restore vision, address any complications that may arise, and enhance the patient's quality of life. The key approaches to management typically involve observing the condition. administering corticosteroids. performing decompression if necessary, or utilizing a combination of these options (19). It's worth noting that different experts may hold varying opinions on which method is most suitable for each type of (TON). In some studies, it is observed that, based on the assumption, some patients may have spontaneous vision recovery due to natural healing processes or collateral circulation. Mild visual loss in patients may need treatment in cases of presentation contraindications to corticosteroids or preference for surgery or a noninvasive management (20). We need to keep in mind that it is advised to have Observation towards patients may also be indicated for patients with direct (TON) from penetrating injury or complete transection of the optic nerve, as these cases have poor prognoses regardless of treatment. Corticosteroids medications that have inflammatory properties. They reduce swelling as well as enhance blood flow. Corticosteroids are given during indirect (TON), mainly injury caused by ischemia and inflammation rather than mechanical injury. Corticosteroids are typically administered through a route, with doses for brief period. Other drugs Methylprednisolone 30mg/per kg body weight, administered through an infusion at a rate of 5.4 mg per kg/h for a period lasting between 24-48 hours. Corticosteroids may be indicated for indirect (TON) patients due to blunt trauma, early presentation

(within 8 hours), moderate to severe visual loss, or preference for medical management. Corticosteroids may also be indicated for patients with direct (TON) due to compression of the optic nerve by a hematoma or a fracture fragment, as these cases may benefit from reducing the pressure on the nerve (21). The surgical decompression procedure involves making an incision to alleviate nerve pressure by removing blockages or creating space around the nerve. To decompress the nerve, some intervening procedure including external ethmoidectomy, trans-ethmoidal sphenoidotomy, endoscopic endonasal optic nerve decompression, or transcranial optic canal decompression (22, 23). Surgical decompression may be indicated for patients with direct (TON) due to compression of the optic nerve by a hematoma or a fracture fragment, early presentation (within 24 hours), severe visual loss, or failure of corticosteroids. Surgical decompression may also be indicated for indirect (TON) patients due to optic canal fracture or hematoma, progressive visual loss, or preference for surgical management. The combination of corticosteroids and surgical decompression is a multimodal approach that focuses on reducing (TON)'s inflammatory and mechanical aspects, which is very significant to do so. The combination may be indicated for patients with mixed or uncertain etiology of (TON), refractory worsening visual loss, or preference for aggressive management. The combination may also be indicated for patients with direct (TON) due to compression of the optic nerve by a hematoma or a fracture fragment, as these cases may benefit from both reducing the pressure and removing the obstruction on the nerve. Endoscopic optic nerve decompression is used for minimal invasion of any optic nerve decompression. Trans-sphenoethmoidal and trans-sphenoid are the two main surgical endoscopic approaches. When (TON) does not respond to steroids, EOND may be beneficial, and if it is applied before irreversible nerve damage occurs, it may prevent permanent disability. EOND should be performed within one week after an injury in patients with confirmed optic canal fractures as a safe technique.

There are no guidelines for (TON) treatment strategies when steroid treatment fails, and all treatments are based on specialist experience when steroid treatment is unsuccessful. In a study, Sun et al. have shown that there were three (18.75%) cases of sixteen patients with partial recovery of VA after surgical decompression. No improvement in VA was observed for the remaining patients (81.25%) operating more than ten days after injury (24). The efficacy and safety of each modality of management for (TON) are uncertain and controversial, as no randomized controlled trial or meta-analysis compares them in terms of visual outcome. The majority of the evidence relies on looking at cases, observing studies, or expert opinions. However, it's important to note that these sources come with limitations. Some of these limitations include biases in selecting cases, factors that can cause confusion in interpreting results sample sizes, variations in patients and treatments, a lack of criteria and methods, and differences in the length of follow-up periods.

# Conclusion

Diagnosing and managing (TON) can be difficult due to the absence of signs and reliable predictors of prognosis. The available treatment options for (TON), including observation, corticosteroids, surgical decompression, or a combination of these approaches, are surrounded by controversy. Nonetheless, there is no evidence that any particular treatment surpasses the outcomes achieved through observation in terms of visual recovery. Thus, further research is necessary to shed light on indirect (TON)'s origins and progression while devising therapeutic strategies for this debilitating condition.

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# Conflict of interest

There is no conflict of interest

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#### Ethical consideration

Non applicable

# Data availability

Data that support the findings of this study are embedded within the manuscript.

### Author contribution

All authors contributed to conceptualizing, data drafting, collection and final writing of the manuscript.

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