KESANS: International Journal Of Health and Science

e-ISSN: 2808-7178, p-ISSN: 2808-7380

Web: http://kesans.rifainstitute.com/index.php/kesans/index



Traumatic Optic Neuropaty

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Article Information Submitted: 02 December

Accepted: 31 December

2021

Online Publish: 20 January 2022

Abstrack

TON or traumatic optic neuropathy is a condition that can cause blindness which can be caused by trauma to the orbital area or head trauma. In its classification, TON is divided into two, namely direct and indirect. Direct TON or direct TON, is usually caused by a decrease or loss of a person's visual ability and the chance of recovery is smaller than indirect TON. This direct TON usually occurs when the optic nerve is laced with surrounding bone or the cause can be anatomical structural abnormalities. TON treatment can be done in two ways (1) Treatment with high doses of corticosteroids (2) with surgery.

Keywords: traumatic optic neuropathy, indirect trauma, direct trauma, treatment of ton

Introduction

TON or traumatic optic neuropathy is a condition that can cause blindness which can be caused by trauma to the orbital area or head trauma. In its classification, TON is divided into two, namely direct and indirect. Direct TON or direct TON, is usually caused by a decrease or loss of a person's visual ability and the chance of recovery is smaller than indirect TON. This direct TON usually occurs when the optic nerve is laced with surrounding bone or the cause can be anatomical structural abnormalities (Karimi et al. 2021).

The incidence rate of TON in the general population is 1 in 1,000,000 populations (Mahayani, Triningrat, and Manuaba 2017). In some case about 5% are caused by head trauma and 2.5% by facial fractures (Karimi et al. 2021). Based on the symptoms, patients with TON experience sudden, severe and unilateral visual disturbances. Not infrequently can also cause a decrease in visual ability caused by disorders of the optic nerve (Jang 2018). Based on the examination, the diagnosis of TON can be made with CT scans and MRIs related to radiological examinations, and related to visual acuity examinations can use visual acuity examinations, RAPD or relative afferent pupillary defects, visual fields and others (Jang 2018).

Method

The writing of this article uses different types of sources derived from scientific journals. Source searches are conducted on online portals of journal publications such as the National Center for Biotechnology Information/NCBI (ncbi.nlm.nih.gov) and Google Scholar (scholar.google.com). The keywords used are "Traumatic Neuropathy", "Neuropathy Trauma", "Optic Nerve Neuropathy", "Traumatic Optic Neuropathy" and "Treatment for Traumatic Optic Neuropathy"

Definition

Traumatic optic neuropathy (TON) is a disorder or disease caused by damage to the optic nerve due to trauma and occurs due to acute axon damage and causes visual disturbances in the form of visual field defects, color vision disorders to decreased visual acuity which can lead to permanent loss of vision (Mahayani, Triningrat, and Manuaba 2017). Traumatic optic neuropathy (TON) is a vision threatening disorder that can be caused by eye or head trauma and can be categorized into two, namely direct and indirect. Direct TON can be associated with severe visual loss and also a lower chance of recovery compared to indirect TON (Harris and Miller 2017).

Epidemiology

Traumatic optic neuropathy (TON) is usually caused by visual impairment after blunt force trauma or head trauma. The overall incidence of TON is 0.7-2.5%. Indirect TON has a higher prevalence than direct, and it occurs in 0.5-5% of all patients with closed head trauma and 2.5% of patients with facial bone fractures. The most common

was the intracanalicular portion of the indirect TON (71.4%) and then to the orbital apex (16.7%). Intracranial and orbital apex were found in 11.9% of cases (Karimi et al. 2021).

TON has a predominance of gender, 80% of whom are male patients with a mean age of 31 years, and 21% are younger at 18 years. Experienced falls 26%, motor vehicle accidents 21%. It often also occurs in trauma situations in 63% motorized traffic accidents and the main cause is usually falls. However, only 2.3% of patients with head trauma experienced head trauma (Karimi et al. 2021).

TON is an uncommon cause of vision loss due to blunt force trauma in the UK, with a population minimum prevalence of 1 in 1,000,000. Most of the affected patients are young adult males (79-85%) in their 30s. The most common causes of TON are 49% motorcycle accidents, 27% falls and 13% assault/robbery. The population in children, most of the cases occur due to falling 50% and traffic accidents when crossing the road 40% (Yu-Wai-Man, 2015). Indirect traumatic optic neuropathy (ITON) for the case population there are two populations, namely pediatric and adult cases in the UK. For the whole case of TON itself, approximately 1 million cases. About 80% occur in male patients, the majority of whom suffer from these cases due to relatively minor head injuries (Singman et al. 2016).

Etiology

Traumatic optic neuropathy (TON) is a disease caused by damage to the secondary optic nerve system caused by trauma and occurs due to acute axon damage and also causes visual disturbances in the form of visual field defects, visual disturbances when seeing colors to decreased visual acuity and can cause loss of ability. permanent vision (Shabrina and Suhartono, 2021). Other etiology can be due to blows by blunt objects, traffic accidents, falls, violent acts that hit the head, and sports injuries that can cause injury or trauma to the head (Harris and Miller 2017).

Pathogenesis

Injury to the microvascular area of the optic nerve will lead to bleeding into the optic nerve and its sheath. Areas of visual focus on axonal structures may also be impaired by indirect trauma. In this condition, the optic nerve splits into two segments. The distal segment undergoes Wallerian degeneration after separation from the soma cells. While the proximal segment is connected to the nerve soma cells become swollen and form a retraction ball. Soma cells then undergo apoptosis after trauma about 6-24 hours after trauma. Apoptosis of soma cells is characterized by ischemia of the optic nerve (Van Stavern 2014).

Pathophysiology

Based on the mechanism of trauma TON is divided into 2 mechanisms, namely primary and secondary. Primary TON is usually described as direct or direct trauma and indirect or indirect (due to blunt trauma) (Harris and Miller 2017). The cause of TON can be caused by many factors, such as trauma that can affect the axons of ganglion cells

which can cause neuronal loss (Yu-Wai-Man 2015). The direct primary cause according to Steinsapir and Goldberg in Harris and Miller, 2017 that "direct trauma is an open wound in which objects from the outside penetrate into the tissue that will have an impact on the optic nerve. While indirect primary trauma occurs when the force of impact or impact on the skull, the energy will be absorbed by the optic nerve.

A. Primary mechanism

As explained above, the mechanism of trauma to the optic nerve is divided into two, namely direct and indirect trauma. Direct trauma can occur spontaneously and cause irreversible trauma depending on the severity of the injury. This is thought to be due to axon transection in retinal ganglion cells. Direct ischemia will cause necrosis due to damage to the microcirculation. On treatment, direct trauma to the optic nerve has a poor prognosis. Especially if the traumatized eye has no perception of light.

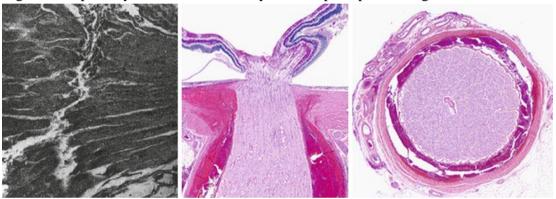


Figure 1. Direct trauma to the optic nerve

Indirect trauma to the optic nerve can occur due to various mechanisms. This indirect trauma can include temporary concussion, permanent concussion, laceration of nerve fibers (not the entire nerve) and direct necrosis/bruising. In particular, the forehead or the temporal region when hit, the impulse will travel to the posterior region through the optic canal. This explains why there is an injury to the optic canal area.

Blunt trauma to the frontal area of the head is more likely to cause indirect TON than trauma to the occipital region. In addition to the skull or facial bones, direct trauma to the eyeball can also cause damage to the optic nerve. For example, in the case of blunt trauma, it is not caused by a direct blow to the facial bone area but by the wave effect of the object.

B. Secondary mechanism

This mechanism is caused by damage disorders such as vasospasm, edema, hemorrhage, and local compression of blood vessels or necrosis of nerve cells. In some cases it can cause cell death due to oxygen free radicals, release of arachidonic acid, activation of bradykinin, intracellular disturbances, production of excitatory amino acids and other disorders. In this secondary mechanism, more emphasis is placed on indirect trauma to the optic nerve, such as ischemia. For prevention strategies, it is usually used to reduce the inflammatory cascade or ischemia and cell apoptosis (Harris and Miller 2017).

Next there is the location of trauma that can cause TON. This is related to the extent of trauma that occurs to the optic nerve. Clinically it can occur in the anterior and posterior or can occur in both. For the anterior part of the optic nerve starting from where the central retinal artery enters and the central retinal vein exits the nerve, about 8-10 mm posterior to the eyeball.

a. Trauma to the anterior

It can be caused by direct or indirect trauma from a collision or blow by a blunt object. The signs will appear swelling of the optic disc resulting in a series of symptoms such as decreased acuity, poor or absent color vision, visual field disturbances, and relative afferent pupillary defects if the trauma is unilateral or asymmetric. Usually accompanied by trauma to the associated blood vessels. This, in turn, may lead to retinal ischemia or infarction, central retinal vein occlusion, anterior ischemic optic neuropathy, or a combination of these phenomena.

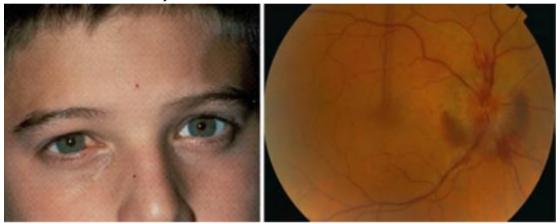


Figure 2. Example trauma to the anterior eye

b. Trauma to the posterior

Posterior TON is more common than anterior TON either the cause can be direct or indirect trauma. On the prognosis, for posterior trauma due to direct trauma tends to be worse than that caused by indirect trauma. The optic canaliculus is the part that is often the site of damage to the posterior TON. In some conditions it can also occur in the intracranial part of the nerve that passes through the falciform ligament structure of the retina (Harris and Miller 2017).

Clinical Manifestations

Optical Coherence Tomography(OCT) is one of the supporting modalities that is growing very rapidly and widely and helps in diagnosis and therapy in the field of ophthalmology. The latest technology from OCT is Spectral Domain OCT (SDOCT). SDOCT examination in TON cases is very useful because it is non-invasive and also with this latest technology, the axial resolution is up to 5-7 m, capable of producing clear images layer by layer of the retina so that damage can be detected early and can even be detected before symptoms occurs (Shabrina and Suhartono, 2021).

In patients with craniofacial trauma to the optic nerve in a normal head, reduced vision and an afferent pupillary defect are usually associated with indirect TON. There are clinical features that can diagnose TON, such as:

- 1. Head trauma
- 2. Relative Afferent Pupillary Defect (RPAD)
- 3. Variable degrees of vision loss
- 4. Color vision impairment
- 5. Different degrees of visual field defects

RPAD is one of the clinical features of mild TON and may be the only clinical finding prior to optic nerve atrophy. It was also found that RAPD was negative in cases of bilateral symmetry and should be considered. Visual Acuity (VA) of visual acuity can range from normal to no light perception, and 40-60% of cases have it or even worse (Jang 2018). In most cases the posterior part of the optic nerve is damaged, but often the optic disc is normal. On funduscopic examination, hemorrhage due to trauma to the optic nerve in the head area can be confirmed as an early indication of TON symptoms.

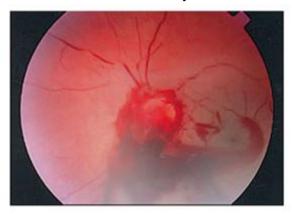


Figure 3. Trauma to the optic nerve on funduscopic examination

Treatment

Corticosteroid therapy for TON became popular in the early 1980s with the aim of reducing edema and secondary post-injury inflammation. Dosage for therapy also varies depending on usage. Dosage ranging from 1-2 mg / kg / day by oral or IV administration to high doses at a dose of 1,000 mg / day. The corticosteroid used is methylprednisolone. In single high-dose use, the initial administration can be 30 mg/kg in a bolus followed by 5.4 mg/kg/hour for 24-48 hours of administration (Harris and Miller 2017). Other sources also discuss surgery as a therapy for TON with indications and contraindications described. Surgical therapy is performed, usually in the form of optic canal decompression or OCD (Yu et al. 2020).

For indications for TON surgery 1) History of traumatic head and face injuries with and without optic canal injury; 2) progressive vision loss unrelated to non-traumatic intraocular lesions; (3) lack of evidence of damage or avulsion of the intracranial portion of the optic nerve; 4) Prolonged absolute latency or reduced amplitude in preoperative

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visual arousal potential (VEP) scans; 5) failure to respond to steroid therapy; and 6) the presence of bone fragments pressing on the optic nerve or a hematoma near the optic nerve. For surgical contraindications to TON therapy 1) complete disturbance of the optic chiasm; 2) complete atrophy of the optic nerve; 3) Carotid cavernous fistula; and 4) Inadequate medical conditions to perform general anesthesia (Oh, Yeo, and Hwang 2018).

Actually the TON therapy technique with surgery is considered better. This is because the procedure is carried out according to the indications for the patient. Optimal surgical techniques for TON therapy are currently being developed by ophthalmologists, neurosurgeons and head and neck surgeons. Complications of steroid use are still rare. However, there is no precise evidence that steroid use is associated with improved visual acuity in TON patients. In TON patients due to head injury, it is not recommended to use high-dose steroid therapy (Karimi et al. 2021). The method of post-therapy evaluation in TON patients. The method commonly used is the visual analog scale (VAS). This method is the gold standard for evaluating TON in the form of a questionnaire that shows the patient's subjective feelings about the therapy that has been obtained (Yao et al. 2017).

Conclusion

Traumatic optic neuropathy (TON) is a disease caused by secondary optic nerve system damage. The causes can be due to blows by blunt objects, traffic accidents, falls, violent acts that hit the head, and injuries during sports that can cause injury or trauma to the head. Traumatic optic neuropathy (TON) is divided into two categories, namely, direct traumatic optic neuropathy and indirect traumatic optic neuropathy (Karimi et al. 2021). This TON needs to be treated to reduce the poor prognosis. However, until now for the management of TON there are no standard guidelines that are used either in medicine or decompression. Treatment of TON is divided into two, namely, (1) Treatment with high doses of corticosteroids (2) by surgery.

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