Neuro-Ophthalmology Society Journal, Case Report of Neuro Ophthalmology 2025, Volume 05, Number 01.
e-ISSN 2775-474X

COMPLETE VISUAL RESOLUTION WITH CORTICOSTEROID TREATMENT IN TRAUMATIC OPTIC NEUROPATHY: A CASE REPORT

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Background
Traumatic optic neuropathy (TON) is an uncommon vision-threatening disorder that can be caused by ocular or head trauma. Incidence of TON is 0.7–2.5%, and indirect TON has a higher prevalence. Purpose of this case report is to present the successful treatment in TON using corticosteroids.
Case Presentation
A 19 years-old female came with chief complaint of suddenly blurred vision since 2 weeks ago in right eye. Her head was hit hard by the wall. Complaints accompanied by intermittent headaches, no complaints of nausea and vomiting. Visual acuity was hand movement and relative afferent pupillary defect in the right eye, Funduscopy within normal limit. Examination of Ishihara 0/38 plates and confrontation test defects in the superior quadrant. She was treated with Methylprednisolone high dose injection. Visual outcome during treatment to 5/7.5 after five days follow up
Conclusion
TON is an uncommon vision-threatening disorder that should be considered in a patient with ocular or head trauma and decreased VA. Detection of an afferent pupillary defect in the presence of an intact globe and clear media strongly suggests TON, and neuroimaging must be performed in this clinical setting. High dose steroids optimized the visual acuity, without any adverse effects
Keywords
Traumatic Optic Neuropathy, TON, Corticosteroids, *Visual Outcome*

BACKGROUND

Traumatic optic neuropathy (TON) is a vision-threatening disorder that can be caused by either ocular or head trauma and is categorized into direct and indirect TON [1, 2]. Direct TON is frequently associated with severe visual loss and a lower chance of recovery compared to indirect TON. [2] Direct TON often occurs when the optic nerve is lacerated with bone fragments or when contusion or concussion causes anatomical disruption. [2, 3] In contrast, indirect TON often occurs when a blunt head or ocular traumatic stress is transmitted through the oculofacial soft tissues and skeleton to the optic nerve; this damages the integrity of the optic nerve, leading to mild-to-severe vision loss. [2, 4, 5] It usually occurs at the junction of the intraorbital and intracanalicular segments causing compression and disruption of the pial vessels, thereby reducing the vascular supply of the optic nerve [6, 7] .

TON has a gender predominance. Up to 80% of patients with TON have been reported to be male with a median age of 31 years, and 21% are younger than 18 years [11, 15] . Having a fall (26%), motor vehicle accidents (21%), and assaults (21%) are common etiologies of TON in the general population. However, in trauma settings, motor vehicle accidents (63%) and falling down are the main etiologies [11, 15] . TON occurs in 0.4% of patients with any kind of trauma [15] . There is a prominent association between TON and head injury, wherein all patients with TON have head injuries (two-thirds of them have a significant head

injury). However, only 2.3% of the patients with head trauma experience concomitant TON [15] . Epidemiologic features of TON in pediatric patients are similar to those in adults [16] . Having a fall (50%) and motor vehicle accidents (40%) are the most common causes of TON in the pediatric population [17] .

TON cases can be categorized as primary or secondary. Mechanical shearing of the optic nerve axons and contusion necrosis due to immediate ischemia from damage to the optic nerve microcirculation are primary mechanisms, while apoptosis of both injured and initially intact adjacent neurons is the mainstay of secondary TON [2, 4] . Many patients have the involvement of both mechanisms to a certain degree. Purpose of this case report is to present the successfull treatment in TON using corticosteroids.

CASE PRESENTATION

A 19 years-old female patient came with chief complaint of blured vision in right eye. The right eye suddenly blurred since 2 weeks ago,the left side of the head was hit hard by the wall by patient's father. Three days after that, the vision of the right eye suddenly darkened. Complaints accompanied by intermittent headaches, no complaints of nausea and vomiting. The patient does not smoke and has no history of alcohol consumption. From the history of trauma there is a head trauma hitting the wall 2 weeks before entering the hospital. Denied history of diabetes and hypertension, no history of wearing glasses. Before going to the Soetomo Hospital, the patient went to the BDH Hospital and received oral methyl prednisolone therapy but there was no improvement.

Physical examination revealed that he was compos mentis. Vital signs and all general examinations were within normal limit. Motoric examinations in four extremities were normal. Cranial nerve examinations showed no signs of lateralization. Ophthalmology examination revealed his visual acuity was 2 meters counting fingers on the right eye and 5/5 on the left eye. Intraocular pressure on both eyes were 17 mmHg on both eyes. Hirschberg test showed orthotropia. Colour vision examination using Ishihara test there was color vision was decreased 0/38 plate on the right eye and 38/38 plate on left eyes. Eye movement was in normal limit on both eye and pain was negative, WFDT examination far and near was in normal limit. Confrontationt test revealed defect in superior quadrant on the right eye and counting finger all quadrant on the left eye.

Anterior segment examination with slit lamp biomicroscopy, relative afferent pupillary defect (RAPD) was positive on right eye and others anterior segment within normal limit. Posterior segment examination revealed within normal limit on both eye (Fig.1).

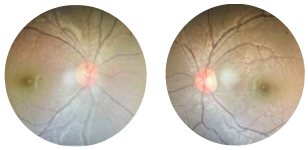

Figure 1 . Posterior segment examination of both eye within normal limit, disk margin was firm and normal color. (Picture taken with patient's family consent. Courtesy of RSUD dr Soetomo)

The patient has had other follow-up examinations, imaging head CT scan from BDH hospital revealed the sulci and gyri appear shallow and narrow suggesting a brain oedema (Fig. 2). Visual field testing was done with humprey visual field analyzer, from the examination of the right eye it is decreased in all quadrant (Fig. 3), while in the left eye it is within normal limits. Based on Optical Coherence Tomography (OCT) there was a decrease in the thickness of the central macula while the optic disc cube and ganglion cells were within normal limits (Fig. 4).

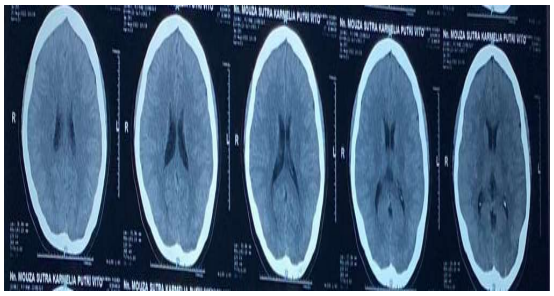

Figure 2. Head CT Scan found shallow sulcus and narrow gryus suggesting brain oedema.

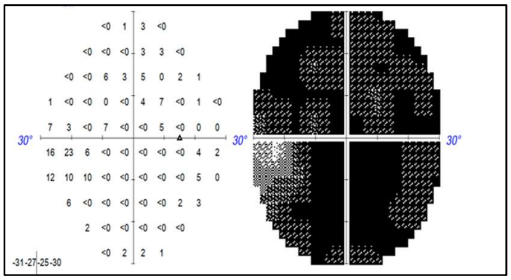

Figure 3. Visual field examination with Humprey Visual Field Analyzer decreased in all quadrant on right eye

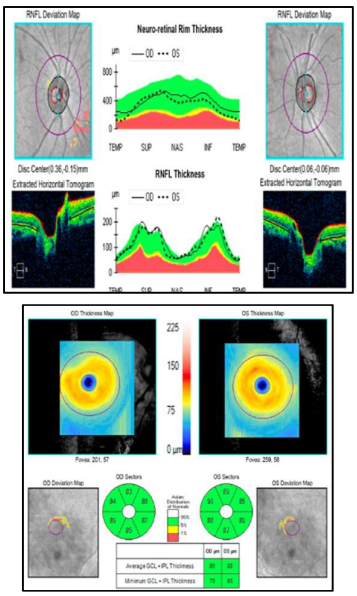

Figure 4. Optical Coherence Tomography within normal limit

The patient's blood laboratory tests were done to evaluate systemic disease for the consideration of treatment option and the result within normal limit.

Based on clinical and ancillary tests result, the patient was diagnosed with Right eye Traumatic Optic Neuropathy. The patient was treated with methylprednisolone high dose injection intravenous 1gram/days divide into 250mg every six hours for three days, omeprazole intravenous twice a day, citicholine tablets 500 mg twice a day orally.

Follow up on the second day of treatment, visual acuity improved to three meter count fingers with a positive RAPD result in the right eye, the therapy given was continued. On the third day of follow-up, the patient felt that his vision was getting better with his visual acuity of four meters counting fingers, the therapy was continued.

Follow-up on the fourth day found visual acuity in the right eye which was getting better to 5/30 pinhole 5/20, for continued therapy with high-dose intravenous corticosteroids. On the fifth day of treatment, the visual acuity of the right eye became 5/12 and the patient felt that his vision was getting clearer.

Follow-up on the sixth day showed visual acuity in the right eye which was getting better to 5/8 pinhole 5/7.5, with the results of the confrontation examination, counting fingers were found in all quadrants of the right eye field. The patient is planned for an imaging examination in the form of an MRI brain to help establish the diagnosis if there any anomalies in the brain. The patient was planned outpatient to receive oral therapy in the form of methylprednisolone 32 mg every 12 hours. Humprey Visual Field Analyzer showed that the visual field was getting better when compared to the first examination (Fig. 9). the results of the color vision examination showed significant changes, the patient was able to read all the plates on the ishihara book.

The patient follow up to the outpatient clinic 2 weeks later, the visual acuity on right eye returned to 5/5 and the visual field also returned to normal followed by improvement in color vision function (Fig. 5)

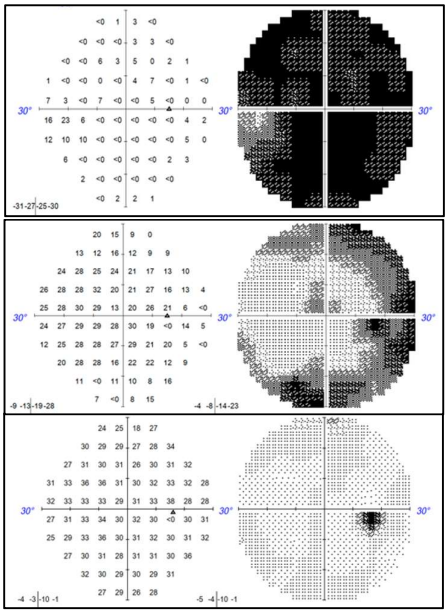

Figure 5. Significant improvement on right eye visual field examination with HFA.

DISCUSSION

Traumatic optic neuropathy (TON) is a vision-threatening disorder that can be caused by either ocular or head trauma and is categorized into direct and indirect TON [1, 2]. Direct TON is frequently associated with severe visual loss and a lower chance of recovery compared to indirect TON [2] . Direct TON often occurs when the optic nerve is lacerated with bone fragments or when contusion or concussion causes anatomical disruption [2, 3] . In contrast, indirect TON often occurs when a blunt head or ocular traumatic stress is transmitted through the oculofacial soft tissues and skeleton to the optic nerve; this damages the integrity of the optic nerve, leading to mild-to-severe vision loss [2-7].

TON is an unusual cause of visual impairment after blunt or penetrating head trauma. The overall incidence of TON is 0.7–2.5% [8–11] . 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 [5, 12] . Intracanalicular part is the most common site of indirect TON (71.4%), followed by the orbital apex (16.7%). Involvement of both the intracanalicular segment and orbital apex was found in 11.9% of the cases. The intracranial portion of the optic nerve adjacent to the falciform ligament is another common site for optic nerve traumatic injury [13, 14] .

TON occurs from either direct or indirect trauma and both primary and secondary mechanisms of damage have been proposed. In direct trauma, stress is applied directly to the ON and is often when orbital fracture fragments lacerate the optic nerve or when mechanical contusion/concussion. The ON commonly sustains indirect trauma, where stress is transmitted through the oculofacial soft tissues and skeleton. The resultant coup– contrecoup forces damage the nerve at transitions between mobile and fixed segments. Commonly, this occurs at the junction of the intraorbital and intracanalicular segments. This results in compression and disruption of pial vessels within the canal, limiting vascular supply of the ON.[22]

TON has a gender predominance. Up to 80% of patients with TON have been reported to be male with a median age of 31 years, and 21% are younger than 18 years [11, 15] . Having a fall (26%), motor vehicle accidents (21%), and assaults (21%) are common etiologies of TON in the general population. However, in trauma settings, motor vehicle accidents (63%) and falling sdown are the main etiologies [11, 15] . TON occurs in 0.4% of patients with any kind of trauma [15] . There is a prominent association between TON and head injury, wherein all patients with TON have head injuries (two-thirds of them have a significant head injury). However, only 2.3% of the patients with head trauma experience concomitant TON [15] . Epidemiologic features of TON in pediatric patients are similar to those in adults [16] . Having a fall (50%) and motor vehicle accidents (40%) are the most

common causes of TON in the pediatric population [17]

The pathophysiology of TON is not yet fully understood, but several mechanisms have been proposed. TON cases can be categorized as primary or secondary. Mechanical shearing of the optic nerve axons and contusion necrosis due to immediate ischemia from damage to the optic nerve microcirculation are primary mechanisms, while apoptosis of both injured and initially intact adjacent neurons is the mainstay of secondary TON [2, 4, 18] . Many patients have the involvement of both mechanisms to a certain degree.

An essential part of the pathop-hysiology of indirect TON is the effect of traumatic loads on the biomechanical response of the cranial contents. One study using holographic interferometry on human skulls suggested that damage to the frontal region deforms the ipsilateral orbital roof, causing damage to the optic nerve and its supporting vasculature, especially where the nerve enters the optic canal [7] . Based on an anatomic study of cadaveric orbits and optic nerves, direct shearing injury to axons, disruption of the blood supply, and pressure from microhematomas and edema due to the damage of anastomoses running between the dura and pia are the possible mechanisms of optic nerve damage [19]

Another mechanism that is thought to be involved in TON is diffuse axonal injury. Detrimental inertial forces to the head cause diffuse axonal damage, which is associated with poor neurological outcomes. Following head injury, axons of the brain white matter become rapidly deformed, resulting in axonal cytoskeleton damage and impaired axoplasmic transmission [20] .

In patients with craniofacial trauma and normal globe and optic nerve head appearance, any evidence of optic nerve dysfunction (reduced vision and an afferent pupillary defect) suggests the diagnosis of indirect TON [2, 4] . Clinical findings that help diagnose TON include (1) ocular injury, (2) a relative afferent pupillary defect (RAPD), (3) variable degrees of vision loss, (4) color vision disorder, and (5) different degrees of visual field defects. RAPD is a valuable finding, and in cases with mild TON, it may be the only clinical finding before overt optic nerve atrophy. The fact that RAPD is negative in bilaterally symmetric cases should be considered. Visual acuity (VA) may range from normal to no light perception, and 40–60% of cases have light perception or worse at the time of first ophthalmic visit. Although the poor VA of the patients may not allow the ophthalmologist to attain valuable results, automated visual field testing should be offered in feasible circumstances [15].

There is a controversy about the role of neuroimaging in TON diagnosis. Some physicians prefer to perform computed tomography (CT) scan and magnetic resonance imaging (MRI) in all patients, while the others preserve imaging modalities for cases with progressive visual impairment or when therapeutic interventions are being considered [18] . As such, patients with head or oculofacial trauma and simultaneous symptoms of optic nerve damage (unilateral or bilateral decreased VA, visual field defect, and an afferent pupillary defect on examination) should undergo urgent radiological investigations [19] .

The next ancillary test for diagnose TON is OCT, several studies using optical coherence tomography (OCT) have shown retinal nerve fiber layer thinning in patients with TON. However, given that this finding may not be detected in the early stages and it is difficult to sit the patient up to do the OCT imaging, the value of OCT in the diagnosis of TON is reduced. OCT may be valuable in the long-term follow-up to show the progression of optic nerve injury over time [19–21] . From the results of the OCT examination in this patient, it was found thinning of the retinal RNFL which supports the direction of traumatic optic neuropathy.

Although the visual acuity of patients with TON may be too poor which reduces the likelihood of achieving an acceptable outcome, automated visual field testing should be considered in all patients [11] . There is no specific visual field defect in patients with TON and any visual field impairment has been reported in patients; however, arcuate, central, and hemianopic field defects may be seen [19] . It is well documented that the visual field is severely compromised at baseline when it is compared with normal subjects, but a comparison of baseline visual field and long-term follow-up visit shows a significant improvement in visual field extension [14] .

Medical Treatment of TON

A visual recovery rate of about 50% is expected following conservative management in indirect TON, where baseline VA plays the main role in the prediction of final visual outcome [22, 15–17] . To estimate the golden time of medical or surgical treatment, a longitudinal study by Kanamori et al. was performed to analyze the decrease in ganglion cell population and nerve fiber layer thickness following TON [18] . They reported that the decrease started two weeks after trauma and stopped changing after 20 weeks. Accordingly, it was suggested that the treatment should be started within 20 weeks following the incidence of TON.

The pharmacological rationale of using corticosteroids for TON arose from their benefits in the management of CNS injuries in animal models [13, 19] . According to these studies, it was hypothesized that steroids exert neuroprotective effects through their antioxidant properties [20] . Animal models of steroid efficacy for the treatment of TON, however, yielded inconclusive observations. Ohlsson et al. failed to show any effectiveness of steroid therapy on retinal axon and ganglion cell survival [21] . In another study, it was found that steroids exacerbated axonal loss following optic nerve damage in a dose-dependent fashion [12] . However, Lew et al. reported an improved optic nerve blood flow following high-dose corticosteroid therapy in 10 rabbits with experimental TON [13] .

Complications of steroid and EPO treatment are rare; however, there is no definite evidence of any benefits of these drugs in terms of VA improvement in patients with TON. Thus, clinicians should be aware of the risk of severe side effects with an aggressive treatment protocol, especially when the effectiveness of treatment is under debate. The Corticosteroid Randomization after

Significant Head Injury (CRASH) study was terminated prematurely due to the increased rate of death in the high-dose corticosteroid group [18] . The Optic Neuritis Treatment Trial reported two cases of acute psychosis and acute pancreatitis in the steroid treatment group, both resolving without sequelae [19] . Transient hypotension has been reported in studies with EPO, which can be hazardous for patients with multiple trauma and unstable medical conditions [16]

CONCLUSION

TON is an uncommon vision-threatening disorder that should be considered in a patient with ocular or head trauma and decreased VA. Detection of an afferent pupillary defect in the presence of an intact globe and clear media stronggly suggests TON, and neuroimaging must be performed in this clinical setting. Although there is no definitive treatment for TON, the use of Corticosteroids can be beneficial in some patients. The use of corticosteroids is still unclear, but based on the literatures it has shown significant results in the use of corticosteroids in cases of traumatic optic neuropathy

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