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Case Report

Direct Traumatic Optic Neuropathy Secondary to Harpoon Injury

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Abstract

Traumatic Optic Neuropathy (TON) is a severe vision threatening condition that develops following ocular and cranial trauma. It is classified into direct and indirect injury regard to mechanism of occurence. Low-velocity, small diameter penetrations may cause significant orbital and even intracranial damages, despite negligible entry wounds through the eyelids or conjunctiva. Tough sclera and abundance of intraorbital fat tissue, partially allows for movement, protect the globe from dangers and lead to delays in diagnosis. Decreased visual acuity and relative afferent pupillary defect (RAPD) were the only signs of optic nerve damage in our 56-year-old female patient, who was evaluated as direct TON as a result of a harpoon injury. Conjunctival nasal laceration and corneal epithelial defect were present. Eye movements were free in all gaze. Indirect ophthalmoscopy and Orbital Computed Tomography (CT) were normal. We had a lucky! response with the oral steroid treatment that we started on the fifth day. We evaluated TON mechanisms of occurence and treatment options in our case. We aimed to draw attention to the careful evaluation of visual acuity and RAPD and the use of imaging methods; in order not to miss orbital and cranial damage in eyelid and conjunctival lacerations in the medial canthus region.

Keywords: *Traumatic Optic Neuropathy (TON); Relative Afferent Pupillary Defect (RAPD); Computed Tomography (CT); Harpoon Injury*

Introduction

Traumatic Optic Neuropathy (TON) is a severe vision threatening condition that develops following ocular and cranial trauma. It is classified into direct and indirect injury regard to mechanism of occurence. Direct optic nerve injury is a rare codition and develops in a way that disrupts its anatomical and functional integrity with lead or sharp instruments. It is assorted: optic nerve avulsion, transection, optic nerve sheath haemorrhage, orbital haemorrhage, and orbital emphysema [1]. Penetrating injuries of small diameter and low velocity may cause significant orbital and even intracranial damages, despite the seemingly negligible entrance through the eyelids or conjunctiva. The abundance of intraorbital fat surrounding the globe partially allows for movement and protects from open glob injury. Intraorbital penetrating injuries that occur without open globe injury can easily be overlooked.

Case Report

A 56-year-old female patient applied to the emergency department with complain of right eye injury from a harpoon. On eye examination, visual acuity in her right eye was counting fingers from one meter. Relative afferent pupillary defect was uncertain. Ocular movements were independent to all direction. On the biomicroscopy, corneal epithelial defect and medial conjonctival tear were examined. There was no impairment of optic disc and retina. She has no history of chronic systemic disease. Computed tomography scan of the orbit was unremarkable (Figure 1). It indicated no scleral or intracranial damage. We considered the visual impairment is due to corneal epithelial defect and conjonctiva was repaired with few sutures.

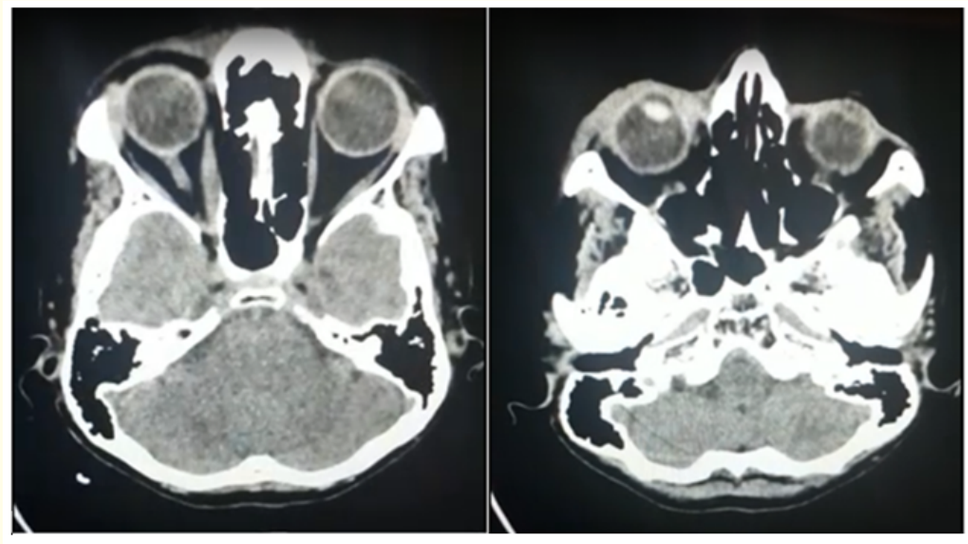

Figure 1

Five days later the patient was applied visual loss. Visual acuity was only light perception without projection. There was relative afferent pupillary defect. Optic coherance tomography (OCT) revealed reduction on retinal nerve fiber layer to the second eye (Figure 2). The patient was diagnosed as traumatic optic neuropathy and we started 1 mg/kg peroral metilprednisolon and acetyl salicylic acid. Rapid visual increment was obtained, visual acuity was 1.3 logMAR followed 1 week and 0.2 logMAR followed 2 week. Peroral steroid therapy was tapered and stopped,at the end of this two weeks. On the fundus examination, temporal of optic disc was pallor. For 2 years follow-up the findings weren't changed.

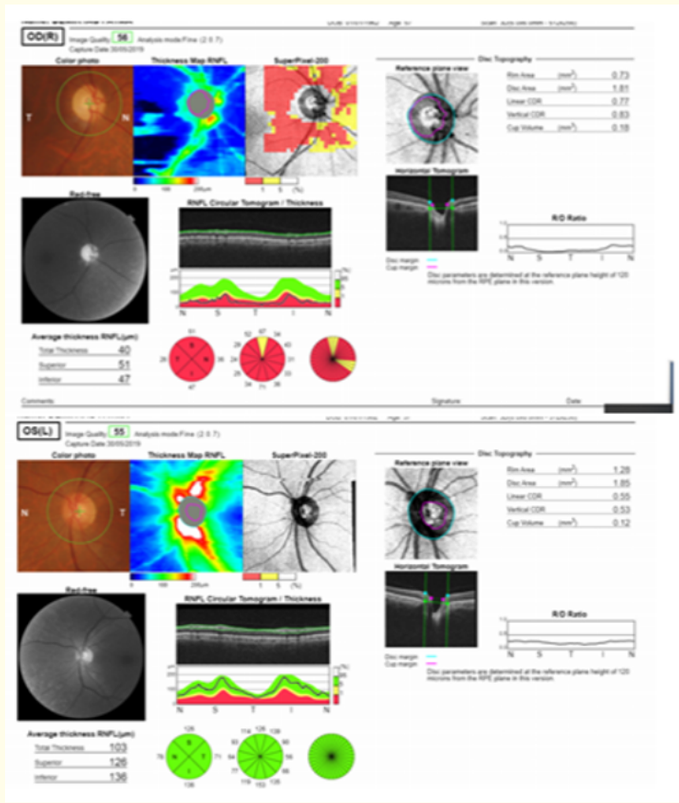

Figure 2

Discussion

Direct TON is a rare codition and develops in a way that disrupts its anatomical and functional integrity with lead or sharp instruments. It is usually associated with severe, permanent visual loss with a minimal chance for recovery. Differentially tissue integrity does not deteriorate in indirect TON, it occurs when orbital or cranial forces are transmitted to the optic nerve caused by blunt traumas. The pathogenesis of indirect TON is still unclear with several possible mechanisms responsible for visual deficit. Mechanical and ischemic damage occur in both direct and indirect TON. It is accepted that this damage occurs by primary and secondary mechanisms. Contusion of optic nerve axons and necrosis due to ischemia are responsible for the primary mechanism and the swelling of the optic nerve in the optic canal, which does not have the ability to expand, and aggravation of ischemia, vasoconstriction, and irreversible damage to the initially mild injured axons constitutes the secondary mechanism [2]. Sometimes direct and indirect mechanisms may play a role in the injury. We evaluated our case direct TON due to the causative object of trauma but the normal appearance of the optic nerve at the first examination and visual improvement with corticosteroids is seen mostly in indirect TON. Also, absence of penetrating injury on CT scans supports indirect origin of TON.

Another transorbital injury from a harpoon presented by P C Windle-Taylor. This case had penetrated the sphenoid sinuses was removed without complication. He had a good recovery except the inferotemporal field defect, ecreased visual acuity with optic atrophy, a sensory pupillary defect and right ptosis. Some aspects of this case fairly similar to our case [3].

Negligible eyelid or conjunctival injuries may obscure deep penetrations. Therefore it can be sutured without the need for detailed examinations. Traumas with small-diameter and low-velocity objects may damage to intraorbital and other intracranial structures. The cone shape of the orbit and mobility of the globe direct penetrating objects toward the apex. Optic canal, superior and inferior orbital fissure are naturally passageways to the cranial cavity. Periorbital traumas that appear mild, without damage to the bone can cause intracranial injury. The scleral coat and motility of the globe protect it from penetration [4]. Likely in our case small medial conjonctival laceration was caused to dissappear transorbital deep injury. Cleary., *et al.* presented two case with penetrating orbital trauma by stiletto, both entering via small eyelid wounds [5].

Turbin and ass investigated 38 cases with occult and non-occult transorbital intracranial injuries. All optic canal injuries were non-occult, and had entrance through the medial or canthal regions. Optic nerve, ophthalmic artery and symphatetic nerves passing through the optic canal are vital structeres for visual function. Therefore the cases with visual loss who has medial conjunctival and canthal wounds should carefully evaluate in terms of optic canal damage [4].

Generally, the fundus examination is normal initially except for presence of relative afferent pupillary defect who has indirect TON. Various electrophysiological methods such as the visual-evoked potential (VEP), flash and pattern electroretinography (ERG) can be used for predict visual outcome after ocular injuries [6]. If the evaluation of imaging revealed that cranial radiography is insufficient, in these cases, performing an orbital computed tomography scan is necessary to detect acute orbital hemorrhages and bony fractures. Magnetic resonance imaging can be used after exclusion of metallic foreign body for detect foreign bodies despite a previously negative CT. Also it can show if there is damage to optic nerve, surrounding blood vessels and soft tissues [7].

Although spontan recovery may ocur in some cases with traumatic optic neuropathy; treatment options contain mainly various dose of steroid regimens, surgical optic canal decompression and combinations. Corticosteroids can reduce the nerve cell death caused by edema and vasospasm induced ischemia [2]. Also they have antioxidant properties by reducing the formation of free radicals that causes lipid peroxidation on axon cell membranes. The Second National Acute Spinal Cord Injury Studies compared the effects of placebo or methylprednisolone administered within 12 hours of injury and found out that treatment with methylprednisolone results in a significant visual improvement in the group treated within the first 8 hours after trauma [8]. A randomized, double-blind, placebo controlled clinical trial compared the effects of placebo or intravenous high dose corticosteroid therapy in the treatment of indirect TON. In this study, no difference was found in visual recovery between the effects of high dose corticosteroid therapy and placebo [9]. The international optic nerve trauma study group also found out neither corticosteroid therapy nor optic canal decompression surgery is benefit [10]. The adverse effects of especially high dose steroids should also keep in mind. The Corticosteroid Randomisation After Significant Head Injury (CRASH)

study evaluated the effectiveness and safety of steroids in patients with acute traumatic brain injury. This study found out the risk of death was higher in the steroid group than in the placebo group at 6 months of follow-up [11].

In studies on neuroprotection of the retinal ganglion cell found out that the optic nerve crush is prior by an unrelated injury to another part of the central nervous system, for example, the spinal cord, the following T cell-mediated protective immunity results in a significant increase in retinal ganglion cell survival. This beneficial effect was not affected by injection of low dose methylprednisolone (1 mg/kg), but was inhibited after high dose (30 mg/kg) [12]. There is no consensus the dosage of corticosteroids for the treatments of TON. Because of the permanenet and severe visual loss risk the high dosage steroid regimens may be more prefered.

In indirect TON, the optic nerve appears normal in early period, but begins to fade after 3-5 weeks. According to a study that analysed of optic nerve retinal nerve fiber layer (RNFL) thickness, circumpapillary RNFL and ganglion cell complexes began to decrease starting two weeks after trauma and macular thickness decreased after four weeks. Also this study showed that circumpapillary RNFL and GCC no longer change after 20 weeks. Therefore the treatment of TON should be performed within 20 weeks after trauma [13]. Our case was treated low-dose corticosteroid, although the treatment was begun late (5 days later from accident) visual acuity was improved. Rapid diagnosis and early treatment are very important in the treatment of TON. The superiority of late-onset treatment over placebo is controversial.

Erythropoietin (EPO) regulates the red blood cell production and has neuroprotective effect in central nervous system. Traumatic optic neuropathy treatment trial (TONTT) compared the effect of eritopoetin, steroid, and observation. Three groups showed a significant improvement ofvisual acuity but there was not significantly different between the groups [14]. It has been shown in experimental studies that Minocycline, an antibiotic, prolongs the survey of retinal ganglion cells by delaying apoptosis. Other treatment options for TON: glutamate inhibitors, nerve growth factors, crystallins, nitric oxide, tumour necrosis factor-α (TNF-α) and hyperbaric O_2 [15]. We used acetyl salicylic acid also, for contribution to the prevention of possible microvascular ischemia.

Some indications have been established for optic nerve decompression surgery. These are traumatic face and head injury with and without optic canal injury; progressive visual impairment unexplained by intraocular lesions; no evidence of optic nerve avulsion or damage; decrease in amplitude or prolongation of latency in visual evoked potential; unresponsive to steroid therapy and a hematoma near the optic nerve or bony fracture compressing the optic nerve [16]. Transorbital, transcranial and endonasal approaches can be chosen for the surgery in apporiate cases.

Conclusion

In conclusion, orbital injuries from sharpen, small-diameter objects like a harpoon must evaluate carefully although small entrance wounds. The imaging techniques are useful distinguishing the occult traumas, so delayed treatments can be prevented. More studies are needed to determine treatment protocols.

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