Case Report

Visual Loss Due to Optic Nerve Injury with Blunt Trauma: A Case Series

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ABSTRACT

Vision impairment resulting from optic nerve damage poses a significant clinical challenge, often stemming from both direct and indirect traumatic injuries. In this article, we address the complexities involved in diagnosing and managing optic nerve damage, particularly in cases of blunt trauma-induced compression. We highlight the potential delay in diagnosis due to concurrent life-threatening conditions and the difficulties in assessing severely affected patients. Additionally, we present a case series involving three individuals who experienced vision loss secondary to optic nerve compression following blunt trauma. Through this analysis, we underscore the importance of timely recognition and appropriate management strategies in optimizing outcomes for patients with optic nerve injuries.

Key Words: Optic neuropathy, Steroid, Trauma, visual outcome

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INTRODUCTION

The eye and surrounding soft tissues, encompassing a segment of the optic nerve, are safeguarded within the bony orbit. Increased internal pressure within the orbit can lead to compression of the optic nerve and diminished blood supply to the orbital region, both of which can precipitate injury. Failure to mitigate elevated pressure and restore adequate blood flow may culminate in irreversible vision loss, particularly in acute cases where irreversible damage can occur within 90 to 120 minutes.[1]

The patient with optic nerve compression may have a history of trauma, surgery, proptosis, visual complaint (e.g., blurred or double vision and peripheral vision loss, slow, intermittent or sudden visual loss, disturbed colour vision), ocular pain, high intraocular pressure, vomiting and nausea. The patient can have a stiff eye that resists the eye's retropulsion. However, it is significant to stress that if there is even the slightest chance of globe rupture, this should not be performed.[2]

The concept of primary and secondary trauma has been proposed, suggesting that the pathophysiology of indirect traumatic optic neuropathy (TON) is likely multifaceted. Immediately following trauma, a subset of retinal ganglion cell axons undergoes irreversible destruction, resulting in neuronal death.[3,4] Subsequent to this initial insult, the optic nerve may experience swelling due to direct mechanical injury and vascular ischemia within the confined space of the optic canal. This cascade of events sets the stage for a downward spiral towards apoptotic cell death, exacerbated by the development of compartment syndrome, further compromising the already compromised blood supply to surviving retinal ganglion cells.[5,6]This case series highlights the importance of timely diagnosis and management of three cases of traumatic optic neuropathy.

CASE 1

A 32-year old man who met an RTA presented one day after loss of vision in his left eye. On examination, visual acuity was 6/6 and hand movements in the right and left eye respectively. He denied history of

unconsciousness but felt disoriented. Slit lamp examination of left eye showed minor subconjunctival haemorrhage as well as upper and lower lid hematoma. The anterior chamber and lens were clear, and fluorescein staining was negative. Mild corneal edema with high IOP of 41mmHg was recorded in the left eye. Left RAPD was positive. Due to the high IOP and corneal edema, fundus view was not clear. In order to lower the IOP, antiglaucoma medications (Alphagan, Xalatan, and Cospt) were started. After two days, the pressure in left eye was reduced to 25mmHg. Fundoscopy revealed macular hemorrhage. Orbital X-ray did not show bone fracture. Mega dose of steroids was started (15 mg/kg Methylprednisolone IV). After two weeks with use of antiglaucoma and systemic steroids his BCVA in improved to 6/36 from hand movement.

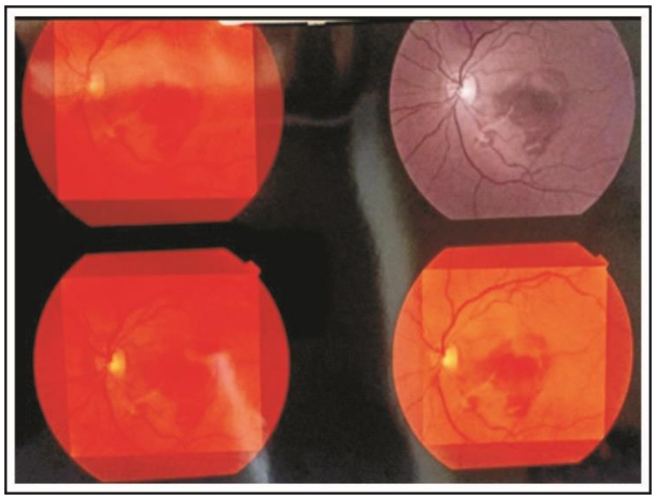

Figure1: Fundus photograph of left eye showing macular hemorrhage.

CASE 2

A 22-year old female was hit in the right eye by a ball. She presented 12 hours later with complaint of loss of vision in her right eye. On examination she had light perception in the right eye and 6/6 in the left eye. Extra ocular movements and IOP were normal in both eyes. Upper and lower lid hematoma was present in the right side. There was sub-conjunctival haemorrhage with soft tissue swelling as well. Fundoscopy revealed no abnormality of retina and optic nerve head. She was given Maxidex eye drops (8 hourly) and intravenous Solucortef. After one week her vision improved to 6/9.

CASE 3

An 18-year old male presented one week after a road traffic accident. He complained of loss of vision in his right eye. On examination there was no perception of light in the right eye and 6/6 in the left eye. The patient had severe subconjunctival haemorrhage as well as upper and lower lid hematoma. The anterior chamber and lens were not clear due to corneal edema. Right fundus showed macular hemorrhage. X-ray orbit showed fracture of great wing of sphenoid on the affected side. There was also a break along anterior and right posterolateral wall of maxillary sinus. A mega dose of steroids was given to the patient right away.

DISCUSSION

Interventions aimed at halting the detrimental cycle of vision loss and preserving the remaining retinal ganglion cells that have survived the initial injury typically involve optic nerve decompression through medical or surgical means.[7] Steroid regimens can be categorized as: low dosage (100 mg), moderate dose (100-499 mg), high dose (500-1999 mg), extremely high dose (2000-5399 mg), and mega dose (> 5400 mg) depending on the first daily dose of methyl-prednisolone. A course of intravenous methylprednisolone in the extremely high-to megadose range is the most popular steroid therapy in TON.[8]

It is found that baseline visual acuity is the most significant predictor of end outcome, with a visual recovery rate of 40–60% recorded for indirect TON cases managed conservatively. Initial and final visual acuities are significantly correlated, and patients who reported no light sensitivity upon presentation almost always had little to no visual improvement.[9] Other indicators of poor prognosis include loss of consciousness, failure to recover vision beyond 48 hours, and a lack of visual evoked responses with signs of orbital bone fractures on X-ray/CT scan. Occurrence of an optic canal fracture is also an indicator of poor visual prognosis. Direct TON is a unique category that causes severe, irreversible vision loss with little chance of recovery and no intervention has been shown to be beneficial.

There are significant methodological problems with every published case series in TON. Majorities are modest retrospective studies with insufficient sample sizes for thorough statistical analysis, and the lack of sufficient randomization adds the additional

risk of selection bias. The large variety of steroid regimens employed and the varying period permitted prior to treatment beginning make it extremely difficult to compare the results, even qualitatively.[10]

Based on current evidence, the therapeutic efficacy of corticosteroids in managing traumatic optic neuropathy (TON) remains unverified. If corticosteroids are contemplated for TON treatment, caution is advised, particularly in cases of concurrent traumatic brain injury or when patients present 8 hours or more after the initial injury. The optimal dosage of steroids for TON treatment remains unclear in the literature.[10]While high-dose steroids have reported beneficial outcomes in select cases, significant complications associated with their use in trauma scenarios must be considered. Decision to utilize corticosteroids in TON management should be individualized based on clinical judgment and patient-specific factors.

In our case series, case 1 and 2 had good visual recovery with moderate use of steroid. Orbital scan showed no bone fracture. However, in case 3 prognosis was poor because of orbital fracture.

Our first two cases presented within 24 hours of RTA and we immediately started with heavy dose of intravenous steroids. CT scan of one patient later on did not show any sign of fracture while the other patient did not go through any diagnostic CT scan. The third patient presented after one week of RTA with NPL and his CT scan showed fracture of right great wing of sphenoid and cortical break along anterior and right posterolateral wall of maxillary sinus. The patient had poor prognosis.

CONCLUSION

We have highlighted the importance of early diagnosis and treatment of optic neuropathy using intravenous steroids. It shows that optic nerve compression due to blunt trauma can be reversible if patient presents well in time and the dose of intravenous steroids is given in moderate to heavy doses.

Conflict of Interest: Authors declared no conflict of interest.

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Authors Designation and Contribution

M. Afzal Bodla; Professor: *Concepts, Design, Manuscript editing, Manuscript review.*

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Nalain Syedah; Optometrist and Ophthalmic diagnostic specialist: *Manuscript preparation, Manuscript editing.*