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An unusual turn of Visual events following head injury : A Case Report

Dr.Sravanthi Chintapalli[1], Dr.P. Saraswati[2], Dr.Kola Vijaya Sekhar M.S[3], Dr.G.Satyavathi M.S[4]

[1]Postgraduate, [2]Postgraduate, [3]Professor, [4]Associate Professor, Department of Ophthalmology, Guntur medical College, Guntur, Andhra Pradesh, India

Abstract: *Traumatic optic neuropathy is known to occur following blunt trauma to the eye and the orbit. This may occur due to compression of the nerve by bony fragments or soft tissue swelling, concussion of the nerve, haemorrhage in or around the optic nerve sheaths and interference with the neural blood supply. The latter is one of the least documented causes of traumatic optic neuropathy. In most of the cases, associated ocular injury, severe visual loss, and opaque media interfere with establishment of the exact etiopathology. The present article describes a case report of a 35 year old male who had an unusual course of traumatic optic neuropathy.*

Key words: *Traumatic Optic Neuropathy, Road traffic accident, Multiple Cranial Nerve Palsies, Optic atrophy*

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I. Introduction

Traumatic optic neuropathy (TON) is a serious vision threatening condition that can be caused by ocular or head trauma. Indirect damage to the optic nerve is the most common form of TON occurring in 0.5% to 5% of all closed head trauma cases. Although the degree of visual loss after indirect TON may vary, approximately 50% of all patients are left with 'light perception' or 'no light perception' vision, making TON a significant cause of permanent vision loss(1).

Of the various forms of vision loss due to trauma, optic nerve injuries are some of the most difficult to diagnose and treat. Hippocrates may have been the first to recognize this condition when he identified the phenomenon of acute and delayed vision loss after injuries placed to and slightly above the brow. It is now recognized that the brow and other facial eminencies are most frequently the site of impact in patients with posterior indirect traumatic optic neuropathy (TON)(2).

II. Case Report

A 35year old male had been in a road traffic accident on 25th october 2020, he sustained injuries over bridge of nose and right jaw, as per the patient. He had ignored immediate medical consultation to the injuries. On 27th october 2020, he had 2 episodes of projectile vomiting, loss of consciousness for 15 minutes and bleeding from ear and nose. He got admitted in the Neurosurgery Department on the same day. During his stay in the hospital, he developed Right eye ptosis and restricted extra ocular movements not associated with pain with apparently normal Left eye. He was diagnosed with Isolated Right IIIrd cranial nerve palsy with Bilateral papilloedema. On MRI, the findings were benign intracranial hypertension and small chronic infarctvin left side of pons. He was started on Tab. Wysolone 10mg for 15 days. He was discharged on 3rd November 2020 with medication. There was slow gradual improvement in his condition over next 1 week. On 10th November he developed urinary retention for the following 3 days, due to which he discontinued his medication. He was admitted in General Surgery Department on 13th November. He had then complained of diminision of vision in right eye and headache, and referred for ophthalmology opinion.

	RIGHT	LEFT
VISUAL ACUITY	PL +	6/24 with pinhole 6/12
HEAD POSTURE	Midline	
FACIAL SYMMETRY	Mouth deviation towards left side	
OCULAR ADNEXA	Severe ptosis	Normal
GLOBE POSITION	Outward and downward	normal
EXTRA-OCULAR MOVEMENTS	All movements restricted except abduction	All movements are full, free and painless
CONJUNCTIVA	Quiet	Quiet
CORNEA	Clear , reduced corneal sensation +	Clear
SCLERA	Normal	Normal
ANTERIOR CHAMBER	AC = 1 PACD	AC = 1PACD

PUPIL	Fixed, Mid dilated, not reacting	Wernicke's hemianopic pupil
LENS	Clear	Clear
FUNDUS	Media - clear Optic disk- normal size, shape, margins - slight elevation C:D - 0.3:1 Vessels - mild venous engorgement + Macula - FR + Background - normal	Media - clear Optic disk- normal size, shape, margins - slight elevation C:D - 0.3:1 Vessels - mild venous engorgement + Macula - FR + Background - normal
VISUAL FIELD	Bi-nasal hemianopia	
OTHER SIGNS	Loss of sensation on right half of face	

Fig 1

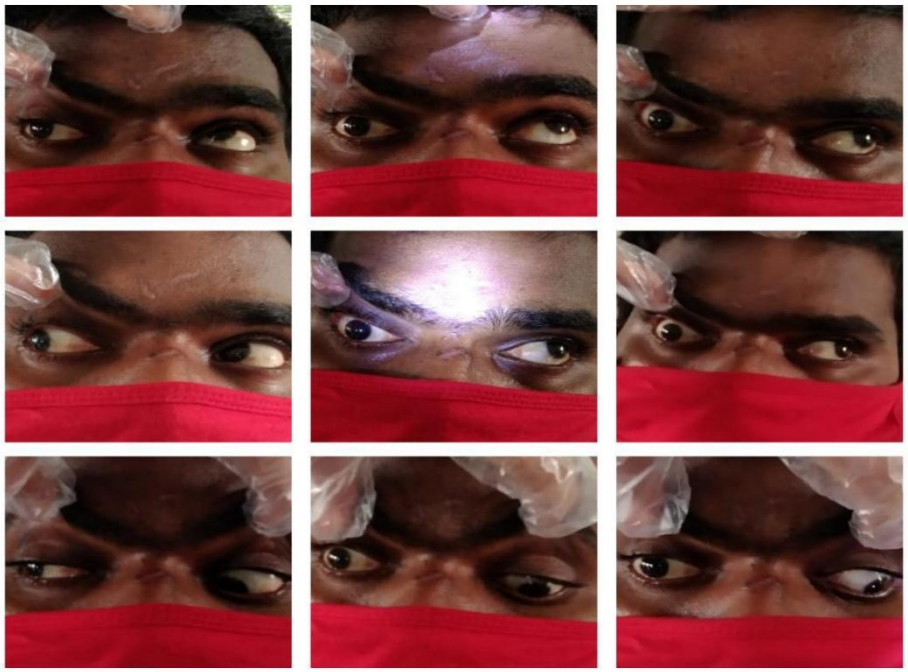

Fig 2

The patient was then referred for neurologist opinion, where he was advised MRI brain. On MRI, a chronic infarct in pons was found whose cause was attributed to hastened atherosclerosis due to chronic smoking and alcoholism. He was then started on Tab. WYSOLONE 20mg for 1 week followed by 10mg for 1 week with timely follow up.

On review after 3 weeks, there was drastic improvement in RE vision to 6/9 and extraocular movements but there is a gradual diminision in the LE vision to 6/24, with field restriction - nasal > superior and inferior field > temporal and LE RAPD Grade I. On fundoscopy, LE progressing optic atrophy was observed. The patient was reviewed periodically by a neurologist and he was started on ecospirin 150mg, atorvas 20mg and pregabalin 75mg for 15 days

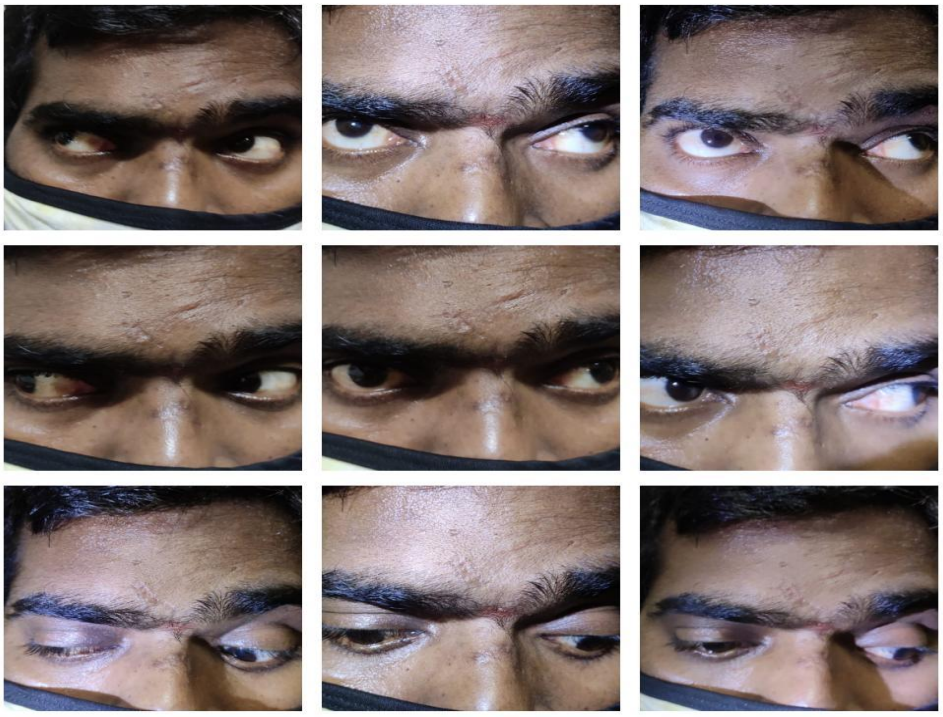

Fig 3

On review after 3 months (Fig 3), both eyes vision had improved to 6/6' which on spectacle correction with -0.50 spherical lens improved to 6/6. But the Left eye showed mild exotropia, previously mentioned pattern of visual field restriction, RAPD Grade I with optic disc temporal pallor on fundoscopy. On this review, the neurologist conducted a VEP which came out normal and had adviced the patient to continue pregabalin 75mg.

III. Discussion

Head trauma may manifest as an ophthalmic condition.Ophthalmic signs and symptoms may be the initial presentation of head trauma, or whereby the patient first presents to an ophthalmologist or evaluation of possible ophthalmological signs and symptoms may be first noted by the neuro-surgeon/ neurologist and then subsequently sent to an ophthalmologist. Ophthalmologists encounter injuries not only to the globe and ocular adnexal structures but also to the anterior and posterior visual pathways, the pupillomotor pathway, the cranial nerves, the supranuclear and the intranuclear pathways.[3-9]

Traumatic optic neuropathy can take many forms. *Direct* injuries are caused by projectiles or penetrating objects that enter the orbit. Self-inflicted optic nerve trauma can also occur. *Indirect* injuries are caused by forces that are transmitted to the optic nerve from the globe and orbit. Examples of this type include posterior indirect traumatic optic neuropathy and optic nerve avulsion. The latter may occur with only minor damage to the front of the eye if it is rotated forcefully, causing separation of the optic nerve as it exits the globe. Ophthalmoscopically, avulsion is often accompanied by intraocular hemorrhages, which may preclude viewing the nerve head. The visual prognosis following optic nerve avulsion is poor. Also recognized are a traumatic form of ischemic optic neuropathy and optic nerve dysfunction due to optic nerve sheath or orbital hemorrhages.[2]

Posterior indirect traumatic optic neuropathy is traumatic loss of vision that occurs without external or initial ophthalmoscopic evidence of injury to the eye or the nerve. This condition occurs in patients of all ages, and, as with any traumatic condition, it is most common in young men. It is estimated that 2–5% of patients with head injuries have TON, and therefore, based on the incidence of head injuries, there are four or five cases of TON/100 000 population per year. Frontal blows are most common. Patients need not have loss of consciousness, and the entity certainly occurs in situations of relatively minor head trauma.[2]

Optic atrophy may be from injury of the optic nerve head; however because of anterograde and retrograde degeneration, it may reflect upstream injury of the retinal ganglion cells or downstream injury of the posterior optic nerve, optic chiasm, or optic tract.[2]

Traumatic Visual Loss

Optic Nerve Injury - Optic nerve trauma is often associated with severe head trauma. Most cases of traumatic optic neuropathy are associated with motor vehicle accidents which produce high energy deceleration-type head trauma[4,5,6,7] Patients with unilateral traumatic optic neuropathy demonstrate a relative afferent pupillary defect and decrease in vision. The diagnosis of traumatic optic neuropathy should not be made in the absence of these two findings. The nerve can be injured at any point between the optic chiasma and the globe. The fundus picture may be variable with an absolutely normal looking optic nerve to one that looks visibly affected

Injury of the intraorbital optic nerve[11]: Traumatic optic neuropathy caused by injury of the intraorbital portion of the optic nerve may result from direct or indirect mechanisms. It may occur as a result of :

• Contusion of the nerve in penetrating orbital trauma
• Traction on the nerve caused by severe axial globe displacement
• Compression of the nerve from space occupying intraorbital hemorrhage or sequestrated air
• Avulsion from the globe with blunt orbital injuries
• Direct trauma to the optic nerve causing hemorrhage within the sheath

Optic disc swelling and hemorrhage, in addition to retinal whitening suggestive of central retinal artery occlusion may be observed. Immediate surgical intervention with optic nerve sheath fenestration has been reported to relieve compression of the anterior optic nerve and to improve optic nerve function.[5,6,7]

Injury of the intracanalicular and intracranial optic nerve: Injury to the intracanalicular portion of the optic nerve is the most common cause of traumatic optic neuropathy. This segment of the optic nerve is protected within the bone of the optic canal, within the canal, and the dura of the optic nerve is tightly adherent to the bone of the canal. The intraorbital and intracranial segments of the optic nerve are less tightly held in position, hence with forceful trauma the orbital and intracranial contents shift, thus applying tractional forces on the tightly bound intracanalicular portion of the optic nerve. This may lead to both shearing of axons and interruptions of the vascular supply of the optic nerve. If a comminuted fracture is present, the displaced fragments may impinge on the optic nerve. In addition to the optic nerve, the ophthalmic artery enters the orbit through the optic canal within dural investments of the floor of the canal. Disruption of the pial vessels, which supply the intracanalicular portion of the optic nerve, induces optic nerve ischemia and axonal necrosis. Secondary edema of the optic nerve may ensue and create a compartment syndrome within the canal.[11]

Optic Chiasm Injury - Head injury severe enough to cause chiasmal trauma is often life-threatening. Multiple cranial neuropathies, traumatic optic neuropathy and hypothalamic dysfunction may occur in association with chiasmal trauma. Traumatic injury of the optic chiasm will result in complete bitemporal hemianopia. The mechanism of injury, may be contusion necrosis, disruption of blood supply or penetrating trauma. In addition to a bitemporal hemianopia, optic atrophy may develop approximately six to eight weeks after the traumatic event.[11]

Binasal hemianopia due to lateral chiasmal compression. This is rare. Lateral compression of the chiasm can be caused by aneurysm of one carotid compressing the chiasm against opposite carotid artery. Bilateral compression is also possible by atherosclerotic, inelastic carotid arteries. However, binasal defects are usually due to a disease process at the level of the optic disk such as glaucoma.[10]

Postchiasmal Visual Pathway Injury - Structures of the postchiasmal visual pathways include the optic tracts, lateral geniculate nuclei, the geniculocalcarine radiations, and the occipital cortex. Postchiasmal lesions, unless bilateral should not produce decrease in visual acuity unless they are associated injury to structures of the anterior visual system. All post chiasmal lesions result in contralateral homonymous visual defect. Lesions more posterior in the postchiasmal pathways, nearer the occipital cortex, are usually more congruous.[11]

Optic Tract Lesions - These are characterized by:

a. Incongruous homonymous hemianopia.
b. Bilateral retinal nerve fiber layer or 'bow-tie' optic atrophy and contralateral relative afferent pupillary defect (if associated with temporal field loss).[11]

Lateral Geniculate Nucleus Lesions - Isolated lesions of the lateral geniculate nucleus are rare. Two types of visual-field loss are possible:

a. An incongruous homonymous hemianopia.
b. A congruous homonymous horizontal sectoranopia.

Fibers of the inferior hemiretina course anteriorly from the lateral geniculate nucleus into the temporal lobe as Meyer's loop fibers. Fibers from the superior hemiretina course directly posterior into the parietal lobe on their way to the occipital cortex. This superior-inferior separation of the fibers is the basis of topographic diagnosis of posterior visual lesions. Injury to the temporal lobe thus produce homonymous visual field defects that are denser above, and parietal lobe lesions produce homonymous defects that are denser below.[11]

Occipital Lobe Lesions - These produce congruous homonymous hemianopias. As a rule, lesions superior to the calcarine fissure yield inferior quadrantanopic defects and those below the calcarine fissure produce superior defects. Lesions of the optic tract, temporal lobe, and occipital lobe may produce homonymous hemianopia. Thus, it is not a localizing finding.[11]

PATHOPHYSIOLOGY OF TRAUMATIC OPTIC NEUROPATHY

It is believed that the damage in traumatic optic neuropathy is caused by a primary and secondary mechanism of injury. While the optic nerve can be injured anywhere along its course, the most common site of injury is the intracanalicular part followed by the intracranial portion. Blunt trauma to the frontal bone results in forces being transmitted to the fixed intracanalicular segment of the optic nerve which can result in a fracture of the optic canal. As mentioned earlier, the tight adherence of the optic nerve's dural sheath to the periosteum within the optic canal causes the optic nerve to be vulnerable to the impact of skull injuries. Hemorrhage, either within the optic nerve sheath or in the orbital cavity can cause loss of optic nerve function.[11]

Mechanical forces are considered to be the primary mechanism of injury. These forces cause lacerations, partial or complete avulsion of the retrobulbar nerve, contusion necrosis, and disruption of the nerve's vascular supply, resulting in hemorrhages, and thereby cause permanent damage.[11]

Once the vascularity of the optic nerve is disturbed, the secondary mechanisms of injury come into play. Edema sets in soon after and this in turn further compromises the vascular supply by causing a rise in intraluminal pressure. Secondary mechanisms that have been studied include numerous pathways for the generation of free radicals and arachidonic acids, lipid peroxidation, production of inflammatory mediators such as bradykinin, loss of calcium homeostasis with disruption of cellular function, glutamate-induced excitotoxicity, cell-mediated inflammation, and initiation of neuronal apoptosis.[3] Most treatment modalities revolve around limiting the secondary injury with the hope of rescuing those axons which have survived the initial trauma. Direct traumatic optic neuropathy is less common because the laxity of the intraorbital optic nerve allows for both absorption and deflection of the penetrating object. The resilience of the dura to penetration also offers further protection.[11]

Diagnostic studies/neuroimaging. Optic canal fractures are identified in only approximately one-third of patients, and therefore identification of a fracture is *not* necessary for the diagnosis. Coronal CT images are best for identifying these fractures. The optic canal is located in the superior posterior lateral aspect of the sphenoid sinus.[2]

Differential diagnosis. Admittedly, ophthalmoscopic evaluation is difficult in patients with altered consciousness, as their pupils cannot be dilated while pupillary status is monitored in acute hospital settings. Important entities that need to be considered in the differential diagnosis include preexisting optic neuropathy, traumatic retinal injury, and factitious visual loss since many patients are seeking secondary gain in the setting of the injury. The absence of an afferent pupillary defect would support nonphysiologic visual loss.[2]

FUNDUS CHANGES in secondary optic atrophy the margins of the disc are hazy, and gliosis is seen overlying the disc. In young patients, it is often possible to see evidence of mild optic atrophy, manifested by the loss of Gunn's dots. These are small bright reflections in a hexagonal pattern that are best seen one to two disc diameters from the disc. The indentations in the internal limiting membrane produced by Müller's cell endfeet serve as a concave mirror to reflect the light of the ophthalmoscope. These reflections are lost with nerve fiber layer loss. Unfortunately, Gunn's dots are obliterated by various other retinal conditions as well as by age.[2]

Treatment. Review of several series reveals that onequarter to one-half of patients improve spontaneously.There are two available options for treatment. Corticosteroids can be used to treat optic nerve swelling and inflammation. Alternatively, since the nerve is swollen within the confines of a tight bony canal, removal of those bones to decompress the canal and prevent compression may also improve the condition. In a comparative, nonrandomized study comparing observation with treatment with corticosteroids or optic canal decompression, no clear benefit was found for either of these modalities. Vision improved by three Snellen lines in 57% of the untreated group, 32% of the surgery group, and 52% of the steroid group. Based on careful review of the literature (Cochrane database), no treatment has been shown to be convincingly effective. Therefore, there is no standard of care for the treatment of TON, and observation alone is reasonable.[2]

The use of megadose steroids is extrapolated from traumatic spinal cord injury studies. Bracken et al and Braughler and Hall in the National Acute Spinal Cord Injury Trial showed that megadose steroids were effective in reducing permanent deficits in patients with spinal cord injury. Glucocorticoid activity is less important in the purported mechanism than the ability of these agents in high doses to scavenge free radicals and prevent lipid peroxidation, perhaps a final pathway in white matter injury. Importantly, however, there is some recent evidence that steroids may be detrimental in patients with head injuries. Experimental work in rats suggests that methylprednisolone may exacerbate axonal loss following crush injury in the optic nerve. Results from the Corticosteroid Randomization after Significant Head Injury (CRASH) Study suggest that high-dose steroids are associated with increased mortality when given in the context of head injury.The mortality rate following the injury was 21.1% in the steroid group and 17.9% in the placebo group, refuting previous smaller studies that reported improved survival following steroid treatment for head injury. Experimental work is currently being conducted to examine the ability of *N*-methyl-d-aspartate receptor antagonists, lazaroids, or 21-aminosteroids with and without vitamin E analogs, calcium channel blockers, and GM1 gangliosides to prevent lipid peroxidation and free radical damage in nerve tissue. These compounds may some day prove to be effective medical treatments of TON.[2]

Both transcranial and extracranial surgical decompression of the optic canal have also shown promise. Uncontrolled and retrospective studies have demonstrated an approximately 70% improvement rate in patients who have extracranial surgery performed via a transethmoidal transsphenoidal route. Endoscopic approaches are also available. The complication rate is very low with no major morbidity or death. However, since the carotid canal sits next to the optic canal, this procedure should not be performed by inexperienced surgeons. Optic canal decompression is indicated when there is radiologic evidence of a bony fragment or hematoma impinging on the optic nerve. It can also be considered in patients in whom surgery is being performed to repair other facial fractures, or in patients with vision loss that deteriorates while on steroids. It should only be considered in patients who are conscious and can understand the potential risks and benefits of this procedure that has unproven efficacy.[2]

In general, we often choose observation and discourage any treatment beyond moderate doses of steroids in most cases. This is particularly true in any patient in whom diagnostic ambiguity exists (simultaneous globe injury or limited examination secondary to cooperation). Each institution should try to identify a team including a (neuro-ophthalmologist, neuroradiologist, and otolaryngologist to care for patients with traumatic optic neuropathy.[2]

IV. Conclusion

This case, in its initial phase, presented with signs and symptoms suggesting impending right eye traumatic complication. After treatment for 3 weeks with oral steroids, the right eye had come back to its near normal state. The interesting and unusual finding after treatment was the gradual decrease in vision in the left eye, which was quiet previously and had only a hint of papilloedema on presentation. On fundus examination, a finding of gradual paling of the optic disc which may result in an optic atrophy in long run was the peculiar finding in this case. The cause of this finding couldn't be found in any literature till now.

The reason behind presenting this case is to stimulate a practitioner to examine every aspect of both the eyes and to keep an eye out for such unusual findings.

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