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Bilateral Proptosis and Optic Nerve Stretching Following Blunt Traumatic Head Injury A Case Report and Review of Relevant Literature

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Abstract **Case Report**

Optic nerve injury is a rare complication of head trauma, occurring in 0.5% to 5.0% of all traumatic head injuries. We report a case of bilateral proptosis and optic nerve stretching following blunt head injury. We present a unique case of a 31 year old male who sustained traumatic head injuries after being hit by a train. He had contusions in the frontal lobes in conjunction with posttraumatic subarachnoid and intraventricular hemorrhage. In addition to suffering from extensive maxillary facial and orbital fractures involving maxillary sinuses, superior alveolar ridge, frontal sinuses, and nasal bone, he also had bilateral proptosis and optic nerve stretching. His optic nerve injuries were managed conservatively. A left frontal decompression craniotomy and evacuation was performed for refractile intracranial hypertension. Subsequently he was discharged to a long term rehabilitation facility with a GCS score of 11. Optic nerve injuries can occur due to primary axonal damage at the time of impact or due to secondary mechanisms involving edema and compression. Optic nerve injuries may easily be overlooked during initial evaluation of severe head and multisystem trauma, but techniques such as the swinging flashlight test and visual evoked potential help evaluate the extent of these injuries. Although the prognosis depends on the initial visual acuity on presentation, the treatment for optic nerve injury includes observation, medical treatment with high dose steroids, and surgical approaches such as decompression of the optic canal and nerve sheath fenestrations. There is no significant benefit of surgical management over observation alone. Optic nerve injury can easily be overlooked in post-traumatic patients with severe head injuries or altered metal status. Proper evaluation, timely diagnosis and appropriate treatment can potentially improve the long term outcome in such patients.

Keywords: Bilateral Proptosis, Optic nerve injury, intraventricular hemorrhage.

INTRODUCTION

Optic nerve injuries after head injury are rare, occurring in 0.5% to 5.0% of all traumatic head injuries [1]. Optic nerve injuries can occur due to primary axonal damage at the time of impact or due to secondary mechanisms involving edema and compression [1, 2]. In closed head trauma, optic nerve injuries may occur due to transmission and concentration of the impact at certain segments along the optic nerve [3].

Optic nerve injuries may easily be overlooked during initial evaluation of severe head and multisystem trauma, but techniques such as the swinging flashlight test and visual evoked potential (VEP) help evaluate the extent of these injuries [1, 4]. Following proper diagnosis, these injuries can be appropriately managed conservatively or surgically [1].

We report a case of bilateral proptosis and optic nerve stretching following blunt head injury.

CASE REPORT

A 31year old male presented to the emergency department as level 1 trauma after being hit by a train. Trauma workup was completed per ATLS protocol. On arrival to the trauma bay the GCS was 3 and the patient was in obvious distress. The patient was emergently intubated by RSI (Rapid sequence intubation) to secure airway. Vital signs were recorded as BP of 150/100 mmHg, pulse of 72, and respiration rate of 16 saturating 95 with 6L nasal cannula. An eFAST examination was completed along with a pelvic examination which were essentially normal. The injuries were severe head

trauma, open clavicle fracture, and extensive maxillofacial injuries. The left pupil was dilated and unresponsive to light. There was obvious anisocoria. Blood was noted in the left hemotympanum and the left naris. The left forehead had a 4 cm stellate laceration with a depressed skull fracture and leakage of brain matter.

The patient was resuscitated and infusion of mannitol was started. Central lines and OGT and foley were placed. An axial non contrast head CT revealed hemorrhagic contusions in the frontal lobes in conjunction with posttraumatic subarachnoid and intraventricular hemorrhage (Figure 1). There was mass effect from the left frontal temporal lobe resulting in effacement of left lateral ventricle and rightward midline shift. Multiple extensive maxillary facial and orbital fractures were seen involving the anterior and posterior wall of left maxillary sinus, the anterior wall of the right maxillary sinus, the superior alveolar ridge on the right side, and the posterior wall of the left orbit, bilateral orbital roof, the lamina papyracea bilaterally, the nasal bone, the anterior and posterior walls of the frontal sinuses (Figure 2). There were comminuted fractures involving left frontal bone, comminuted and depressed fracture involving the left squamous temporal bone. There was bilateral orbital emphysema. The visualized globes, ocular lenses, extra ocular muscles and optic nerves were unremarkable.

The patient was transferred to the ICU in critical condition. Patient was placed on full ventilator support and started on Fentanyl and propofol drips.3% hypertonic saline was initiated to reduce cerebral edema. Neurosurgery, orthopedic surgery, and ENT were consulted. A Camino was inserted and intracranial pressure (ICP) monitoring was instituted with a cerebral perfusion pressure goal of 70. Keppra was administered for seizure prophylaxis. Q 1 hour neuro monitoring was ordered. A repeat scan was ordered 6 hours later to evaluate the evolution of the ICH (Intracranial hemorrhage)

A CT scan repeated 3 days later to evaluate change in neuro status showed bilateral proptosis with stretching of the optic nerves and mild deformity of the left globe (Figure 3). ICP had increased from 18 to 21. Ophthalmology was consulted and noted conjunctival chemosis in addition to the left eye being proptotic, 6 mm dilated, and fixed (Figure 4). The intraocular pressure was 17.3 in the left eye, and 20.6 in the right. Fundus examination showed normal optic discs with no signs of edema. Macula, retinal periphery, and retinal arteries appeared normal without retinal tears or vitreous hemorrhage. Decision was made to continue with conservative management with close monitoring. He was started on artificial tears and topical antibiotic ointment to prevent exposure keratitis.

A head CT done on hospital day 5 showed increased mass effect, causing increased rightward midline shift. A left frontal decompression craniotomy and evacuation was performed for refractive intracranial hypertension. Following reduction of ICP and improving level of consciousness (GCS 7) the Camino was removed on day 8. An IVC filter was placed. The patient underwent tracheostomy and percutaneous endoscopic gastrostomy placement on day 11. On day 12, the patient underwent open reduction of right-sided Lefort I and II fractures and intermaxillary fixation. He was discharged on post trauma day 18 to a long term care and rehabilitation facility. His best GCS score recorded on discharge was 11.

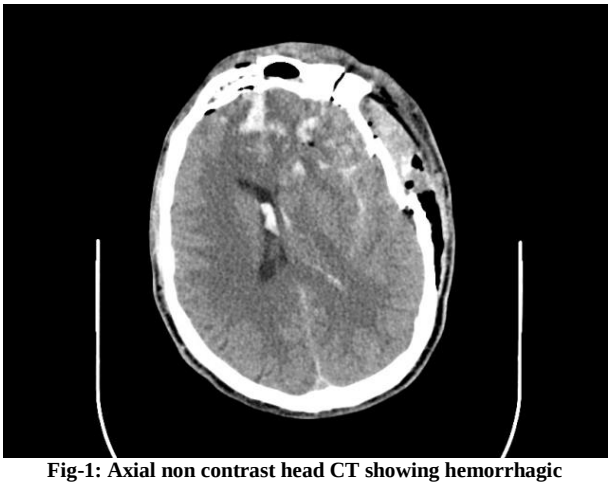

Fig-1: Axial non contrast head CT showing hemorrhagic contusions in the frontal lobes in conjunction with posttraumatic subarachnoid and intraventricular hemorrhage

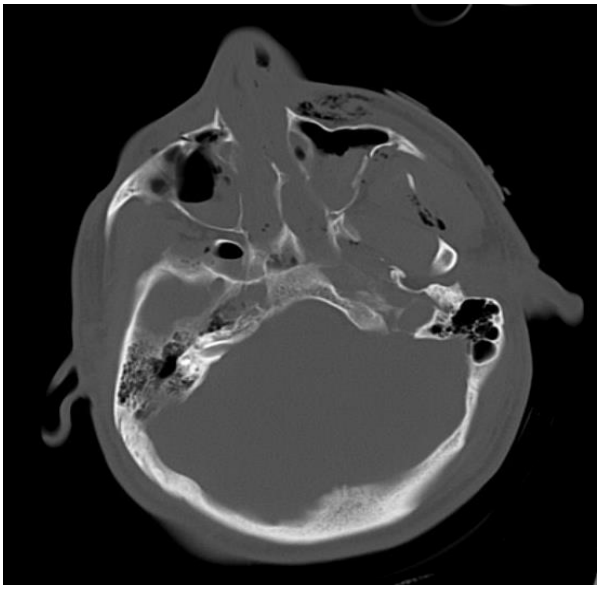

Fig-2: Head CT showing multiple extensive maxillary facial and orbital fractures

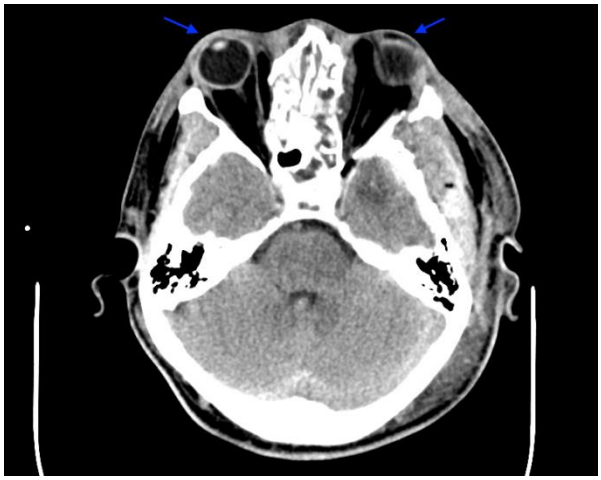

Fig-3: Bilateral proptosis (arrows) with stretching of the optic nerves and mild deformity of the left globe

Fig-4: Bilateral eyes appear proptotic .Significant conjunctival chemosis present in the left eye

DISCUSSION

Traumatic optic neuropathy following blunt head trauma is a rare complication [1]. The optic nerve consists of the axons of the ganglion cells of the retina encased in sheaths continuous with the meninges and can be divided into four segments: intraocular (1 mm), infraorbital (23 -30mm), intracanalicular (8 mm), and intracranial (15 mm) [5]. The intraocular segment is susceptible to avulsion injuries, the intraorbital segment to compressive neuropathy with proptosis and increased IOP, and the intracanalicular segment to shearing, stretching, or torsion given its fixed attachment with the dural sheath [5].

Optic nerve injuries can occur as a result of primary and secondary mechanisms [1]. Primary mechanism involves permanent axonal injury at the time of impact due to contusion, mechanical shearing, laceration, avulsion, and ischemic necrosis [1,2]. Secondary mechanism involves further axonal damage that occurs due to edema, ischemia, thrombosis, and infarction after the initial impact [2,6]. Traumatic optic nerve injury can be classified as direct (penetrating objects) and indirect injuries (transmission of impact in a closed head injury) [1] with the former having a worse prognosis [1].

In a comprehensive review article, Bhateo points out that while the mechanism of optic nerve injury may be clear in direct penetrating trauma to the orbit and globe, it is somewhat unclear in closed head trauma [2]. Anderson et al presented seven cases of sudden unilateral blindness following frontal lobe trauma [3]. The impact on the frontal region of the head is transmitted and concentrated in the optic foreman and is absorbed by the fixed canalicular portion of the optic nerve causing optic nerve stretching, tearing, torsion, and convulsion [3]. These injuries may occur as a result of bony distortion by forces away from the original site of impact [3]. Gross et al inferred that a blow to the frontal region causes an acute descending of the orbital wall and, upon the release of the load, the soft tissues in the orbit oscillate for a longer period of time in comparison to the skeletal optic canal, causing the optic nerve to stretch [7]. This damages the microvasculature and nerve fibers comprising the optic nerve, leading to its compression in the intracanalicular segment [7].

Optic nerve injuries in trauma patients with serious concomitant head injuries may be easily overlooked during the initial evaluation [5]. It may be delayed by weeks or months due to severe multi system trauma which may require sedation and mechanical ventilation [1]. It is important that visual functioning be assessed using appropriate techniques [4,9]. Patients with optic nerve injuries present with visual loss, dilated pupil, and afferent pupillary defect (Marcus Gunn pupil) in the affected eye [5]. Evaluation should start with a careful history and documentation of the time interval between injury and visual examination since delayed visual defects can be detected in some case [1,9]. If the patient is alert and responsive, visual acuity should be tested using a Snellen chart or a Rosenbaum chart, finger counting, or light perception testing [4]. Pupillary reactivity should be tested using the swinging flashlight test [1, 4]. Pupillary response is the most reliable test for detecting the degree of optic nerve injuries [8]. This test is particularly useful in unresponsive patients with unilateral optic nerve injury but is less helpful in patients with bilateral optic nerve injuries [1]. Funduscopic exam to assess optic nerve injury is usually unremarkable immediately after trauma since optic nerve atrophy does not become apparent until 4 to 6 weeks later [2].

In cases of bilateral optic nerve injuries or altered mental status, visual evoked potential (VEP) can provide an objective evaluation of the visual system [1, 4]. VEP measures the electrical signal recorded at the scalp over the occipital cortex in response to light stimuli. The precise origin of the VEP is unclear, but it reveals the integrity of the afferent visual pathway. However, for most clinical situations, VEP usefulness is limited by the use of sedation or in patients on medications that diminish the VEP [1].

In cases of unilateral optic nerve injury, flash visual evoked potentials (FVEP) can be used. FVEP utilizes a ratio between the patient's uninjured eye and injured eye [1]. This ratio shows the degree of afferent optic nerve injury. The advantage of FVEP is that the ratio helps to eliminate confounding factors, including sedation, allowing its use in additional clinical situations [1].

Although the prognosis depends on the initial visual acuity on presentation, the treatment for optic nerve injury includes observation, medical treatment with high dose steroids, and surgical approaches such as decompression of the optic canal and nerve sheath fenestrations [1]. Although conservative management can lead to a 40% to 60% improvement rate [1], medical and surgical approaches are useful [1]. High dose steroids reduce the inflammatory responses that could potentially lead to secondary edema while surgical decompression release the tension on the optic nerve [1]. The International Optic Nerve Trauma Study (IONTS) showed no significant benefit of surgical or medical management over observation alone [10]. Surgery is indicated when bony fragments are present or when there is progressively deteriorating vision in the affected eye after it was documented to be good initially after head trauma [5].

CONCLUSION

Optic nerve injury can easily be overlooked in post-traumatic patients with severe head injuries or altered metal status. It is important these patients undergo appropriate evaluation for optic nerve injuries. Proper evaluation, timely diagnosis and appropriate treatment can potentially improve the long term outcome in such patients.

REFERENCES

1. Aziz KM, Alexander KY, Chen D, Sekula Jr RF. Management of Cranial Nerve Injuries. InSchmidek and Sweet Operative Neurosurgical Techniques 2012 Jan 1 (pp. 2329-2338). WB Saunders.
2. Col Harjinder S Bhateo. Trauma to the cranial nerves. *Indian Journal of Neurotrauma*. 2007;4(2): 89-100..
3. Anderson RL, Panje WR, Gross CE. Optic nerve blindness following blunt forehead trauma. Ophthalmology. 1982 May 1;89(5):445-55.
4. Kline LB, Morawetz RB, Swaid SN. Indirect injury of the optic nerve. Neurosurgery. 1984 Jun 1;14(6):756-64.
5. Thamnuraj AV. Head injury & neuro-ophthalmology. *Neurosurgery on the web*. Online.
6. Walsh FB, Lindenberg R. In, Die Veranderugen ges. Schnervan beci indirectem trauma. Entiwicklung and Fortschritt in der Angenheilkundge-Fortbilung skurs tur Aurgenarze. Hamburg, 1962. Fredinand, Enke Verlag, 1963.
7. Gross CE, DeKock JR, Panje WR, Hershkowitz N, Newman J. Evidence for orbital deformation that may contribute to monocular blindness following minor frontal head trauma. Journal of neurosurgery. 1981 Dec 1;55(6):963-6.
8. Edmund J, Godtfredsen E. Unilateral optic atrophy following head injury. Acta ophthalmologica. 1963 Dec;41(6):693-7.
9. Greenberg RP, Becker DP, Miller JD, Mayer DJ. Evaluation of brain function in severe human head trauma with multimodality evoked potentials: Part 2: Localization of brain dysfunction and correlation with posttraumatic neurological conditions. Journal of neurosurgery. 1977 Aug 1;47(2):163-77.
10. LEONARD A. The treatment of traumatic optic neuropathy. Ophthalmology. 1999;106:1268-77.