Traumatic chiasmal syndrome: A meta-analysis

Loganathan Vellayan Mookan, Philip A. Thomas*, Ankit Anil Harwani

Institute of Ophthalmology, Joseph Eye Hospital, PB 138, Tiruchirapalli 620001, Tamil Nadu, India

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ABSTRACT

Purpose: To report a case presenting with bitemporal hemianopia due to traumatic chiasmal syndrome after head injury, and to compare the findings with individual case reports published in the literature.

Methods: A detailed search was made in PubMed, MedIND, Taylor and Francis online and Wiley online library databases for individual case reports of traumatic chiasmal syndrome. All the case reports were read in full and the findings summarized in a table, which included details of the case who presented with bitemporal hemianopia as an index case.

Results: All published cases of traumatic chiasmal syndrome appear to share some common features, such as injury to the frontal bone and fracture of the anterior skull base. Bitemporal hemianopia and visual acuity have a variable presentation, and do not appear to correlate with severity of injury. Isolated bitemporal hemianopia is rare and clinical improvement may or may not occur.

Conclusions and importance: Traumatic chiasmal syndrome should be considered as a differential diagnosis in patients presenting with bitemporal hemianopia after head injury causing frontal and anterior skull base fracture.

1. Introduction

Bitemporal hemianopia is a hallmark of optic chiasmal injury, because of the peculiar arrangement of the fibres of the optic nerves; various lesions, including tumour, inflammation, demyelination, ischemia and infiltration, can affect the optic chiasma.1 Traumatic chiasmal syndrome, which occurs due to an injury at the level of crossing fibres in the optic chiasma following trauma to the head, is characterised by bitemporal hemianopia or scotomata. Traumatic chiasmal syndrome is a rare occurrence, since only severe impact will damage the anatomically-privileged chiasma, and it is difficult to survive such an impact. The first case of traumatic chiasmal syndrome was reported by Nieden in 1883; several cases have since been reported. The current brief report describes a patient who presented with traumatic chiasmal syndrome, and compares the findings observed with those of individual cases reported in the literature.

2. Material and methods

A patient presenting with traumatic chiasmal syndrome was investigated and a systematic literature search was performed in the PubMed, MedIND, Taylor and Francis online and Wiley online library databases using the search term: “traumatic chiasmal syndrome” and “chiasmal injury”. Only individual case reports of traumatic chiasmal syndrome were selected to be included in this report, and all the case reports were read in full. Case reports with no follow-up findings were excluded. A table was prepared to provide an overview and to compare the salient aspects of the published case reports with the case being presented in this report.

3. Results

A total of 9 case reports were selected; the salient findings in these 9 cases are summarized in Table 1, in addition to details of our case. An analysis of the clinical details revealed that eight out of 10 cases had injury due to road accident while riding a motor vehicle, while one had closed head injury when the bus he was travelling in was targeted by a bomb and one had an accidental fall. The visual acuity after trauma ranged from normal to counting fingers. The extent of field defect, bitemporal hemianopia, was uncertain, being either complete or incomplete, with or without macular splitting. An analysis of the radiological reports revealed that all the cases had fracture of the frontal bone, along with the involvement of the anterior base of the skull. Magnetic resonance imaging (MRI) details were provided in five case reports, which showed some abnormality in the chiasma; the MRI report of the case being presented here did not reveal any chiasmal

* Corresponding author.

E-mail addresses: drvml1940@gmail.com (L. Vellayan Mookan), thomasdiagnosticcentre@gmail.com (P.A. Thomas), ankitanil_harwani@yahoo.co.in, harwaniankit@gmail.com (A.A. Harwani).

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| Sr. no. | Case reported by | Age | Sex | Mode of injury | Visual acuity | Visual field defect | Other deficits | CT/X-ray Findings | MRI findings | Outcome |
|--------|----------------|-----|-----|----------------|---------------|-------------------|---------------|-----------------|-------------|---------|
| 1 | Index case of the current report | 39 | M | Motorcycle accident | RE 6/6, LE 6/12 | Bitemporal hemianopia | Anosmia, CSF rhinorrhea | CT: Comminuted frontal and anterior skull base fractures, bilateral frontal lobe hemorrhagic contusions and multiple frontal aeroceles | Normal optic chiasma (Fig. 2) | Vision improved to 6/9 in LE, Visual fields improved in both eyes (Fig. 3) |
| 2 | Dutta et al. | 25 | M | Closed head injury | RE 6/12, LE 6/60 | Bitemporal hemianopia | Intermittent CSF rhinorrhea | CT: Orbital roof and basi sphenoid fracture | Fronto gyrus rectus herniation in sphenoid sinus with chiasmal edema | Marginal visual improvement |
| 3 | Hughes et al. | 45 | M | Road accident | BE 6/9 | Complete bitemporal hemianopia | Bilateral anosmia, complete facial paralysis on the right with loss of taste and mild left hemiparesis | X-ray: Fracture of frontal bone | No improvement |
| 4 | Hughes et al. | 31 | M | Head on motor crash | RE 6/18, LE 1/60 | RE – upper temporal quadrant defect LE – upper nasal island of vision only | 3rd nerve paresis | X-ray: Fissured fracture in the left frontal bone, traversing the left frontal sinus, also involving left anterior and middle fossa | Significant improvement |
| 5 | Logan et al. | 33 | M | Road accident | RE 6/9, LE Counting fingers | Bitemporal hemianopia | CSF rhinorrhea, bilateral anosmia, LE limitation of ocular movements except lateral movements | Multiple frontal fracture, anterior fossa fracture involving sphenoid and ethmoid air sinuses | No improvement |
| 6 | Mohindra et al. | 17 | M | Accidental fall | RE 6/60, LE 6/9 | Bitemporal hemianopia | None reported | CT: Speck of air at interpeduncular and chiasmatic cistern | Isolated contusion of optic chiasma | No improvement |
| 7 | Resneck et al. | 50 | M | Automobile accident | BE 6/6 | Bitemporal hemianopia | Absence of olfactory functions | X-ray: Fracture of right superior orbit CT: Pneumocephalus and fracture of sella turica | No improvement |
| 8 | Tang et al. | 17 | M | Motor cycle accident | BE 20/25 | Bitemporal hemianopia | Diplopia with esophoria, diabetes insipidus, decreased thyroid stimulating hormone and testosterone levels | Multiple facial and skull fractures, including fracture of lesser wing of the sphenoid | Hemorrhagic contusion of the frontal lobes, mild swelling on the left side of the optic chiasm | No change |
| 9 | Vora et al. | 26 | M | Motor vehicle accident | RE 6/6, LE 6/36 | Complete bitemporal hemianopia | Midline frontal bone fracture, basi sphenoid fracture, intraparenchymal hematoma in left frontal region | Central chiasmal contusion with thinning of nasal fibres | Vision improved to 6/6 both eyes but no improvement in fields |
| 10 | Yazici et al. | 20 | M | Motor vehicle accident | BE 20/20 | Bitemporal hemianopia | Exotropia with RE fixation. | Fracture frontal, ethmoid and maxillary, fracture in the sellar region of sphenoid bone, air in the orbit and cranium | Thinning, shape deformity in the optic chiasm | No change in visual fields |

**Abbreviations:** RE = right eye; LE = left eye; BE = both eyes; M = male; CT = computed tomography; MRI = magnetic resonance imaging; CSF = cerebrospinal fluid.
damage. Out of 10 cases, six did not show any improvement in the visual manifestations. Only one case presented with isolated bitemporal hemianopia, while the other cases had other accompanying manifestations, such as cerebrospinal fluid rhinorrhea, anosmia, 3rd nerve palsy and extraocular muscle injury.

In our case, it is not clear why visual loss was gradually progressive. In previously reported cases, it appears that the onset of the visual loss was shortly after the trauma, and there was no progression several months thereafter, unlike the observation in our case.

4. Discussion

The optic chiasma is a structure located along the visual pathway at the point where the optic nerves of the right eye and left eye join each other. The nasal fibres of the retina decussate at the optic chiasma. It is the arrangement of the fibres at the chiasma that accounts for the typical visual field defects seen when a lesion occurs at the site. The most common field defect observed in patients with optic chiasmal injury is bitemporal hemianopia, although the field loss can be of varying degrees, as seen in this report. Traumatic chiasmal syndrome is a rare entity; in fact, the patient being described is the first such case documented among patients with trauma to the head who have been examined and treated at our institution.

In a study by Mejico et al., the most frequent cause of bitemporal hemianopia was found to be pituitary adenoma. A bitemporal field defect due to optic chiasmal damage after head injury occurs in 0.3% of cases. The field defect that develops after traumatic chiasmal syndrome is variable, and can manifest as either complete monocular blindness or incomplete subtle scotomas which may or may not involve the macula, suggesting that there is no specific pattern of field defects, as seen in this report. Traumatic chiasmal syndrome is usually seen following a fracture of the frontal bone along with multiple cranial fractures. Similar observations were made in the case being presented in the current report.

Two main types of head injury have been described, namely, contact injury and acceleration/deceleration injury. A contact injury is produced by direct impact, while acceleration/deceleration injury occurs following violent motion of the head, which results in subdural haemorrhage and diffuse axonal injury. Following trauma, the axons either undergo primary axotomy, which implies complete transection of the nerve due to mechanical shearing, or secondary axotomy, which implies slow degeneration of the nervous tissue.

It was previously believed that the principal mechanism involved in chiasmal injury following trauma to the head involved tearing of the crossing fibres due to mechanical stretching of the chiasma. However, Traquair et al. suggested that the pial vessel, which supplies the chiasma, undergoes tearing due to stretching, resulting in the functional impairment of the optic chiasma. Hughes et al., who were unable to
detect any anatomical disruption of the chiasma itself, opined that the damage was probably due to compromised blood supply to the central area of the chiasma, thus substantiating the theory suggested by Traquair et al. However, the chiasma is known to be richly supplied by anastomoses between branches of the anterior communicating artery with the chiasmal artery, anterior cerebral artery, internal carotid artery, posterior communicating artery, anterior choroidal artery, middle cerebral artery and prechiasmal arcade; hence, the vascular theory for chiasmal damage may not completely explain why the lesion occurs. Hassan et al., in their case report, opined that the mechanism of the injury to the optic chiasma following trauma to the head may vary from individual to individual, being due to one or more of the following: i) mechanical stretch or mechanical tear; ii) contusion haemorrhage; iii) contusion necrosis or avulsion. In contusion necrosis, there is disruption of axons, followed by breakdown of the axonal membrane and sparing of the other fibres, remains unclear.

Very rarely, isolated (without accompanying manifestations) bitemporal hemianopia is seen in patients with traumatic chiasmal syndrome, as seen in only one previous case report. Other possible manifestations of traumatic chiasmal syndrome, some of which have been described in the case reports analyzed here, include first cranial nerve palsy, third cranial nerve palsy, fourth cranial nerve palsy, sixth cranial nerve palsy, seventh cranial nerve palsy, eighth cranial nerve palsy, cerebrospinal fluid rhinorrhoea, carotid cavernous fistula, carotid aneurysm, panhypopituitarism, diabetes insipidus, meningitis, intrasellar hematoma and pneumatocele.

Savino et al., in their study on 11 patients with traumatic chiasmal syndrome, opined that the severity of visual field defect does not necessarily correlate with the severity of the head injury or associated neurological deficits. The loss of visual field seldom improves; it either remains stationary or may worsen. Out of 10 cases listed in Table 1, six cases did not show any improvement in the visual fields. However, some cases might show improvement in the visual acuity and visual field, as seen in three of the case reports and in the index case. Also, Anderson et al. reported a case which showed improvement in the visual field. The exact cause for this improvement cannot be ascertained; however, if the mechanism of injury had been due to vascular compromise, then re-establisment over time of vascular channels or development of new anastomoses between the arteries supplying the chiasma might have improved the functionality of the optic chiasma.

The investigation of choice for traumatic chiasmal syndrome is magnetic resonance imaging. However, as seen in the case being presented in the current report, the MRI may not show any anatomical abnormality of the chiasma that could explain the bitemporal field defect. Hence, it is not always necessary to perform MRI unless there are other neurological deficits. Ostri et al., in their case report, concluded that measurement of the retinal nerve fibre layer thickness by optical coherence tomography can be an effective, non-invasive tool in diagnosing an optic chiasmal lesion. Due to the paucity of cases being reported, management guidelines for traumatic chiasmal syndrome are yet to be established. The mecobalamin capsules which were prescribed to the index case in this report may not have contributed to the improvement of the visual acuity and visual fields, as its role in human subjects is inconclusive. Patients should be followed up regularly to check for any new neurological signs or symptoms other than involvement of the optic pathway.

To summarize, traumatic chiasmal syndrome should be suspected in a patient with bitemporal hemianopia occurring after trauma to the frontal bone associated with anterior skull base fracture, with or without other accompanying manifestations.

5. Conclusion

Although traumatic chiasmal syndrome is rare, it is important to identify this entity when it does occur; it should be considered as a differential diagnosis in a patient with bitemporal field loss after head trauma. Thus, unnecessary neurological diagnostic and investigative procedures can be avoided in such patients.

Patient consent

Consent to publish the case report was not obtained, because this case report does not contain any personal information that could lead to the identification of the patient.
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Authorship

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References

1. Miller NR, Levin LA. Topical diagnosis of chiasmal and retrochiasmal disorder. In: Miller NR, Levin NJ, eds. Walsh and Hoyt's Clinical Neuro-ophthalmology. sixth ed. Philadelphia: Lippincott Williams & Wilkins; 2005:503–573.
2. Datta SGS, Pathak HC. Traumatic chiasmal syndrome. J Neurotrauma. 2009;6(2):137–140.
3. Hughes EBC. Indirect injury of the optic chiasma—a case report. Br J Ophthalmol. 1945;29(12):429–432.
4. Hughes EBC. Injury to the optico-chiasmal junction—a case report. Br J Ophthalmol. 1943;27(8):367–371.
5. Logan WC, Gordon DS. Traumatic lesions of the optic chiasma. Br J Ophthalmol. 1967;51(4):258–260.
6. Mohindra S, Sharma M, Mohindra S. Traumatic chiasmal syndrome. Br J Neurosurg. 2012;26(6):872–874.
7. Resneck JD, Lederman IR. Traumatic chiasmal syndrome associated with pneumocephalus andellar fracture. Am J Ophthalmol. 1981;92(2):233–237.
8. Tang RA, Kramert IA, Schiffer J, Woom C, Hayman LA. Pardo G. Chiasmal trauma: clinical and imaging considerations. Surv Ophthalmol. 1994;38(4):381–383.
9. Vora TK, Ravi RR. Traumatic chiasmal syndrome: a midline axonal injury. Neuro Ophthalmol. 2015;39(5):253–256.
10. Yezici B, Kivanc SA. Isolated bitemporal hemianopsia due to traumatic chiasmal syndrome. Ulus Travma Ve Acil Cerrahi Derg. 2016;22(1):97–99.
11. Mejico LJ, Miller NR, Dong LM. Clinical features associated with lesions other than pituitary adenoma in patients with an optic chiasmal syndrome. Am J Ophthalmol. 2004;137(5):908–913.
12. Laueran AB. Traumatic bitemporal hemianopsia. Acta Ophthalmol. 1971;49(1):134–142.
13. Savino PJ, Glaser JS, Schatz NJ. Traumatic chiasmal syndrome. Neurology. 1980;30(9):963–970.
14. Hassan A, Crompton JL, Sandhu A. Traumatic chiasmal syndrome: a series of 19 patients. Clin Experiment Ophthalmol. 2002;30(4):273–280.
15. Traquair HM, Dott NM, Russell WR. Traumatic lesions of the optic chiasma. Brain. 1935;58(3):398–411.
16. Hughes B. Indirect injury of the optic nerves and chiasma. Bull Johns Hopkins Hosp. 1962;111:98–126.
17. François J, Neetens A, Collette JM. Vascularization of the optic pathway. Br J Ophthalmol. 1956;40(12):730–741.
18. Anderson DL, Lloyd LA. Traumatic lesions of the optic chiasma: a report of four cases. Can Med Assoc J. 1964;90:110–115.
19. Ostri C, Damgaard B, Hamann S. Optical coherence tomography documenting retinal nerve fiber loss in traumatic optic chiasmal syndrome. Acta Ophthalmol. 2012;90(8):792–794.