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ISOLATED COMPLETE BITEMPORAL HEMIANOPIA IN TRAUMATIC CHIASMAL SYNDROME

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ABSTRACT
A 48 Year Old man presented with an isolated complete Bitemporal Hemianopia after sustaining a closed head trauma in an accidental fall one month after injury without visual acuity defect and without neurological complications as a rare case of traumatic chiasmal syndrome and its pathogenic mechanisms discussed.

INTRODUCTION

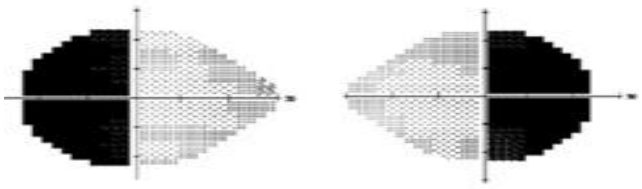
Chiasmal Syndrome comprises various signs and symptoms associated with lesions of optic chiasma. Trauma is one of the rare aetiological factors of chiasmal syndrome because few patients survive after the severe impact [1]. Various visual field defects including Bitemporal Hemianopia, temporal Hemianopia and quadrantanopia have been reported [2]. However complete Bitemporal Hemianopia without profound visual loss as is seen in the present case is rare. Frequently associated neurological complications include cranial nerve palsies, diabetes insipidus, cerebrospinal fluid rhinorrhea, panhypopituitarism and carotid cavernous fistula [3].

CASE REPORT

A 48 Year Old man sustained a closed head trauma in an accidental fall. One month after the accident he presented to us with a chief complaint of lateral blindness in both eyes. His best corrected visual acuity in both eyes was 6/6. Humphrey visual field analyser showed a complete Bitemporal Hemianopia visual field defect (Fig. 1). Other than the visual field defect no focal neurological deficits including cranial nerve palsies, diabetes insipidus, cerebrospinal rhinorrhea and panhypopituitarism were detected. Fundus examination was normal with normal optic discs. Pupils in both eyes were normal in size and reacting sluggishly. CT Scan reveals multiple fractures in facial bones involving walls of left orbit and both paranasal sinuses. Small contusion in left orbito frontal lobe was seen. Pockets of Pneumocephalus in frontal region and in basal cisterns were seen.

MRI Images reveal a collection in left frontal region, extra axial indenting the left frontal lobe – 44 x 27 MM with internal air fluid level. Evidence of parenchymal contusion was present in left orbito frontal lobe with subtle mass effect over left optic foramen and both optic nerves in pre chiasmatic region. No radiological abnormalities of the visual pathways were detected.

Fig. 1. Humphrey automated static perimetry shows complete bitemporal hemioanopia in grey scale



DISCUSSION

The pathogenicity of indirect injury limited to the decussating fibers of chiasma while sparing those that do not cross remains complex and unclear. Rand provided photographic evidence of sagittal tearing of the chiasma. Copez suggested that the tear injury of the chiasma results from anteroposterior distortion of the skull with separation of the optic canals. Traquire, Dott and Russell stated tearing of the pial vessels supplying the chiasma produced an interruption of function and softening of the chiasma. Walsh and Gass say compression of the chiasma occurs by the cyrus recti of frontal lobe which herniates into chiasmal cistern as a consequence of diffuse brain swelling. Other authors reported basilar skull fracture involving sella turcica and producing pneumocephalus with CAT scan in cases of traumatic chiasmal syndrome. Pneumocephalus damages chiasma either due to its compression effect or by the development of cystic pocket that evolves into adhesive arachnoiditis of optic chiasma. Regardless of pathogenicity of traumatic chiasmal syndrome awareness of its presence is important to the patient. The physician must maintain fluid, electrolyte balance, anticipate possible hormonal deficiency and avoid unnecessary neurosurgical intervention. An assessment of the status of point of fixation is important with respect to long term disability. Bitemporal field defects resulting in loss of corresponding points in visual space leads to loss of depth perception.

CONCLUSION

Isolated complete Bitemporal hemianopia without profound visual loss and without associated neurologic complications including cranial nerve palsies, diabetes insipidus, cerebrospinal rhinorrhea, panhypopituitarism and carotid cavernous fistula is very rare as in the present case.

The mechanism of injury resulting in bitemporal hemianopia is quite complex varying from stretching of decussating nasal fibres to actual rupture in chiasma to compression by haematoma, pneumocephalus and arachnoiditis due to fracture of bones of paranasal sinuses especially sphenoid sinus.

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