Case Report

A Traumatic Bitemporal Hemianopia with Macular Sparing

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Traumatic chiasmal syndrome is a rare complication of closed head trauma. It often presents as bitemporal hemianopia and may be associated with other neurological signs. The authors report a case of a 47-year-old man who had sustained severe frontal head trauma from a motor vehicle accident that caused multiple cranial fractures and prolonged loss of consciousness. He was subsequently diagnosed with traumatic chiasmal syndrome. Tangent field testing revealed bitemporal hemianopias with some macular sparing. Macular sparing was not found on the central 30-2 pattern of Humphrey visual field test.

Keywords: *Traumatic chiasmal syndrome, Bitemporal hemianopia; Macular sparing, Microsaccades*

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Bitemporal hemianopia, the sine qua non of chiasmal syndrome, can vary in expression from a subtle bitemporal arcuate scotoma to a dense bitemporal hemianopia[1,2]. Variances may resemble other visual field defects, such as junctional scotoma[1]. Traumatic chiasmal syndrome secondary to closed head trauma is a rare event[2-4]. It may occur in isolation, or it may be associated with neurological signs that indicate damage to other structures in the region of the anterior cranial fossa or at the base of skull (e.g., diabetes insipidus, panhypopituitarism, anosmia, deficits of cranial nerves II, III, IV, VI or VII, deafness, CSF rhinorrhea and/or otorrhea, carotid aneurysm, carotid-cavernous fistula, meningitis, pneumatocele, or intrasellar hematoma)[1,2,4-8]. Delayed onset seesaw nystagmus can also develop in cases of traumatic chiasmal syndrome[2,9-11]. Eggenberger achieved moderate improvement of seesaw nystagmus using a combination of clonazepam and baclofen[10], while Frisen and Wikkelso reported a temporary cessation of nystagmus after ethanol ingestion[9].

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The present case report involves a traumatic chiasmal syndrome presenting as bitemporal hemianopia with macular sparing.

Case Report

A 47-year-old male patient presented to the Doheny Eye Institute in 1999. He reported that he had suffered severe head trauma in a motor vehicle accident in 1997, and during his hospital stay was placed in a drug-induced coma for three and a half weeks. Computarized tomograms (CT) of his head taken during his hospital stay revealed extensive, multiple comminuted fractures of the right frontal bone, right orbital roof, and right paranasal sinus, as well as bifrontal hemorrhagic contusions, a right frontal epidural hematoma, and a basal subarachnoid hemorrhage.

When the patient was first seen at the Doheny Eye Institute, he complained of a variable binocular diplopia. He said the images could overlap, separate, or slide by each other, depending upon how tired he was and which direction he was looking. Humphrey visual fields showed bitemporal hemianopia (Fig. 1). Based on the test results and his history of head trauma, he was diagnosed with traumatic chiasmal syndrome.

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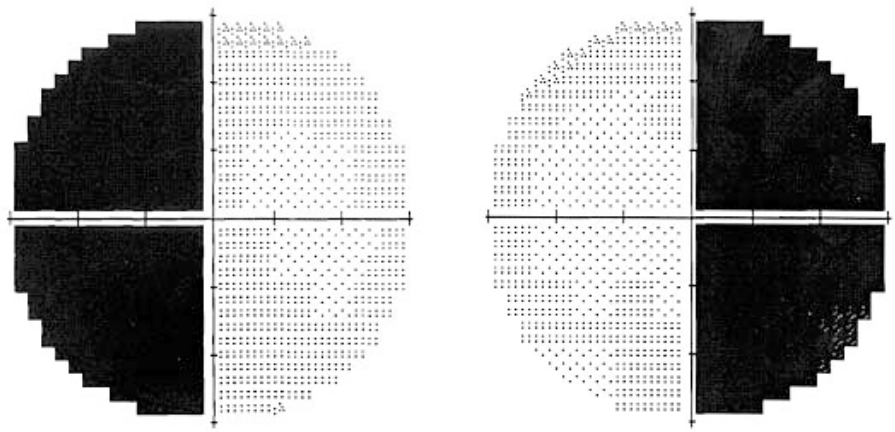

Fig. 1 Humphrey visual fields showed bitemporal hemianopia with macular splitting in 1999

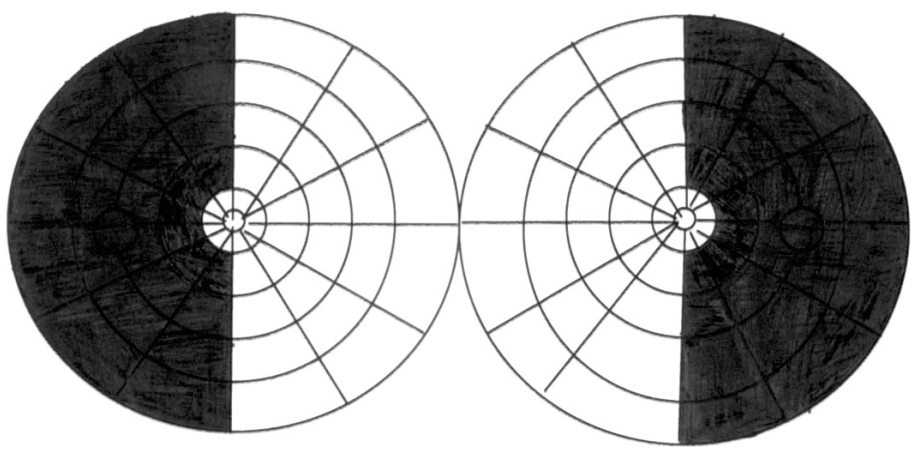

Fig. 2 Tangent field testing performed with a 3-mm white target at 2000 mm revealed bitemporal hemianopia with some macular sparing in 2002

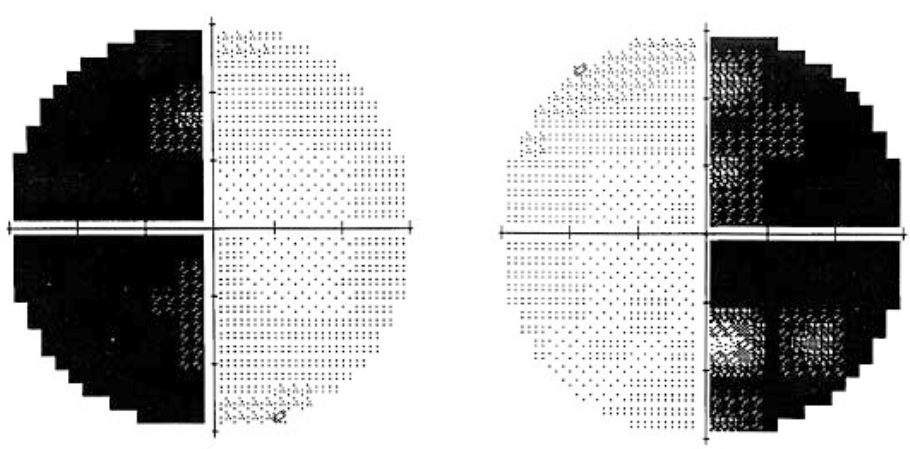

Fig. 3 Humphrey visual fields showed bitemporal hemianopia with macular splitting in 2002

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In 2002, the patient returned to Doheny with a chief complaint that he could not see well at a distance. His best corrected visual acuities were 20/20 OD and 20/30 OS.

External eye examination was normal and slit-lamp examination was unremarkable. Intraocular pressures were 16 mmHg in both eyes. Ocular motility examination revealed 7 diopters of left hypertropia in primary gaze and full ductions and versions. Neuropsychological, generalized neurological, and facial neurological examinations were grossly normal. Amsler grid testing showed a small paracentral temporal loss in the left eye. Tangent field testing performed with a 3-mm white target at 2000 mm revealed bitemporal hemianopia with some evidenced of macular sparing (Fig. 2). However, a Humphrey visual field test showed bitemporal hemianopia without macular sparing (Fig. 3). Fundus examination showed marked bilateral bow-tie optic atrophy.

The clinical findings and visual field testing led to the diagnosis of traumatic chiasmal syndrome was made. The patient was reassured that the visual loss was not progressive and he was prescribed 7 diopters of prism equivalent to base down in the left eye together with his refraction.

Discussion

Closed head trauma is a rare cause of chiasmal injuries[2-4], which are often manifested as bitemporal hemianopia. Traumatic chiasmal syndrome usually follows severe frontal head trauma accompanied by multiple cranial fractures and prolonged loss of consciousness[2,3,6-8], but the resultant degree of visual loss does not necessarily reflect the severity of the craniocerebral trauma[6]. Injuries associated with traumatic chiasmal syndrome are usually to the frontal region, although temporal or parietal region and occipital injuries are described in few rare cases[2,4,7]. The two most common causes of such injuries are falls and motor vehicle accidents[7]. Magnetic resonance imaging (MRI) is the best method for identifying chiasmal abnormalities[4].

The pathogenesis of indirect injuries that affect only the decussating fibers of the optic chiasm and spare the nondecussating fibers remains controversial. Several possible mechanisms of injury have been suggested by various authors. It has been suggested that shearing of the internal carotid arteries and of their branches that supply the chiasm may induce ischemia and softening[2,7]. It has been argued that rupture of fine vessels that supply the median chiasmal region could affect the crossing fiber[2]. These authors have also ascribed the phenomenon to rupture of Françios' chiasmal artery, a branch off of the anterior communicating artery that supplies the median region of the chiasm[2]. Notwithstanding, these explanations have not been pathologically demonstrated. The authors agree with those that propose that the great force of the injury may cause a sagittal tearing and stretching of the chiasmal crossing fibers[2,3,8]. A shearing force that reaches the chiasm may cause contusion necrosis at the submicroscopic level[1,2,13]. This is thought to be the most common mechanism responsible for traumatic chiasmal syndrome[12].

Traumatic chiasmal syndrome can present with either incomplete or complete bitemporal hemianopia, and also with macular splitting or sparing[2,5-7,14,15]. Complete bitemporal hemianopia is associated equally with either macular sparing or macular splitting, while macular sparing is often seen in incomplete bitemporal hemianopia[2].

In macular sparing hemianopias the hemifield loss spares the central 5 degrees or less of the visual field[13,15,16]. It is difficult to explain why all of the crossing fibers would be destroyed except for the macular fibers. Some authors have suggested that macular sparing may not really exist, but may be an artifact caused by unsteady fixation[2,14-16], because of either small, undetectable shifts of gaze fixation or released attention of the subject during long examinations[15]. Haidinger brushes and the Maxwell spot can be used for macular field examination to determine whether there is macular sparing or splitting in hemianopia. These methods have been to demonstrate true macular splitting in some patients who had traumatic bitemporal hemianopia[14,15].

Traumatic chiasmal syndrome may demonstrate macular sparing due to microsaccades. These small involuntary movements are a prominent feature of human eye movement patterns during fixation[17-20]. They occur 1-3 times each second[17-18]. The amplitude varies, ranging from seconds to minutes of arc[17,18,21,22]. Microsaccades appear to be of significant importance to vision under stabilized conditions. When the target image does not move on the retina, it will normally fade and disappear from view within several seconds. Microsaccades return the retinal image by sweeping the target image rapidly back and forth to stimulate retinal receptors[17,19,20]. However, microsaccades can be voluntarily suppressed[18,19,22]. They are probably not important for the successful completion of finely guided visuomotor tasks[18,22].

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True macular sparing may be explained by the fact that crossing fibers in the chiasm are supplied by the superior hypophyseal artery or by a branch of the posterior communicating artery. In traumatic bitemporal hemianopia with macular sparing, these arterial branches might be spared, while the chiasmal artery and the branches from Dawson's arcade that supply the remaining crossing fibers are injured[2].

The retinotopic map of the visual field in V_1 shows that the fovea is represented at the occipital pole while the far periphery is represented at the anterior margin of the calcarine fissure[23]. In addition, the area representing the fovea covers a much larger area in V_1 than the area representing the periphery[23]. It is often suggested that this high degree of redundancy of the macula, allows for central vision to be preferentially preserved.

In the present case report, the patient had classic traumatic chiasmal syndrome after a severe frontal head trauma accompanied by multiple cranial fractures and prolonged loss of consciousness. Tangent field testing revealed a bitemporal hemianopia with macular sparing. This phenomenon is likely from pseudomacular sparing due to unsteady fixation or to microsaccades occurring during fixation. Tangent field testing, it is does not permit accurate accounting of whether the patient is really holding the fixation point at all times. The authors did not find macular sparing on the central 30-2 pattern of Humphrey visual field test in this patient because in this pattern, the testing point steps every 6 degrees[24], whereas the macular sparing was less than 5 degrees from fixation. Amsler grid, threshold Amsler grid or a central 10-2 pattern is more useful for patients with central islands of field remaining or defects very close to fixation[24,25].

The pathogenesis of traumatic chiasmal syndrome remains uncertain. However, it is important to recognize bitemporal hemianopia as a consequence of closed head trauma. This may permit the anticipation of hormonal deficiencies which requires close monitoring for fluid and electrolyte balance. Such patients with a nonprogressive cause of chiasmal field loss need not be subject to invasive neurological or neurosurgical investigations.

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ลานสายตาผิดปกติแบบ Bitemporal hemianopia โดยมี macular sparing จากอุบัติเหตุ

นิพนธ์ จิรภาไพศาล, Alfredo A Sadun

Traumatic chiasmal syndrome เป็นภาวะแทรกซ้อนที่เกิดจากอุบัติเหตุที่ศีรษะซึ่งพบได้น้อย ผู้ป่วยจะมาด้วยอาการ bitemporal hemianopia และอาจสัมพันธ์กับอาการทางระบบประสาทอื่น ๆ ผู้เขียนได้รายงานผู้ป่วยชายอายุ 50 ปี ได้รับอุบัติเหตุที่ศีรษะอย่างรุนแรงจากอุบัติเหตุรถมอเตอร์ไซค์ ทำให้กะโหลกศีรษะแตกหลายตำแหน่งและหมดสติเป็นเวลานาน ผู้ป่วยได้รับการวินิจฉัยเป็น traumatic chiasmal syndrome การตรวจลานสายตาโดยวิธี Tangent field พบ bitemporal hemianopias ร่วมกับ macular sparing โดย macular sparing ไม่พบในการตรวจลานสายตา โดยวิธี Humphrey แบบตรงกลาง 30-2

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