# Traumatic Chiasmal Syndrome Associated With Midline Basilar Skull Fractures

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We studied two young males who had visual field defects consistent with optic chiasmal injury after blunt frontal head trauma. One patient also had a unilateral optic neuropathy. Long-term follow-up disclosed complete bitemporal hemianopsias in these patients. Both patients had midline basilar skull fractures that traversed the midclivus through the sella turcica floor, dorsum sellae, and sphenoid sinus. Magnetic resonance images did not identify intrachiasmal hemorrhage as the cause of the visual field defect. We believe this fracture pattern, in conjunction with the magnetic resonance image findings, suggests tearing of the optic chiasm on a microscopic, if not macroscopic, scale as the cause of the complete bitemporal hemianopsia.

**T**<sub>RAUMATIC</sub> CHIASMAL SYNDROME is a rare condition manifested by the onset of a bitemporal hemianopsia after head trauma, usually frontal. Since its recognition in 1883 by Nieden<sup>1</sup> approximately 100 cases have been reported.<sup>2,3</sup> Multiple mechanisms for injury to the optic chiasm that result in bitemporal hemianopsia have been proposed. These proposed mechanisms include chiasmal infarction,<sup>4,5</sup> compressive injury from intrachiasmal hemorrhage,<sup>6,7</sup> and direct tearing or stretching of the chiasm.<sup>8,11</sup>

We studied two cases of traumatic chiasmal syndrome with computed tomographic and magnetic resonance images.

## **Case Reports**

#### Case 1

A 21-year old man struck his forehead after being projected from an all-terrain vehicle. Examination disclosed a cerebral hemorrhage, basilar skull fracture, and LeFort II midfacial fracture with minimal displacement.

Twelve days after the injury, his mental status improved and he complained of decreased vision in his left eye and decreased hearing in his right ear. This complaint prompted the first ophthalmic examination since the accident. Visual acuity without correction was R.E.: 20/ 20 and L.E.: 20/200 with eccentric fixation in his nasal field. A relative afferent pupillary defect was noted in the left eye. Ocular motility was full and facial nerve function was intact bilaterally. He had no enophthalmos, malocclusion, or midfacial hypermobility. Ophthalmoscopy demonstrated normal retinal perfusion and no pallor or edema of the optic nerve head. The patient was treated with intravenous methylprednisolone, 1 g, every six hours for 72 hours.

Goldmann visual field testing, before corticosteroid administration, demonstrated a left nasal island and a complete right temporal hemianopsia (Fig. 1). Audiometry demonstrated a profound right sensorineural hearing loss.

Computed tomography (Fig. 2) demonstrated a longitudinal midclival fracture that traversed the clivus, sella turcica floor, and tuberculum sellae. No optic nerve canal fractures were noted. A LeFort II fracture with comminuted maxillary fractures and a minimally telescoped nasoethmoid fracture were present. Magnetic resonance imaging (Fig. 3) demonstrated a swollen optic chiasm located just beneath a right-sided hypothalamic hemorrhagic contusion. The hemorrhage also involved the gyrus rectus of the frontal lobe. Hemorrhage was also seen in the sphenoid sinus and along the tentorium.

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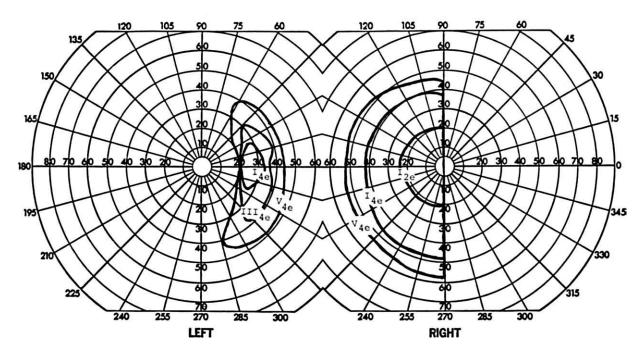


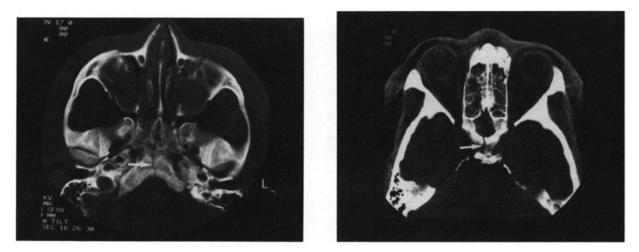
Fig. 1 (Heinz, Nunery, and Grossman). Left nasal island and a complete right temporal hemianopsia. Visual acuity is L.E.: 20/200 and R.E.: 20/20.

Nine months after the injury visual acuity was R.E.: 20/20 and L.E.: 20/20. The left relative afferent pupillary defect had resolved. There was bilateral optic nerve pallor distributed in a band distribution. No extraocular muscle restriction or diplopia, enophthalmos, malocclusion, or midfacial flattening were noted. Goldmann visual field examination demonstrated complete bitemporal hemianopsia (Fig. 4).

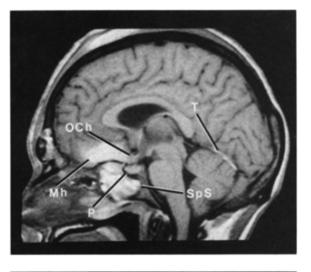
## Case 2

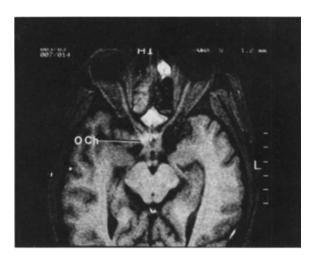
A 15-year-old boy, who had been an unrestrained driver in a motor vehicle accident, sustained a frontal head impact. When we examined him five days after the injury, he denied any visual problems.

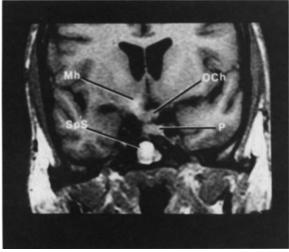
Visual acuity was 20/20 in both eyes with no afferent pupillary defect present. Confrontational visual field demonstrated a bitemporal hemianopsia; slit-lamp examination disclosed



**Fig. 2** (Heinz, Nunery, and Grossman). Left, Axial computed tomography scan slices at the clivus level, and right, at the sella turcica level. A midclival fracture (arrows) longitudinally traverses the clivus, sella turcica floor, and tuberculum sellae. Comminuted maxillary and ethmoid fractures are also present.







bilateral subconjunctival hemorrhages; and indirect ophthalmoscopy demonstrated normal retinal perfusion and no pallor of the optic nerve head. Goldmann visual field examination demonstrated a complete bitemporal hemianopsia (Fig. 5).

Computed tomography (Fig. 6) disclosed a sagittally oriented fracture that traversed the midclivus, through the sella turcica floor and sphenoid sinus, through the planum sphenoidale and dorsum sellae. A swollen optic chiasm was seen on magnetic resonance imaging (Fig. 7).

Traumatic chiasmal syndrome was diagnosed. Cerebrospinal fluid rhinorrhea persisted ten days after injury. The patient developed meningitis, which was culture-positive for *Staphylococcus aureus* and was treated effectively with intravenous antibiotics. Diabetes insipidus was noted and resolved within three weeks after the injury. **Fig. 3** (Heinz, Nunery, and Grossman). Top left, Sagittal; top right, axial; and bottom left, pituitary level coronal magnetic resonance  $T_1$ -weighted scans. The swollen optic chiasm (OCh) is seen just beneath the right-sided hyperintensive hypothalamic hemorrhagic contusion. The hemorrhage methemoglobin (Mh) content is responsible for the  $T_1$  bright signal. The hemorrhage is shown involving the frontal lobe gyrus rectus on the sagittal sequence. There is hemorrhage in the sphenoid sinus (SpS) and along the tentorium (T). The pituitary gland (P) is seen adjacent to the swollen optic chiasm. The infundibular stalk is obscured.

Six months later, his visual acuity remained 20/20 in both eyes. The bitemporal hemianopsia had not improved. Bilateral optic atrophy had developed in a band distribution.

## Discussion

Traumatic chiasmal syndrome is diagnosed by an immediate bitemporal hemianopsia after head trauma. The bitemporal visual field loss may be partial, but complete bitemporal hemianopsia is common.<sup>7,12</sup> Complete bitemporal hemianopsia is rarely seen except after trauma.<sup>12</sup> Compressive lesions of the chiasm more likely produce relative bitemporal hemianopsia.<sup>12</sup>

Laursen,<sup>7</sup> in 1971, reported that 60% of patients with traumatic chiasmal syndrome had complete bitemporal hemianopsias. Other in-

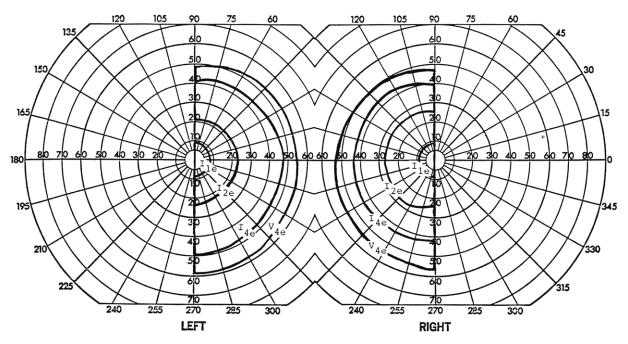


Fig. 4 (Heinz, Nunery, and Grossman). Complete bitemporal hemianopsia. Visual acuity is 20/20 in both eyes.

vestigators<sup>6</sup> had similar findings. Both our patients had complete bitemporal hemianopsias.

The combination of optic neuropathy and contralateral chiasmal injury was noted by Duke-Elder and others.<sup>4,6,7,13</sup> One of our patients (Case 1) initially had a complete right temporal hemianopsia and left optic neuropathy with a small nasal island of vision. Over the next three months, the optic neuropathy completely resolved, disclosing complete bitemporal hemianopsia.

Traumatic chiasmal syndrome may be noted either in isolation or associated with other neurologic signs and symptoms. Associated find-

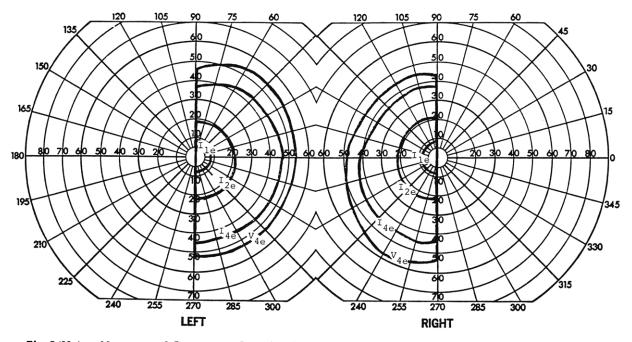
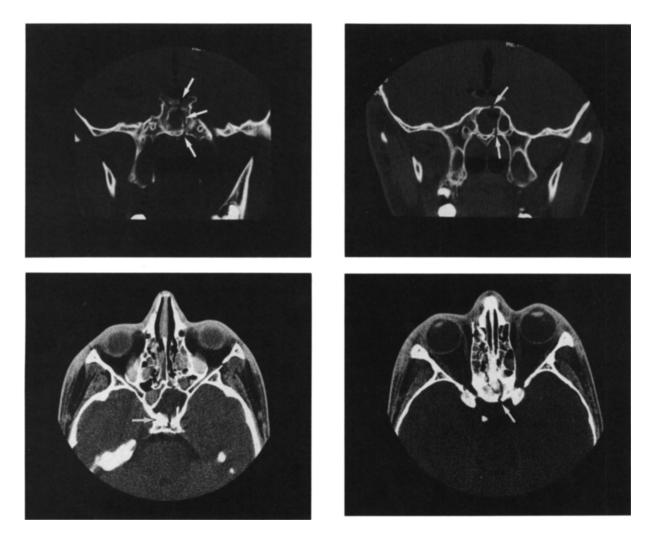


Fig. 5 (Heinz, Nunery, and Grossman). Complete bitemporal hemianopsia. Visual acuity is 20/20 in both eyes.



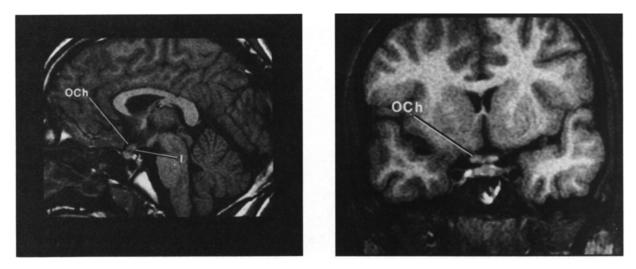
**Fig. 6** (Heinz, Nunery, and Grossman). Top left, Coronal clivus level; top right, sella turcica-sphenoid sinus level; bottom left, axial sella turcica floor; and bottom right, planum sphenoidal level computed tomographic scans. The fracture traverses the midclivus through the sella turcica floor and sphenoid sinus through the planum sphenoid (arrows). The optic canals are not involved.

ings result from damage to structures in the region of the anterior cranial fossa or base of the skull. These include diabetes insipidus, anosmia, cranial nerve defects (II, III, IV, V, VI, VII), deafness (VIII), cerebrospinal fluid rhinorrhea and otorrhea, carotid aneurysm, carotid cavernous fistula, meningitis, panhypopituitarism, intrasellar hematoma, and pneumatocele.<sup>6</sup>

Diabetes insipidus occurred in both our cases and resolved within three weeks. Similarly, seven of the 11 patients with traumatic chiasmal syndrome described by Savino, Claser, and Schatz<sup>6</sup> developed diabetes insipidus, which resolved spontaneously. Sensorineural hearing loss from eighth cranial nerve injury was found in our first case, whereas cerebrospinal fluid leak and meningitis occurred in the second case.

Traumatic chiasmal syndrome may also be associated with cranial fractures and prolonged loss of consciousness.<sup>3,7</sup> Altered mental status may delay recognition of the bitemporal hemianopsia, especially when the patient retains central vision.<sup>4</sup> Neither of our patients complained of visual field defects until it was demonstrated on examination, although the first patient (Case 1) recognized his neuropathic visual loss in the left eye.

The cause of traumatic chiasmal syndrome has inspired speculation. The possibility of chiasmal infarction as a cause of traumatic bitem-



**Fig. 7** (Heinz, Nunery, and Grossman). Left, Sagittal; and right, pituitary level coronal magnetic resonance scans. The optic chiasm (OCh) is swollen and is seen closely abutting the infundibular stalk (I) and the sagittal images.

poral hemianopsia<sup>4,5</sup> became less attractive when the rich anastomotic and multiple blood supply to the chiasm was demonstrated.<sup>14</sup>

Intrachiasmal compressive hemorrhage damaging chiasmal crossing fibers within the noncompliant dural covering<sup>6,7</sup> may also be unlikely in view of the relative sparing of uncrossed fibers, as seen in most patients with traumatic chiasmal syndrome. Crompton<sup>15</sup> performed autopsies on 84 patients with histories of anterior visual pathway injury, and found that of the 16 patients with chiasmal injury, none had hemorrhages in the chiasm consistent with compression or loss of vision. They attributed vision loss in these patients to shearing lesions of the nerve. Intrachiasmal hemorrhage could not be documented on computed tomography or magnetic resonance imaging in either patient, although hemorrhage was seen in adjacent structures (Fig. 3).

Sagittal tearing or stretching of the chiasm has been demonstrated at autopsy, and has long been held as a possible cause for traumatic chiasmal syndrome.<sup>4,8</sup> Logan and Gordon<sup>16</sup> described a patient with traumatic chiasmal syndrome and facial and basilar skull fractures. On craniotomy, they noted a fracture of the planum sphenoid and a displaced tuberculum sellae with resultant stretching of the chiasm. They concluded that the sudden stretching of the chiasm may have caused the chiasmal injury.

Coppez<sup>9</sup> and Osterberg<sup>10</sup> produced sagittal tearing of the chiasm with frontal head injury in cadaver studies. Gurdjian and Webster<sup>11</sup> demonstrated the force of a frontal blow is transmitted to the spheno-orbital roof to the base of the skull in cadavers. This force then causes separation of the skull, in the midline, in a bilaterally outward direction with resultant fracturing of the clivus and sella turcica. Gurdjian and Webster<sup>11</sup> concluded that these shearing forces could be transmitted to the chiasm, resulting in preferential stretch injury to the crossing fibers. Rand<sup>8</sup> emphasized that with a separation of the optic foramina, the optic chiasmal fibers, being weaker than the optic nerves, tear first.

In both of our cases, the precipitating trauma was blunt frontal head trauma. This mode of injury is consistent with Gurdjian and Webster's theory on chiasmal injury<sup>11</sup> and is found in 73% to 96% of traumatic chiasmal syndrome patients.<sup>3,6,17</sup>

Computed tomography (Figs. 2 and 6) in both our cases demonstrated sagittal midline fractures of the clivus and sella turcica, consistent with a midline separation of the skull. The ability of the clivus to separate widely at impact and then recoil is illustrated by the occasional occurrence of the vertebral or basilar arteries becoming trapped in these types of fractures, resulting in death.<sup>18-20</sup> In 1988, Joslyn, Mrivis, and Markowitz<sup>21</sup> published five reports of sagittal clivus fractures. They found no previous reports demonstrating computed tomography findings of these fractures. Four of five patients in their series died within 30 days of the injury.

Our two cases demonstrate the sagittal nature of the clivus and sella turcica fractures in asso-

ciation with complete bitemporal hemianopsias, and illustrate the computed tomographic and magnetic resonance imaging findings associated with the fractures. Our cases suggest tearing or stretching with microtearing of the chiasmal crossing fibers as the mechanism of injury. The magnetic resonance (Figs. 3 and 7) imaging in these cases is not consistent with intrachiasmal hemorrhage being the cause of chiasmal injury.

This syndrome has not previously been associated with a distinctive pattern of basilar skull fracture, even though Gurdjian and Webster's<sup>11</sup> studies predicted a fracture pattern similar to that found in our patients. We believe that with sagittal clivus and sella turcica fractures, the chiasm is torn on a microscopic, if not macroscopic, scale with development of a complete bitemporal hemianopsia. Traumatic chiasmal syndrome, with this unique fracture pattern, may occur more frequently than previously recognized because of an associated decreased level of consciousness and high mortality rate in this injury.

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