Trauma of Optical Nerve Associated to Diencephalic Injury. Case Report

Wilson Enrique Fuentes Galvis1*, Carlos Eduardo Bermúdez Medina1, Carlos Martin Moreno Arias2, Montserrat Carulla Fornaguera2 and Wagib Abwmar Yaber3

1Resident III Year Ophthalmology, San Martin University Foundation, Bogotá Headquarters, Colombia
2Ophthalmologist - Supra - Specialist in Pediatric and Strabismus, Central Police Hospital Bogotá, Colombia
3Neurologist, Central Police Hospital Bogotá, Colombia

*Corresponding Author: Wilson Enrique Fuentes Galvis, Resident III Year Ophthalmology, San Martin University Foundation, Bogotá Headquarters, Colombia.

Received: November 06, 2019; Published: December 10, 2019

Abstract

The trauma of the chiasm and optic nerve is characterized by immediate onset of bitemporal hemianopsia after head trauma. It is a rare clinical entity that occurs after a severe trauma frontofacial. Few survive to the severity of the impact. Patients who survive usually have suffered injuries and cranial nerve deficits hypothalamus.

Much of the damage also involves the intracranial optic pathways, damage to the backs of the visual pathway may be underdiagnosed in patients with this type of trauma. The MRI is the key to display lesions mode suprasellar structures.

Keywords: Chiasm and Optic Nerve Magnetic Resonance; Bitemporal Hemianopsia

Introduction

Since the first report by Nieden in 1883, more than 170 cases described in the literature have been recorded [1-11].

The injuries in this pathology are caused by: traffic accidents by 37%, motorcycle accidents 21%, fall from their own height 21%, by bicycle 11%, fall from a horse to 5%, and another 5%. About two thirds of patients with optic chiasma syndrome have skull fractures. Of these, 21% are frontal fractures, 16% basal, and 31% both frontal and basal. Closed head trauma usually occurs in 32% of patients, and most of them have intracranial hemorrhages [12].

Etiology

One of the mechanisms presented is the mechanic, in this there is a stretch and tear of the optic nerve and the chiasma which is associated with compression by bone fragment. They are often associated with tearing of blood vessels, hemorrhage and causing secondary ischemic damage. The visual deficit is instantaneous and irreversible, due to an axonal lesion that can be focal or diffuse [13].

The other mechanism is contusion necrosis that occurs due to kickback that occurs in the face of the fracture. This contusion necrosis seems to affect the central chiasmatic fibers, while hemorrhage and compression damage mainly affects the lateral and inferior fibers of the chiasma [1].

Trauma of Optical Nerve Associated to Diencephalic Injury. Case Report

Clinic

In most patients their visual acuity is affected, bitemporal hemianopia is found in the visual fields [12], the examination of the optic discs shows temporal pallor. There are some associated deficits such as: alteration of the cranial nerves, anosmia, ocular motility defects, deafness, diabetes insipidus, rhinorrhea, cavernous carotid fistula, carotid aneurysm, meningitis, panhypopituitarism, intrasellar hematoma, Pneumatocoele [2].

Oculomotor nerve deficit is common, third and sixth cranial nerve paralysis was present in 10 of the 19 cases in the study: Traumatic chiasmal syndrome: a series of 19 patients [12].

Trochlear nerve lesions occurred in four patients [12], in due course, 21% of the patients in the study required surgical correction of strabismus.

In the study: Traumatic chiasmal syndrome: a series of 19 patients. 12 was found in one of the cases, a severe basal fracture producing a sixth bilateral cranial nerve alteration, in the same way paralysis of the seventh and eighth cranial nerves.

Within other clinical associations we find the involvement of the interstitial nucleus of Cajal and adjacent prectal nuclei, producing nystagmus in See-Saw [14,15]. It is also common to find the development of a cavernous carotid fistula associated with pulsatile proptosis.

As for the endocrine abnormalities that may occur, diabetes insipidus occurs in 30% of patients with chiasma and optic nerve trauma [5]. These patients experience polyuria, and often show high plasma osmolarity with elevated sodium in plasma.

In addition to diabetes insipidus, patients develop impaired pituitary function, altering cortisol and thyroxine levels.

Diagnosis

It is very important to perform a complete physical examination of visual acuity, ocular motility, evaluation of eye reflexes, evaluation of optical discs, campimetric studies. Magnetic resonance imaging is very useful for identifying alterations at the level of the chiasma and optic nerve, as well as associated brain lesions. During the evolution of the clinical picture, it is vitally important to rely on laboratories that provide us with information on probable endocrine alterations.

Treatment

As for treatment, most patients require neurosurgical management, as for ophthalmological management, emphasis is placed on the management of strabismus and nystagmus.

In the Traumatic chiasmal syndrome: a series of 19 patients12 study, it was found that 4 of the 19 cases required strabismus surgery.

Clinical Case

A 29-year-old male patient, from Tarqui- Huila, who consulted for a traffic accident as a motorcycle driver. Admitted to the emergency department with diagnoses: Severe craniocerebral trauma, bilateral malar fracture and mandibular fracture.

When assessed by ophthalmology, the external examination was found: mild left palpebral ptosis, bimemoral hemianopia confrontation campimetry, visual acuity in right eye 20/140 and left eye 20/200 that do not correct with pinhole.

Pupillary reflexes: with afferent pupillary defect. Eye motility: vertical nystagmus in both eyes, exotropia of 40 diopters in the left eye, biomicroscopy: normal.

On examination of the fundus: temporary pallor of 25% in the right eye and 50% in the left eye, rest of the normal fundus.

The following diagnostic impressions are made: Sequelae of severe craniocerebral and craniocerebral trauma, acquired vertical nystagmus of left predominance, left exotropia of 40 diopters, Paralysis of the third left cranial nerve, traumatic optic neuropathy, contusion of optic chiasma.

Computerized visual fields are performed finding congruent bitemporal hemianopia that suggests chiasmatic lesion. Face tomography is performed with sagittal and coronal reconstruction in 3 dimensions finding left frontal fracture, left maxillary fracture, roof fracture and left orbital floor, frontal sinus fracture and left maxilla. Fracture of the minor wing of the sphenoid is evident with bone fragments that move fracture over the left optic nerve, observing the collapsed optic nerve, pinched at the apex of the orbit. MRI of the brain is performed, finding in T2 large left frontal cystic encephalomalacic area that extends basally to both sides compromising optic thalamus and midbrain, compatible with traumatic injury in the basal frontal region. (commitment III ventricle). Assessed by neurosurgery, he interrogates rhinolychia, finding in cisternotac bone defect of continuity in the screened lamina of the ethmoid, they diagnose traumatic optic neuropathy, orbital trauma and central nystagmus.

Maxillofacial performs fracture fixation. It is subsequently valued by ophthalmology and strabismus finding.

<table>
<thead>
<tr>
<th>Physical Exam</th>
<th>Right eye</th>
<th>Left eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVSC</td>
<td>20/40</td>
<td>20/200</td>
</tr>
<tr>
<td>Refraction</td>
<td>+1.50(-0.25) x 0</td>
<td>+2.50(-2.00) x 0</td>
</tr>
<tr>
<td>Subjective</td>
<td>+1.00 (-0.50) x 0 20/20</td>
<td>+1.00(-3.00) x 0 20/70</td>
</tr>
<tr>
<td>PPM</td>
<td>Nystagmus</td>
<td>XTI 40 DP Nistagmo</td>
</tr>
<tr>
<td>Cover test</td>
<td></td>
<td>XTI 40 DP</td>
</tr>
<tr>
<td>Deductions/Versions</td>
<td>Unrestricted</td>
<td>Restriction Adduction</td>
</tr>
<tr>
<td>Pupil reflexes</td>
<td>Dpa</td>
<td>Dpa</td>
</tr>
<tr>
<td>Eyelids</td>
<td>Normal</td>
<td>Ptosis Leve</td>
</tr>
<tr>
<td>Eye back</td>
<td>Pallor Temporal Nerve Optical</td>
<td>Pallor Temporal Nerve Optical</td>
</tr>
</tbody>
</table>

Management begins with botulinum toxin in all extra-ocular muscles left eye, finding poor therapeutic response. A table of measures is carried out evidencing incompetent deviation.

Subsequently, surgery is carried out and retro-insertion of the left lateral rectus of 10 mm is performed, residual exotropia was found in the immediate postoperative period.

It is assessed by neurosurgery who monitors with MRI and pituitary hormone titration, finding clinically anosmia and polyuria and alterations levels of cortisol and thyroxine (T3-T4).

Discussion

Chiasma and optic nerve trauma has been cited as a cause of disability visual in 0.3% of head injuries [5], clinically the Bitemporal hemianopia is absolute, unlike compression, the syndromes chiasmatic and other inflammatory, which usually cause defects bitemporal incomplete [8].

In this case report, we found a patient with oculomotor nerve disorders and diencephalic disorder due to nystagmus and endocrinological involvement (anosmia and diabetes insipidus). These findings are consistent with other reports [2,4].

Figure 1: Computerized visual fields both eyes.

Figure 2: TAC face with sagittal and coronal reconstruction in 3 dimensions.
Figure 3: Bone window CT.

Figure 4: a: TAC, axial section. Right and left maxillary sinus fracture. b: TAC, axial section. Fracture minor wing of the left sphenoid, with optic nerve deformity.

Figure 5: CT scan, coronal section. Sphenoid sinus fracture, left optic nerve collapse.

Figure 6: Magnetic resonance imaging (MRI) of simple brain, coronal section T2. Wide left frontal encephalomalacia area that extends basally both sides.

Figure 7: Brain MRI, T2 sagittal section. Left frontal hyperintense area that extends basally and compromises the optic thalamus and midbrain. Commitment of the III ventricle.

Figure 8: Look positions.

Figure 9: a: Residual left exotropia. b: Residual left exotropia measured with prisms.
**Figure 10:** MRI, T2 axial section, hyperintense optic chiasma lesion.

**Figure 11:** MRI, T2 axial section, intermesencephalic lesion that extends to the Silvio aqueduct.
Despite having managed for nystagmus with botulinum toxin, it is not corrected due to brain damage.

**Conclusion**

Bitemporal hemianopia after craniocerebral trauma is the main sign of chiasmatic alteration. Magnetic resonance imaging is the most useful imaging study. Endocrine and ocular motility deficits, in particular, often require continuous review.

**Bibliography**


**Volume 11 Issue 1 January 2020**

©All rights reserved by Wilson Enrique Fuentes Galvis, et al.