Asymmetrical bilateral optic neuropathy. Case report

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Abstract

Background: Diagnosis of traumatic optic neuropathy is difficult during primary care as a result of the absence of optic disk changes or coexisting injuries that may be more apparent. Although there are functional signs that suggest its presence, the lack of a comparison parameter may further prevent its detection. A case of bilateral traumatic optic neuropathy is analyzed to emphasize the need for functional evaluation in the injured eye.

Clinical case: A 34-year-old male presented with severe head trauma, loss of consciousness and type III Lefort facial fracture as a result of a vehicular accident. Seventy two hours after the accident, the left eye had only light perception and an afferent pupillary defect. The right eye had visual deficiency not explained by anterior segment injuries. Therapy was aimed at the left eye and, during follow-up, involvement of the right eye was identified very late. The patient's evolution is analyzed, as well as the causes that prevented early detection of traumatic optic neuropathy in the right eye.

Conclusions: Functional evaluation must be used to search for traumatic optic neuropathy in every injured eye. The absence of ocular fundus abnormalities does not rule out the disease, especially when visual deficiency exists. Detection of patients with a positive pupil during initial evaluation of trauma is required in order to refer the patient in a timely manner to the ophthalmologist. The specialist should identify atypical cases and therapy should be initiated as early as possible.

Key words: ocular trauma, optic atrophy, traumatic optic neuropathy.

Introduction

Traumatic optic neuropathy is defined as a lesion of the optic nerve secondary to trauma.1,2 It may cause posttraumatic amaurosis, be associated with cranial and facial trauma and be overlooked. Its incidence is 2-5% in patients with facial trauma.1,2

Its most common form is indirect damage to the optic nerve, which affects between 0.5 and 5% of all cases of closed head trauma.3,4 It has been described that the most common location of the indirect lesion of the optic nerve is the optic canal.5

Although the most common form of presentation is unilateral, the bilateral form may imply greater difficulty for its detection and timely treatment.

The objective of this report was to analyze the case of a patient with traumatic optic neuropathy with bilateral asymmetric involvement in order to emphasize the need to functionally evaluate the injured eyes.

Clinical case

We present the case of a 34-year-old male with a history of cranial trauma and loss of consciousness as a result of an automobile accident. The following abnormalities were identified at primary care: wound in the right nasolabial sulcus and left eyelid, facial instability in the frontonasal junction and right orbit, and absence of the left papillary response. A severe craniofacial trauma with a type III Lefort facial fracture was diagnosed. Antisepsis and closure of the facial wounds was performed and the patient was referred to a tertiary level hospital.

The hospital emergency department to which the patient was referred received him 24 h after the accident. The patient received a Glasgow coma scale score of 15, and generalized facial edema, otorrhagia, nasal and mandibular instability and plegia of the left eye were identified. Fracture of both orbits was identified with a Waters radiographic projection, and a type III Lefort facial...
Fracture was confirmed. To rule out the presence of a cerebrospinal fluid (CSF) fistula, evaluation from Maxillofacial Surgery and Neurosurgery was requested.

The Maxillofacial Surgery Department found the patient to have a “dish face” sign, interciliary and bilateral malar emphysema, collapse of the nasal bridge and floating jaw. X-rays demonstrated a right temporal fracture and displacement of the frontomalar suture.

The Neurosurgery Service confirmed the diagnosis of CSF fistula, found no neurological compromise and stated that it was not possible to assess the fundus of the eye, pupillary reflexes or cranial nerves except V, which was “preserved.” On CT scan, a left temporal fracture was identified and the patient was hospitalized for 48 h surveillance because of the fistula and fracture of the anterior floor.

During his hospital stay, at 72 h since the accident occurred, it was identified that the left eye only perceived light and had limitation of eye movements in supraduction and adduction. Seven days after the accident, progressive decrease in visual acuity in the left eye was reported, and an ophthalmological consultation was requested.

The Ophthalmology Service found visual acuity in the right eye of finger counting at 1.5 m without improvement. The left eye had no light perception and had an afferent pupillary defect.

Intraocular pressure was 12 mmHg in the right eye and 10 mmHg in the left. There was a hypotropia of the left eye with limited mobility for supraduction, abduction, adduction and infraduction. There was a left predominant bilateral chemosis and left corneal de-epithelization. No other alterations in the anterior ocular segment were reported.

The fundus of the right eye was normal. In the left eye there was papillary hyperemia, intra- and preretinal hemorrhage, and retinal edema in the papillomacular bundle. In the CT scan, compression of the left papillomacular bundle was found (Figure 1).

Diagnosis of traumatic left optic neuropathy was made. The Neurology Service scheduled the patient for an optic nerve decompression, which was canceled because it was believed that the functional prognosis was poor. The patient was intervened for rigid internal fixation of the facial fractures.

During follow-up, the patient developed visual loss in the right eye, the best corrected acuity in this eye was 20/40, and the left eye had no light perception. There was papillary pallor in the fundus of the right eye, papillary pallor on the left, and gliosis over the temporal vessels, which partially included the papillomacular bundle (Figure 2). Right eye campimetry identified a residual island of the central visual field (Figure 3).

With the data of the latest examination, diagnosis of traumatic posterior optic neuropathy of the right eye was made as well as traumatic anterior optic neuropathy of the left eye, with secondary bilateral optic atrophy. The best optical correction was adapted for the visual rehabilitation of the right eye.

**Discussion**

Traumatic optic neuropathy is not often reported in ocular trauma (0.5-5%), but it may cause blindness in structurally stable eyes. If detected early, the prognosis improves as the damage may be limited and even reverted. Data that suggest the existence of an optic neuropathy are reduced visual acuity, visual field defects, dyschromatopsia and afferent pupillary defect.

With direct trauma to the nerve there is edema of the optic disc, hemorrhage or avulsion of the optic nerve; with indirect trauma the disc may be normal at the beginning and develop progressive pallor until damage is irreversible.

Traumatic optic neuropathy affects vision to varying degrees, but when it is unilateral there is always an afferent pupillary defect of the affected eye, which occurs if the magnitude of the signal transmitted by the affected optic nerve is less than that of the contralateral nerve.

**Figure 1.** Facial and anterior cranial floor fractures; compression of the optic nerve due to displacement and bone fragment in the left optic canal. Computerized axial tomography: right side of figure shows the patient's facial fractures; left side of figure shows the compression zone of the optic nerve due to displacement of bone fragments to the optic canal.

**Figure 2.** Bilateral traumatic optic neuropathy: papillary pallor in the right eye, papillary pallor and gliosis in the left eye.
A positive pupil indicates damage to the optic nerve if the traumatized eye is "positive pupil." A positive pupil indicates the afferent pupillary defect as a sign with prognostic value for final visual outcome and qualifies its presence in the anterior ocular segment. In patients with ocular trauma and loss of consciousness, a positive pupil may be the only manifestation of optic nerve damage. Although posttraumatic total retinal detachment may also cause it, a positive pupil indicates damage to the optic nerve if the fundus reflex is normal. This damage may also coexist with retinal lesions that are more obvious; therefore, the diagnosis is based on functional findings (positive pupil) rather than anatomic.

The pupil may be negative if there is bilateral symmetrical traumatic optic neuropathy, which is unusual. In these cases there is no afferent pupillary defect because the transmission through both optic nerves is similar.

The patient’s clinical condition was not considered a diagnostic problem in the left eye: he presented with a closed globe trauma caused by a blunt object, with decreased visual acuity, afferent pupillary defect and involvement of the posterior zone. The right eye also presented with closed globe trauma caused by a blunt object; visual acuity was also deficient, there was no afferent pupillary defect and there were lesions in the posterior zone of the eye.

Traumatic optic neuropathy in our patient was evident in the left eye: he presented with a closed globe trauma caused by a blunt object, with decreased visual acuity, afferent pupillary defect and involvement of the posterior zone. The right eye also presented with closed globe trauma caused by a blunt object; visual acuity was also deficient, there was no afferent pupillary defect and there were lesions in the posterior zone of the eye.

The alteration analyzed developed in a patient with facial fractures, but the bilateral condition must be sought in all cases. Identification of traumatic optic neuropathy may also be difficult in patients with only one eye or when the contralateral eye has a condition that restricts vision more than the trauma itself.

Evolution of the disease makes it necessary to add the positive pupil to the two priorities of care in ocular trauma (visual deficiency and open globe).

In conclusion, disability caused by traumatic optic neuropathy may be minimized if the tools for its detection are available. With this in mind, the primary care physician can apply the identification of the priorities of care in ocular trauma, document the damage and refer the patient in a timely manner. The ophthalmologist must implement early treatment and use the appropriate diagnostic resources to identify the disease in atypical cases.
References


