AMAUROSIS SECONDARY TO FRONTAL TRAUMATISM

AMAUROSIS SECUNDARIA A TRAUMATISMOFRONTAL

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ABSTRACT

Case report: We present the case of a patient diagnosed with amaurosis of the right eye secondary to a right frontal contusion. The energy of the impact was projected from the orbital ceiling to the minor wing of the sphenoid bone. This bone was fractured, thus reducing the optic canal diameter and damaging the optic nerve.

Discussion: In our case, we describe a mixed mechanism of injury, that is to say, a frontal contusion indirectly transmitted to the optic canal and a direct lesion of the optic nerve secondary to the movement of the minor wing of the sphenoid bone into the optic canal (Arch Soc Esp Oftalmol 2006; 81: 115-118).

Key words: Amaurosis, traumatism, optic nerve, optic canal.

RESUMEN

Caso clínico: Se describe el caso de un paciente que sufre amaurosis del ojo derecho secundaria a traumatismo contuso frontal derecho. La energía se proyectó por el techo de la órbita hacia el ala menor del esfenoides, cuya fractura redujo el diámetro del canal óptico lesionando el nervio óptico.

Discusión: El mecanismo de producción en el caso clínico que se presenta es mixto al tratarse de un traumatismo frontal cerrado que se ha transmitido indirectamente hacia el canal óptico y una lesión directa del nervio óptico al desplazarse el ala menor del esfenoides en el interior del canal óptico.

Palabras clave: Amaurosis, traumatismo, nervio óptico, canal óptico.
INTRODUCTION

Optic nerve (ON) traumatism cause severe lesions with visual sequels which are usually irreversible. These sequels occur in relationship to high energy traumatisms like traffic accidents or aggressions, with greater prevalence in men under 35 and with a frequency of 5% of closed head traumatisms (1-3).

Traumatisms can be classified on the basis of different criteria. According to the type, they can be direct (penetrating wounds which directly damage the ON), indirect (the lesion is due to a transmission of the impact forces towards the ON) or combined. According to the location of the lesion, they may involve one of the four segments in which the path of the ON is divided: intraocular, intraorbitary, intracanalicular and intracranial (4). The cellular lesion mechanisms can be primary or immediately after the impact, and secondary. The primary mechanisms produce direct axonal lesion, with irreversible necrosis of the ON axons. The secondary mechanisms cause edema of the ON which produces a compartmental syndrome because the optic channel (OC) is a non-extendable structure (4).

CLINICAL CASE

A 76-year old patient who went to the urgency ward due to front right head injury with loss of consciousness caused by a casual fall in her home. Upon recovering consciousness, the patient mentioned loss of vision in her right eye (RE).

The patient’s history included Type 2 Diabetes Mellitus in treatment with oral antidiabetics, and arthrosis. She did not refer other personal or family history of relevance.

In the exploration, she exhibited an infra-ciliary deep, bruising wound and a diffuse right front orbitary hematoma (fig. 1). Visual acuity (VA) included no perception of light with a relative afferent pupillary defect (RAPD) in the RE and of 0.6 in the left eye. The anterior pole was normal as well as the IOP and the eye fundus.

A cranium CAT with orbitary sections was normal and the nuclear magnetic resonance imaging (MRI) exhibited an increase of peri-neural liquid in the sheath of the right ON (figs. 2 and 3).

In view of the persisting symptoms, a sagittal reconstruction of the optic canal of the helicoidal TC was performed, identifying a linear fracture of the right orbit roof extending to the right sphenoid minor wing in the region of the OC, with minimum sinking of the lateral fragment which reduced the internal diameter of the OC (fig. 4 and arrow).
Evoked visual potentials were made utilizing single-eye stimulus (Medelec® Synergy. Oxford. Instruments, England) with Googles at 2 cycles/second, evidencing a large asymmetry in the morphology of responses to stimuli in both eyes, suggesting absence of transmission of visual stimuli in the right ON.

The patient was diagnosed with traumatic optic neuropathy (TON) with combined production mechanism because it was a closed frontal traumatism which was indirectly transmitted to the OC as well as a direct lesion of the ON due to the displacement of the sphenoid minor wing inside the OC.

Treatment was established with mega doses of corticoids (1 g of methylprednisolone followed by 250 mg every 6 hours during 48 hours), without exhibiting improvement.

**DISCUSSION**

This case is infrequent because it is caused by a traumatism of moderate intensity. In addition, this impact has projected its energy through the orbit roof to the sphenoid minor wing, its fracture and displacement towards the OC causing the ON damage.

The lesion occurred in the intra-canalicular segment of the ON in which the peristium of the OC is firmly adhered to the duramater of the ON. This anatomic characteristic makes it very sensitive to indirect transmitted traumatic impacts (4).

In the diagnostic, it is essential to perform imaging tests, mainly CT with study of the orbit and the

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**Fig. 3: MRI with increase of peri-neural liquid. Sagittal section.**

**Fig. 4: CAT scan showing fracture of the sphenoid minor wing in the optic canal (OC). The arrow shows the fracture of the sphenoid minor wing, with its displacement causing reduction in OC diameter.**

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OC. CT is superior to MRI for evaluating bone fractures, although the former provides more information about adjacent soft tissue.

The NOT treatment must be individualized according to the characteristics of each case. In the indirect NOT the therapeutic approach is highly controversial because it has not been possible to demonstrate the benefit of the various treatments. High dosages of corticoids can be utilized, or surgical decompression, or even abstention and observation (4), although the International Optic Nerve Trauma Study concluded that the final VA differs very little with any therapeutic option (5). The case presented here was treated with high dosage of corticoids without improvement.

In conclusion, we must suspect a lesion of the ON in all patients with visual deficit, RAPD and TCE history, even though the usual imaging test yield normal results.

**REFERENCE**