Short communication

Traumatic optic neuropathy: To treat or not to treat? Report of two cases☆,☆☆

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ABSTRACT

Case reports: We present two cases of blunt ocular trauma with clinical findings and complementary tests compatible with traumatic optic neuropathy (TON), and progression to sectorial optic atrophy.

Discussion: TON has an essentially clinical diagnosis, and in the last few years has been treated with high doses of intravenous steroids, based on weak evidence of the benefit of the steroids in cases of medular trauma. Nevertheless, subsequent studies have concluded that there is a relatively high proportion of cases in all series with spontaneous visual recovery, and there was no convincing evidence of a benefit of steroids compared to observation only.

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Neuropatía óptica traumática: ¿tratar o no tratar? A propósito de dos casos

RESUMEN

Casos clínicos: Se presentan dos casos de traumatismo ocular contuso con hallazgos clínicos y pruebas complementarias compatibles con neuropatía óptica traumática (NOT), y evolución a atrofia sectorial de la papila.

DisCUSión: La NOT tiene un diagnóstico primordialmente clínico y se ha tratado en los últimos años con altas dosis de corticoesteroides intravenosos, basándose en una evidencia débil de beneficio de los esteroides en traumatismos medulares. Sin embargo, estudios posteriores llevan a concluir que existe en todas las series una proporción relativamente alta...
**Introduction**

Traumatic Optic Neuropathy (NOT) is a term that refers to any optic nerve injury secondary to penetration or concussive cranial traumatism. Diagnostic of NOT is not easy due to the frequent presence of brain damage, while confusing, incomplete literature does not render matters easier. This paper presents 2 cases of NOT with a similar and positive clinical evolution derived from different therapeutic approaches.

**Case reports**

**Case 1**

A 19-year-old male patient who visited our service after a traffic accident with a concussive traumatism over the left eye (LE). Baseline visual acuity (VA) was finger counting and in addition the patient exhibited hyphema, central corneal edema and ruptured iris sphincter. The retina was within its plane and did not exhibit details. After resolving the corneal edema and the anterior chamber inflammation caused by bleeding, residual post-traumatic midriasis was observed (Fig. 1). Funduscopy and macular optic coherence tomography did not reveal pathological findings (Fig. 2), with VA being 0.5. Computerized campimetry revealed a small corneal defect. The Ishihara test in LE was 15/20. In the presence of a clinical diagnostic compatible with retrobulbar NOT, it was decided to keep the patient under observation. Decimal VA increased to 0.9, dyschromatopsia disappeared, residual minimum central scotoma and partial optic atrophy remained.

**Case 2**

A 40-year-old male patient reported with concussion trauma in LE due to tennis size ball, with VA of 0.6, a small level of hyphema and nasal papilla edema revealed by funduscopy (Fig. 3). The Ishihara test gave a result of 18/20 while campimetry uncovered an inferior altitudinal defect (Fig. 4). It was decided to treat with methyl prednisolone at a dose of 250 mg every 6 h IV during 3 days as well as 80 mg of oral prednisone per day the following week and alternating for 3 subsequent days. The patient evolved towards the normalization of the anterior segment and resolution of the optic nerve edema,
with a final decimal VA of 0.8 and papillary sector atrophy (Figs. 5 and 6).

Discussion

With a prevalence of 2–5% in facial trauma cases, NOT is an infrequent cause of visual loss. It generally occurs in the context of cranium traumatism due to traffic accidents (50%), falls (25%) or sports accidents (10%). It is described mostly in males with a mean age of 30 years.

Optic nerve damage can be direct or indirect. The direct mechanism comprises nerve avulsion or rupture and orbital hemorrhage or emphysema. Indirect damage is the result of the transmission of compression stress in concussion traumatisms. In general, direct damage is most severe while diagnostic delays are more frequent in the indirect injuries. Another classification is based on the location of the injury, i.e., papillary, intra-orbital, intracanalicular or intracranial.

Clinically, we can find relative afferent pupil defect, VA involvement which can range from slight or nonexistent to lack of light perception, dyschromatopsia, variables defects in campimetry, optic nerve with edema and/or hemorrhage in the anterior optic neuropathies or with normal appearance if the involvement is retrobulbar. The diagnosis, essentially clinical, can be supplemented with imaging tests such as magnetic resonance or computerized tomography. NOT evolves
towards optic atrophy which is partial as in our 2 cases or total in the weeks following the injury.

Therapeutic options involve systemic corticosteroids with variable duration and administration,2 surgical decompression of the optic channel and combination of steroids and surgery, as well as observation of the patient without medical or surgical therapy. In recent years many of these cases have been treated with high doses of intravenous corticosteroids on the basis of the NASCIS II and III studies which in the nineties demonstrated a benefit of steroids over placebo in patients with spinal cord damages under 8 h of evolution.2 Reviews on this topic emphasized a percentage of spontaneous visual recovery in all cohorts, concluding that there is insufficient evidence to state whether neither steroids or optic channel surgery should be considered as gold standard treatments for NOT. The findings by the Steinsapir4 group in 2000 are significant: in rat studies, the group found an exacerbation of axonal loss with corticoids treatment in a non-insignificant and dosage-dependent magnitude. In addition, the first randomized study carried out by Entezari et al.5 in 2007 matched previous studies in that steroids treatment does not improve VA against placebo. However, the initial VA, the improvement thereof in the first 48 h in the severity of the relative afferent pupil defect are established as predictive VA improvement factors.

Our clinical experience in the reported cases point to similar evolutions both in VA and the final funduscopic appearance in the patient treated with methylprednisolone as well as in the one who did not receive systemic medical therapy. These findings match the results of larger studies, which concluded that there is no significant benefit for intervention versus observation, although we consider that each case should be studied individually.

Fig. 5 – Residual partial optic atrophy.

**Fig. 6 – Optic coherence tomography of the retina nervous fiber layer showing loss of superior and nasal fibers in the atrophic papilla (left eye).**

REFERENCES