

BILATERAL SIXTH NERVE AND LEFT THIRD NERVE PALSY AFTER HEAD TRAUMA

PARÁLISIS DEL VI PAR BILATERAL Y III PAR IZQUIERDO POSTRAUMÁTICO

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ABSTRACT

Case report: To describe a case of combined bilateral cranial nerve palsy of traumatic origin. To determine the lesions that produce the symptoms is useful to define the final prognosis and the best treatment.

Discussion: We report the case of a patient who developed a bilateral sixth nerve and left third nerve palsy after head trauma. The underlying lesion was a diffuse axonal injury. After an observation period during which no spontaneous improvement occurred, we administered botulinum toxin with a successful clinical result. Bilateral combined traumatic cranial nerve palsies are rare. When a diffuse axonal injury is present, the chance of spontaneous resolution is poor (*Arch Soc Esp Ophthalmol 2006; 81: 41-44*).

Key words: Oculomotor palsy, sixth nerve palsy, third nerve palsy, head trauma, diffuse axonal injury.

RESUMEN

Caso clínico: Describir un caso de parálisis bilateral combinada de origen traumático. Conocer las lesiones que ocasionan la clínica nos es útil para inferir el pronóstico final y tratamiento más adecuado.

Discusión: Presentamos un paciente con una parálisis del III par izquierdo y VI par bilateral de origen traumático. El daño anatomofuncional era una lesión axonal difusa. Tras un período de observación sin presentar mejoría se realizó una inyección de toxina botulínica con la que mejoró clínicamente. Las lesiones de pares craneales combinadas y bilaterales traumáticas son infrecuentes. Si son por un daño axonal difuso las expectativas empeoran.

Palabras clave: Parálisis oculomotora, parálisis VI par, parálisis III par, trauma craneal, daño axonal difuso.

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INTRODUCTION

The lesions of the sixth cranial pair are very frequent, the most usual cause being trauma (1) due a lesion against the petrous portion of the temporal bone followed by inflammation (otitis, mastoiditis), vascular lesions (ACV) and tumors (cephalic trunk gliomas). Any displacement of the brain can cause a stretching of the nerve and lead to palsy. About 10% of cases present bilateral involvement, among which traumatic etiology is less frequent and comes after tumors, vascular and demyelinating lesions. Associated damages of other cranial pairs are even less frequent.

CASE REPORT

Seventy-two year old male patient suffering serious crano-cephallic trauma due to a traffic accident, exhibiting at admission a score of 8 in the Glasgow coma scale, with serious associated trauma in the thorax and pelvis which required oro-tracheal intubation and thoracic drainage. Cranial CAT evidenced intraventricular bleeding and a slight hemorrhage in the right front parietal sub-arachnoid region, which evolved as subdural hygromas. No lesions were found in the posterior fossa. The facial CAT evidenced multiple fractures of the facial mass, including the roof and medial wall of the left orbit.

When the patient left the ICU and was clinically stable, the ophthalmologic study evidenced a VA of 0.7 in both eyes. The biomicroscopy showed moderate phacosclerosis without the presence of other alterations. The funduscopy evidenced the casual finding of myelinated nervous fibers in both eyes, with normal papilla and without alterations of the posterior pole or peripheral lesions. As regards the intrinsic motility study, anisochoria appeared with midriasis of the left eye. Extrinsic motility showed a complete paralysis of both abductor nerves and the and the common left ocular motor nerve (including phtosis) (fig. 1). Clinically, this expressed as torticollis (fig. 2) as well as diplopia in all positions, thus being incapacitating.

As none of the described lesions radiologically supported the clinical findings or were susceptible to surgical treatment, it was decided to complete the study by means of a cranial MR to find a physiopathological explanation for the clinical findings. Said scan revealed the existence of subcortical pete-



Fig. 1: Detail of the patient. Primary gaze position and versions at diagnosis. Paralysis of the Sixth bilateral pair and Third left pair.

chiae and focal lesions of the corpus callosum, all this compatible with the diagnostic of diffuse axonal damage. No structural lesions were found in the cord of the encephalon (fig. 3 a and b).

After four months of evolution, the patient presented combined paralysis which did not show signs of clinical improvement. As a therapeutic procedure a botulin toxin injection was made (5 UI Botox) in both medial rectae. The clinical improvement was sustained for three months (fig. 4) before the symptoms reappeared.

DISCUSSION

Eye motor paralysis are usually due to direct damage over cranial pairs, encephalon nuclei or,

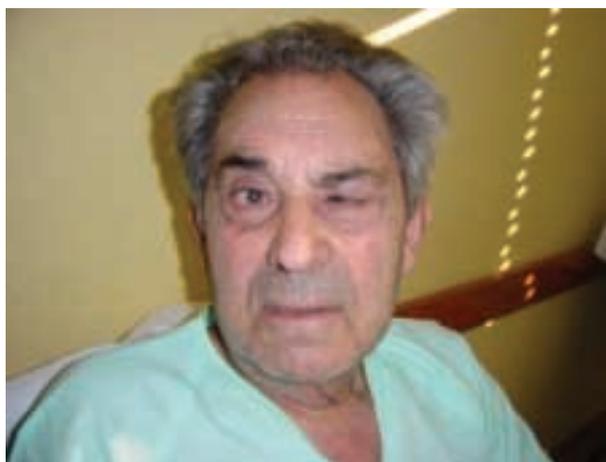


Fig. 2: Basal situation of the patient. A marked compensating torticollis is appreciated.

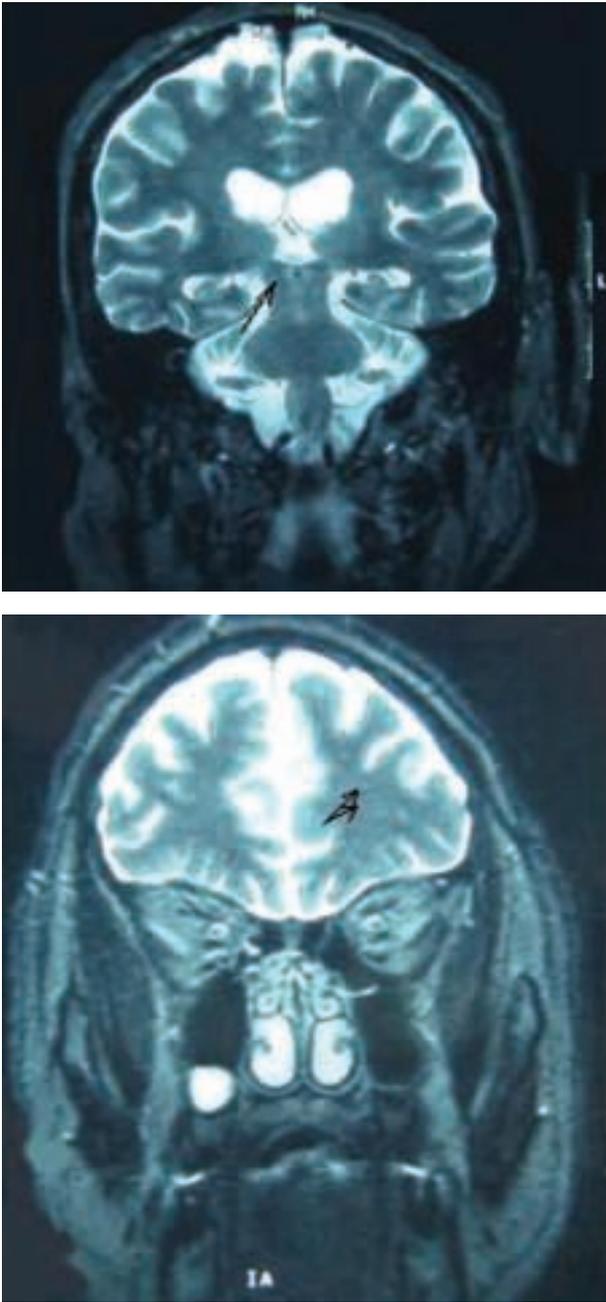


Fig. 3: Pondered crown cranial MR in T2. Hyper-intense bilateral pointy lesions are observed as the arrows show in the mesencephallic periaqueductal location (a) and the subcortical region of both frontal lobes (b). Both lesions indicate diffuse axonal damages.

less frequently, to diffuse axonal damage (2). The latter damage consists in a lesion due to secondary shearing in cranial traumatism, usually with an acceleration-deceleration mechanism. Clinically it



Fig. 4: Detail of the patient condition after two months since the Botox injection in both medial rectae.

appears in patients with a low level of consciousness and multifocal neurological damage. Typically, it manifests in radiological studies as small areas of hemorrhage in the corpus callosum, the dorsal-lateral mesencephalone and the subcortical white substance. The prognosis for these patients is highly variable and depends on the extension of the initial involvement. The neurological damage is usually unrecoverable (3).

Unilateral traumatic cases resolve spontaneously in 72% of cases against 12% of bilateral cases (6-month follow-up period) (4).

As regards treatment of bilateral paralysis of the sixth cranial pair, some authors consider it best to wait 6 months before utilizing non-surgical therapeutic measures such as toxin injection in case a spontaneous improvement appears. In patients who do not improve and present a deviation of >10 prismatic dioptries, surgery is indicated after 6-12 months of the lesion, although lesser delays produce better prognosis. The surgical options are mainly two: retro-insertion of the medial rectum together with resection of the lateral rectum, or substitution techniques with transposition of the vertical muscles. In our case this was not indicated due to the involvement of the Third pair.

The conservative treatments as well as the toxin injection do not seem to have a significant influence on the final recovery (5) although they assist a temporary improvement of symptoms.

Bilateral lesions of cranial pairs due to trauma are infrequent and require the exclusion of possible coincident pathologies.

MR can be decisive to help infer the mechanism which produced the damage and the functional prognosis of the patient. but when diffuse axonal damage has occurred involving neurological deficit recovery can hardly be expected.

The injection of Botox is useful to alleviate symptoms, although a definitive solution usually requires surgical intervention.

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