Traumatic Bilateral Abducens Nerve Palsy

Paralisia Traumática do VI Par Bilateral

Ana Marta[1,2], Sofia Maia[1], Vasco Miranda[1,2], Ricardo Parreira[1], Pedro Menéres[1,2]

[1] Centro Hospitalar Universitário do Porto EPE, Porto, Portugal
[2] Instituto de Ciências Biomédicas Abel Salazar, Porto, Portugal

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ABSTRACT

INTRODUCTION: Our purpose was to report a case of severe traumatic bilateral sixth nerve palsy.

We describe a clinical case documented with photographs on different gazes; Hess screen charts and Goldmann visual field tests over time.

CASE REPORT: A 42-year-old male was involved in a high-energy car crash (240 km/h). Nine days after, when transferred to our hospital, he presented with severe bilateral sixth nerve palsy, with limitation in the abduction of right and left eye, without crossing the midline. He was submitted to 3 rounds of medial rectus botulinum toxin (BTX) injections. The first done 1 month and the last 6 months after the accident. He achieved good results with no diplopia and no abduction limitations.

CONCLUSION: Although the bilateral involvement and the severity of the abduction deficit in sixth nerve palsy are independently associated with a poor outcome, our clinical case had a good outcome with total and definitive recuperation of severe bilateral palsy after medial rectus BTX injections started in the acute phase.

KEYWORDS: Abducens Nerve Injury/drug therapy; Botulinum Toxins/therapeutic use.

RESUMO

INTRODUÇÃO: Nosso objetivo é relatar um caso de paralisia traumática grave do VI par bilateral. Descrevemos um caso clínico documentado com fotografias em diferentes posições do olhar; gráficos do ecrã de Hess e testes de campo visual de Goldmann durente o período de seguimento.

RELATO DE CASO: Um homem de 42 anos envolveu-se num acidente de carro de alta energia (240 km/h). Nove dias depois, quando foi transferido para nosso hospital, apresentou quadro de paralisia grave do sexto nervo bilateral, com limitação na abdução do olho direito e esquerdo, sem cruzar a linha média. Ele foi submetido a 3 injeções de toxina botulínica do reto medial (BTX). O primeiro feito 1 mês e as últimas 6 meses após o acidente. O doente obteve bons resultados sem diplopia e sem limitações de abdução.

CONCLUSÃO: Embora o envolvimento bilateral e a gravidade do déficit de abdução na paralisia do VI par estejam independentemente associados a um mau prognóstico, o caso clínico apresentado obteve um bom desfecho com recuperação total e definitiva da paralisia bilateral grave após injeções de BTX no reto medial iniciadas na fase aguda.

PALAVRAS-CHAVE: Toxinas Botulinicas/uso terapêutico; Traumatismo do Nervo Abducente/tratamento farmacológico.

INTRODUCTION

The sixth cranial nerve (abducens nerve) is responsible for the innervation of the lateral rectus muscle, whose action consists of the abduction of the eyeball. Paresis of the VI cranial nerve is common in clinical practice, especially in an emergency setting, and according to most series, it is the most common paresis of the three oculomotor nerves,[1] probably due to its extremely long intracranial course,[2] its angulation at the base of the skull (weakest point) and its vulnerability in situations of increased intracranial pressure. The lesion of this nerve can occur anywhere along its course between the sixth nerve nucleus in the dorsal pons and the lateral rectus muscle within the orbit.[3]

The etiology of the sixth cranial nerve paresis series available in the literature is variable, in particular that of isolated cases. Regarding traumatic causes, although unilateral sixth cranial nerve palsy is reported to occur in 1% to 2.7% of all head traumas, bilateral sixth cranial nerve palsy after head trauma is an extremely rare condition[4] and is typically associated with an additional intracranial or cervical spine injury. Probably, bilateral cases are rare because the force required is usually incompatible with survival.[5] The purpose of this article is to report a case of traumatic isolated bilateral sixth nerve palsy with a good outcome.

CASE REPORT

A 42-year-old male was admitted to a hospital after a car accident associated with a high-energy impact (240 km/h) during a race car practice (Day 0 – D0). On admission, he presented with headache, diplopia, dysarthria, dysphagia, and facial paresis. Computed tomography (CT) of the head showed acute blood contamination of the 4th ventricle and peritroncular cisterns, with no more anomalies. Magnetic resonance imaging (MRI) of the brain showed posterior cerebral hemispheric laminar subdural hematoma, intraventricular hemorrhage (4th ventricle), and subarachnoid hemorrhage, but excluded diffuse axonal lesions. CT angiography of the carotid and CT of the chest, abdomen, and pelvis showed no acute injuries.

After 9 days of admission (D9), he was transferred to our hospital with clinical improvement, except for the headache and diplopia. The patient was prescribed with alternating occlusion. On examination, his visual acuity was normal as was the anterior segment, intraocular pressure, and fundus examination. He had a bilateral sixth nerve palsy with mild lateral torticollis when fixing with the right eye (RE) and more significant when fixing with the left eye (LE) and esotropia. There was a limitation in the abduction of RE and LE, without crossing the midline (Fig. 1A). The diplopia was nullified with 85 diopter base-out prism in primary position for distance with dominant eye fixation. For near distance, he had no diplopia up to 4 cm. At that time, the Hess screen was not recordable, given the severity of the abduction limitation. (Fig. 2A). He maintained alternating occlusion and, 1 month after the accident (Month 1 - M1), he was treated with the first injection botulinum toxin (BTX) on the right medial rectus (RMR) and left medial rectus (LMR), 5 units (U) in each muscle. One month after the 1st injection (M2) (Fig. 1B), the patient had ptosis and subconjunctival hemorrhage at RE, Lang 550′ at 30 cm, no torticollis with RE fixating, limitation in the abduction of RE and LE crossing midline slightly and without diplopia up to 30 cm. Two months after the first injection (M3), he clinically worsened with torticollis again, limitation in the abduction of RE and LE without crossing midline again and without diplopia only up to 10 cm. For this reason, the patient was submitted to the second injection of BTX on the RMR (5U) and LMR (5U). One month after the second injection (M4), the patient had mild ptosis on the LE, no torticollis, limitation in the abduction of the RE (without cross midline) and a slight limitation in the abduction of the LE (crossing midline), and no diplopia up to 100 cm. Nevertheless, binocular visual fields showed important diplopia areas (Fig. 3A). 2 months after the second injection (M5), he clinically worsened with torticollis again, limitation in abduction (Fig. 2B) of the RE (crossing only 2 mm beyond midline), and without diplopia up to 100 cm, leading to a third injection of BTX on RMR (5U) and LMR (2.5 U). One month after the third injection (M7), he presented slight ptosis on the RE, no torticollis, no important limitation in the abduction, and no diplopia in the primary position. The Hess screen charts 3 months after third BTX (M9) (Fig .2C) and the Goldmann visual field tests 3 months (Fig. 3B)

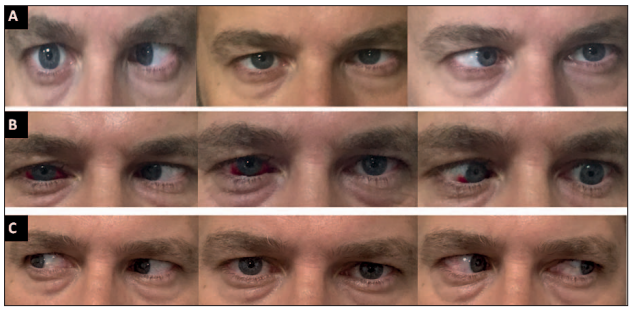

Figure 1. Photographs on right gaze, primary position and left gaze, before 1st injection of botulinum toxin (BTX) (September 2017) (A), at 1 month after 1st BTX injection (November 2017) (B) and at 14 months after 3rd BTX injection (May 2019) (C).

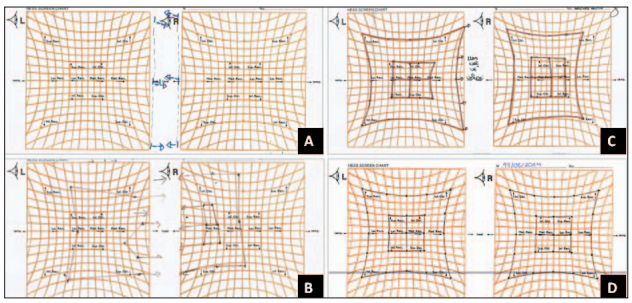

Figure 2. Hess screen charts before 1st injection of botulinum toxin (BTX) (September 2017) (A), 2 months after 2nd BTX (February 2018) (B), 3 months after 3rd BTX (June 2018) (C), 14 months after 3rd BTX (May 2019) (D).

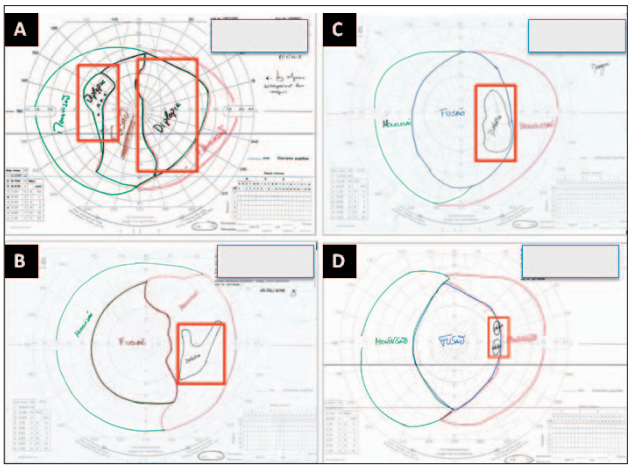

Figure 3. Goldmann visual field tests 1 month after 2nd injection of botulinum toxin (BTX) (January 2018) (A), 3 months after 3rd BTX (June 2018) (B), 5 months after 3rd BTX (August 2018) (C) and 14 months after 3rd BTX (May 2019) (D). Diplopia areas are surrounded by red rectangles.

and 5 months after third BTX (Fig. 3C) showed a favourable evolution. At 14 months after third injection (M20), he maintained the clinical improvement (Fig. 1C), without abduction limitation on Hess screen chart (Fig. 2D), and without diplopia (Fig. 3D). He returned to race at the top world division he was previously racing on.

DISCUSSION

Traumatic sixth nerve palsy may resolve spontaneously, but recovery is more common in unilateral cases.[6] The severity of the abduction deficit and bilateral involvement, like in our case, are independently associated with poorer outcomes, with or without conservative therapy or acute injection of BTX.[7]

BTX can be used in paralytic strabismus. In the acute phase in adults, although it had no effect on spontaneous recovery in the nerve palsy, it can rapidly relieve symptomatic diplopia, like we partially achieved in our clinical case, and might also prevent contracture of the unopposed medial rectus. More recently, Hung *et al*[8] showed that BTX can facilitate recovery of acute traumatic complete sixth nerve palsy in severely injured patients. In a pediatric population, it can prevent loss of binocularity and development of suppression and allow amblyopia treatment in large angles.[9] In a chronic phase, if the patient feels better after BTX, we can do it regularly. If BTX is not enough, strabismus surgery might be needed in the chronic phase.[10] However, in this case, as we noted clinical improvement after each injection, we did 3 BTX injections, with a good clinical outcome after the third BTX, performed 6 months after the accident. The patient did not require additional treatment, kept improving, obtaining a definitive recovery only with repeat BTX injections, and was able to resume his race car career without limitations.

It is known that the medial rectus contracture prevents recovery of the abducents palsy, particularly in long standing cases or when recovery takes more time. In this particular case, we believe that BTX played a role not only in velocity of recovery, but also in final functional status. By preventing the contracture of the medial rectus that would naturally take place in such a long recovery, BTX allowed the progressively greater strength of lateral rectus to efficiently show itself, instead of being masked by the contracted medial rectus.

Is there any decrease in the strength of the medial rectus after the end of BTX temporary effect that may contribute to the balance of oculomotor control system? It is known that the longer the period of muscle denervation, the higher the likelihood of muscle atrophy.[11] However, atrophy of the fibers of extraocular muscles after BTX has not been seen in a significant degree.[12] During the end of the acute phase of BTX muscle paresis, oculomotor adaptive mechanisms may act for the maintenance of the newfound binocular state by the formation of new neuromuscular junctions, recovery of poisoned ones, or both, resulting in reorganization in the extraocular muscles and a reduction in sustained muscle force after long-term BTX.[13]

We want to emphasize that: recoveries sometimes can surprise us because they are longer than usual; and in these cases, we believe there is a role for repeated BTX. If the patient has increasing functional benefit from BTX, we can consider maintaining BTX treatments, as what happened in our case, having performed the last injection 6 months after the accident with good results.

In conclusion, in cases of severe bilateral palsy, treatment with repeated injections of BTX should be considered, before considering surgery, when there are clinical signs of progressive recovery with the treatments.

RESPONSABILIDADES ÉTICAS

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Corresponding Author/
Autor Correspondente:

Ana Luísa Ambrósio Marta
Department of ophthalmology, Centro Hospitalar e Universitário do Porto
Largo do Prof. Abel Salazar, 4099-001 Porto, Portugal
analuisamarta2@gmail.com

ORCID: 0000-0003-3495-4649