Botulinum toxin and facial palsy. Our experience

María Luisa Navarrete Álvaro, a,*, Josefina Junyent, b and Luisa Torrent c

a Servicio de Otorrinolaringología, Hospital Universitario Vall d’Hebron, Barcelona, Spain
b Servicio de Rehabilitación, Hospital Universitario de Bellvitge, Barcelona, Spain
c Servicio de Rehabilitación, Hospital Universitario Vall d’Hebron, Barcelona, Spain

Received December 9, 2009; accepted February 9, 2010

Abstract

Introduction and objectives: Therapeutic indication of peripheral facial paralysis depends on the degree of nerve injury. Severe facial palsy (electroneuronographic study less than or equal to 10%) leads to healing with sequelae. The sequelae of facial paralysis are contractures, hemifacial spasm and synkinesis. Our purpose was to demonstrate that these patients could benefit from rehabilitation treatment.

Methods: We present a study of 48 patients with severe peripheral facial paralysis. They were treated from the beginning of reinnervation with botulinum toxin and facial exercises according to the Wisconsin School.

Results: The subjective efficacy of rehabilitation is high.

Conclusions: Rehabilitation treatment can inform patients about their chances of recovery, give them control over and quality of facial expression and help to achieve greater facial symmetry. These factors provide better functionality and quality of life.

© 2009 Elsevier España, S.L. All rights reserved.

Keywords

Facial palsy; Rehabilitation treatment; Synkinesis; Botulinum toxin

PALABRAS CLAVE

Parálisis facial; Sincinesias; Tratamiento rehabilitador; Toxina botulínica

Toxina botulínica y parálisis facial. Nuestra experiencia

Resumen

Introducción y objetivos: La indicación terapéutica de la parálisis facial periférica depende del grado de lesión nerviosa. Una parálisis facial severa (electroneuronografía menor o igual al 10%) evoluciona hacia una curación con secuelas. Las secuelas de una parálisis facial son las contracturas, el espasmo hemifacial y las sincinesias. El propósito de este trabajo es demostrar que este tipo de pacientes puede beneficiarse de un tratamiento rehabilitador.

Métodos: Presentamos un estudio con 48 pacientes afectos de parálisis facial periférica severa. Fueron tratados a partir del inicio de la reinervación motora facial mediante ejercicios faciales según la Escuela de Wisconsin y toxina botulínica.

Resultados: La eficacia subjetiva de la rehabilitación es alta.

*Corresponding author.
E-mail address: mlna@telefonica.net (M.L. Navarrete Álvaro).

0001-6519/$ - see front matter © 2009 Elsevier España, S.L. All rights reserved.
Introduction

Severe facial paralysis, that is, those cases whose electroneurographic (ENoG) studies show nerve degeneration greater or equal to 90%, evolve towards a partial cure with sequelae. The sequelae of facial paralysis are contractions, hemifacial spasm and synkinesis. Synkinesis is the most common sequel; it consists of the association of an abnormal involuntary movement accompanying a voluntary movement.

The aim of this study was to observe the aesthetic and functional results in these patients through rehabilitation and use of botulinum toxin, and to show that these patients could benefit from physiotherapy treatment.

The general pattern of rehabilitation in facial paralysis includes a general treatment based on the functional profile. This includes techniques that facilitate movement, inhibition of abnormal motor patterns and complex movement coordination.

Neuromuscular retraining does not completely restore the function. Consequently, the use of botulinum toxin implements good functional and aesthetic results.

Botulinum toxin decreases aberrant contractions of facial muscles by blocking acetylcholine at the synapse level. It temporarily paralyses the target areas of synkinesis (usually orbicularis oculi and mental) for a period of 4-6 months. Combined with neuromuscular retraining, it offers a “time of opportunity” during which the patient can practice the movement patterns without interference by the synkinesis.

Methods

We studied 48 patients with severe facial paralysis, that is, with more than 90% axonal degeneration in the ENoG study. All the paralyses were forms of Bell palsy. The distribution of the sample by gender was 14 males and 34 females, with a mean age of 48 years.

The patients were received at the facial palsy unit upon onset of paralysis. The examiner assessed the degree of facial involvement using the House-Brackman scale and the Facial Grading System, which measures facial nerve function at rest, with isolated voluntary movements and in relation to synkinesis (Figure 1). We also used a visual analogue scale to assess prospects for recovery, the final aesthetic result and the degree of patient satisfaction. Patients were studied further using our facial paralysis protocol (ENT-otoneurological and electrophysiological study).

During the evolution, the checks were carried out every month, 3 months, 6 months and 12-18 months until recovery. Physiotherapy and treatment with botulinum toxin was planned from the start of nerve reinnervation.

All patients underwent a personalised program of facial exercises according to the facial-muscular scheme (Wisconsin school) (Figure 2). A program of 8 sessions of treatment at the hospital was established, with each session lasting 1 hour and with a complementary home exercise program. Botulinum toxin was indicated in all patients once the beginning of the sequel (synkinesis) was observed both clinically and electrophysiologically. The frequency of administration was every 16 weeks. We used Botox 100 units with 2cc SSF, insulin syringe (1 ml) and a 26G needle. The patients received 2.5 units (0.05 ml) injected at each point in the muscle groups part of the synkinesis (Figure 3), in areas of hypertonia (caused pain) and in healthy hemifacial muscles that caused noticeable asymmetries.

The results were obtained through percentage and averaged calculation.

Discussion

Severe peripheral facial palsy involves an aesthetic problem associated to a functional problem with impaired verbal communication. The consequences of these events can be physical and psychosocial.

The physical consequences are paralysis (hypotonic phase with lower eyelid ectropion) and the hypertonia phase (sustained muscle contraction) with the appearance of aberrant movement or synkinesis. At this point the patient usually complains of stiffness, tension and even pain in the area. It occurs due to a failed reinnervation or reorganisation of the facial nucleus (it does not recognise afferent stimuli from the 5th cranial nerve) creating a distorted response. The clinical manifestations include defects of eye closure and oral-motor impairment (speech, mastication, periodontal problems, etc.). Psychosocial consequences are the poor correlation between the degree of objective dysfunction and psycho-emotional involvement.

Conclusiones: El tratamiento rehabilitador permite informar al paciente sobre sus posibilidades de recuperación, tener un control y calidad de la mimica facial, lograr una mayor simetría facial y con ello dar una mejor funcionalidad y calidad de vida.

© 2009 Elsevier España, S.L. Todos los derechos reservados.
Botulinum toxin and facial palsy. Our experience

This study is focused on severe cases of facial paralysis where rehabilitation treatment can be a good solution.

The general rehabilitation pattern in these patients includes general treatment based on facilitating movement techniques, inhibiting abnormal motor patterns and coordinating complex movements. These are slow and gradual movements in which the patient observes him- or herself and changes angles, tensions and speed of execution. Movements are short to limit recruitment of motor units and to preserve isolated and symmetrical responses of the facial muscles.2

The characteristics of the facial muscles are the lack of muscle spindles, presence of small motor units, high resistance to degeneration, presence of flat muscles and presence of cortical connections for voluntary and spontaneous expressions.4 Some authors maintain that if voluntary actions are repeated they could be acquired voluntarily, but this is questionable.5

Given that neuromuscular retraining does not fully restore facial function, the addition of botulinum toxin therapy can help to obtain better results.

The goals of rehabilitative treatment are to improve motor control and quality of movement, the acceptance of the deficit by the patient and to improve the quality of life by increasing functionality. Still, we must be realistic and make it clear that no method of treatment restores full functionality if there is axonal injury.6,7

Classical facial rehabilitation is based on muscle contractions in mass (gross movements) such as moving the platysma, raising eyebrows and laughing with both sides at once. It creates a non-specific movement with a high recruitment of motor units that favour abnormal movement patterns and facial asymmetry.

At the moment, specific movement attracts the most interest: it is important to recruit only functional muscle motor units, which will be the really useful ones. The Baillet and Bach-Rita (Wisconsin) neuromuscular re-education programs conducted in this study work with brain plasticity and the acquisition of new motor behaviours.8

General principles of rehabilitation treatment are high motivation, high level of patient collaboration, slow execution and making small, symmetrical movements. Effective rehabilitation is based on feedback that is both visual and sensory, as well as electromyographic (the advantage of visual feedback is that it is immediate). Feedback increases the activity of weak muscles, decreases the activity of overactive muscles and improves coordination of muscle groups (agonists / antagonists).

The home program is carried out after 6-8 sessions in the hospital. The hypotonic treatment phase consists of eye care...
with Ophthalmology consultation, reducing hyperactivity of the healthy side and improving symmetry with the mobility of tissue in a rotary endo-exobuccal manner. All this in conjunction avoids the elongation of affected muscles.

At this stage, the botulinum toxin is injected into the unaffected side at a dose of 2.5 IU per point. The toxin is used in the frontal, or zygomatic, area, in very superficial nasolabial folds (using less than 2.5 IU) and the mentalis in the chin. Treatment in the phase of synkinesis and hypertonia has the main goal of reducing hypertonia through endobuccal mobilisation, seeking painful points (digital pressure stronger than in the hypotonic phase). Relaxation should be practiced using feedback from a mirror or EMG.

At this stage, the botulinum toxin allows movement patterns to be individualised. The corrugator is infiltrated in the affected side (up to 2 points), the superior orbicularis oculi (internal and external edges with a minimal dose),
Botulinum toxin and facial palsy. Our experience

the inferior orbicularis oculi of the affected side (inner and bottom edges), the chin and the platysma. The final clinical outcome is always better than the neurophysiological.

Conclusions

1. The evolution of Bell’s palsy is resolution in 67%-84% of cases according to different authors.
2. Complete facial paralysis with ENoG<10% always evolves sequelae and should therefore be treated immediately.
3. Treatment of severe FP must be aimed towards protecting the eye and maximising the recovery of facial expression, oral communication and feeding. The objectives are to achieve a normal appearance and thus reduce secondary anxiety.
4. Active exercise techniques are the most effective, either with biofeedback or mirror control. The final clinical outcome is always better than the neurophysiological.
5. The subjective effectiveness of rehabilitation is high, and even more so if the use of the toxin is added.

Conflict of interests

The authors declare no conflict of interests.

References