

Efficacy of Levetiracetam in Hemifacial Spasm: A Case Report

Pietro Biagio Carrieri, MD, Maria Petracca, MD, and Silvana Montella, MD

Abstract

Objective:

Safety and efficacy of levetiracetam in a man with hemifacial spasm (HFS).

Methods and Results:

The present work reports the case of a 54-year-old man with a 5-year history of left-sided HFS who, after treatment with levetiracetam (dosage, 500 mg bid), showed a marked improvement in condition. After 7 months of therapy with levetiracetam, the patient remains symptom free with no adverse drug reactions.

Conclusions:

Levetiracetam proved its effectiveness and safety in the treatment of a case of HFS. Nevertheless, there is a need for further controlled studies with larger samples.

Key Words: botulinum toxin, hemifacial spasm, levetiracetam

(*Clin Neuropharmacol* 2008;31:187–188)

Hemifacial spasm (HFS) consists of unilateral involuntary and paroxysmal contractions of facial muscles innervated by the seventh cranial nerve. Its incidence rate in the United States is 0.5 case per 100,000 persons for ages 60 to 79 years.¹

Primary HFS is generally caused by vascular compression of the nerve's root exit zone from the brainstem. Often, vascular loops can be demonstrated by imaging studies; in addition, patients who are not affected by organic disease in which the results of imaging studies are normal are diagnosed as having primary HFS. Secondary HFS is associated with space-occupying lesions in the brainstem or posterior cranial fossa, traumatic events, demyelinating disorders, and Bell palsy. Primary HFS initially involves periocular muscles and then

spreads to the neighboring facial muscles (orbicularis oris and platysma); secondary HFS simultaneously involves the upper and the lower facial muscles. This different clinical presentation is related to causative differences between the 2 forms and to the organization of facial nerve motor fibers. Because facial nerve motor fibers are topographically organized at the root entry zone, primary HFS gradually involves facial muscles, whereas damage of the temporal portion of facial nerve, where fibers become more diffusely arranged, involves all facial muscles at the same time.² Although HFS is a benign condition, it can cause significant cosmetic and functional disability, and patients rarely recover spontaneously.

Levetiracetam is indicated for the treatment of partial seizures with or without secondary generalization in epilepsy, but some reports indicate its effectiveness in the treatment of tardive dyskinesia.^{3,4} Moreover, Deleu⁵ described 2 cases of idiopathic HFS in which levetiracetam (dosage, 500 mg 3 × a day) proved efficacious; in this report, both patients had been previously treated with botulinum toxin type A with a short period of relief and disabling adverse effects.

We report the case of a patient with HFS who responded favorably to levetiracetam.

MATERIALS AND METHODS

In June 2006, we examined a man (age, 54 years) with a 5-year history of left-sided HFS. The symptoms of HFS developed gradually. Initially, they involved upper facial muscles. After 1 year, they spread to lower facial muscles; there was no association with

Dipartimento di Scienze Neurologiche—Università di Napoli "Federico II," Naples, Italy.

Address correspondence and reprint request to Pietro Biagio Carrieri, MD, Dipartimento di Scienze Neurologiche—Università di Napoli "Federico II," Via S Pansini 5, 80131 Naples, Italy; E-mail: carrieri@unina.it

Copyright © 2008 by Lippincott Williams & Wilkins

Bell palsy. The patient presented involuntary tonic-clonic contractions of the eye and mouth orbicular muscles and the platysma; involuntary eye closure interfered with vision. He was hypertensive in treatment with nifedipine (dose, 30 mg/die). Blood test results were normal. Magnetic resonance angiography of the cerebellopontine angle had been also performed and revealed no pathological elements. The patient had been previously treated with clonazepam (dosage, 12 mg bid) for 2 months with no results.

RESULTS

The patient was treated with levetiracetam (dosage, 250 mg bid) for 2 weeks with no benefit. We decided to change the trial dosage to 500 mg bid, and we observed a gradual regression of symptoms during a 3-week period. After 5 weeks of therapy with levetiracetam, involuntary movements disappeared at all. Three months later, the patient spontaneously reduced the dosing of levetiracetam to 250 mg bid for 2 weeks with the reappearance of the disorder; rechallenge with levetiracetam resulted in total regression of visible spasm.

After 7 months of therapy, the patient remains symptom free with no adverse drug reactions.

DISCUSSION

The 2 most commonly used treatments for HFS are botulinum toxin injections and surgical microvascular decompression. Botulinum toxin injections around the eye and cheek result in symptomatic relief and have no serious adverse effects, but they need to be repeated every few months indefinitely with high cost.⁶ Surgical microvascular

decompression also offers relief and, unlike botulinum toxin treatment, it provides a permanent solution with a small chance of serious complication.¹ There are also some reports about the efficacy of gabapentin or pregabalin in the treatment of HFS.⁷⁻⁹

The temporal relationship between drug exposure and clinical effects has shown the usefulness of levetiracetam in the management of our case of HFS. Levetiracetam can be a suitable therapeutic option for patients who are intolerant or resistant to botulinum infiltrations¹⁰ and is an interesting alternative to repeated botulinum injections. Additional studies to confirm the efficacy of levetiracetam in the treatment of HFS are necessary.

REFERENCES

1. Rahman EA, Trobe JD, Gebarski SS. Hemifacial spasm caused by vertebral artery dolichoectasia. *Am J Ophthalmol* 2002;133:854-856.
2. Colosimo C, Bologna M, Lamberti S, et al. A comparative study of primary and secondary hemifacial spasm. *Arch Neurol* 2006;63:441-444.
3. Meco G, Fabrizio E, Epifanio A, et al. Levetiracetam in tardive dyskinesia. *Clin Neuropharmacol* 2006; 29:265-268.
4. Konitsiotis S, Pappa S, Mantas C, et al. Levetiracetam in tardive dyskinesia: an open label study. *Mov Disord* 2006;21(8):1219-1221.
5. Deleu D. Levetiracetam in the treatment of idiopathic hemifacial spasm. *Neurology* 2004;62: 2134-2135.
6. Costa J, Espirito-Santo C, Borges A, et al. Botulinum toxin type A for hemifacial spasm. *Cochrane Database Syst Rev* 2005;25:CD004899.
7. Patel J, Naritoku DK. Gabapentin for the treatment of hemifacial spasm. *Clin Neuropharmacol* 1996; 19(2):185-188.
8. Daniele O, Caravaglios G, Marchini C et al. Gabapentin in the treatment of hemifacial spasm. *Acta Neurol Scand* 2001;104(2):110-112.
9. Urban PP. Pregabalin as add-on treatment to botulinum toxin in idiopathic hemifacial spasm. *Neurology* 2006;66(11):1781.
10. Dressler D, Bigalke H, Benecke R. Botulinum toxin type B in antibody-induced botulinum toxin type A therapy failure. *J Neurol* 2003;250(10):1263-1265.