Botulinum Toxin for Facial Spasms

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Introduction

Normally, our facial muscles are under our voluntary control. We can smile when we want, and raise our eyebrow at our wish. In disorders such as facial spasms, this voluntary control is lost, and the nerves fire on their own: much like a short-circuiting wire that sparks spontaneously. This produces unwanted facial twitching and grimaces on the patient’s face. Not only can these be socially bothersome, but when they are too close to the eyes, it can lead to functional blindness since the patient cannot keep the eyes open at their own will. Benign Essential Blepharospasm is one such disorder that affects both the eyes. Hemifacial spasm affects one half of the face.

Myokimia and Self-limiting twitches

Not all cases are spasms. Eyelid twitching simply means that the muscles under your eyelid skin are contracting. In perfectly normal people, they may begin to fire on their own. The commonest causes of occasional twitch that may persist for a few days are insufficient sleep, stress, altered sleep cycles such as time zone travels, and night time jobs (several IT professionals are at risk). A disturbance of the body fluid-electrolyte balance (recent diarrhoea, or fever, or reduced water intake) can also lead to myokimia. The nerves can get hyperexcitable with caffeinated drinks, and can lead to myokimia. Most often, this twitch is self limiting, and resolves spontaneously without any treatment.
Facial dystonias

Botulinum toxins have revolutionized the treatment of patients with facial dystonias. The success rate is reported to be over 90%. Facial dyskinesias most commonly faced by the ophthalmologist include Benign Essential Blepharospasm (BEB), Hemifacial spasms, Orbicularis myokimia, Meige syndrome and Apraxia of Eyelid opening (Figure 1).

![Figure 1: Easy algorithm to understand facial dystonias. Myokimia involves a single muscle fascicle; Benign Essential Blepharospasm (BEB) involves upper half of face, and by definition is bilateral; Hemifacial spasm (HFS) involves one half of the face; and Meige syndrome is a BEB with lower facial dystonia: thereby involving the entire face.]

Benign Essential Blepharospasm (BEB) is an involuntary and repetitive bilateral spasmodic contraction of the orbicularis oculi muscle (Figure 2), and is often progressive. It usually presents in the 4th to 6th decade, with early symptoms of irritation and discomfort in the eyelids causing an increase in the blink rate, which increases in one or two years to forceful involuntary closure of eyelids. The etiology of blepharospasm is considered to be an organic dysfunction of the rostral brainstem. Treatments that

![Figure 2: Middle aged man with BEB, pre and post-Botulinum toxin injection]
have been tried for BEB include central nervous system depressants (diazepam and clonazepam), orbicularis myectomy and selective facial nerve neurectomy. However, patient acceptance is highest with botulinum toxin chemodenervation.

Reflex blepharospasm, caused by dry eye or ocular surface pathology can mimic BEB. It can be associated with spastic lower eyelid entropion which in turn induces ocular surface damage, and the vicious cycle continues. It is typically relieved by instillation of topical anaesthetic. Meige Syndrome is a form of cranial dystonia characterized by the presence of bilateral dystonic spasms of the orbicularis oculi (blepharospasm) with concurrent dystonia of the lower face, in the form of lip pursing, chewing, or jaw opening movements.

**Hemifacial spasm** (Figure 3) is characterized by repetitive unilateral periodic tonic contractions of ipsilateral facial muscles. It begins in middle age, and is more common in females than males. It generally results from mechanical-vascular compression of the seventh cranial nerve root in the cerebello-pontine angle. Less than 1% are caused by posterior fossa tumors, therefore a Magnetic Resonance Imaging may be indicated in patients with Hemifacial spasms to rule out this rare cause. Treatments that have been tried for HFS include carbamazepine, clonazepam, nerve blocks, myectomy, selective facial nerve neurectomy and neurosurgical microvascular decompression. Although neurosurgical procedure may be the definitive form of treatment, botulinum toxin injections are effective in controlling the spasms.

*Figure 3: Middle aged woman with Left HFS, pre and post-Botulinum toxin injection*
Injection Sites and Technique

Patients with BEB typically require 5-8 sites of injection per side of the face (Figure 4). Each site can receive 2-5 units of Botulinum toxin as per the dilution. Repeat injections every 3-4 months.

Hemifacial spasms receive 8-10 sites of injection over the ipsilateral hemiface (Figure 5). The periorbital sites remain the same as BEB, but the mid and lower facial sites are decided by the involved muscles.

HFS has a longer spasm-free interval of 4-6 months. Patients are evaluated two to four weeks after their initial injections to assess the efficacy, and side effects or complications. Further injections may be given at that time, or a note may be made to inject more or less toxin into the areas that are under or over paralyzed respectively.

Summary

These disorders generally have no permanent cure, or this option is too complex, such as a brain surgery, in case of Hemifacial spasm. Botulinum toxin causes relaxation of these overacting muscles, thereby allowing them to lead a normal life for a period of 3-4 months. It can then be repeated for continued effect. When Botox is not effective (which may happen in a minority of patients), a surgical weakening of these muscles (myectomy) is required.