Botulinum toxin treatment in patients with hemifacial spasm

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Abstract  Hemifacial spasm is nearly always a unilateral disease of the facial musculature and is characterized by involuntary tonic or clonic cramps that considerably reduce the affected patient’s quality of life. In the past, a number of different conservative and operative therapeutic procedures have been applied for the treatment of hemifacial spasm. In many cases these attempts failed to control the disease permanently or resulted in unwanted, sometimes strong, side effects. We report our own experiences with botulinum therapy in 29 patients with hemifacial spasm (78 therapeutic sessions). In our patients the mean duration of an effect after treatment with botulinum toxin was 18.2 weeks. Side effects were rare. Our results since 1990 at the University of Göttingen demonstrate that subcutaneous application of toxin from Clostridium botulinum to involved facial muscles represents a reliable method for successful treatment of hemifacial spasm.

Key words  Hemifacial spasm · Botulinum toxin · Clostridium botulinum

Introduction

The clinical findings in hemifacial spasm are characterized by involuntary tonic or clonic spasms of all muscles of one side of the face receiving their innervation from the facial nerve. Spasms usually start periorbitally [4] and may spread caudally in the further course of the disease. This can take any period of time, ranging from hours to years. In a small number of cases, the disease is bilateral. The mean age of patients is about 45 years [7], with women suffering more often than men. Occurrence is very rare in childhood.

Different pathogenic approaches have been taken to elucidate the etiology of hemifacial spasm, although its interpretation as a psychogenic disease [7] has been abandoned. Among those clinicians looking for an organic cause of hemifacial spasm, most authors agree today on an intracranial irritation of the facial nerve caused by a pulsating vascular loop [13].

The therapeutic concepts suggested for hemifacial spasm are manifold. They range from medical treatment with such drugs as carbamazepine [1], alcohol blocks or targeted surgical transections of the facial nerve [9] to myectomies of the periorbital muscles [14]. Jannetta et al. [13] have proposed a therapy based on the relief of intracranial nerve irritation by a vascular loop (mostly the anterior inferior and posterior cerebellar arteries). They then performed surgical decompression of the facial nerve, placing an interposition graft between the vascular loop and facial nerve. This operation has a low recurrence rate, which according to several authors ranges from 3% to 25% [10, 18]. Its disadvantage lies in the potential side effects of the surgery involved with persistent impairment of hearing and facial nerve paralysis predominating.

A completely different therapeutic approach was first reported in 1981 by Scott [22]. As a purely symptomatic therapy, a targeted, selective weakening of the muscles affected by the hemifacial spasm was performed by subcutaneous injections of botulinum toxin (BT). The main advantage of this technique was its easy performance. Complications, if occurring at all, were only temporary. Its disadvantage was that the effect of BT was not permanent, necessitating repetition of therapy after varying time intervals. Since 1990, patients with hemifacial spasm have been treated at the University ENT Hospital in Göttingen by subcutaneous injections of BT.

Patients and methods

From September 1990 until February 1993, 29 patients suffering from hemifacial spasm were treated by injections of botulinum toxin type A (Botox, Allergan, USA) at the University ENT Hospital, Göttingen. Their ages ranged from 34 to 81 years, with a median of 61.5 years and a mean of 59.5 years. Nineteen patients were women, and 10 were men.
A total of 78 therapeutic sessions were performed with all patients. Individual patients received Botox doses between 5 and 40.5 units (mean = 22.25 units) by injection into the region of the affected facial muscles. The number of injection points varied from a minimum of 2 to a maximum of 17 sites (mean, 10.6 injection points).

Figure 1 is a schematic illustration of the positions of the individual injection points. The doses applied at these sites varied between 1.25 and 5 units of BT. Solutions used for injection were prepared by dissolving 100 units of lyophilized Botox in 4 ml of 0.9% saline (final concentration, 2.5 units/0.1 ml solution).

In order to evaluate therapeutic success, all patients were asked before and after the first therapeutic session to rate the degree of their impairment according to a quantitative, subjectively graded scale. This scale distinguishes between four different grades of hemifacial spasm, beginning with a stage of rare contractions up to a stage of nearly continuous spasms [6, 21]. Following BT treatment, patients were hospitalized for 2 days for observation. The patients themselves determined the interval between the first and second injections, depending on the severity of the symptoms.

**Results**

Hemifacial spasm occurred on the left side of the face in 13 cases (45%), and on the right side in 16 cases (55%). The time lags between initial disease symptoms and first toxin application, as revealed by history, varied from 1 to 33 years for all 29 patients. Altogether 35 previous therapeutic attempts by various techniques had been attempted before patients presented for further treatment. Among the prior approaches used, drug therapies (33%) and acupuncture (29%) were the most frequent.

**Table 1** Factors triggering contractions in hemifacial spasm

<table>
<thead>
<tr>
<th>Factor</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light</td>
<td>6 patients</td>
</tr>
<tr>
<td>Stress</td>
<td>11 patients</td>
</tr>
<tr>
<td>Cold</td>
<td>1 patient</td>
</tr>
<tr>
<td>Innervation</td>
<td>8 patients</td>
</tr>
<tr>
<td>Reading</td>
<td>2 patients</td>
</tr>
<tr>
<td>Exposure to wind</td>
<td>1 patient</td>
</tr>
</tbody>
</table>

After careful history, trigger factors causing contractions of the facial muscles were determined in 14 patients (Table 1). The most common factor was a stress reaction (78.6% of cases).

Nineteen of the 29 patients (65.5%) subjectively rated their spasm-induced facial impairments before and after the first BT injection (Fig. 2). Administration of the toxin resulted in a self-assessed alleviation of symptoms in 18 cases (94.7%). Only one patient did not feel any difference in his spastic attacks before and after treatment. In this case, however, symptoms were rather slight prior to onset of therapy and consisted mainly of rare contractions which could not be further influenced by treatment with toxin. The frequency of contractions was reduced to significantly fewer spasms in 14 patients (73.7%).

Table 2 presents the results of BT treatment. The onset of the effect produced occurred on average 4.7 days after treatment. The average time during which the BT effect was at its optimum was 12.6 weeks. A further, although weaker, effect extending beyond this optimum period was reported by patients to last for an additional 5.6 weeks. On average the total effective time following BT application was 18.2 weeks. Additionally, 9 of the patients showed a reduction of spasms in mimetic muscles in regions where no injections were given.