BOTULINUM TOXIN IN THE TREATMENT OF HEMIFACIAL SPASM

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ABSTRACT

Thirteen patients with disabling idiopathic hemifacial spasm received botulinum toxin A injections to the affected muscles. Previous treatments, including one posterior fossa decompression, had no sustained benefit. There was excellent response in all the patients with improvement in social disability. Local side effects were mild and transient and include mild facial weakness. One patient had mild diplopia. The benefit lasted more than three months. Reinjection resulted in identical efficacy. Botulinum toxin A injection is a useful therapy for Hemifacial spasm.

Keywords : Hemifacial spasm, botulinum toxin

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INTRODUCTION

Idiopathic hemifacial spasm (HFS) is an involuntary movement of the face characterised by episodic clonic and tonic twitches of muscles innervated by the facial nerve on one side of the face. Theories for the aetiology and mechanism of the spasm are not agreed upon, and this is reflected by the varied therapeutic options which include medications[1-3], nerve blocks[4,5] and surgery[6-10]. The most recent development in the medical therapy of HFS is the use of injected botulinum toxin into the offending muscles. We report our experience in the usage of botulinum toxin in Asians.

PATIENTS AND METHODS

Thirteen patients with disabling HFS were treated with botulinum toxin injections. Criteria for inclusion include an otherwise normal neurological examination, symptoms of at least 6 months duration, moderate or marked disability, and a lack of response to previous treatments. The investigational nature of the treatment was explained, and informed consent was obtained from all patients.

There were eight females and five males aged 42-67 years. All patients had no other neurological abnormality. The mean duration of symptoms before treatment was 4.6 years (range 8 months to 15 years). Five patients had spasms on the right side and eight on the left. All patients had been treated previously without lasting benefit. The treatments included medications (anticonvulsives, anxiolytics, and tranquilizers), acupuncture in two cases and in one case, posterior fossa surgery five months prior to injection.

The patients were examined, rated, and videotaped before injections and at monthly intervals. Severity of spasms, frequency of spasms, degree of functional disability, and following injections, degree of improvement were assessed according to standard protocols (Figs. 1 & 2).

Fig 1
Hemifacial Spasm Rating Scale

SEVERITY	FREQUENCY
0 = None	0 = Absent
1 = Intermittent	1 = < 25% of waking time
2 = Mild. No eye closure	2 = 25-50% of waking time
3 = Moderate. Eye closure	3 = 50-75% of waking time
4 = Marked spasm. Eye closure	4 = > 75% of waking time

Fig 2
Functional Scale

IMPROVEMENT SCALE	FUNCTIONAL DISABILITY SCALE (eat, sleep, talk, watch, concentration)
0 = No change	0 = None
1 = Mild improvement	1 = Mild
2 = Moderate improvement	2 = Moderate (Interferes)
3 = Marked improvement	3 = Severe
4 = Worse	

Botulinum Toxin A (BToxA) was supplied in the form of 0.05ug toxin-haemagglutinin complex per vial. It was reconstituted immediately before injections with 1.0 ml normal saline and then further diluted in 9.0 ml saline to give a concentration of 5.0 ng/ml. Intramuscular injections were given at 3 points in the orbicularis oculi and 2 points in the facial musculature (Fig. 3) with a 27 gauge

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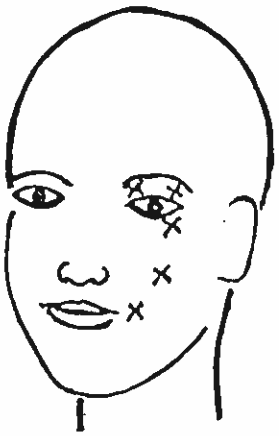
Fig 3
Injection Sites



I/M 0.1 - 0.2 ml/site

needle using an insulin syringe. Four patients received 0.2 ml per injection site with a total dose of 5.0 ng. The rest received injections of 0.1 ml per site with a total dose of 2.5 ng per subject.

RESULTS

All 13 patients had substantial relief following the first injection. No patient required another injection to achieve an initial response. Relief was manifest in the reduction of the severity of the spasms and the frequency of the spasms. Seven patients had complete relief of spasms (Figs. 4 & 5). One month after the initial injection, ten patients reported marked improvement while three had moderate improvement. Six patients had mild residual disability (Fig. 6). Effects of the toxin were noticed 48-72 hours after the injections,with a maximum effect at 5-7 days. Duration of the beneficial effects of the first injection lasted from 3 to 5 months.

Ten patients received a second injection of botulinum toxin. At the time of reinjection, the severity of the spasms in all patients were less than the pre-injection status. Beneficial effects of the reinjection were similar to the first injections.

Side Effects

One patient who received 0.2 ml injections had a mild diplopia which lasted four weeks. Lower eyelid edema was present in eight patients. Facial weakness was present in ten patients, the severity of which was dose related. One patient who had mild facial weakness did not realise her facial weakness till it was pointed out to her by the investigator. No patients in this series had significant ptosis. There was no exposure keratitis nor excessive tearing. The side-effects were well tolerated by all patients.

DISCUSSION

HFS is a disabling involuntary movement characterised by paroxysms of muscle twitches on one side of the face. Affecting mostly middle-aged patients, it begins as twitches in one eye and progresses to involve other parts of the facial musculature in ever increasing severity and frequency. In severe cases it may lead to closure of the eyes. Longstanding cases may develop mild ipsilateral facial weakness[11].

Fig 4
Results – Severity of Spasms

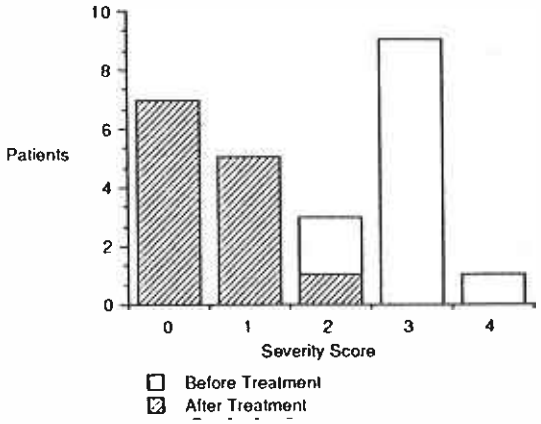


Fig 5
Results – Frequency of Spasms

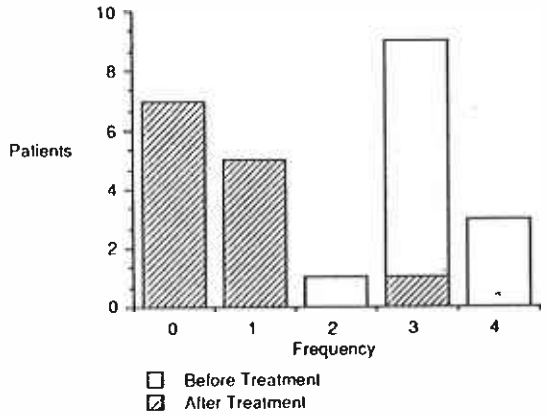


Fig 6
Results – Functional Scale

IMPROVEMENT SCALE	FUNCTIONAL DISABILITY SCALE
0 = 0	0 = 0 (7)
1 = 0	1 = 2 (6)
2 = 3	2 = 9 (0)
3 = 10	3 = 2 (0)
4 = 0	( ) after treatment

The cause of HFS is often unknown. Surgeons have reported vascular structures lying across the root entry zone of the facial nerve. Separation of these offending vessels from the nerve have been reported to abolish or reduce the spasm[6-10]. Since medical therapy is often ineffective, treatment is usually directed towards neurosurgical procedures. HFS is a chronic disorder and a cause of social embarrassment. It interferes with activities of daily living but has no risk to life. Neurosurgical procedures, while offering the possibility of cure, is not without risk. In addition, surgery carries a recurrence rate of up to 60%[12]. The one patient in this study who had microvascular decompression still had marked disability 5 months after surgery although he did have some benefit from the treatment.

Local injections of BToxA is a successful alternative to surgery for what is essentially a social handicap[13-15]. The toxin was uniformly effective in relieving the spasms in all our patients, with significant improvement in social disability. Local side effects were mild, transient and well

tolerated. Other studies reported a somewhat higher rate of ptosis which was not seen in our patients. There were no systemic effects although abnormal single fibre electromyography was reported in some distant muscles following injections of more than 245 units (or 100ng)[16]. This was not associated with clinical weakness nor abnormality in routine electrophysiology.

Botulinum toxin produces its effects by causing weakness locally. It is taken up by the presynaptic terminals and causes weakness by interfering with release of acetylcholine. Return of function is due to resprouting of nerve terminals[17,18]. Apart from its direct effect on reducing spasm intensity it also causes a yet unexplained reduction in spasm frequency.

Besides a high rate of success in HFS, the major advantages are its low cost and low complication rate. The disadvantage is the necessity for reinjections due to loss of efficacy after 3-5 months. The other yet known factor is the long term effectiveness. However, studies conducted since the early eighties have shown a continuing usefulness in HFS and other form of focal dystonias like blepharospasm torticollis, and other conditions of pathological sustained muscle contractions. We conclude that BToxA injection is a useful symptomatic therapy for HFS and should be considered a viable alternative to neurosurgical procedures.

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