Treatment of unilateral masseter hypertrophy with botulinum toxin in two cases

İki olgunun tek taraflı masseter hipertrofisinin botulinum toksinin tedavisi

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Botulinum toxin is a new method for treating masseteric hypertrophy which offers many advantages over conventional surgical treatment. We describe the successful outpatient medical management of two patients with masseteric hypertrophy using botulinum toxin type A. No significant side effects have occurred and the patients are satisfied cosmetically six months after the treatment.

Key Words: Masseter muscle/pathology; hypertrophy/etiology; botulinum toxins/therapeutic use.

Masseteric hypertrophy (MH) is recognized as an asymptomatic enlargement of one or both masseter muscles. The enlargement usually represents a work hypertrophy whose most frequent cause is clenching. Bruxing during sleep, constant gum chewing, and mastication of hard foods can also lead to MH.1 Dysfunction of temporomandibular joint, malocclusion and periodontal breakdown have also been implicated.2

Most cases of MH are bilateral and symmetric, but unilateral occurrence also can be seen when patients chew or clench primarily on one side.3 Therapy is generally not necessary for MH. However, because of myofacial pain problems and cosmetic concerns, intervention may be required. This report describes the successful treatment of two patients with unilateral MH using botulinum toxin type A (BT).

Case 1– A 25-year-old woman presented to our clinic with a left sided swelling around the angulus of mandible of undetermined cause (Fig. 1a). History indicated that the swelling was first noted 5 years ago by the patient herself. The swelling had never caused any discomfort. However, there had been a gradual increase in size during this period. She had a habit of chewing gum usually on left side and bruxing during sleep.
Palpation indicated that the swollen tissue was normal in tone and non-tender. When the patient asked to clench, the swelling became accentuated and firm. The patient dental status was excellent and examination by the dentist revealed no pathology.

Because the patient was a photomodel she requested cosmetic treatment due to facial asymmetry.

The patient was asked to clench her jaws, and 5 units of BT were deposited percutaneously within the left masseter muscle at six different sites with the guide of ultrasonography. The patient was seen for follow-up examination every month. Within six months masseter muscle atrophy occurred and facial asymmetry was no longer visible (Fig. 1b). Patient stated that she no longer bruxes at night very happy with cosmetic appearance of her face.

**Case 2**– A 14-year-old asian girl presented to our clinic with swelling around the left angulus of mandibula (Fig. 2a). Questioning indicated that swelling was first noted 2 years previously. The

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**Fig. 1** - (a) The patient’s appearance prior to her treatment for masseteric hypertrophy. (b) The patient’s current appearance following her treatment for masseteric hypertrophy with BT (Case 1).

**Fig. 2** - (a) Appearance prior to treatment for masseteric hypertrophy of case 2. (b) The patient’s current appearance following her treatment for masseteric hypertrophy with BT.
patient denied bruxism, chewing gum and excessive clenching of food on one side. The patients dental status was excellent. Because the patient was concerned about the cosmetic facial asymetry, treatment was initiated with BT. Five units of BT were injected to 6 different sites of greatest muscle bulging. Although cosmetically acceptable appearance obtained after 6 months a second BT treatment was performed due to residual facial asymetry (Fig. 2b).

DISCUSSION

Conventional treatment of MH consist of resection of the muscle and reduction of any bony hyperostosis. Surgical reduction can be performed from an intra or extra-oral route. Endoscope assisted intraoral approach for masseteric hypertrophy has been described lately. Many conservative treatments including occlusal adjustment, splint therapy, relaxation therapies, medical therapies with spasmyotics and antidepressant have been nearly always unsuccessful. In addition many surgeons are hesitant to recommend conventional surgery for this benign, although distressing condition. Particular problems associated with surgery are:

1. Difficulty in judging the correct amount of muscle to resect,
2. Occasional need for an extra-oral incision,
3. Risk to the lower branches of the facial nerve, and,
4. Considerable post-operative morbidity with pain, swelling, hemorrhage and trismus which may persist for months.

Recently BT has been suggested for the management of MH. This potent neurotoxin is a protein produced by the anaerobic bacteria Clostridium botulinum. The toxin exerts its action by permanently binding to motor end plate at the neuromuscular junction which prevents the release of acetylcholine from the neuromuscular cleft. This accounts for the delayed onset of action of the toxin which is usually between 2 and 4 days after administration, as storage vesicles of acetylcholine within the presynaptic motor end-plate are used up.

Although the toxin binds permenantly to the neuromuscular junction, recovery of muscle function does occur. The process by which function returns was demonsntrated by Duchen who showed that new neuromuscular junctions are formed by a process of presynaptic axonal sprouting. The duration of muscle paralysis is usually between 2 and 4 months with gradual recovery of full function thereafter.

Drug resistance due to antitoxin antibody formation was initially considered to be a potential problem, however this has not been the case in clinical practice with only sporadic reports of clinical resistance to the effects of repeated large doses of BT. No serious side effects of BT therapy have been reported to date and those side effects which have occurred are minor such as local bruising or unwanted spread of action to adjacent muscles. In case unwanted accidental overdoses of BT are given to adjacent muscles, patients occasionally develop severe and prolonged dysphagia. After an accidental overdose, antitoxin may neutralise the toxin if given within a few hours, but it will be ineffective after a day or so as the toxin will have been taken up by nerve terminals. Unfortunately, side effects of BT are usually apparent only after a week or more, when antitoxin will be useless.

BT is expensive and available only in ampoules of 100 units. The solution for injection is prepared by diluting the BT with normal saline. Unfortunately BT has a very short shelf-life. Once the BT has been mixed with saline, its use within 6 hours has been suggested. However Garcia and Fulton have indicated that with refrigeration the reconstituted solution can be maintained for 1 month without a loss of potency.

Muscle growth is dependent on two processes: hyperplasia-an increase in cell number; and hypertrophy-an increase in cell size, in general, muscle fiber number is set at birth. In a histopathologic study it was noted that the picture was not one of classic fibre hypertrophy. Rather, the impression was of an increase in the numbers of muscle fibers suggesting hyperplasia. In this case unlike the clinical impression it is expected that the muscle may hypertorhies again after the effect of BT has been lost. This may be due to cessation of the habits causing the muscle enlargement by BT.

The medical treatment of MH would be preferable to surgical procedure requiring an inpatient stay. These cases suggest that BT may well be effective in the management of MH.
REFERENCES