

SENSORY TRICK: INFILTRATION OF ITS TRIGGER POINT WITH BOTULINUM NEUROTOXIN IN CERVICAL DYSTONIA

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INTRODUCTION

Cervical dystonia (CD), a hyperkinetic motor syndrome, frequently associates pain and the presence of a sensory trick (ST), as sensory phenomena (1).

GA may be present in primary and secondary dystonia (2), and its mechanism of action is unknown. Intramuscular (IM) infiltration of botulinum neurotoxin (BoNT) is an efficacious therapy for CD (3) and some neuropathic pain syndromes, such as diabetic neuropathy and trigeminal neuralgia (4).

The influence of BoNT on GA has not been investigated before. Thus, the aim of this work has been to find out whether BoNT, applied subcutaneously (SC) at the point that triggers GA, could improve the results obtained with IM BoNT in patients with CD.

PATIENTS AND METHODS

Nine patients with CD treated with IM BoNT from seven to twelve years, identified a sensory trick (table 1). With IM BoNT, five patients were free of dystonia while sitting, while four had persistent, albeit improved, dystonia. In addition, seven patients experienced pain.

In addition to the usual dose of IM BoNT, every patient received SC BoNT at the trigger point of the ST, during eight to twenty-four months. No changes in IM BoNT or oral drug dosages were introduced during the study period.

Patients	7 female, 2 male
Age	35-80
Aetiology of CD	Idiopathic (8) Secondary to chronic neuroleptic treatment (1)
Cervical Dystonia	Torticollis (5) Retrocollis (3) Antecollis (1)
Other abnormal movements	Oromandibular dystonia (1) Opisthotonic back arching (1) Laterocollis Shoulder elevation
Pain	7
Geste Antagoniste	Chin (4) Neck (3) Occiput (1) Shoulder (1)
IM BoNT	Botox, 150-400 u Dysport, 300-900 u
Interval between IM BoNT	12 weeks
SC BoNT at trigger point of GA	Botox, 20 u Dysport, 40 u

Table 1

RESULTS

Among five patients with a normal neck posture when seated, while receiving IM BoNT, one reported disappearance of torticollis during gait, with BoNT infiltration at the trigger point of the ST, while in two others, the interval between IM BoNT infiltrations could be delayed to sixteen weeks, and in another, to eighteen. One patient, among four with an abnormal neck posture during IM BoNT, attained a normal neck position sitting, and two others experienced amelioration in neck posture. Pain was decreased in five out of seven instances (table 2).

The referred to improvements were apparent from the first SC infiltration of BoNT, and have remained constant through more than three years of follow-up. No side effects were noted.

DISCUSSION

SC infiltration of BoNT at the trigger point responsible for GA resulted in additional benefit in seven out of nine patients treated with IM BoNT: improvement or normalization of anomalous neck postures while seated, decreased or suppressed dystonia when walking, prolongation of the interval between IM BoNT infiltrations and partial relief of pain, were all noted. Besides, no worsening of dystonia, or any other side effects, were noted. Two patients did not benefit from SC BoNT: patient 5 was already asymptomatic receiving IM BoNT, and patient 9 experienced no improvement in dystonia or pain; tardive dystonia (patient 9) has been reported to be resistant to BoNT therapy (5), and ceased in this case after withdrawal of antipsychotic drugs.

These results, though favourable, must be regarded as preliminary, considering the observational nature of this work. Nevertheless, they support the idea that modification of peripheral proprioceptive feedback may bring about a significant reduction of abnormal movements and pain in CD. As a consequence, research into methods capable of modifying sensory inputs from the trigger point of GA would be desirable.

CONCLUSIONS

Manipulation of the sensory trick in CD with BoNT may prove a useful, safe and simple method of reducing abnormal movements by itself, or of increasing the benefit provided by IM BoNT infiltration or other therapies. Adequate clinical trials, designed to test the feasibility of this hypothesis, are needed.

REFERENCES

1. Fahn S, Jankovic J. Dystonia: phenomenology, classification, etiology, pathology, biochemistry and genetics. In: Fahn S, Jankovic J, editors. Principles and practice of movement disorders. Philadelphia: Churchill Livingstone Elsevier, 2007: 309-343.
2. Muller J, Wissel T, Masuhr F, et al. Clinical characteristics of the geste antagoniste in cervical dystonia. J Neurol 2001; 248: 478-482
3. Fahn S, Jankovic J. Treatment of dystonia. In: Fahn S, Jankovic J, editors. Principles and practice of movement disorders. Philadelphia: Churchill Livingstone Elsevier, 2007: 309-343: 345-367.
4. Francisco GE, Tan H, Green M. Do botulinum toxins have a role in the management of neuropathic pain?: a focused review. Am J Phys Med Rehabil 2012; 91: 899-909.
5. Godeiro-Junior C, Felício AC, De Carvalho Aguiar P, Borges V, Silva SMA, Ferraz HB. Neuroleptic-induced tardive cervical dystonia: clinical series of 20 patients. Can J Neurol Sci 2009; 36: 222-226.

	Dystonia with IM BoT	Dystonia with SC + IM BoT	Neck pain with IM BoT	Neck pain with SC + IM BoT
Patient 1	Normal neck sitting, torticollis walking	No torticollis walking.	Neck pain.	Neck pain decreased.
Patient 2	Normal sitting. Interval: 12 weeks	Interval: 16 weeks	Neck pain.	Neck pain decreased.
Patient 3	Normal sitting. Interval: 12 weeks	Interval: 16 weeks	Neck pain.	Neck pain decreased.
Patient 4	Normal neck sitting. Interval: 12 weeks	Interval: 18 weeks.		
Patient 5	Normal neck sitting.	Unchanged		
Patient 6	Torticollis sitting.	Normal neck sitting.	Neck pain.	Neck pain decreased.
Patient 7	Retrocollis sitting.	Neck posture improved.	Neck pain.	Neck pain decreased.
Patient 8	Anterocollis sitting.	Neck posture improved.	Neck pain.	Unchanged
Patient 9	Retrocollis sitting.	Unchanged	Neck pain	Unchanged

Table 2