The Role of Kinesiotaping Combined With Botulinum Toxin to Reduce Plantar Flexors Spasticity After Stroke

Evrim Karadag-Saygi,[1] Koza Cubukcu-Aydoseli,[1] Nilufer Kablan,[1] and Demet Ofluoglu[2]

[1]Department of Physical Medicine and Rehabilitation, Marmara University School of Medicine, Istanbul, Turkey; [2]Department of Physical Medicine and Rehabilitation, Alman Hospital, Istanbul, Turkey

Purpose: To evaluate the effect of kinesiotaping as an adjuvant therapy to botulinum toxin A (BTX-A) injection in lower extremity spasticity. **Methods:** This is a single-center, randomized, and double-blind study. Twenty hemiplegic patients with spastic equinus foot were enrolled into the study and randomized into 2 groups. The first group (n=10) received BTX-A injection and kinesiotaping, and the second group (n=10) received BTX-A injection and sham-taping. Clinical assessment was done before injection and at 2 weeks and 1, 3, and 6 months. Outcome measures were modified Ashworth scale (MAS), passive ankle dorsiflexion, gait velocity, and step length. **Results:** Improvement was recorded in both kinesiotaping and sham groups for all outcome variables. No significant difference was found between groups other than passive range of motion (ROM), which was found to have increased more in the kinesiotaping group at 2 weeks. **Conclusion:** There is no clear benefit in adjuvant kinesiotaping application with botulinum toxin for correction of spastic equinus in stroke.
Key words: *botulinum toxin A, hemiplegia, kinesiotaping, spasticity*

Spasticity is a disorder characterized by a velocity-dependent increased resistance to passive stretch.[1] It is a common complication of stroke that can lead to impaired gait characteristics related to equinus deformity in the lower extremity.[2] There are many different options in the treatment of spasticity secondary to stroke. Most of the current options such as antispastic drugs, physical therapy agents, and surgical procedures have disadvantages like generalized weakness or lack of long-term efficacy.[3]

Botulinum toxin A (BTX-A) is a potent neurotoxin that blocks local synaptic transmission at cholinergic terminals.[4] Because it is a safe and effective option in the treatment of poststroke spasticity, it has become a useful therapeutic alternative for spasticity management. The main advantage of BTX-A treatment is that it has focal, selective, and reversible effects in the injected muscles with low incidence of adverse effects.[3]

Several attempts have been made by previous investigators to increase the efficacy and effective period of BTX-A by using combination therapies such as serial casting, electrical stimulation, taping, and therapeutic exercise.[3,4] Although there is no agreement as to which treatment is the most effective after the injection, taping is proposed to be effective. Kinesiotape, an alternative taping technique, was introduced by Kenzo Kase in 1996. It is thin, latex free, anti-allergenic and can be stretched in the longitudinal axis. Therefore, less mechanism constraints are seen compared with conventional tape.[5] Therapeutic effects of kinesiotaping include decreasing pain, increasing muscle strength, improving blood and lymph circulation, and repositioning the subluxated joints by relieving abnormal muscle tension.[6] Kinesiotaping is currently used in rehabilitation as an adjuvant therapy method due to positive effects on pain and gait pattern.[5,7] Although the exact mechanism of action is not clear, neurofacilitation and mechanical restraint have been proposed as possible underlying mechanisms.[5,8] The aim of this study is to assess the additive effect of adjuvant kinesiotaping on BTX-A injection in stroke patients.

Method

Patients

This is a single-centered, randomized, and double-blind study. Twenty hemiplegic patients

Top Stroke Rehabil 2010;17(4):318–322

doi: 10.1310/tsr1704-318

with equinus foot associated with plantar flexor spasticity (Modified Ashworth Scale [MAS] grade 2–4) and poststroke duration of ≥6 month who were able to walk 10 m with or without assistance were included in the study. Subjects were excluded if they had plantar flexion contracture, cognitive impairment, previous surgery of the plantar flexor muscles on the affected side, BTX-A treatment within previous 12 months, or allergy to taping. The study was approved by local ethics committee and written informed consent was obtained.

Injection technique

Eligible patients were randomized into 2 groups. The first group received BTX-A injection (Botox®, Allergan) and taping, and the second group received BTX-A injection and sham taping. Both medial and lateral heads of the affected gastrocnemius muscle were injected using motor point localization by peripheral electrical stimulation. Repetitive monopolar stimulation technique was employed, using a single channel (maximum current of 5mA) Stimuplex nerve stimulator for all patients. Depending on spastic hypertonia grade, the dose ranged between 75 and 100 IU for each muscle head and diluted with 2 mL of saline to obtain a concentration of 5 unit/0.1 mL. Flexion-extension was performed to enhance the spreading of BTX-A. All the injections were performed by a single investigator (E.K.S.).

Taping

Taping was performed by a physiotherapist who is certified for this method. Subjects were taped in accordance to Kenzo Kase's *Kinesiotaping Manual.* The taping was applied with the ankle in neutral position in 4 steps. Patients were in supine position in the first step and prone position in the other 3 steps of taping. The first strip of tape was placed from the anterior midfoot, stretched approximately to 120% of its maximum length and attached just below the fibular head over the tibialis anterior muscle. The second strip began from the heel and attached medial and lateral heads of gastrocnemius muscle. The third strip originated at the arch and stretched slightly above both the medial and lateral malleolus. The last strip stretched across the anterior ankle, covering both the medial and lateral malleolus (**Figure 1A**). All patients were taped right after the injection. Just after the first week, old tapes were removed and new ones were placed with the same method. Sham taping was also done on the same days and with the same kind of material on ineffective parts of the muscles (**Figure 1B**).

(A)

(B)

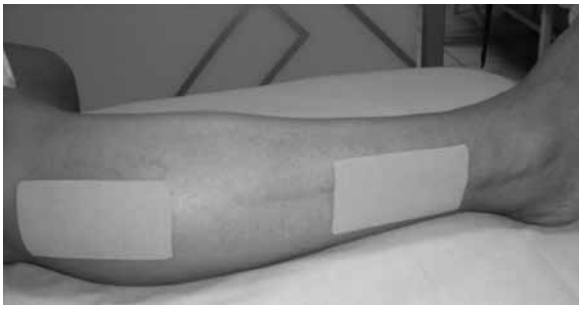

Figure 1: The application of (A) true (B) sham tape. Numbers indicate order of taping.

All patients had undergone intensive rehabilitation treatment during the first months of stroke. Active-assistive range of motion and stretching exercises were given as a home exercise program to both groups. Exercises were assigned twice daily for 20 minutes for 4 weeks.

Clinical assessment

The clinical assessment was done before BTX-A injection and at 2 weeks and 1, 3, and 6 months by an investigator who was blinded to which group patients were allocated (K.C.A.). Passive range of motion (ROM) of ankle was measured with a goniometer while the patient was lying in a supine

position. Ankle plantar flexor muscle tone was evaluated according to modified Ashworth scale (scores 0 to 4). To evaluate gait cycle parameters, patients walked 10 m. Several practice runs were made to ensure the recording of natural gait, and 3 consecutive measurements were taken for evaluation of walking speed in both groups at each assessment. Number of steps and time of walking were recorded and were used to calculate step length and gait velocity. Patients walking with a walking aid had to use it for each assessment.

Statistical analysis

Friedman nonparametric repeated measure analysis of variance (ANOVA) test was used for continuous data to test means equality of measurements. When *P* value was less than .05, Dunn's pairwise multiple comparison tests was performed. Differences between groups were tested by Mann-Whitney *U* test. Statistical significance was defined by *P* value less than .05. All analyses were performed using SPSS 11.5 for Windows (SPSS, Inc., Chicago, Illinois).

Results

All participants completed the second week and first and third month's visits. Results were obtained for 17 patients at the sixth month (9 in kinesiotaping group, 8 in sham group).No serious side effect was seen in any of the patients during the study; only 1 patient from each group complained of transient pain at the injection site. The clinical and demographic characteristics of the subjects are summarized in **Table 1**. The 2 groups were well matched according to baseline characteristics.

Table 1. Baseline characteristics of patients

	Kinesiotaping group	Sham group	*P*
Age, years[a]	63.8 ± 9	57.3 ± 12	.227
Gender, female/male	7/3	5/5	.139
Body mass index, kg/m^2[a]	28.9 ± 4	29.7 ± 4	.654
Paretic side, right/left	3/6	2/6	.815
Age at stroke time, years[a]	60 ± 10	53.5 ± 11	.236
Time since stroke, months[a]	35.2 ± 29	39.4 ± 30	.673

[a]The values are presented as mean ± *SD*.

Median values for clinical and functional scales at baseline and follow-up are presented in **Table 2**. Passive ROM, step length, and gait velocity increased and MAS score decreased during the follow-up period for both groups ($P < .05$). In the kinesiotaping group, patients demonstrated an increase in passive ankle dorsiflexion at the second week and first and third month visits when compared with the baseline ($P < .05$, $P < .001$, $P < .01$, respectively). Sham group also showed improvements in passive ROM for the first and third month visits ($P < .001$). The gains in dorsiflexion started to decline at 3 months, and no significant improvement was detected according to baseline in both groups at

Table 2. Evaluation and progression of modified Ashworth score, passive ankle dorsiflexion, step length, and velocity: median (ranges)

	Kinesiotaping group	Sham group	P
Modified Ashworth score			
Baseline	3 (2 to 4)	3 (2 to 4)	.481
Week 2	2(1 to 4)	2.5 (1 to 4)	.963
Month 1	2 (1 to 4)	2.5 (0 to 3)	.606
Month 3	2 (1 to 4)	2.5 (0 to 3)	.888
Month 6	2 (2 to 4)	2.5 (0 to 4)	.963
Passive ankle dorsiflexion			
Baseline	-5 (-8 to 5)	-5 (-5 to 5)	.963
Week 2	5 (0 to 12)*	0 (-3 to 5)	.015
Month 1	8 (0 to 12)**	5 (5 to 10)**	.167
Month 3	7 (-3 to 10)**	5 (0 to 10)**	.541
Month 6	0 (-5 to 5)	-1 (-5 to 5)	.321
Step length (m)			
Baseline	0.42 (0.28 to 0.50)	0.39 (0.19 to 0.50)	.423
Week 2	0.48 (0.27 to 0.59)	0.40(0.15 to 0.59)	.423
Month 1	0.50 (0.27 to.059)*	0.43 (0.16 to 0.83)	.743
Month 3	0.50 (0.23 to 0.50)	0.44 (0.17 to 0.67)	.998
Month 6	0.48 (0.23 to 0.56)	0.40 (0.16 to 0.67)	.234
Velocity (m/s)			
Baseline	0.65 (0.18 to 0.99)	0.59 (0.13 to 1.00)	.481
Week 2	0.71 (0.21 to 1.07)	0.65 (0.10 to 1.15)	.541
Month 1	0.71 (0.19 to 1.22)**	0.74 (0.17 to 1.45)	.998
Month 3	0.80 (0.18 to 1.20)*	0.75 (0.17 to 1.45)	.888
Month 6	0.76 (0.18 to 1.08)	0.63 (0.17 to 1.45)	.743

*$P < .05$. **$P < .01$.

6 months ($P > .05$). After within-group analysis, the kinesiotaping group showed increase in step length at the first month and increase in gait velocity at first and third months. Observed MAS score decreases were not significant for controls in both groups. Intergroup comparison showed a higher benefit in passive ROM for kinesiotaping group only at 2 weeks ($P < .01$).

Discussion

This study assessed the effect of combined use of BTX-A and kinesiotaping on muscle tone, passive ankle dorsiflexion, and gait in stroke patients. All outcome variables showed similar improvements in both kinesiotaping and sham groups. No significant difference was found between groups other than passive ROM, which was found to have increased more in the kinesiotaping group at 2 weeks.

Different methods to increase the internalization and diffusion of BTX-A so as to enhance its effects have been described. Among these are various rehabilitative technologies, including stretching, functional electrical stimulation, taping, and therapeutic exercises. The sustained stretching of spastic muscles obtained by taping procedure can result in greater internalization of BTX-A (producing a muscular activation by elicitation of tonic stretch reflex) and even a positive action on the rheological properties of spastic muscles. Although combined use of BTX-A with rehabilitative treatments is accepted, there is no agreement about which treatment is the most effective in the early phase after its administration.[9,10] There are only a few studies in the literature about the effect of taping. Reiter et al showed that the combination of low doses of BTX-A with ankle-foot taping was as effective as the injection of higher doses for the reduction of foot inversion.[11] On the same line of evidence, Baricich et al reported that combining BTX administration for the ankle plantar flexors with taping had better results on the MAS and maximum ankle dorsiflexion angle in stance than stretching exercise.[10] Another study compared the efficacy of taping with electrical stimulation and splinting after BTX-A injection for wrist and finger spasticity. Taping was found to be more effective than electrical stimulation and splinting in reducing spasticity.[9] On the other hand, we did not see any significant difference between kinesiotaping and sham groups except passive ROM at 2 weeks.

In the aforementioned studies, the follow-up period ranged from 1 to 3 months. Therefore, the effectiveness of taping in enhancing the efficacy of long-term BTX use could not be evaluated.[9–11] Our study with a follow-up of 6 months can provide some information about the combined effect of BTX-A and taping in the medium and long term. This lengthened period showed that kinesiotaping provided no extra beneficial efficacy to the BTX-A.

Because there are similar improvements in all parameters in both the kinesiotape and sham groups, it seems the observed positive effect is associated with BTX-A injection. From this point of view, to objectively evaluate the effect of kinesiotaping, it is suggested that future studies should harbor a design including just a kinesiotaping group without BTX-A.

There is no consensus on techniques and frequency of tape application in the literature. Reiter et al applied an elastic tape promptly after foot-ankle BTX-A injection, then once a week for 3 times.[11] Baricich et al reported elastic adhesive tape applications to foot and thigh consecutively for 5 days.[10] In another study, adhesive taping after wrist-finger BTX-A injection was put in place for 6 consecutive days.[9] Kinesiotape can stay for 5 to 7 days on the skin,[9,12] so we applied tape immediately after injection to foot-ankle and thigh and reapplied it after 1 week.

Spasticity duration of patients in the present study ranged between 6 to 70 months. There are conflicting data about the relation between the efficacy of BTX-A injection and spasticity duration. Some authors have suggested performing BTX-A injection early after stroke[13,14] whereas others have reported success in patients with long-term spasticity.[3,15,16]

The medial and lateral heads of gastrocnemius muscle were chosen for BTX-A injection, because of its major role in equinus deformity. Although all of the plantar flexor muscles are known to contribute to the ankle plantar spasticity, different injection localizations were selected in studies investigating the effect of BTX-A in combination

with tape. For example, Baricich et al selected gastrocnemius (GCN) muscle whereas Reiter et al selected especially tibialis posterior for injection.[10,11]

The major limitation of our study is its small sample size. Double-blind design and relatively long follow-up period can be viewed as its strengths. Although both groups underwent the same physical therapy program, the amount of activity and exercise in home were not controlled, which could be a potential confounding variable. There are a few studies using tape in the treatment of spastic equinus foot problems of stroke patients and to our knowledge this is the first study using kinesiotape for this purpose.

In conclusion, the application of kinesiotaping combined with BTX-A provided no superior effect compared to sham taping with BTX-A. To fully understand the additive effect of kinesiotape on spastic equinus, further research on a large number of patients is required.

REFERENCES

1. Choi H, Sugar R, Fish DE, Shatzer M, Krabak B. *Physical Medicine and Rehabilitation Pocketpedia.* Philedelphia, PA: Lippincott Williams&Wilkins; 2003:92–96.
2. Hirsch MA, Westoff B, Toole T, Haupenthal S, Krauspe R, Hefter H. Association between Botulinum toxin injection into the arm and changes in gait in adults after stroke. *Mov Dis.* 2005; (8) 20: 1014–1020.
3. Bayram S, Sivrioglu K, Karli N, Ozcan O. Low-dose botulinum toxin with short- term electrical stimulation in poststroke spastic drop foot. *Am J Phys Med Rehabil.* 2006; 85(1):75–81.
4. Bell MS, Vermeulen LC, Sperling KB. Pharmacotherapy with botulinum toxin: harnessing nature's most potent neurotoxin. *Pharmacotheraphy.* 2000; 20:1079–1091.
5. Fu TC, Wong AMK, Pei YC, Wu KP, Chou SW, Lin YC. Effect of kinesio taping on muscle strength in athletes: a pilot study. *J Sci Med Sports.* 2008;11:198–201.
6. Kase K, Tatsuyuki H, Tomoki O. Development of Kinesio™ tape. In: *Kinesio Taping™ Perfect Manual.* Kinesio Taping Association; 1996:6–10, 117–118.
7. Yasukawa A, Patel P, Sisung C. Pilot study: investigating the effects of Kinesio Taping in an acute pediatric rehabilitation setting. *Am J Occup Ther.* 2006;60(1):104–110.
8. Halseth T, McChesney JW, DeBeliso M, Vaughn R, Lien J. The effects of kinesio taping on proprioception at the ankle. *J Sports Sci Med.* 2004;3:1–7.
9. Carda S, Molteni F. Taping versus electrical stimulation after botulinum toxin type A injection for wrist and finger spasticity. A case-control study. *Clin Rehabil.* 2005;19(6):621–626.
10. Baricich A, Carda S, Bertoni M, Maderna L, Cisari C. A single-blinded, randomized pilot study of botulinum toxin type A combined with non-pharmacological treatment for spastic foot. *J Rehabil Med.* 2008 Nov;40(10):870–872.
11. Reiter F, Danni M, Lagalla G, Ceravolo G, Provinciali L. Low-dose botulinum toxin with ankle taping for the treatment of spastic equinovarus foot after stroke. *Arch Phys Med Rehabil.* 1998;79(5):532–535.
12. Yoshida A, Kahanov L. The effect of kinesio taping on lower trunk range of motion. *Res Sports Med.* 2007;15(2):103–112.
13. Burbaud P, Wiart L, Dubos JL, et al. A randomised, double blind, placebo controlled trial of botulinum toxin in the treatment of spastic foot in hemiparetic patients. *J Neurol Neurosurg Psychiatry.* 1996;61(3):265–269.
14. Johnson CA, Burridge JH, Strike PW, Wood DE, Swain ID. The effect of combined use of botulinum toxin type A and functional electric stimulation in the treatment of spastic drop foot after stroke: a preliminary investigation. *Arch Phys Med Rehabil.* 2004;85(6):902–909.
15. Dunne JW, Heye N, Dunne SL. Treatment of chronic limb spasticity with botulinum toxin A. *J Neurol Neurosurg Psychiatry.* 199;58(2):232–235.
16. Hesse S, Krajnik J, Luecke D, Jahnke MT, Gregoric M, Mauritz KH. Ankle muscle activity before and after botulinum toxin therapy for lower limb extensor spasticity in chronic hemiparetic patients. *Stroke.* 1996;27(3):455–460.