SHORT COMMUNICATION

Neurologia i Neurochirurgia Polska
Polish Journal of Neurology and Neurosurgery
2024, Volume 58, no. 1, pages: 127–133
DOI: 10.5603/PJNNS.a2023.0042

ISSN: 0028-3843, e-ISSN: 1897-4260

KinesioTaping: impact on non-motor symptoms in cervical dystonia patients treated with botulinum toxin injection

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ABSTRACT

Aim of the study. To assess whether combined therapy with botulinum toxin injections (BoNT) and KinesioTaping could be helpful in managing non-motor symptoms (NMS) of cervical dystonia (CD).

Material and methods. Seventeen patients with CD were enrolled in this single-centre, prospective, evaluator-blinded, randomised, crossover trial. We compared three forms of treatment: BoNT treatment alone, or combined with KinesioTaping, or combined with ShamTaping. NMS were assessed using the 14-item self-reported questionnaire proposed by Klingelhoefer, the Hospital Anxiety and Depression Scale (HADS) and the Pittsburgh Sleep Quality Index (PSQI).

Results. There were no significant differences between the groups concerning mean results of HADS and PSQI scales, or mean total number of NMS after the procedures. The mean change from baseline HADS and PSQI scores, and total number of NMS after the procedure, also did not differ significantly between groups. ShamTaping combined with BoNT significantly increased the prevalence of pain.

Conclusions and clinical implications. Our study did not confirm the effectiveness of combined therapy of BoNT and KinesioTaping in the management of NMS in patients with CD. Due to a potential negative effect of improper taping on pain in CD, patients with CD should only experience KinesioTaping as an adjunctive therapy, and if it is performed by a trained, experienced physiotherapist.

Key words: cervical dystonia, non-motor symptoms, botulinum toxin injection, KinesioTaping

(*Neurol Neurochir Pol 2024; 58 (1): 127–133*)

Introduction

Cervical dystonia (CD) is the most prevalent form of adult-onset focal dystonia and is considered mostly a motor disorder [1]. Recently, attention has been drawn to the presence of non-motor symptoms (NMS) in the course of the disease.

Thirty-six percent of CD patients experience marked NMS such as sensory and perceptual abnormalities, psychiatric symptoms, pain, sleep impairment, or sexual dysfunction [2]. Pain is the most frequent NMS, reported by as many as 90% of CD patients [3], followed by disrupted sleep with a prevalence of 67.3%. Psychiatric symptoms, such as anxiety and depression, are also common, ranging from 21-65.5% and from 25-47.1% respectively [4]. Compared to motor symptoms, NMS are significantly linked to a poor quality of life [4]. Nevertheless, the relationship between motor symptoms and NMS is still being researched. It has been proposed that motor symptoms and NMS in CD could be explained by a common pathophysiological deficit. In primary CD, the core

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Received: 26.02.2023 Accepted: 5.06.2023 Early publication date: 28.06.2023

abnormality is centred in the cortico-striatal-thalamo-cortical circuits. Its non-motor consequences would be expected given that these circuits have been linked not only to motor, but also to sensory, cognitive, and reward processing [5].

Intramuscular botulinum toxin injections (BoNT) are the treatment of choice for motor symptoms in CD. However, BoNT treatment meets only limited patient satisfaction [6]. The therapeutic response becomes apparent within 1-2 weeks after the BoNT injection, with peak effects at approximately 4-6 weeks and a gradual decline thereafter [7, 8]. Thus, patients with CD treated with BoNT experience a 'rollercoaster' effect, as they receive treatment with waning effectiveness over time that then increases again following the subsequent injection [9]. It might be useful to combine BoNT with an adjunctive therapy for a beneficial synergy.

Only a few studies have investigated the effect of BoNT on NMSs in CD patients [10, 11]. Evidence for the effectiveness of rehabilitation strategies in CD patients is also scarce. Kinesiology taping involves a combination of tension applied along the tape and stretching of the target muscle. That, amongst others, results in a change of recruitment activity patterns of the muscles, and alleviates prolonged muscle contraction and even postural deviation [12].

To the best of our knowledge, the effect of KinesioTaping on NMSs in patients with focal dystonia has so far been analysed in only one study [13].Our current study is a continuation of our recently published research [14]. Our observations help to elucidate the possible role of combined BoNT-A plus KinesioTaping therapy in the management of NMS in patients with CD.

Material and methods

This was a single-centre, prospective, evaluator-blinded, randomised, crossover trial. The participants were recruited from the Movement Disorders outpatient clinic of the Department of Neurology of the University Hospital in Kraków, Poland between January 2019 and January 2021. The study included patients with CD previously treated with BoNT within Poland's National Health Fund (NHF) programme. Patients with segmental, multifocal, generalised dystonia and hemidystonia, with a history of invasive dystonia treatment (deep brain stimulation or neck surgeries), with contraindications for KinesioTaping (wounds, fresh scars, allergies to acrylic glue, tape intolerance), or who were still undergoing diagnostic evaluation for dystonia, were all excluded from the study. All patients provided written informed consent.

We obtained demographic characteristics and a medical history and performed a neurological examination during the initial visit. Participants were then randomly assigned 1:1:1 to one of three groups using a computer-generated random number:

- Group 1: BoNT + KinesioTaping
- Group 2: BoNT + ShamTaping
- Group 3: BoNT + no taping

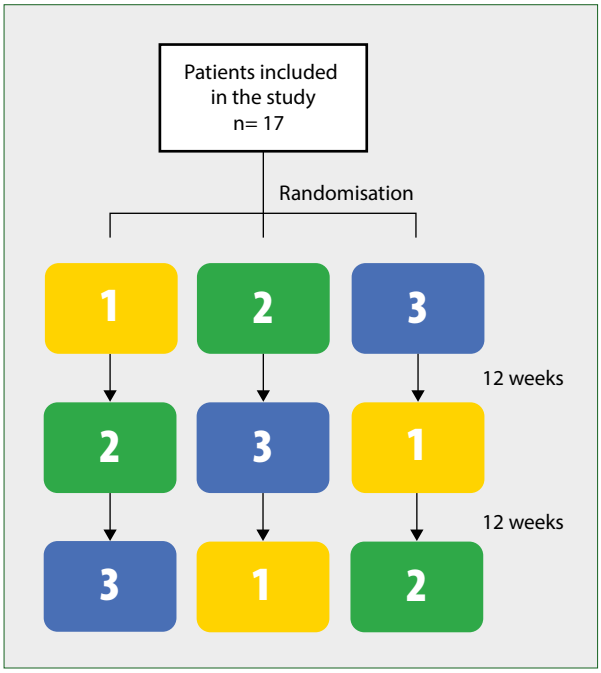

Figure 1. Study design. Group 1 = botulinum toxin injections (BoNT) + KinesioTaping; Group 2 = BoNT + ShamTaping; Group 3 = BoNT

Every 12 weeks, the participants were switched to another treatment group, so as to apply all treatment options to all patients over the course of 36 weeks (Fig. 1). The KinesioTaping and ShamTaping and no taping arms were included to assess the possible placebo effect of the taping.

BoNT injections were received by patients at the beginning of the 12-week cycle. The injection pattern was individual and based on the patient's cervical dystonia subtype according to the collum-caput (*Col-Cap*) concept [15]. In each cycle, patients received the same dose and same brand of BoNT in the same localisation. The injections were ultrasound-guided. BoNT preparations were used, depending on the individual tolerance and availability at the hospital: onabotulinumtoxin type A or abobotulinumtoxin type A.

In Groups 1 and 2, the BoNT injection was followed by KinesioTaping or ShamTaping respectively, which was performed after seven days and continued for four consecutive weeks, with tapes being changed once per week by an experienced physiotherapist informed about the patient's group allocation as described previously [14].

In Group 1, the tape was applied in the direction of fascial restriction. The physiotherapist slid a fascia over an individual muscle or group of synergistically acting muscles and assessed the presence of involuntary movements of the head and neck and the posture of cervical-thoracic (C-Th) spine and shoulder girdle, gluing the tape when an improvement was seen. Additionally, patients were taped using the analgesic (ligament) technique in the area of the C-Th spine or the shoulder

complex (depending on which was subjectively indicated as being more painful by the patient). Analgesic taping was executed with a single transverse application or double cross application (applying the central part of the tape with 75-100% tension and two ends without tension). If a patient did not report any pain, this application was omitted.

In Group 2, patients were taped in a non-therapeutic manner, which means applying the tape without tension and without stretching the muscles or moving the head and neck. The tape was applied in two vertical slices and one horizontal slice glued to the C-Th area of the spine. Patients were unable to feel the difference between KinesioTaping and ShamTaping during application.

Participants were assessed by a neurologist twice per cycle: firstly on the day of the BoNT injection, and secondly during the control visit six weeks later. The patient and the assessing neurologist were unaware of the group allocation. During the assessment visits, information on NMSs was gathered according to a 14-item self-report questionnaire [2]. The patients' mood and the presence of anxiety were assessed using the Hospital Anxiety and Depression Scale. The presence of sleep impairment was evaluated with the Pittsburgh Sleep Quality Index (PSQI). Disease severity was assessed using the Toronto Western Spasmodic Torticollis Rating scale (TWSTRS).

Data was gathered in a database and statistical analysis was performed using a PS Imago Pro 6.0 statistical package. Categorical data was presented as counts and percentages, and continuous data as mean and standard deviation. Chi-Square test was used to compare the prevalence of individual NMS after interventions. McNemar test was used to assess the influence of each intervention on the prevalence of individual NMS within the group. Continuous variables were compared using a nonparametric Kruskall-Wallis test (due to a limited sample size). Differences were considered statistically significant with the two-sided p-value of less than 0.05.

Ethical approval was granted by the institutional review board (opinion number 1072.6120.217.2018).

Results

The groups consisted of 17 patients aged 29–72 with a mean age of 53.5 (± 12.77) years. 13/17 (76.47%) were female. Age at dystonia onset varied from 23 to 58 years with a mean 40.47 (± 11.42) years. Disease duration was 4–47 years with a mean 13.18 (± 12.01) years. Three patients were diagnosed with concomitant depression, for which two were receiving pharmacotherapy and one was in remission. Each patient received a stable dose of BoNT, 14 of the patients were treated with onabotulinum type A (mean dose: 186.4 SD = 31.0 units), and the other three with abobotulinum type A (1,000 units). None was treated with physiotherapy or psychotherapy before or during the study.

Before interventions, 92.16% of patients declared that they experienced NMSs. The most common NMS was fatigue/lack of energy limiting everyday activity. The distribution of individual NMSs before and after intervention in each group is presented in Table 1. The analysis using McNemar test showed that, in Group 2, ShamTaping combined with BoNT significantly increased the prevalence of pain. The procedures in each group had no statistically significant effect on the prevalence of any other NMS. The prevalence of individual NMSs in the control assessment was compared between groups using a Chi-square test. No statistically significant differences were found.

The results of the Kruskall-Wallis test showed that the mean total number of NMSs after interventions and the mean change from baseline number of NMSs after interventions did not differ significantly between groups. There were also no statistically significant differences concerning the mean scores obtained with TWSTRS, PSQI, HADS-A, HADS-D and HADS-total scales after intervention or in the mean change from baseline score after intervention (see Table 2, supplementary material).

Discussion

In this study on NMS of KinesioTaping in patients with CD, we did not observe superior efficacy of taping as an adjunctive therapy to BoNT injection versus BoNT alone. We noticed significant worsening of pain reported by the group treated with BoNT and ShamTaping. Fatigue and lack of energy limiting everyday activity was the most common complaint, with a prevalence of 47.1%. This aligns with the literature, where the prevalence of this symptom ranges from 46% to 64% [16].

Pelosin et al. evaluated the effectiveness of KinesioTaping on NMSs in patients with focal dystonia not treated with BoNT [13]. Compared to ShamTaping, KinesioTaping decreased the subjective sensation of pain and modified the ability of sensory discrimination. CD patients treated with ShamTaping had higher results on the VAS-W scale (assessing the worst pain) after intervention than they did at baseline.

It has been proven that BoNT injection significantly reduces pain associated with CD [17] and other neurological conditions such as migraine [18]. KinesioTaping has been found to be effective in decreasing pain in musculoskeletal disorders [19].

The direct antinociceptive mechanism of BoNT in CD is unclear, although several hypotheses have been proposed. BoNT affects muscle spindles acting as proprioceptors. BoNT-induced relaxation of hypertonic muscles contributes to decompression of nerve fibres, thus decreasing afferent activity of spindles and reducing excitability of motoneurons [20]. Relief of local ischaemia, secondary to muscle relaxation, reduces lactate production, and diminishes traction-related and positional pain [20–22]. BoNT may also inhibit neurogenic inflammation and peripheral sensitisation by inhibiting the release of local neuropeptides involved in pain transmission

Table 1. Impact of treatment on non-motor symptoms in studied group

	Group 1 (BoNT + KinesioTaping)			Group 2 (BoNT + ShamTaping)			Group 3 (BoNT)		
	Before intervention	After intervention		Before intervention	After intervention		Before intervention	After intervention	
Lack of confidence, social withdrawal [n (%)]	8 (47.1%)	**7 (41.2%)**	p = 1.000	7 (41.2%)	**8 (47.1%)**	p = 1.000	8 (47.1%)	**5 (29.4%)**	p = 0.250
Problems with falling and/or staying asleep [n (%)]	7 (41.2%)	**6 (35.3%)**	p = 1.000	6 (35.3%)	**5 (29.4%)**	p = 1.000	7 (41.2%)	**7 (41.2%)**	p = 1.000
Insomnia [n (%)]	2 (11.8%)	**4 (23.5%)**	p = 0.500	3 (17.6%)	**5 (29.4%)**	p = 0.625	3 (17.6%)	**5 (29.4%)**	p = 0.625
Fatigue/lack of energy limiting everyday activity [n (%)]	11 (64.7%)	**8 (47.1%)**	p = 0.375	10 (58.8%)	**12 (70.6%)**	p = 0.500	10 (58.8%)	**10 (58.8%)**	p = 1.000
Problems with gait [n (%)]	3 (17.6%)	**4 (23.5%)**	p = 1.000	0 (0.0%)	**2 (11.8%)**	p = 0.500	4 (23.5%)	**2 (11.8%)**	p = 0.500
Problems with balance [n (%)]	6 (35.3%)	**6 (35.3%)**	p = 1.000	7 (41.2%)	**4 (23.5%)**	p = 0.250	6 (35.3%)	**8 (47.1%)**	p = 0.500
Vertigo/dizziness [n (%)]	6 (35.3%)	**4 (23.5%)**	p = 0.625	5 (29.4%)	**4 (23.5%)**	p = 1.000	7 (41.2%)	**7 (41.2%)**	p = 1.000
Pain (not caused by known comorbidities) [n (%)]	6 (35.3%)	**4 (23.5%)**	p = 0.625	3 (17.6%)	**10 (58.8%)**	p = 0.016	7 (41.2%)	**9 (52.9%)**	p = 0.625
Feeling tired after sleeping for whole night [n (%)]	6 (35.3%)	**6 (35.3%)**	p = 1.000	7 (41.2%)	**7 (41.2%)**	p = 0.727	7 (41.2%)	**5 (29.4%)**	p = 0.625
Anxiety [n (%)]	9 (52.9%)	**9 (52.9%)**	p = 1.000	11 (64.7%)	**11 (64.7%)**	p = 1.000	9 (52.9%)	**10 (58.8%)**	p = 1.000
Lowered mood, depression [n (%)]	2 (11.8%)	**4 (23.5%)**	p = 0.500	6 (35.3%)	**4 (23.5%)**	p = 0.500	5 (29.4%)	**5 (29.4%)**	p = 1.000
Paraesthesia [n (%)]	5 (29.4%)	**4 (23.5%)**	p = 1.000	7 (41.2%)	**9 (52.9%)**	p = 0.687	7 (41.2%)	**5 (29.4%)**	p = 0.625
Dysphagia [n (%)]	3 (17.6%)	**3 (17.6%)**	p = 1.000	3 (17.6%)	**6 (35.3%)**	p = 0.375	4 (23.5%)	**5 (29.4%)**	p = 1.000
Dysarthria [n (%)]	1 (5.9%)	**3 (17.6%)**	p = 0.625	3 (17.6%)	**3 (17.6%)**	p = 1.000	4 (23.5%)	**3 (17.6%)**	p = 1.000
Problems with vision [n (%)]	3 (17.6%)	**3 (17.6%)**	p = 0.625	3 (17.6%)	**3 (17.6%)**	p = 1.000	5 (29.4%)	**4 (23.5%)**	p = 1.000
Mood swings [n (%)]	6 (35.3%)	**5 (29.4%)**	p = 1.000	10 (58.8%)	**7 (41.2%)**	p = 0.375	7 (41.2%)	**6 (35.3%)**	p = 1.000
Any of above symptoms [n (%)]	15 (88.24%)	**16 (94.12%)**	p = 1.000	16 (94.12%)	**16 (94.12%)**	–	16 (94.12%)	**15 (88.24%)**	p = 1.000

Table 2. Mean results of HADS, PSQI, TWSTR and total number of NMS after procedures. Mean differences in results of HADS, PSQI, TWSTR and total number of NMS before and after procedures

	Group 1 (BoNT + KinesioTaping)	Group 2 (BoNT + ShamTaping)	Group 3 (BoNT)	Kruskall-Wallis test
Total number of NMS after intervention	4.71 (+/– 3.58)	5.88 (+/– 3.87)	5.65 (+/– 4.33)	p = 0.646
ΔNMS	–0.24 (+/– 2.7)	0.53 (+/– 2.07)	–0.24 (+/– 2.56)	p = 0.668
Total PSQI after intervention	6.12 (+/– 3.44)	6.18 (+/– 3.38)	5.76 (+/– 3.88)	p = 0.898
ΔPSQI	0.76 (+/– 3.17)	0.18 (+/– 2.74)	–0.35 (+/– 2.52)	p = 0.667
HADS-A after intervention	6.47 (+/– 3.99)	6.53 (+/– 3.63)	5.47 (+/– 4.16)	p = 0.608
HADS-D after intervention	5.06 (+/– 3.07)	5.35 (+/– 4.11)	5.29 (+/– 4.33)	p = 0.987
Total HADS after intervention	11.53 (+/– 6.19)	11.88 (+/– 6.97)	10.76 (+/– 7.54)	p = 0.746
ΔHADS-A	–1.12 (+/– 2.91)	–0.94 (+/– 1.82)	0.06 (+/– 1.78)	p = 0.296
ΔHADS-D	0.00 (+/– 1.23)	–0.12 (+/– 2.09)	0.65 (+/– 2.21)	p = 0.395
ΔTotal HADS	–1.12 (+/– 3.60)	–1.06 (+/– 2.68)	0.71 (+/– 3.44)	p = 0.162
Total TWSTRS after intervention	16.06 (+/– 9.01)	17.76 (+/– 9.40)	14.76 (+/– 8.31)	p = 0.733
ΔTotal TWSTRS	–15.39 (+/– 8.52)	–14.49 (+/– 7.12)	–14.09 (+/– 4.34)	p = 0.771

from sensory nerves (substance P, calcitonin gene-related peptide, glutamate, and transient receptor potential vanilloid) [23–24]. The evidence suggests that changes to the afferent input caused by BoNT may result in short-term and long-term plastic changes in the network associated with pain in CD, causing a therapeutic effect [21]. A recent study has shown that the antinociceptive BoNT effect may last longer than motor improvements [25].

The mechanism of KinesioTaping is not yet fully understood. Sensorimotor, proprioceptive feedback mechanisms, inhibitory and excitatory nociceptive stimuli, and mechanical restraint have all been suggested [26–28]. We hypothesise that incorrect tape application may activate other muscles, such as deep-seated cervical muscles with high densities of muscle spindles. In turn, change in muscle tone may cause excitability of motoneurons and additionally mechanoreceptors and nociceptors activation, thus modifying pain transmission.

Slawek et al. reported depression in 47.5% of CD patients that was improved after BoNT-A treatment [29]. Costanzo et al. reported significant improvements in psychiatric disturbances, pain, and disability, whereas sleep disorders remained unchanged one month after BoNT-A injection in CD patients. Our study did not confirm these results.

In both the aforementioned studies, NMS assessment was performed at baseline (a washout period meaning 16 weeks after the last BoNT-A injection) and one month, or in Costanzo's cohort, three months, after BoNT-A. It is known that BoNT-A's effects wear off at 16 weeks, but that they are still present to a lesser extent after 12 weeks [30]. In the current study, there was no such 'washout period' before the intervention, due to ethical concerns. Poland's National Health Fund Programme approves BoNT injection every 12 weeks in CD patients. Thus, we did not notice any significant change from baseline after the treatment in NMS assessment.

Sleep disturbances are usually persistent and can be due to varying confounding factors such as environmental factors, occupational factors, physiological changes, medical and psychiatric disorders. It is possible that the short-term prospective design of the study looking at them did not allow reliable results to be obtained [31].

This study has some limitations such as a small sample size. Accordingly, this research was designed to be evaluator-blinded, randomised and crossover to overcome the weakness of the small number of patients participating in the study. Its short-term prospective design did not allow us to observe BoNT-induced changes beyond its timeframe. As mentioned above, we did not pursue a 16-week washout period after the previous BoNT injection, and thus we cannot fully exclude BoNT's impact on the study's baseline assessment. Moreover, we used different types of BoNT depending on individual tolerances and medication availability at the hospital, which may have had an impact on the results [32, 33].

To conclude, our study showed that combined BoNT and KinesioTaping therapy was not effective in the management of NMS in patients with CD. It is worth underlining that incorrect KinesioTaping application can actually worsen pain in CD patients.

Clinical implications/future directions

Our results require confirmation in larger studies. Due to a potential negative effect of improper taping on pain in CD, patients with CD should only experience KinesioTaping as an adjunctive therapy and if it is performed by a trained, experienced physiotherapist.

Conflicts of interest: *Malgorzata Dec-Cwiek has received speaking fees from IPSEN Poland sp z o. o., AbbVie sp. z o. o.,*

and Merz Pharmaceuticals. Karolina Porebska has received speaking fees from IPSEN Poland sp z o. o. The other authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.
Funding: *The authors received a grant from CMUJ for the research and/or publication of this article (N41/DBS/000200).*
Acknowledgements: *We thank all those who took part in this project.*

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