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ORIGINAL RESEARCH

Effectiveness of a Treatment Involving Soft Tissue Techniques and/or Neural Mobilization Techniques in the Management of Tension-Type Headache: A Randomized Controlled Trial

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Abstract

Objective: To evaluate the effects of a protocol involving soft tissue techniques and/or neural mobilization techniques in the management of patients with frequent episodic tension-type headache (FETTH) and those with chronic tension-type headache (CTTH).

Design: Randomized, double-blind, placebo-controlled before and after trial.

Setting: Rehabilitation area of the local hospital and a private physiotherapy center.

Participants: Patients (N=97; 78 women, 19 men) diagnosed with FETTH or CTTH were randomly assigned to groups A, B, C, or D.

Interventions: (A) Placebo superficial massage; (B) soft tissue techniques; (C) neural mobilization techniques; (D) a combination of soft tissue and neural mobilization techniques.

Main Outcomes Measures: The pressure pain threshold (PPT) in the temporal muscles (points 1 and 2) and supraorbital region (point 3), the frequency and maximal intensity of pain crisis, and the score in the Headache Impact Test-6 (HIT-6) were evaluated. All variables were assessed before the intervention, at the end of the intervention, and 15 and 30 days after the intervention.

Results: Groups B, C, and D had an increase in PPT and a reduction in frequency, maximal intensity, and HIT-6 values in all time points after the intervention as compared with baseline and group A ($P<.001$ for all cases). Group D had the highest PPT values and the lowest frequency and HIT-6 values after the intervention.

Conclusions: The application of soft tissue and neural mobilization techniques to patients with FETTH or CTTH induces significant changes in PPT, the characteristics of pain crisis, and its effect on activities of daily living as compared with the application of these techniques as isolated interventions.

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Tension-type headache (TTH) is the most prevalent form of benign primary headache.[1] The prevalence of episodic TTH is 33.8% in a year and of chronic TTH 2.3%,[2] with TTH being the second most prevalent pathology in the world.[3] These situations make TTH a shocking disorder in its social and economic aspects.[4]

To explain the whole symptomatology, researchers[5,6] refer to peripheral sensitization that involves myofascial pain in the craniocervical muscule, as well as higher mechanical sensitivity in the nerve trunks. However, as a consequence of the continuous nociceptive afferents, previous studies[7-9] also suggest central

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sensitization with an alteration in the processing and/or inhibitory mechanisms of pain that set the chronic character of this pathology, with the trigeminal caudal nucleus (TCN) being one of the structures that can be sensitized.

Despite the significant effect of TTH, studies[10] to date have not established the best treatment to manage the symptomatology. Although previous studies[11] reported benefits of manual therapy by including soft tissue techniques to manage the myofascial pain, these studies were often low quality, making it difficult to draw clear conclusions.

Neural mobilization techniques intend to improve adaptability, reduce mechanosensitivity, and activate analgesic mechanisms by mechanically stimulating the nerves with palpation, elongation, and sliding.[12-14] In this regard, previous studies[15,16] have shown that increases in mechanosensitivity may induce pain with neuropathic, nociceptive, and mixed characteristics[14] as well as increases in muscle contraction. For this reason, therapies that mechanically stimulate the nervous tissue could decrease the local mechanosensitivity and increase the mechanical tolerance as a consequence of the activation of the central mechanisms of analgesia.[12-14,17-20] However, to our knowledge, no studies to date have included this type of intervention in the management of TTH.

Based on these arguments, the present study aimed to analyze the effects of a protocol involving soft tissue techniques combined or not with neural mobilization techniques in the management of patients with frequent episodic tension-type headache (FETTH) and those with chronic tension-type headache (CTTH). It was hypothesized that the combination of both therapies is more effective in reducing the sensitivity of the neuromusculoskeletal structures and thus improves the central sensitization and chronic trend of this pathology as compared with the isolated techniques.

Methods

Design

The present study refers to a double-blind randomized controlled trial with 4 intervention groups.

Participants

Participants were recruited randomly from the local hospital and other health centers from the region.

The inclusion criteria for this study were as follows: patients aged between 18 and 65 years and diagnosed with FETTH and CTTH with increased pericranial tenderness on manual palpation by neurologists according to the *International Classification of Headache Disorders*.[21]

The exclusion criteria for this study were as follows: patients with impossibility of receiving manual therapy; patients with previous physiotherapy treatment for their TTH; and patients receiving pharmacologic prophylactic treatment 2 months before the beginning of the study.

Patients were told not to take medication unless they had an increase in symptoms with a visual analog scale value ranging from 6 to 7 and then they could take ibuprofen 400mg, 1 or 2 doses maximum to go through the crisis.

This study was performed between December 2, 2013, and March 27, 2015, in the local hospital and a private physiotherapy clinic. Before the beginning of the study, all participants signed an informed consent according to the Declaration of Helsinki.[22] The study was approved by the Clinical Investigation Ethical Committee of the Balearic Islands.

List of abbreviations:

CTTH	chronic tension-type headache
FETTH	frequent episodic tension-type headache
HIT-6	Headache Impact Test-6
PPT	pressure pain threshold
TCN	trigeminal caudal nucleus
TTH	tension-type headache

Interventions

Six 15-minute sessions were given to every patient: 2 in the first week, 2 in the second week, and 1 each in the third and the fourth week. Patients were randomly assigned to group A (placebo superficial massage: n=25), group B (soft tissue techniques: n=25), group C (neural mobilization techniques: n=25), or group D (combined treatment involving soft tissue and neural mobilization techniques: n=25). EpiData software v.4.0[a] was used to randomize the intervention to each participant. The randomization sequence was guarded by an independent collaborator who guaranteed its concealment. Also, every intervention was blinded for both participants and evaluators, and physiotherapists who administered the treatment were blinded to the objectives of the investigation.

Protocol: Placebo superficial massage

A physiotherapist gave a soft and superficial massage while patients were in the prone position. The physiotherapist used ultrasound gel to minimize skin stimulation while performing multidirectional gliding in the thoracic region of the patients' back, without overstepping the D1 spinous process in the cranial direction. The protocol lasted 15 minutes.

Protocol: Soft tissue techniques

A physiotherapist expert in manual therapy treated 5 muscles in the craniocervical region. The protocol lasted 15 minutes (3min in each pair of muscles). Patients should not feel pain greater than 2 points on the visual analog scale (0−10 points). The techniques were randomly applied in the following order: sternocleidomastoid muscle, temporal muscle, suboccipital muscule, masseter muscle, and upper trapezius muscle (fig 1).

Protocol: Neural mobilization techniques

A physiotherapist expert in manual therapy performed 3 neural mobilization techniques, whose performance was always mild, progressive, and slow. The protocol lasted 15 minutes (5min of every mobilization). Patients should not feel pain greater than 2 points on the visual analog scale (0−10 points). The techniques were applied in the following order:

- Mobilization in craniocervical flexion: the physiotherapist performed an anterior rotation of the head, which stimulates the meninges.[23] To increase mechanical stress in the nervous system, patients were asked to do a descent and retropulsion of the shoulders while gradually extending both elbows (fig 2).

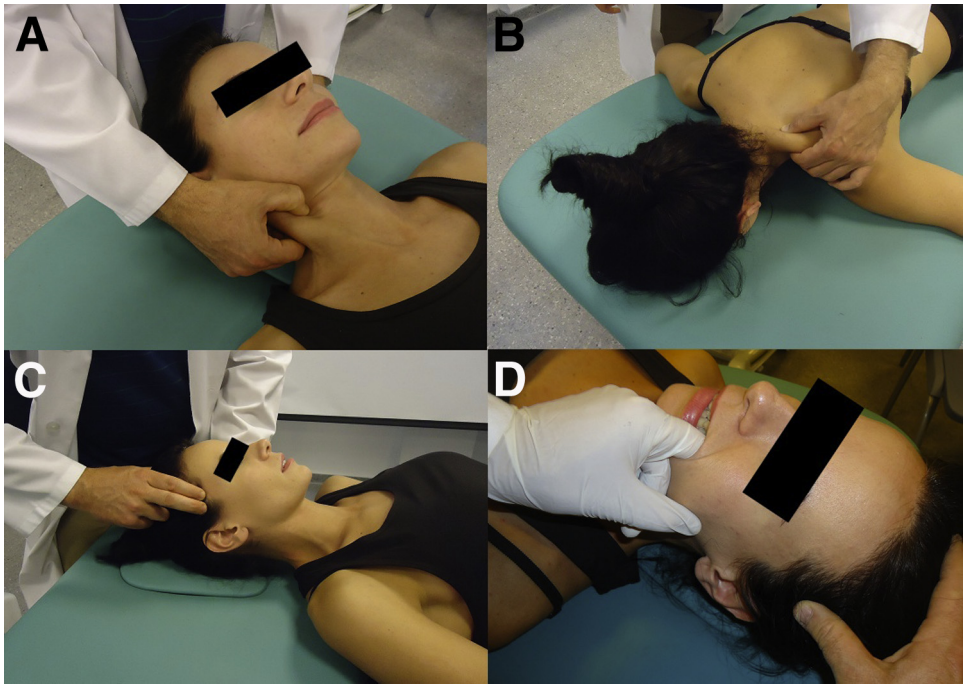

Fig 1 Soft tissue techniques: (A) sternocleidomastoid muscle, (B) upper trapezius muscle, (C) temporal muscle, and (D) masseter muscle.

- Lateral cervical sliding: on the basis of the technique described by Elvey,[24] the physiotherapist laterally slide the cervical region of the patient. The purpose of this movement was to stimulate the brachial plexus. Patients were allowed to move their shoulders. To increase mechanical stress in the nervous system, patients were asked to progressively extend their elbows, followed by forearm supination and a dorsal flexion of the carpal and fingers (fig 3).
- Opening the mouth in craniocervical flexion: the physiotherapist passively held craniocervical flexion with one hand, while the other hand opened the mouth (passive-assisted). The opening of the mouth increases the deformation of the trigeminal nerve, mainly the mandibular branch.[25] To increase mechanical stress in the nervous system, patients were asked to progressively extend their elbows, followed by forearm supination and a dorsal flexion of the carpal tunnel and fingers (fig 4).

Protocol: Combined protocol involving soft tissue and neural mobilization techniques

A physiotherapist expert in manual therapy performed a combination of both protocols: soft tissue and neural mobilization techniques. The protocol lasted 15 minutes. The techniques included were the same as those added in the previous protocols; however, the duration was shorter to adjust the total duration of the protocol to 15 minutes and avoid skewing this protocol's effects: 7.5 minutes of neural mobilization techniques and 7.5 minutes of soft tissue techniques.

Outcome measures

Frequency of crisis

Patients were given 15-day diaries. One diary was given 2 weeks before the first session (premeasurement[26]), another one after the fourth session (evaluation 1h after the latest session), another one after the sixth session (evaluation 15d later), and the last one 15 days after the sixth session (evaluation 30d later). The diary had to be filled every day in the morning, afternoon, and night to inform if they had headache.

Maximal intensity of pain

Patients informed about this variable according to a visual analog scale (0: no pain; 10: maximum pain) placed in the diaries of

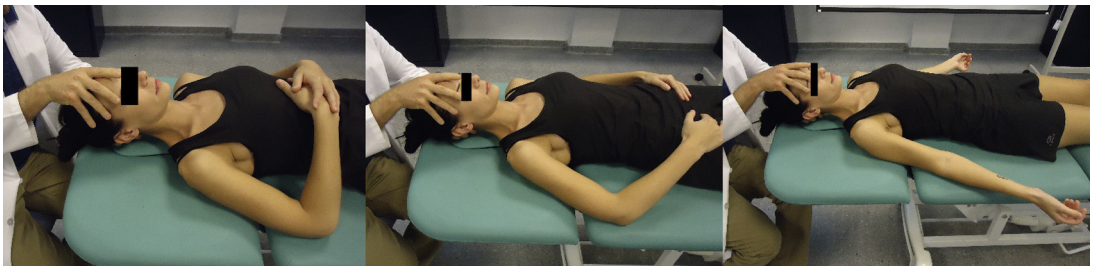

Fig 2 Progression of the mobilization in craniocervical flexion.

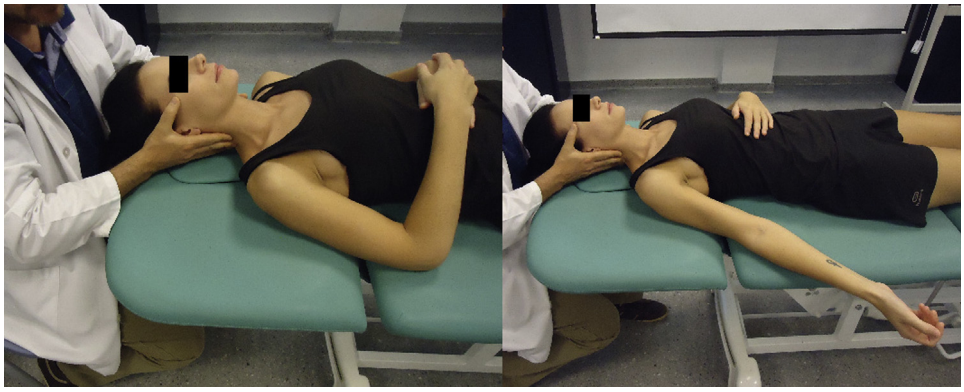

Fig 3 Progression of the lateral cervical sliding.

headache frequency. If they felt headache while filling this information in the diary, they had to record the maximal intensity of that pain perceived during that crisis by using the visual analog scale. The maximal intensity of pain was recorded 3 times every day (morning, afternoon, night), such as the frequency of crisis was collected. The maximal intensity of pain was obtained from the average of the 3 highest values in each diary.[27]

Pressure pain threshold

We used an electronic pressure algometer (Commander Muscle Tester[b]) with a stimulation surface area of 1cm^2. Its reliability and validity have been proven previously.[28] The pressure pain threshold (PPT) was analyzed in 3 points:

1. Temporal muscle (point 1): 3cm above the upper margin of the ear, vertical to the ear canal.[29]
2. Temporal muscle (point 2): 1cm in front of point 1.[29]
3. Supraorbital nerve emerging (point 3): it can be located between the medial third and the middle third of the frontal bone edge.[6]

The PPT was assessed 3 times in each point, with an interval of 30-second rest. To obtain the final measure, the highest trial was discarded and the other 2 trials in each point were averaged.[6,29-31] This variable was evaluated before the beginning of the study, 1 hour after the latest session, 15 days later, and 30 days later.

Headache Impact Test-6

Its reliability to evaluate the effect of headache on patients' activities of daily living has been proven in previous studies.[32] It consists of 6 items with 5 response options: never, 6 points; rarely, 8 points; sometimes, 10 points; very often, 11 points; always, 13 points, with a total score ranging from 36 to 78 points.[33] This variable was evaluated before the beginning of the study, 1 hour after the latest session, 15 days later, and 30 days later.

Statistical analysis

The sample size calculation was performed with GRANMO 7.12 software[c] for the punctuation in the Headache Impact Test-6 (HIT-6) because of its capacity to evaluate the effect of headache on patients' activities of daily living and because of its relation with key aspects of the symptomatology, such as headache severity and quality of life.[33] An α level of .05 and a desired power (β) of 80% with a bilateral contrast were assumed. These assumptions generated a sample size of at least 23 participants per group to detect a minimal difference of 6 between 2 groups and an SD of 5.52. Losses during follow-up were estimated at 10%.

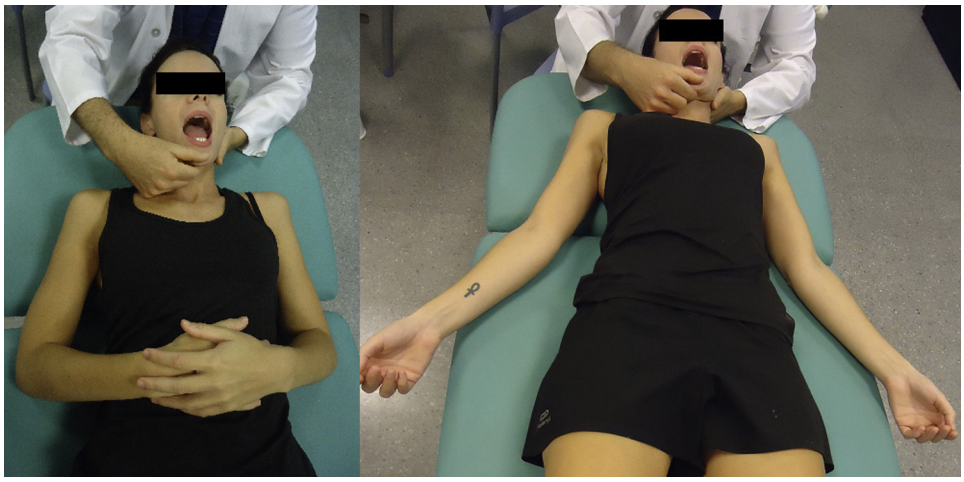

Fig 4 Opening the mouth in craniocervical flexion.

Demographic and clinical characteristics of the groups were compared using 1-way analysis of variance for quantitative variables and the chi-square test for categorical variables.

Mixed model repeated-measures analysis of variance was used to determine whether any change in PPT, frequency and maximal intensity of pain crisis, and HIT-6 values is the result of the interaction between the type of intervention (no treatment, soft tissue treatment, neural mobilization treatment, combined treatment) and time. Analysis included within-patient variables (the time of measurement with 4 levels: before, immediately after, 15d after, 30d after the intervention) and between-patient variables (the intervention with 4 levels: no treatment, soft tissue treatment, neural mobilization treatment, combined treatment).

Cohen's *d* was used to calculate and interpret the effect size of mean differences. The effect size was rated as follows: small (0.2−0.5), medium (0.5−0.8), and large (>0.8).

The percentage of individual patients achieving ≥50% improvements for every group was calculated to determine the clinical relevance of improvements in the frequency of crisis.

Results

Ninety-seven participants (78 women; 19 men) aged 19 to 60 years (mean age, 39.7±11.5y; body mass index, 25.0±3.2kg/m^2) and diagnosed with TTH were included in this study (table 1). All the groups were comparable with respect to the clinical and anthropometric variables (P>.05). The flow diagram is presented in figure 5.

The linear mixed model analysis revealed a significant group × time interaction for PPT in points 1, 2, and 3 (P<.001), in which patients treated with neural mobilization techniques (group C), soft tissue techniques (group B), or the combination (group D) experienced an increase of 41.7% (d=.79), 48.6% (d=.71), and 63.5% (d=.91), respectively, as compared with baseline measurements in point 1 of the temporal muscle, 44.8% (d=.73), 54.0% (d=.80), and 63.4% (d=.97) in point 2 of the temporal muscle, respectively, and 63.0% (d=.86), 48.6% (d=.72), and 67.5% (d=.90) in the supraorbital region, respectively (P<.001). The between-group differences showed that the control group (group A) had statistically significant lower values in all post-intervention measurements than did the rest of the groups (P<.001). Also, in point 2 of the temporal muscle and in the supraorbital region, patients treated with the combined protocol (group D) experienced a significant increase as compared with the rest of the groups at the evaluation 1 hour after the latest session, evaluation 15 days later, and evaluation 30 days later time points (P<.001) (table 2). No difference in PPT was found with respect to only group B or group C (P>.05 for all cases).

The linear mixed model analysis revealed a significant group × time interaction for the frequency and maximal intensity of pain crisis in points 1, 2, and 3 (P<.001), in which patients treated with neural mobilization techniques (group C), soft tissue techniques (group B), and the combined protocol (group D) experienced a maximum reduction of 45.2% (d=1.7), 47.5% (d=2.1), and 57.2% (d=2.1), respectively, as compared with baseline measurements in frequency (P<.001) and experienced a maximum reduction of 37.2% (d=1.6), 30.0% (d=1.9), and 43.6% (d=2.2), respectively, as compared with baseline measurements in maximal intensity. The between-group differences showed that the control group had higher values of frequency and maximal intensity than did the rest of the groups in all the post-intervention measurements (P<.001) and the group receiving the combined treatment had statistically significant lower values than did the soft tissue treatment group (P<.01) (table 3). Participants who received the sham intervention (group A) also showed significant differences as compared with baseline measurements in frequency, with a reduction of 6.9% (d=0.2), and in maximal intensity, with a reduction of 4.1% (d=0.2) (P>.05). No difference in frequency, maximal intensity, or HIT-6 values was found with respect to only group C (P>.05 for all cases).

An additional analysis to calculate the individual percentage of improvements in frequency of crisis showed that 13 participants from group B, 14 participants from group C, and 24 participants from group D achieved ≥50% improvements in the frequency of crisis after the intervention. No participant from group 1 achieved 50% of improvements in the frequency of crisis (supplemental tables S1−S4, available online only at http://www.archives-pmr.org/).

Table 3 also shows that the linear mixed model analysis revealed a significant group × time interaction for punctuation in the HIT-6 (P<.001), with patients receiving soft tissue treatment (group B), neural mobilization treatment (group C), or the combined treatment (group D) experiencing a maximum reduction of 13.1% (d=1.48), 13.5% (d=1.48), and 16.3% (d=1.57), respectively, as compared with baseline measurements. Furthermore, participants who received the sham intervention showed significant differences, with a reduction of 4.7% (d=.53) as compared with baseline measurements (P<.05). The between-group differences showed that the control group (group A) had

Table 1 Anthropometric characteristics of participants

Variable	Group A (n=24)	Group B (n=23)	Group C (n=25)	Group D (n=25)
Sex				
Female	20 (83.3)	17 (74.0)	20 (80.0)	21 (84.0)
Male	4 (16.7)	6 (26.0)	5 (20.0)	4 (16.0)
TTH				
FETTH	14 (58.3)	15 (65.2)	14 (56.0)	13 (52.0)
CTTH	10 (41.7)	8 (34.8)	11 (44.0)	12 (48.0)
Age (y)	40.5±12.0	38.1±10.9	39.4±11.0	40.8±12.1
Height (m)	1.70±0.07	1.66±0.01	1.64±0.08	1.63±0.06
Weight (kg)	69.2±10.2	68.2±11.8	67.9±12.1	66.3±7.8
BMI (kg/m^2)	25.3±3.0	24.7±3.4	25.1±3.3	24.9±3.0

NOTE. Values are mean ± SD or n (%).
Abbreviation: BMI, body mass index.

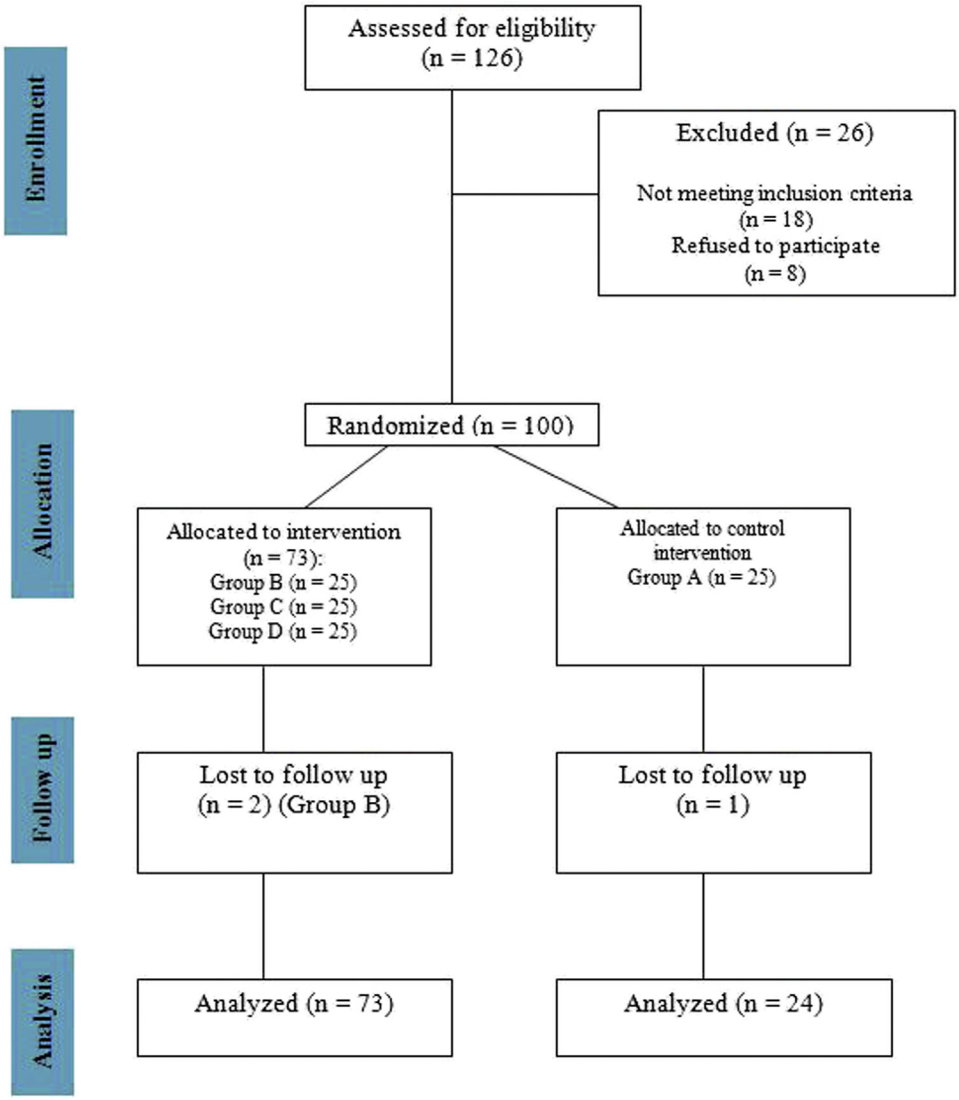

Fig 5 Flow diagram.

statistically significant lower values in all the postintervention measurements than did the rest of the groups ($P<.001$).

Of the 21 participants who took medication in their crisis episodes during the study period, 9 belonged to the control group, 3 to the neural mobilization technique group, 5 to the soft tissue technique group, and 4 to the combined protocol group. All these 21 patients took medication just once during the study, except 4 participants in the control group, who took medication during 2 crisis (3 patients) or 3 crisis (1 patient) episodes.

Discussion

The main finding of the present study was that 4 weeks of treatment, combined or not, using soft tissue and neural mobilization techniques, is effective in improving the PPT in the head region, frequency and maximal intensity of pain crisis, and HIT-6 values of patients with TTH. However, the results showed that the combined treatment is a more effective option in the management of TTH than these techniques applied separately. These findings support previous studies that determined the combination of soft tissue and neural mobilization techniques as the best option to manage patients with TTH[34] and those with other types of headaches.[35] In this regard, neural mobilization techniques combined with soft tissue techniques stimulate the peripheral and central receptors, producing an interaction of the mechanical and neurophysiological factors that could lead to improvements in the mechanosensitivity of these structures and thus a reduction in the pain level of patients with TTH.[36]

In contrast with previous studies,[36-39] a neural mobilization component to the mobilizations was included because of the neural mobilization technique's ability to activate inhibitory mechanisms that modulate the mechanosensitivity of the neuromusculoskeletal tissues.[12-14,17-20]

In this regard, the most extended theory about TTH refers to a peripheral process for the episodic TTH, in which $\geq$1 neuromusculoskeletal structures are sensitized and send nociceptive inputs to the central nervous system.[40,41] This nociceptive inputs from the trigeminal nerve and craniocervical muscles are integrated in the TCN,[42] whose continuous stimulation will sensitize the TCN and the central nervous system.[8,43-46]

Table 2 Values of the pressure pain threshold in the 3 points

Variable	Group A (n=24)	Group B (n=23)	Group C (n=25)	Group D (n=25)
PPT_1 (kg/cm^2)				
Pre	1.9±0.3	2.1±0.4	2.0±0.3	2.0±0.4
Post	2.0±0.4*	3.1±0.4†	2.9±0.3†	3.2±0.4†
$Post_{15days}$	2.0±0.4*	3.1±0.4†,‡	2.9±0.4†	3.2±0.4†
$Post_{30days}$	1.9±0.4*	3.0±0.4†	2.9±0.4†	3.2±0.4†
PPT_2 (kg/cm^2)				
Pre	1.8±0.3	1.9±0.4	1.9±0.4	2.0±0.3
Post	1.8±0.4*	2.9±0.5†	2.8±0.4†	3.3±0.3*,†
$Post_{15days}$	1.8±0.4*	2.9±0.5†	2.8±0.4†	3.3±0.4*,†
$Post_{30days}$	1.8±0.4*	2.8±0.5†	2.8±0.4†	3.3±0.4*,†
PPT_3 (kg/cm^2)				
Pre	1.0±0.3*	1.1±0.3	1.0±0.3	1.2±0.3
Post	1.0±0.3*	1.6±0.3†	1.6±0.3†	2.0±0.4*,†
$Post_{15days}$	1.0±0.3*	1.6±0.3†	1.6±0.3†	2.0±0.4*,†
$Post_{30days}$	1.0±0.3*	1.6±0.3†	1.6±0.3†	2.0±0.4*,†

NOTE. Values are mean ± SD.

Abbreviations: Post, measurement 1h after the intervention period; $Post_{15days}$, measurement 15d after the intervention period; $Post_{30days}$, measurement 30d after the intervention period; PPT_1, pressure pain threshold in point 1 of the temporal muscle; PPT_2, pressure pain threshold in point 2 of the temporal muscle; PPT_3, pressure pain threshold in the supraorbital region; Pre, measurement before the intervention period.

* $P<.001$ compared to the rest of the groups.

† $P<.001$ compared to the baseline measurement.

‡ $P<.01$ compared to the previous measurement.

Therefore, mechanical stimulation of the neuromusculoskeletal tissues that send their inputs to the TCN could decrease these nociceptive inputs and thus the nociceptive information to the central nervous system. Also, this stimulation would activate the inhibitory mechanisms, normalizing the TCN. Also, the reduction in the mechanosensitivity of the nervous tissue can reduce the muscle responses, which intend to protect the nerve tissue against tension and deformation stimulus due to the movement.[15,16]

Previous studies[47] report the clinical relevance of the findings when frequency reduction reaches 50%. Therefore, it is important to highlight the number of patients who achieved a reduction of ≥50% in the frequency of crisis in any of the postintervention measurements, where group D had the higher number with 24 of 25 patients and group A had the lower number with 0 of 24 patients. The large effect size and the 57% reduction shown in the frequency of crisis in patients receiving the combined protocol

Table 3 Values of the frequency and intensity of pain crisis and punctuation in the HIT-6

Variable	Group A (n=24)	Group B (n=23)	Group C (n=25)	Group D (n=25)
Frequency (d/15 days)				
Pre	7.2±2.7	8.6±2.3	7.9±2.7	8.0±2.6
Post	6.7±2.5*	4.7±1.7†	4.2±1.7†	3.5±1.7†,‡
$Post_{15days}$	6.9±2.5*,†	4.7±1.4†	4.3±2.2†	3.5±1.7†,‡
$Post_{30days}$	6.8±2.3*	4.8±1.7†	4.3±2.2†	3.4±1.9†,‡
Intensity (0–10 points)				
Pre	5.6±1.1	4.4±1.1	5.7±0.8	5.1±1.0
Post	5.4±1.2*,†	2.8±1.0†	4.0±1.0†	2.9±1.0†
$Post_{15days}$	5.4±1.0*,†	2.8±0.8†	4.0±0.9†	2.9±1.0†
$Post_{30days}$	5.4±1.1*,†	2.8±1.0†	4.1±0.9†	3.0±1.1†
Punctuation in the HIT-6 (36–78 points)				
Pre	60.0±5.9	60.8±5.7	59.0±5.3	59.7±6.0
Post	57.2±4.5*,†	52.8±5.1†	51.0±5.5†	50.0±6.2†
$Post_{15days}$	57.5±4.8*,†	52.9±4.7†,§	51.8±5.2†	50.0±5.7†
$Post_{30days}$	57.7±5.5*,†	52.9±5.1†	51.7±5.4†	50.3±5.4†

NOTE. Values are mean ± SD.

Abbreviations: d/15 days, days with pain every 15d; Post, measurement 1h after the intervention period; $Post_{15days}$, measurement 15d after the intervention period; $Post_{30days}$, measurement 30d after the intervention period; Pre, measurement before the intervention period.

* $P<.001$ compared to the rest of the groups.

† $P<.001$ compared to the baseline measurement.

‡ $P<.01$ compared to group B.

§ $P<.001$ compared to the previous measurement.

must also be taken into account, in contrast with the 47% and 45% obtained in patients who received soft tissue and neural mobilization treatments separately, respectively. Although groups receiving the techniques separately did not reach clinical significance according to previous studies,[47] they equaled and even overcame the reduction of 40% that is normally achieved thanks to pharmacological treatment.[48] Similar results were found using the HIT-6, where previous studies[49] reported that the reduction should reach 8 points to be clinically relevant. In the present study, the groups receiving neural mobilization techniques or soft tissue techniques separately reached 8 points on the HIT-6 whereas the combined treatment group experienced a reduction of 9.8 points.

Improvements were found even in the control group in variables such as the frequency of crisis and punctuation in the HIT-6, although with less significant differences and small effect sizes. These improvements could be explained by the observational effect (patients being observed in strict investigations may report better outcomes), the placebo effect, a simple random variation, or the normal course of the disorder. These same reasons support similar results obtained in previous studies,[36,39] in which the control group also improved in some variables.

Apart from the variability in the techniques applied, the nature and location of the structures where the technique is applied seems to be important.[50] In this way, the present study managed more types and number of structures than most studies involving combined protocols. In this regard, the inclusion of the neural mobilization component may be other important mechanism to reduce the mechanosensitivity of the TCN, but future studies are needed to confirm its importance.

Study limitations

As limitations, our sample included mainly women, which may affect the results. However, epidemiological studies have determined that women are most likely to suffer from any type of TTH and even a risk factor for this pathology.[51] Therefore, the present study sample may refer to the most representative population suffering from TTH. Other limitations refer to follow-up, which may have been short if the chronic character of TTH is taken into account. Another limitation of our study is the impossibility to blind the physiotherapists with respect to the interventions that they were applying.

With respect to the medication consumption, we highlight that it mainly referred to the initial days of the intervention period in the groups who received intervention protocols. In this regard, we suggest the local effects of the techniques that could remain any minimal inflammatory process as the main reason or even because of any misunderstanding about the level of pain that patients should feel. Conversely, in the control group the ibuprofen intake occurred during the entire study period.

For future studies, it is recommended to increase the duration of follow-up to identify the long-term effects of the treatment, apart from including the neural mobilization techniques in combination with other frequent treatments, such as manipulation or even therapeutic exercise, to obtain the most effective clinical approach to TTH.

Conclusions

A protocol combining soft tissue and neural mobilization techniques is more effective in the management of patients with FETTH and those with CTTH than does the application of these techniques separately.

Suppliers

a. EpiData software v.4.0; EpiData Association.
b. Commander Muscle Tester; J-TECH Medical.
c. GRANMO 7.12 software; IMIM Hospital del Mar.

Keywords

Musculoskeletal manipulations; Nerve tissue; Rehabilitation; Tension-type headache; Therapy, soft tissue

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References

1. Bendtsen L, Jensen R. Tension-type headache: the most common, but also the most neglected, headache disorder. Curr Opin Neurol 2006; 19:305-9.
2. Russell MB. Tension-type headache in 40-year-olds: a Danish population-based sample of 4000. J Headache Pain 2005;6:441-7.
3. Vos T, Flaxman AD, Naghavi M, et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380:2163-96.
4. Linde M, Gustavsson A, Stovner LJ, et al. The cost of headache disorders in Europe: the Eurolight project. Eur J Neurol 2012;19:703-11.
5. Mendoza-Puente M, Oliva-Pascual-Vaca A, Rodriguez-Blanco C, Heredia-Rizo AM, Torres-Lagares D, Ordonez FJ. Risk of headache, temporomandibular dysfunction, and local sensitization in male professional boxers: a case-control study. Arch Phys Med Rehabil 2014;95:1977-83.
6. Fernandez-de-Las-Penas C, Coppieters MW, Cuadrado ML, Pareja JA. Patients with chronic tension-type headache demonstrate increased mechano-sensitivity of the supra-orbital nerve. Headache 2008;48:570-7.
7. Lai TH, Protsenko E, Cheng YC, Loggia ML, Coppola G, Chen WT. Neural plasticity in common forms of chronic headaches. Neural Plast 2015;2015:205985.
8. Ashina S, Bendtsen L, Ashina M, Magerl W, Jensen R. Generalized hyperalgesia in patients with chronic tension-type headache. Cephalalgia 2006;26:940-8.
9. Castien R, Blankenstein A, De Hertogh W. Pressure pain and isometric strength of neck flexors are related in chronic tension-type headache. Pain Physician 2015;18:E201-5.

10. Luedtke K, Allers A, Schulte LH, May A. Efficacy of interventions used by physiotherapists for patients with headache and migraine—systematic review and meta-analysis. Cephalalgia 2016;36:474-92.
11. Fernandez-de-Las-Penas C, Alonso-Blanco C, Cuadrado ML, Miangolarra JC, Barriga FJ, Pareja JA. Are manual therapies effective in reducing pain from tension-type headache? A systematic review. Clin J Pain 2006;22:278-85.
12. Elvey RL. Physical evaluation of the peripheral nervous system in disorders of pain and dysfunction. J Hand Ther 1997;10:122-9.
13. Hall TM, Elvey RL. Nerve trunk pain: physical diagnosis and treatment. Man Ther 1999;4:63-73.
14. Nee R, Butler D. Management of peripheral neuropathic pain: integrating neurobiology, neurodynamics, and clinical evidence. Phys Ther Sport 2006;7:36-49.
15. Coppieters MW, Stappaerts KH, Wouters LL, Janssens K. Aberrant protective force generation during neural provocation testing and the effect of treatment in patients with neurogenic cervicobrachial pain. J Manipulative Physiol Ther 2003;26:99-106.
16. Jaberzadeh S, Scutter S, Nazeran H. Mechanosensitivity of the median nerve and mechanically produced motor responses during Upper Limb Neurodynamic Test 1. Physiotherapy 2005;91:94-100.
17. Brown CL, Gilbert KK, Brismee JM, Sizer PS, Roger James C, Smith MP. The effects of neurodynamic mobilization on fluid dispersion within the tibial nerve at the ankle: an unembalmed cadaveric study. J Man Manip Ther 2011;19:26-34.
18. Coppieters MW, Butler DS. Do 'sliders' slide and 'tensioners' tension? An analysis of neurodynamic techniques and considerations regarding their application. Man Ther 2008;13:213-21.
19. Efstathiou MA, Stefanakis M, Savva C, Giakas G. Effectiveness of neural mobilization in patients with spinal radiculopathy: a critical review. J Bodyw Mov Ther 2015;19:205-12.
20. Nee RJ, Vicenzino B, Jull GA, Cleland JA, Coppieters MW. Neural tissue management provides immediate clinically relevant benefits without harmful effects for patients with nerve-related neck and arm pain: a randomised trial. J Physiother 2012;58:23-31.
21. Headache Classification Committee of the International Headache (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia 2013;33:629-808.
22. General Assembly of the World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. J Am Coll Dent 2014;81:14-8.
23. Breig A. Adverse mechanical tension in the central nervous system: an analysis of cause and effect: relief by functional neurosurgery. Stockholm: Wiley; 1978.
24. Elvey RL. Treatment of arm pain associated with abnormal brachial plexus tension. Aust J Physiother 1986;32:225-31.
25. Von Piekartz H. Craniofacial pain: neuromusculoskeletal assessment, treatment and management. Philadelphia: Elsevier Health Sciences; 2007.
26. Blanchard EB, Andrasik F, Neff DF, Jurish SE, O'Keefe DM. Social validation of the headache diary. Behav Ther 1981;12:711-5.
27. Anderson RE, Seniscal C. A comparison of selected osteopathic treatment and relaxation for tension-type headaches. Headache 2006;46:1273-80.
28. Nussbaum EL, Downes L. Reliability of clinical pressure-pain algometric measurements obtained on consecutive days. Phys Ther 1998;78:160-9.
29. Fernandez-de-las-Penas C, Ge HY, Cuadrado ML, Madeleine P, Pareja JA, Arendt-Nielsen L. Bilateral pressure pain sensitivity mapping of the temporalis muscle in chronic tension-type headache. Headache 2008;48:1067-75.
30. Peddireddy A, Wang K, Svensson P, Arendt-Nielsen L. Stretch reflex and pressure pain thresholds in chronic tension-type headache patients and healthy controls. Cephalalgia 2009;29:556-65.
31. Schmid AB, Brunner F, Luomajoki H, et al. Reliability of clinical tests to evaluate nerve function and mechanosensitivity of the upper limb peripheral nervous system. BMC Musculoskelet Disord 2009;10:11.
32. Kosinski M, Bayliss MS, Bjorner JB, et al. A six-item short-form survey for measuring headache impact: the HIT-6. Qual Life Res 2003;12:963-74.
33. Nachit-Ouinekh F, Dartigues JF, Henry P, et al. Use of the Headache Impact Test (HIT-6) in general practice: relationship with quality of life and severity. Eur J Neurol 2005;12:189-93.
34. Espi-Lopez GV, Gomez-Conesa A, Gomez AA, Martinez JB, Pascual-Vaca AO, Blanco CR. Treatment of tension-type headache with articulatory and suboccipital soft tissue therapy: a double-blind, randomized, placebo-controlled clinical trial. J Bodyw Mov Ther 2014;18:576-85.
35. Bevilaqua-Grossi D, Goncalves MC, Carvalho GF, et al. Additional effects of a physical therapy protocol on headache frequency, pressure pain threshold, and improvement perception in patients with migraine and associated neck pain: a randomized controlled trial. Arch Phys Med Rehabil 2016;97:866-74.
36. Espi-Lopez GV, Rodriguez-Blanco C, Oliva-Pascual-Vaca A, Benitez-Martinez JC, Lluch E, Falla D. Effect of manual therapy techniques on headache disability in patients with tension-type headache: randomized controlled trial. Eur J Phys Rehabil Med 2014;50:641-7.
37. Castien RF, van der Windt DA, Grooten A, Dekker J. Effectiveness of manual therapy for chronic tension-type headache: a pragmatic, randomised, clinical trial. Cephalalgia 2011;31:133-43.
38. Castien RF, van der Windt DA, Dekker J, Mutsaers B, Grooten A. Effectiveness of manual therapy compared to usual care by the general practitioner for chronic tension-type headache: design of a randomised clinical trial. BMC Musculoskelet Disord 2009;10:21.
39. Espí-López GV, Gómez-Conesa A. Efficacy of manual and manipulative therapy in the perception of pain and cervical motion in patients with tension-type headache: a randomized, controlled clinical trial. J Chiropr Med 2014;13:4-13.
40. Andersen S, Petersen MW, Svendsen AS, Gazerani P. Pressure pain thresholds assessed over temporalis, masseter, and frontalis muscles in healthy individuals, patients with tension-type headache, and those with migraine—a systematic review. Pain 2015;156:1409-23.
41. Bendtsen L, Fernandez-de-las-Penas C. The role of muscles in tension-type headache. Curr Pain Headache Rep 2011;15:451-8.
42. Hu JW, Sun KQ, Vernon H, Sessle BJ. Craniofacial inputs to upper cervical dorsal horn: implications for somatosensory information processing. Brain Res 2005;1044:93-106.
43. Bendtsen L. Central sensitization in tension-type headache—possible pathophysiological mechanisms. Cephalalgia 2000;20:486-508.
44. Watson DH, Drummond PD. Head pain referral during examination of the neck in migraine and tension-type headache. Headache 2012;52:1226-35.
45. Bendtsen L, Ashina S, Moore A, Steiner TJ. Muscles and their role in episodic tension-type headache: implications for treatment. Eur J Pain 2016;20:166-75.
46. Fernandez-de-Las-Penas C. Myofascial head pain. Curr Pain Headache Rep 2015;19:28.
47. Castien RF, van der Windt DA, Blankenstein AH, Heymans MW, Dekker J. Clinical variables associated with recovery in patients with chronic tension-type headache after treatment with manual therapy. Pain 2012;153:893-9.
48. Bendtsen L, Jensen R. Pharmacological and non-pharmacological management of tension-type headache. Eur Neurol Rev 2008;3:119-22.
49. Castien RF, Blankenstein AH, Windt DA, Dekker J. Minimal clinically important change on the Headache Impact Test-6 questionnaire in patients with chronic tension-type headache. Cephalalgia 2012;32:710-4.
50. Fernandez-de-Las-Penas C, Courtney CA. Clinical reasoning for manual therapy management of tension type and cervicogenic headache. J Man Manip Ther 2014;22:44-50.
51. Lyngberg A, Rasmussen B, Jorgensen T, Jensen R. Has the prevalence of migraine and tension-type headache changed over a 12-year period? A Danish population. Eur J Epidemiol 2005;20:243-9.

Supplemental Table S1 Individual percentage of improvement in the frequency of crisis compared to baseline in patients of group A

Patient No.	Post	$Post_{15days}$	$Post_{30days}$
1	0.0	20.0	0.0
2	20.0	0.0	0.0
3	28.6	28.6	14.3
4	−20.0	−10.0	−10.0
5	7.7	7.7	7.7
6	−33.3	0.0	−33.3
7	0.0	0.0	0.0
8	0.0	16.7	0.0
9	−25.0	0.0	−25.0
10	28.6	14.3	0.0
11	22.2	22.2	11.1
12	0.0	−20.0	0.0
13	11.1	22.2	33.3
14	27.3	18.2	27.3
15	10.0	0.0	10.0
16	28.6	14.3	28.6
17	−40.0	−20.0	0.0
18	0.0	25.0	0.0
19	−25.0	−25.0	−75.0
20	16.7	16.7	33.3
21	25.0	12.5	12.5
22	0.0	0.0	12.5
23	16.7	0.0	0.0
24	0.0	9.1	0.0

Abbreviations: Post, measurement 1h after the intervention period; $Post_{15days}$, measurement 15d after the intervention period; $Post_{30days}$, measurement 30d after the intervention period.

Supplemental Table S2 Individual percentage of improvement in the frequency of crisis compared to baseline in patients of group B

Patient No.	Post	$Post_{15days}$	$Post_{30days}$
1	36.4	45.5	36.4
2	60.0*	40.0	60.0*
3	71.4*	57.1*	57.1*
4	45.5	36.4	36.4
5	44.4	44.4	55.6
6	60.0*	60.0*	50.0*
7	41.7	50.0*	41.7
8	25.0	37.5	25.0
9	45.5	45.5	45.5
10	16.7	33.3	50.0*
11	20.0	40.0	40.0
12	66.7*	55.6*	55.6*
13	37.5	37.5	37.5
14	54.5*	54.5*	45.5
15	20.0	40.0	40.0
16	27.3	27.3	36.4
17	50.0*	37.5	25.0
18	55.6*	55.6*	66.7*
19	60.0*	40.0	60.0*
20	25.0	25.0	25.0
21	70.0*	60.0*	50.0*
22	36.4	45.5	45.5
23	42.9	57.1*	42.9

Abbreviations: Post, measurement 1h after the intervention period; $Post_{15days}$, measurement 15d after the intervention period; $Post_{30days}$, measurement 30d after the intervention period.

* Improvements that were $\geq$50%.

Supplemental Table S3 Individual percentage of improvement in the frequency of crisis compared to baseline in patients of group C

Patient No.	Post	$Post_{15days}$	$Post_{30days}$
1	44.4	44.4	44.4
2	66.7*	33.3	16.7
3	30.0	40.0	40.0
4	50.0*	37.5	25.0
5	40.0	60.0*	80.0*
6	37.5	50.0*	50.0*
7	77.8*	55.6*	55.6*
8	80.0*	80.0*	60.0*
9	57.1*	57.1*	71.4*
10	36.4	36.4	45.5
11	85.7*	71.4*	57.1*
12	33.3	33.3	25.0
13	28.6	42.9	57.1*
14	50.0*	50.0*	50.0*
15	75.0*	75.0*	50.0*
16	25.0	33.3	33.3
17	85.7*	71.4*	85.7*
18	37.5	37.5	37.5
19	75.0*	50.0*	50.0*
20	54.5*	54.5*	54.5*
21	66.7*	33.3	33.3
22	22.2	33.3	22.2
23	27.3	36.4	45.5
24	46.2	38.5	46.2

Abbreviations: Post, measurement 1h after the intervention period; $Post_{15days}$, measurement 15d after the intervention period; $Post_{30days}$, measurement 30d after the intervention period.

* Improvements that were $\geq$50%.

Supplemental Table S4 Individual percentage of improvement in the frequency of crisis compared to baseline in patients of group D

Patient No.	Post	$Post_{15days}$	$Post_{30days}$
1	83.3*	66.7*	83.3*
2	62.5*	62.5*	62.5*
3	46.2	53.8*	61.5*
4	60.0*	60.0*	80.0*
5	62.5*	50.0*	50.0*
6	57.1*	42.9	57.1*
7	44.4	55.6*	55.6*
8	66.7*	66.7*	55.6*
9	66.7*	50.0*	66.7*
10	42.9	42.9	42.9
11	50.0*	50.0*	50.0*
12	71.4*	57.1*	57.1*
13	58.3*	50.0*	41.7
14	60.0*	40.0	60.0*
15	50.0*	66.7*	50.0*
16	41.7	41.7	50.0*
17	54.5*	45.5	36.4
18	62.5*	75.0*	62.5*
19	80.0*	80.0*	80.0*
20	50.0*	66.7*	83.3*
21	45.5	54.5*	45.5
22	50.0*	33.3	50.0*
23	75.0*	100.0*	100.0*
24	72.7*	72.7*	63.6*
25	44.4	55.6*	55.6*

Abbreviations: Post, measurement 1h after the intervention period; $Post_{15days}$, measurement 15d after the intervention period; $Post_{30days}$, measurement 30d after the intervention period.

* Improvements that were $\geq$50%.