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The Effectiveness of a Manual Therapy and Exercise Protocol in Patients With Thumb Carpometacarpal Osteoarthritis: A Randomized Controlled Trial

STUDY DESIGN: Double-blind, randomized controlled trial.

OBJECTIVE: To examine the effectiveness of a manual therapy and exercise approach relative to a placebo intervention in individuals with carpometacarpal (CMC) joint osteoarthritis (OA).

BACKGROUND: Recent studies have reported the outcomes of exercise, joint mobilization, and neural mobilization interventions used in isolation in patients with CMC joint OA. However, it is not known if using a combination of these interventions as a multimodal approach to treatment would further improve outcomes in this patient population.

METHODS: Sixty patients, 90% female (mean ± SD age, 82 ± 6 years), with CMC joint OA were randomly assigned to receive a multimodal manual treatment approach that included joint mobilization, neural mobilization, and exercise, or a sham intervention, for 12 sessions over 4 weeks. The primary outcome measure was pain. Secondary outcome measures included pressure pain threshold over the first CMC joint, scaphoid, and hamate, as well as pinch and strength measurements. All outcome measures were collected at baseline, immediately following the intervention, and at 1 and 2 months following the end of the intervention. Mixed-model analyses of variance were used to examine the effects of the interventions on each outcome, with group as the between-subject variable and time as the within-subject variable.

RESULTS: The mixed-model analysis of variance revealed a group-by-time interaction ($F = 47.58$, $P<.001$) for pain intensity, with the patients receiving the multimodal intervention experiencing a greater reduction in pain compared to those receiving the placebo intervention at the end of the intervention, as well as at 1 and 2 months after the intervention ($P<.001$; all group differences greater than 3.0 cm, which is greater than the minimal clinically important difference of 2.0 cm). A significant group-by-time interaction ($F = 3.19$, $P = .025$) was found for pressure pain threshold over the hamate bone immediately after the intervention; however, the interaction was no longer significant at 1 and 2 months postintervention.

CONCLUSION: This clinical trial provides evidence that a combination of joint mobilization, neural mobilization, and exercise is more beneficial in treating pain than a sham intervention in patients with CMC joint OA. However, the treatment approach has limited value in improving pressure pain thresholds, as well as pinch and grip strength. Future studies should include several therapists, a measure of function, and long-term outcomes. Trial registration: Current Controlled Trials ISRCTN37143779.

LEVEL OF EVIDENCE: Therapy, level 1b.

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KEY WORDS: *arthritis, CMC, joint mobilization, neural mobilization*

The carpometacarpal (CMC) joint of the thumb plays a vital role in the function of the hand. The first CMC joint is frequently affected by osteoarthritis (OA), a degenerative condition resulting in deterioration of the joint surfaces and eventual bone remodeling.[2] The consequence of CMC joint OA is severe pain, leading to considerable limitations in function and disability, and a substantial societal burden.[2,15,42,44] Patients with CMC joint OA represent the cohort of individuals with upper extremity arthritis most likely to undergo surgical intervention.[3] However, previous studies examining the effectiveness of surgical intervention have reported varied levels of benefits in terms of pain reduction and improved function, and have demonstrated an adverse event rate of between 10% and 22%, depend-

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ing on the procedure used.[53] Therefore, it remains important to determine the most effective management strategies for improving pain and function in a population with CMC joint OA.[10]

Patients with CMC joint OA are often encountered in physical therapy practice. Typical physical therapy management strategies for this population include manual therapy and exercise. Manual therapy interventions for this population include both joint[49-51] and neurodynamic mobilization techniques.[48,52] Recently published randomized controlled clinical trials[49,51] have demonstrated that 4 to 6 sessions of joint mobilizations over a 2-week period resulted in significantly greater improvements in pressure pain thresholds (PPTs) measured over the first CMC joint and scaphoid bone than a sham intervention (nontherapeutic ultrasound).

In a single-cohort design study, Villafañe et al[48] examined the outcomes of patients with CMC joint OA who were treated with a neural mobilization technique purported to bias the median nerve. Although PPTs measured over the first CMC joint improved after the intervention, no changes in PPTs were identified when measured over the scaphoid or hamate, and there was no improvement in grip or pinch strength. A subsequent randomized controlled trial[48] comparing the effectiveness of a neural slider mobilization technique targeting the radial nerve[11] to a placebo intervention demonstrated greater improvements in PPT measured over the first CMC joint and greater improvements in pinch strength in the intervention group. However, all these studies investigated the effects of a single intervention approach, which does not represent typical clinical practice in the management of these patients. Joint mobilization techniques, traction, and glide are often used in this population to stretch the joint capsule to improve physiological accessory motions,[34] to improve limited range of motion, and to reduce pain.[28,36]

Hand exercises for CMC joint OA are aimed at maximizing pain-free range of motion, increasing functional strength, maintaining joint stability, and avoiding fixed deformities of the thumb.[47] The use of exercise for patients with CMC joint OA has recently been questioned, based on the results of a clinical trial by Rogers and Wilder[37] demonstrating that exercise was no better than a sham intervention in this population. However, an earlier study by Stamm et al[45] showed that exercise resulted in greater improvements in grip strength and function when compared to no intervention. It should be noted that the aforementioned studies had the patients perform the exercises at home; therefore, the level of compliance with the exercise program is not clear. It is not known whether a similar program performed under the direct supervision of a physical therapist would result in similar outcomes.

To the best of the authors' knowledge, no previous studies have examined the effectiveness of a multimodal approach that includes manual therapy and exercise in patients with CMC joint OA. Therefore, the purpose of this randomized controlled trial was to examine the effectiveness of the application of joint mobilization, neural mobilization, and exercise compared to a placebo intervention in patients with CMC joint OA.

METHODS

Design

We conducted a double-blind, randomized controlled trial. Informed consent was obtained from all participants, and the study protocol was conducted according to the Declaration of Helsinki. The protocol (number 93571/c) was approved by the Local Ethical Committee in Azienda Sanitaria Locale 3, Collegno, Italy (nursing home). The study was registered after completion at the Current Controlled Trials trial-registration website (ISRCTN37143779).

Participants

Sixty participants, 65 to 90 years of age, were recruited for the study from January 2012 to April 2012. All subjects were right-hand dominant. A diagnosis of CMC joint OA was established by a hand surgeon. Each patient underwent subjective and physical examination, performed by a physical therapist experienced in musculoskeletal physiotherapy and was evaluated for inclusion/exclusion in the study. Participants were asked not to take analgesics, muscle relaxants, or anti-inflammatory drugs for 24 hours prior to the examination.

To be included in the study, the participants needed to have a history of repetitive use of their dominant hand (eg, former factory worker) and a diagnosis of stage III or IV secondary CMC joint OA in the dominant hand, according to the Eaton-Littler-Burton classification system based on radiographic findings.[27] Patients were excluded if they scored greater than 4 points on the Beck Depression Inventory[55] or greater than 30 points on the State-Trait Anxiety Inventory.[1] Patients with a medical history of carpal tunnel syndrome, surgical interventions to the first CMC joint, De Quervain tenosynovitis, bilateral symptoms, or degenerative or nondegenerative neurological conditions in which pain perception was altered were excluded. None of the individuals in this study had received prior interventions for CMC joint OA , and they were, therefore, naïve to the treatment they received.

Outcome Measures

Current Pain The primary outcome measure was pain intensity of the first CMC joint, which was assessed with a visual analog scale (VAS). The VAS is a 10-cm line, anchored with 0 at one end, representing no pain, and 10 at the other end, representing the worst pain imaginable.[9] Pain was assessed by having the participant perform a key pinch between the thumb and the index finger. The VAS was selected as the primary outcome measure, based on its ability to detect changes (minimal clinically important difference, 2.0 cm).[18,19]

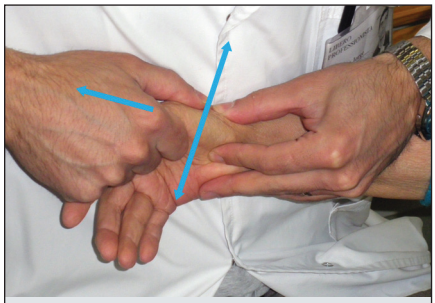

FIGURE 1. Posterior/anterior glide with distraction of the first carpometacarpal joint (**ONLINE VIDEO**).

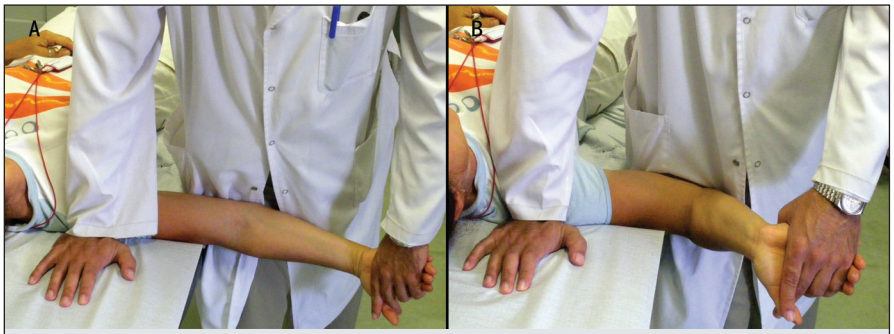

FIGURE 2. Nerve slider exercise used to target the median nerve. (A) Shoulder girdle depression, glenohumeral abduction, lateral rotation, supination of the forearm, elbow extension, and wrist, thumb, and finger flexion. (B) Shoulder girdle depression, glenohumeral abduction, lateral rotation, supination of the forearm, elbow flexion, and wrist, thumb, and finger extension (**ONLINE VIDEO**).

Pressure Pain Thresholds The PPT is the minimal pressure recorded when the sensation of pressure changes to pain. This was assessed using a mechanical algometer (Wagner Instruments, Greenwich, CT).[35,57] The device consists of a 1-cm^2, round, rubber disc attached to a force gauge (kg). The tester, using the algometer, applied pressure at a rate of approximately 0.1 kg/cm^2/s until the onset of pain. This procedure was repeated 3 times. The mean of the 3 measurements was calculated and used for the primary data analysis. A 30-second rest period was given between each measurement. Previous publications have reported an intraexaminer reliability of this procedure ranging from 0.6 to 0.97 and an interexaminer reliability ranging from 0.4 to 0.98.[21] Walton et al[54] reported the minimal detectable change for PPT measurements over the cervical spine and tibialis anterior muscle in patients with acute neck pain; however, no normative data for PPT assessed over the locations used in patients with CMC joint OA have been reported in the literature.

To investigate the hypoalgesic effects of the intervention, PPT was assessed at 3 predetermined locations[48]: the lateral epicondyle, the first CMC joint at the center of the anatomical snuff box, and the unciform apophysis of the hamate bone.

Pinch Strength Pinch strength between the index finger and thumb was evaluated with the participant in the sitting position, with the shoulder adducted and in neutral rotation and the elbow flexed to 90°.[22,25,32] Two measurements were taken using a mechanical pinch gauge (Fabrication Enterprises, Inc, White Plains, NY). The reliability of pinch strength measurement has been found to be high (intraclass correlation coefficient [ICC] = 0.93).[39]

Grip Strength Grip strength was assessed with a grip dynamometer (Fabrication Enterprises, Inc) and the patient in a sitting position. This procedure has been shown to have a precision of ±3%.[38,40,43] The reliability of grip strength measurements has been reported to be high (ICC = 0.82-0.97).[39]

All outcome measures were captured at baseline, immediately postintervention, and at 1 and 2 months postintervention by an assessor blinded to group assignment. The sequence of testing for the outcome measures was randomized among participants. The trial was designed according to the CONSORT publishing guidelines.[38]

Randomization

After the completion of all baseline measurements, using a computer program (http://www.graphpad.com/quickcalcs/randomize1.cfm), subjects were randomly assigned by an external assistant to 1 of 2 groups: an experimental group that received a multimodal treatment protocol for CMC joint OA–related pain or a placebo group that received detuned ultrasound therapy. The participants in both groups were treated by a clinician with postgraduate orthopaedic manual therapy training and more than 8 years of clinical experience in the management of CMC joint OA pain. The physical therapist was blinded to all data that were collected for the study. All participants received 12 treatment sessions scheduled on separate days, at least 48 hours apart and at the same time of day, 3 days per week for 4 weeks. Treatment was only applied to the affected hand. All outcomes were collected by an external assessor blinded to the treatment allocation of the participants.

Multimodal Treatment Intervention

Patients in the experimental group received a multimodal treatment intervention consisting of joint mobilization, neurodynamic intervention, and exercise. Each patient received 12 sessions over a period of 4 weeks (3 sessions per week).

Joint Mobilization We applied a grade 3 posterior/anterior glide with distraction technique to the first CMC joint, as described by Kaltenborn.[49] The therapist grasped the right-thumb metacarpal bone of the patient with his right thumb and index finger and distracted the joint, retracting the thumb and gliding the first metacarpal bone in a posterior/anterior direction (**FIGURE 1**, **ONLINE VIDEO**).[49] The technique was applied for 3 minutes, followed by a 1-minute rest period. The mobilization sequence was repeated 3 times.

Neurodynamic Techniques A passive "nerve slider" neurodynamic technique, purported to bias the median nerve, was applied (**FIGURE 2**, **ONLINE VIDEO**).[11] A nerve

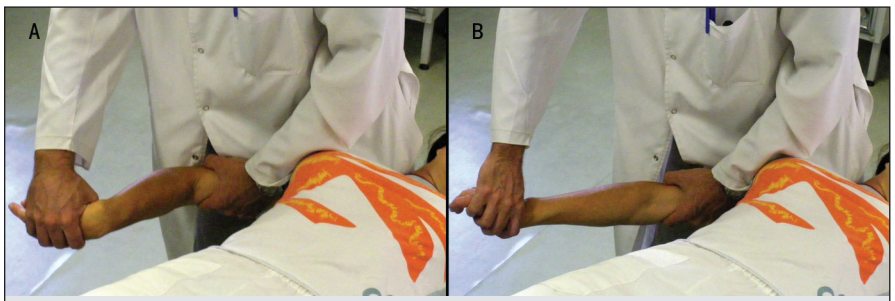

FIGURE 3. Nerve slider exercise used to target the radial nerve. (A) Shoulder girdle depression, glenohumeral medial rotation, pronation of the forearm, elbow flexion, and wrist, thumb, and finger flexion. (B) Shoulder girdle elevation, glenohumeral medial rotation, supination of the forearm, elbow extension, and wrist, thumb, and finger extension (**ONLINE VIDEO**).

TABLE 1 Hand Exercises

Exercise	Description
Tabletop	The hand and wrist are held in a neutral position; the subject flexes the second to fifth MCP joints only, then returns to neutral.
Small fist	From neutral position, the subject flexes at the second to fifth proximal interphalangeal joint and distal interphalangeal joint only, then returns to neutral.
Large fist	From neutral position, the subject flexes all joints to form a fist, then returns to neutral.
Okay signs	From neutral position, the subject flexes to form an "O" with the tip of the thumb to the tip of each finger, in turn, returning to neutral after each.
Finger spread	From neutral position, the hand is placed on a flat tabletop and the fingers are spread apart as wide as possible before returning to neutral.
Thumb reach	From neutral position, the subject reaches across the palm of the hand and touches the tip of the thumb to the fifth MCP joint and then returns to neutral.
Gripping	Subject holds the Thera-Band Hand Exerciser ball in the palm of the hand and squeezes until the ball is about 50% depressed.
Key pinch	Subject holds the Thera-Band Hand Exerciser ball between the side of the thumb and the side of the index finger and squeezes until the ball is about 50% depressed.
Fingertip pinch	Subject holds the Thera-Band Hand Exerciser ball between the tip of the thumb and the tip of the index finger and squeezes until the ball is about 50% depressed; this is repeated for digits 3 to 5.

Abbreviation: MCP, metacarpophalangeal.

slider is a maneuver that produces a sliding movement of the neural structures in relation to their anatomical adjacent tissues. It involves a combination of movements of surrounding joints, such that tension increases on one end of the nerve and is relieved on the other end.

For this technique, the patient was positioned in supine and the therapist was seated. The sequence of movement performed on the patient by the physical therapist for initial positioning for the nerve slider technique was shoulder girdle depression; glenohumeral abduction and lateral rotation; supination of the forearm; and wrist, thumb, and finger extension. The nerve slider technique for the median nerve consisted of alternating a combination of elbow extension (which increases tension on the median nerve) and wrist flexion (which decreases tension on the median nerve) movement with a combination of elbow flexion (decreasing tension) and wrist extension (increasing tension) movement. The range of motion used at the wrist was from 0° to 60° of extension and at the elbow was from 15° to 90° of flexion,[52] depending on tissue resistance. The nerve slider technique was performed twice for 5 minutes each time, with a 1-minute rest between sets. Speed and amplitude of movement were adjusted so that no pain would be induced by the technique.

For the radial nerve, the sequence of movement performed on the patient by the physical therapist for initial positioning consisted of shoulder girdle depression, glenohumeral medial rotation, pronation of the forearm, elbow extension, and wrist, thumb, and finger flexion.[48] Finally, ulnar deviation of the wrist was added.[12,41] The slider neurodynamic technique for the radial nerve consisted of alternating the combination of shoulder depression (loads the radial nerve) and elbow flexion/wrist extension (unloads the radial nerve) with shoulder elevation (unloading) and elbow-wrist extension (loading) (**FIGURE 3**, **ONLINE VIDEO**). We decided to use the nerve slider directed at the radial nerve, as this is the nerve that innervates the thumb. Similar to the technique for the median nerve, 2 sets of 5 minutes, with a 1-minute rest between them, were used.

We selected a protocol-based treatment approach, standardizing the interventions for all included patients, rather than an impairment-based approach. It has recently been reported that in nearly 50% of clinical trials, the interventions are not described in enough detail to allow for direct replication by clinicians and researchers.[23,24] Therefore, we decided that an impairment-based approach would not allow for enough accurate description of the selected techniques (rate, direction of force, amplitude) for clinicians to be able to perform the identical procedure in their own clinical practice. Hence, we decided to use a standardized treatment approach in this clinical trial.

Exercise Patients in the experimental group received the same standardized exercise protocol as that described by Rogers and Wilder[37] (**TABLE 1**). The first 6 exercises consisted of active range-of-motion movements of the hand that were designed to improve joint flexibility. The remaining 3 exercises were designed to strengthen grip and pinch strength by us-

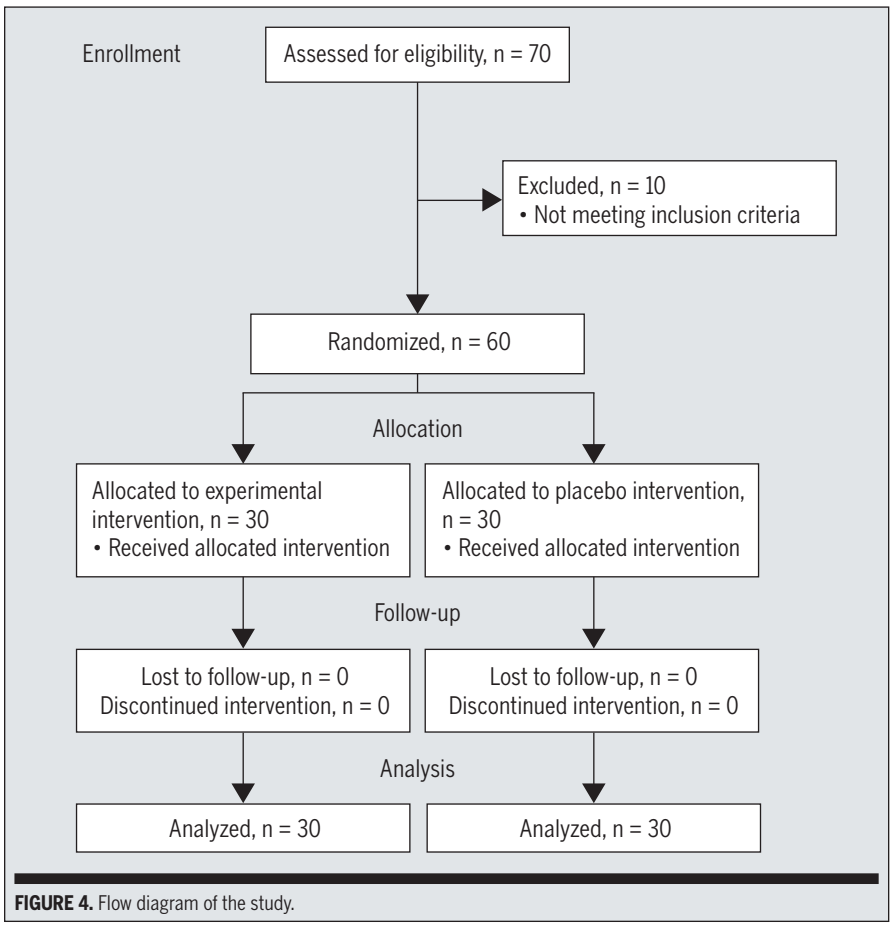

FIGURE 4. Flow diagram of the study.

ing a nonlatex polymer ball (Thera-Band Hand Exerciser; The Hygenic Corporation, Akron, OH). The Hand Exercisers were color coded to indicate approximate resistance provided at 50% compression (yellow, 0.68 kg; red, 1.36 kg; green, 2.27 kg; and blue, 3.64 kg).[37] All participants started with a yellow ball, and, depending on clinical presentation, subjects could be assigned to use more than 1 colored ball. Subjects began with 10 repetitions for the first 4 sessions, progressed to 12 repetitions for the next 2 sessions, then to 15 repetitions for 2 sessions, and finally to 20, if able, for the last 4 sessions.[37]

Placebo Intervention

Patients in the placebo group received the same number of treatment sessions of a similar duration as those in the experimental group, but received only inactive doses of pulsed ultrasound with an intensity of 0 W/cm^2 and gentle application of an inert gel for 10 minutes to the hypothenar area of the symptomatic hand.[4,13,26] This placebo protocol has been successfully used in our previous studies.[48,49,51]

Sample-Size Calculation

The sample-size and power calculations were performed with ENE 3.0 software (Universidad Autónoma de Barcelona, Barcelona, Spain). The calculations were based on detecting a mean difference of 2.0 cm (minimal clinically important difference) on a 10-cm VAS, assuming a standard deviation of 2.0 cm, a 2-tailed test, an alpha level of .05, and a desired power of 90%. The estimated desired sample size was 22 individuals per group. To accommodate expected dropouts before study completion, a total of 30 participants were included in each group.

Statistical Analysis

Data were analyzed using SPSS Version 18.0 (SPSS Inc, Chicago, IL), conducted following an intention-to-treat analysis using the last-value-forward method. The results are expressed as means, standard deviations, and/or 95% confidence intervals (CIs). The Kolmogorov-Smirnov test showed a normal distribution of the data. ICCs and standard error of measurement (SEM) were calculated to determine intraexaminer reliability of our PPT data, based on the 3 trials performed at each location at baseline.

Potential differences in baseline demographic and clinical variables between groups were analyzed using independent Student *t* tests for continuous data and chi-square tests of independence for categorical data. For the main outcome of the study, a 2-by-4 mixed-model analysis of variance (ANOVA) was used to examine the effects of treatment on pain intensity at each measurement occasion (the dependent variable), with group (experimental and control) as the between-subject variable and time (baseline, postintervention, and 1 month and 2 months postintervention) as the within-subject variable. For the secondary outcomes of the study, separate 2-by-2-by-4 mixed-model ANOVAs, with group (experimental and control) as the between-subject factor, and side (ipsilateral and contralateral to the CMC joint OA) and time (baseline, postintervention, and 1 month and 2 months postintervention) as within-subject factors, were conducted to examine the effects of the intervention on PPT and pinch and grip strength (the dependent variables). The main hypothesis of interest was the group-by-time interaction. Post hoc comparisons were conducted with Bonferroni correction. Between-group effect sizes were calculated by using the Cohen *d* coefficient.[14] An effect size greater than 0.8 was considered large, around 0.5 moderate, and less than 0.2 small.[14] The statistical analysis was conducted at a 95% confidence level, and $P<.05$ was considered statistically significant.

RESULTS

Seventy (n = 70) consecutive individuals with CMC joint OA were screened for eligibility criteria. Sixty patients (mean ± SD age, 82 ± 6 years; 90% female) satisfied all eligibility criteria, agreed to participate, and were randomized to the control (n = 30) or experimental (n = 30) group. The reasons for ineligibility were bilateral pain symptoms (n = 5), no confirmation of the diagnosis with radiographs (n = 3), and the concurrent presence of De Quervain tenosynovitis (n = 2). **FIGURE 4** provides a flow diagram of subject recruitment and retention through the study. All subjects were right-hand dominant and were affected on the right side. Baseline features of both groups were similar for all variables (**TABLE 2**). No adverse effects were detected during or after the application of the treatment, and none of the subjects started drug therapy during the study.

TABLE 2 Baseline Demographics for Both Groups*

	Experimental Group (n = 30)	Placebo Group (n = 30)	*P* Value
Age, y	82 ± 2	83 ± 1	.61
Gender (female/male), n	27/3	24/6	.47
Pain (visual analog scale, 0-10), cm	5.0 ± 0.3	5.0 ± 0.2	.89
PPT, kg/cm^2			
Lateral epicondyle, affected side	5.9 ± 0.3	5.5 ± 0.4	.45
Lateral epicondyle, nonaffected side	5.1 ± 0.3	5.1 ± 0.4	.87
Carpometacarpal joint, affected side	3.3 ± 0.2	3.4 ± 0.2	.64
Carpometacarpal joint, nonaffected side	3.2 ± 0.2	3.3 ± 0.2	.91
Hamate bone, affected side	5.5 ± 0.4	5.5 ± 0.3	.96
Hamate bone, nonaffected side	5.4 ± 0.4	5.5 ± 0.3	.78
Tip pinch and grip strength, kg			
Tip pinch, affected side	2.3 ± 0.2	2.3 ± 0.3	.99
Tip pinch, nonaffected side	2.3 ± 0.2	2.1 ± 0.2	.56
Grip strength, affected side	10.6 ± 1.0	10.7 ± 1.2	.97
Grip strength, nonaffected side	10.3 ± 1.1	10.1 ± 1.6	.91

Abbreviation: PPT, pressure pain threshold.
**Values are mean ± SD with the exception of gender.*

First CMC Joint Pain Intensity

The 2-by-4 mixed-model ANOVA indicated a significant group-by-time interaction (F = 47.58, *P*<.001) for pain intensity. Post hoc analysis indicated that the patients with thumb CMC joint OA receiving the multimodal intervention experienced a significantly greater reduction in pain compared to those receiving the placebo intervention immediately postintervention (experimental group mean, 3.7; 95% CI: 2.4, 3.8; placebo group mean, 0.3; 95% CI: 0.0, 0.3; difference between groups, 3.4; 95% CI: −4.6, −3.2), as well as at the 1-month follow-up (experimental group mean, 3.7; 95% CI: 2.8, 4.1; placebo group mean, 0.3; 95% CI: 0.2, 0.8; difference between groups, 3.4; 95% CI: −4.7, −3.3) and 2-month follow-up (experimental group mean, 3.7; 95% CI: 2.9, 4.2; placebo group mean, 0.3; 95% CI: 0.2, 0.9; difference between groups, 3.4; 95% CI: −4.8, −3.3) periods (all, *P*<.001) (**TABLE 3**). Between-group effect sizes were large at all follow-up periods (*d*>1.5).

Pressure Pain Thresholds

The ICCs for intraexaminer reliability of PPT measurements ranged from 0.84 to 0.92 for the affected side and from 0.84 to 0.92 for the unaffected side. The SEMs ranged from 0.51 to 0.65 kg/cm^2 for both sides.

For PPTs measured over the lateral epicondyle, there was no significant group-by-time-by-side (F = 0.75, *P* = .5), group-by-time (F = 1.46, *P* = .23), side-by-time (F = 0.60, *P* = .62), or group-by-side (F = 0.55, *P* = .82) interaction. There was also no significant main effect for time (F = 1.81, *P* = .15). There was a significant main effect for side (F = 14.40, *P*<.001), with higher PPT measured over the lateral epicondyle of the affected hand as compared to the nonaffected hand (**TABLE 3**).

For PPTs measured over the first CMC joint, the 2-by-2-by-4 ANOVA revealed no significant group-by-time-by-side (F = 0.44, *P* = .72), group-by-time (F = 1.1, *P* = .35), side-by-time (F = 1.10, *P* = .35), or group-by-side (F = 0.21, *P* = .65) interaction (**TABLE 3**). There was also no significant main effect for time (F = 1.79, *P* = .15) or side (F = 1.21, *P* = .28).

For PPTs measured over the hamate bone, the 2-by-2-by-4 ANOVA revealed no significant group-by-time-by-side (F = 1.36, *P* = .25), side-by-time (F = 3.19, *P* = .02), or group-by-side (F = 0.32, *P* = .57) interaction. There was a significant group-by-time interaction (F = 3.19, *P* = .025), with the patients receiving the experimental protocol exhibiting greater PPT over the hamate bone, as compared to those receiving the placebo intervention, immediately after the intervention (*P*<.005) but not at the 1- and 2-month follow-ups (**TABLE 3**).

Pinch and Grip Strength

The ICCs for intraexaminer reliability of measurements of tip pinch and grip strength were 0.81 and 0.72 for the affected arm and 0.81 and 0.73 for the unaffected side, respectively. For tip pinch and grip strength, the SEMs were 0.75 kg and 4.02 kg in the affected arm, respectively, and 0.51 kg and 4.47 kg in the unaffected side, respectively.

For tip pinch strength, the 2-by-2-by-4 ANOVA revealed no significant group-

TABLE 3 Pain and Pressure Pain Threshold Data*

Group	Preintervention	Postintervention	1 mo Postintervention	2 mo Postintervention
Pain				
Experimental	5.0 ± 0.3	1.9 ± 0.3†	1.5 ± 0.2†	1.5 ± 0.2†
Placebo	5.0 ± 0.2	4.9 ± 0.2	4.4 ± 0.3	4.4 ± 0.3
Between-group differences	0.0 (−0.1, 0.2)	3.0 (2.6, 3.8)	2.9 (2.2, 3.7)	2.9 (2.3, 3.8)
PPT, kg/cm^2				
Lateral epicondyle, affected				
Experimental	5.9 ± 0.3	6.3 ± 0.4	5.8 ± 0.3	5.7 ± 0.3
Placebo	5.5 ± 0.4	5.6 ± 0.4	5.8 ± 0.3	5.5 ± 0.4
Between-group differences	0.4 (−0.7, 1.3)	0.7 (−0.3, 1.7)	0.0 (−1.0, 0.9)	0.2 (−0.8, 1.1)
Lateral epicondyle, nonaffected				
Experimental	5.1 ± 0.3	5.8 ± 0.3	5.5 ± 0.4	5.6 ± 0.3
Placebo	5.1 ± 0.4	5.1 ± 0.3	5.4 ± 0.3	4.9 ± 0.3
Between-group differences	0.0 (−0.9, 0.9)	0.7 (−0.3, 1.5)	0.1 (−0.8, 1.1)	0.7 (−0.2, 1.5)
Carpometacarpal joint, affected				
Experimental	3.3 ± 0.2	3.7 ± 0.2	3.7 ± 0.3	3.7 ± 0.3
Placebo	3.4 ± 0.2	3.4 ± 0.2	3.4 ± 0.2	3.4 ± 0.2
Between-group differences	−0.1 (−0.6, 0.4)	0.3 (−0.3, 0.9)	0.3 (−0.3, 0.9)	0.3 (−0.4, 0.9)
Carpometacarpal joint, nonaffected				
Experimental	3.2 ± 0.2	3.5 ± 0.2	3.4 ± 0.4	3.3 ± 0.2
Placebo	3.3 ± 0.2	3.3 ± 0.2	3.4 ± 0.2	3.2 ± 0.2
Between-group differences	−0.1 (−0.6, 0.5)	0.2 (−0.3, 0.9)	0.0 (−0.6, 0.7)	0.1 (−0.5, 0.7)
Hamate bone, affected				
Experimental	5.5 ± 0.4	6.5 ± 0.4‡	6.0 ± 0.3	6.1 ± 0.4
Placebo	5.5 ± 0.3	5.5 ± 0.3	5.7 ± 0.3	5.4 ± 0.3
Between-group differences	0.0 (−1.0, 0.8)	1.0 (−0.1, 1.8)	0.3 (−0.6, 1.2)	0.7 (−0.3, 1.6)
Hamate bone, nonaffected				
Experimental	5.4 ± 0.4	5.9 ± 0.4	5.9 ± 0.4	5.9 ± 0.4
Placebo	5.5 ± 0.3	5.6 ± 0.3	5.4 ± 0.3	5.6 ± 0.3
Between-group differences	−0.1 (−1.2, 0.9)	0.3 (−0.7, 1.3)	0.5 (−0.5, 1.5)	0.3 (−0.7, 1.3)

Abbreviation: PPT, pressure pain threshold.
**Values are mean ± SD, except for between-group differences, which are mean (95% confidence interval).*
†*Significantly different from preintervention (P<.05).*
‡*Significant difference between groups at that measurement occasion (P<.05).*

by-time-by-side (F = 0.4, *P* = .75), group-by-time (F = 0.57, *P* = .64), side-by-time (F = 0.85, *P* = .47), or group-by-side (F = 0.80, *P* = .64) interaction. There were also no significant main effects for time (F = 0.8, *P* = .49) or side (F = 0.05, *P* = .81).

For grip strength, the 2-by-2-by-4 ANOVA revealed no significant group-by-time-by-side (F = 1.2, *P* = .31), group-by-time (F = 0.57, *P* = .64), side-by-time (F = 0.85, *P* = .47), or group-by-side (F = 0.66, *P* = .58) interaction, and no main effects for time (F = 0.27, *P* = .85) and side (F = 0.46, *P* = .5) (**TABLE 4**).

DISCUSSION

This randomized controlled trial examined the effects of joint mobilization, neural mobilization, and exercise on a patient population with CMC joint OA at short-term follow-up. The results demonstrated that patients receiving a multimodal intervention of manual therapy and exercise exhibited significantly greater improvements in pain compared to those who received a placebo intervention. It is interesting to note that the between-group differences for pain improvements and the lower-bound estimate of the 95% CI exceeded the reported minimal clinically important difference of 2.0 cm.[18,19] We believe that this provides evidence to support the use of this multimodal approach in patients with CMC joint OA.

In contrast to the differences between groups for pain, there was no difference between groups for PPT after the intervention, except when measured over the hamate immediately after treatment. The current study was powered to detect

TABLE 4 Tip Pinch and Grip Strength Data*

Group	Preintervention	Postintervention	1 mo Postintervention	2 mo Postintervention
Tip pinch, affected, kg				
Experimental	2.3 ± 0.2	2.3 ± 0.2	2.3 ± 0.2	2.3 ± 0.2
Placebo	2.3 ± 0.3	2.2 ± 0.2	2.2 ± 0.2	2.3 ± 0.2
Between-group differences	0.0 (−0.6, 0.6)	0.1 (−0.5, 0.7)	0.1 (−0.5, 0.7)	0.0 (−0.6, 0.6)
Tip pinch, nonaffected, kg				
Experimental	2.3 ± 0.2	2.4 ± 0.2	2.2 ± 0.2	2.2 ± 0.2
Placebo	2.1 ± 0.2	2.2 ± 0.2	2.1 ± 0.2	2.2 ± 0.3
Between-group differences	0.2 (−0.4, 0.7)	0.2 (−0.4, 0.8)	0.1 (−0.5, 0.7)	0.0 (−0.6, 0.7)
Grip strength, affected, kg				
Experimental	10.6 ± 1.0	11.4 ± 1.1	11.5 ± 1.1	11.1 ± 1.0
Placebo	10.7 ± 1.2	10.6 ± 1.2	10.6 ± 1.2	10.6 ± 1.2
Between-group differences	−0.1 (−3.2, 3.1)	0.8 (−2.4, 4.0)	0.9 (−2.3, 4.1)	0.5 (−2.4, 3.5)
Grip strength, nonaffected, kg				
Experimental	10.3 ± 1.2	10.0 ± 1.1	10.3 ± 1.1	10.2 ± 1.1
Placebo	10.1 ± 1.6	10.0 ± 1.4	10.0 ± 1.4	9.9 ± 1.4
Between-group differences	0.2 (−3.4, 3.8)	0.1 (−3.7, 3.4)	0.3 (−3.6, 3.6)	0.3 (−3.5, 3.6)

**Values are mean ± SD except for between-group differences, which are mean (95% confidence interval).*

changes in pain, as measured by the VAS, and not PPT. A larger sample size might have detected significant changes in PPT between groups. Previous studies examining the effects of either joint mobilization[49-51] or nerve mobilization[49,51] in this same population have found a difference between groups for PPT measurements. However, in these studies, the differences in PPT did not exceed the SEM and thus were within measurement error and likely not clinically meaningful. Our findings are similar to those of a study performed in individuals with carpal tunnel syndrome, in which patients who received neural mobilization did not exhibit changes in PPT different from those receiving a sham intervention.[6] However, as the authors reported, this might have been the result of differing patient expectations, which were not measured in the current study.[6]

We did not observe differences in PPT values between hands. These findings are in agreement with emerging evidence suggesting that OA-related pain cannot be attributed exclusively to local joint nociceptors (peripheral sensitization processes), because central sensitization is also present. Therefore, discrepancies between sensory and motor outcomes are expected in this population. Considering the above, the current treatment approach provides little value in inducing mechanical pain hypoalgesia associated with an increase in PPT. Similarly, without the inclusion of a healthy group, we cannot determine whether PPTs found in our sample of patients were normal. The presence of normal PPTs would also explain the lack of effect, as no improvement could have been made. Future studies are required to determine the presence of pressure pain sensitivity in this patient population. It is possible that if the patient had received manual therapy directed at the cervical spine, a change in PPT might have been produced. Numerous studies have shown that interventions directed to the cervical spine exert a mechanical hypoalgesic effect by increasing PPT.[20,21,31] The increase in PPT over the hamate on the affected side immediately postintervention is consistent with the results of Moss et al,[33] who showed that joint mobilization of the tibia or the femur for 9 minutes in patients with knee OA had an immediate, local hypoalgesic effect. These techniques are frequently performed and are often included as an integral part of the rehabilitation program.[16,17]

Despite the fact that patients in the experimental group performed exercises to improve pinch and grip strength for 4 weeks, no changes were identified. These findings are consistent with the results of Rogers and Wilder,[37] who used the same exercise program in this population. In contrast, the authors of a recent review[46] concluded that the current literature supports the use of orthoses, exercises, application of heat, and joint protection education combined with adaptive equipment to improve grip strength and function in patients with OA of the hand. Discrepancies between exercise programs could be related to the fact that not all regimens are developed based on a clinical and biomechanical analysis of the pathology.[47] Future studies should investigate which exercise regimens are the most appropriate for reducing pain and improving function in patients with CMC joint OA. It should be noted that in our population, despite the pain related to the CMC joint, there were no apparent strength deficits based on the similar strength values between the affected and nonaffected

sides through the course of the study.

Determining the exact reason for the reduction in pain experienced in the treatment group is beyond the scope of this study. However, it has recently been speculated that joint mobilization may exert its effects through a neurophysiological rather than a biomechanical response.[5,8] It has been suggested that supraspinal pain-inhibitory areas, including the periaqueductal gray matter, can be stimulated by joint mobilization.[5,56] It has also been shown that with arthritis, mechanical loading of the involved tissues resulted in smaller amounts of substance P released from the dorsal root ganglia and spinal cord, at least in the rat model.[32] The reason patients' perception of pain was significantly better but the PPTs were not better requires further elaboration. As mentioned by Bialosky et al,[6] this might be directly related to patient expectations, and the treating therapist's attitude could have impacted the outcomes.[48] It is also possible that psychosocial issues may be associated with differing outcomes of pain responses. A recent clinical trial involving patients with low back pain showed that psychological factors were not correlated with the pain response.[7] However, these researchers used temporal summation rather than PPTs to report the pain response in their study. It is possible that manual therapy has different effects on delta fiber–mediated pain compared to c-fiber pain. This hypothesis requires further scientific investigation.

There are a number of limitations to this study that must be considered. We did not use an outcome measure of function, which would have provided an indication as to the effectiveness (or lack thereof) of the current treatment approach for improving function in individuals with CMC joint OA. Additionally, because only 1 therapist performed all the interventions, the generalizability of the results may be limited. Because the follow-up period was limited to 2 months, we cannot be certain if the pain reduction would last beyond that time. The patients all received the same standard treatment and might have been either overtreated or undertreated as a result, without specifically addressing impairments unique to each patient. Future randomized clinical trials should use multiple therapists, include a measure of function, and collect data at a long-term follow-up.

CONCLUSION

This study provides evidence that a multimodal intervention consisting of joint mobilization, neural mobilization, and exercise is beneficial to reduce pain in patients with CMC joint OA. However, the treatment approach did not produce change in PPTs or pinch and grip strength. ◉

KEY POINTS

FINDINGS: The application of a multimodal manual therapy intervention of joint mobilization, neural mobilization, and exercise is beneficial to reduce pain in patients with CMC joint OA. No changes in PPT and motor function were observed.

IMPLICATIONS: Physical therapists should consider using these interventions for the management of CMC joint OA–related pain.

CAUTION: We only assessed short-term outcomes and did not assess whether the changes in pain translated into better function.

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