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Subacromial Corticosteroid Injection for Poststroke Shoulder Pain: An Exploratory Prospective Case Series

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Abstract

Objective: To assess the effectiveness of subacromial corticosteroid injections for poststroke shoulder pain.

Design: Exploratory, prospective case series.

Setting: Ambulatory setting, university-affiliated hospital.

Participants: Stroke survivors (N=10) with pain in the hemiparetic shoulder.

Intervention: Consecutive stroke survivors with evidence of supraspinatus impingement, supraspinatus tendonitis, or subacromial bursitis received subacromial corticosteroid injections.

Main Outcome Measures: The primary outcome measure was the Brief Pain Inventory (BPI) question 12 (BPI 12), which assesses “worst pain” in the previous 7 days. Secondary measures included BPI question 15, which assesses present pain and BPI question 23 (BPI 23), which assesses pain interference with 7 daily activities. Outcomes were assessed at baseline, weekly for the first 4 weeks and then at 8 and 12 weeks postinjection.

Results: Repeated measure analysis of variance revealed significant within group time effect for BPI 12 ($F=7.7$, $P<.001$). Based on absolute means, the largest therapeutic benefit was seen by the second week postinjection with partial loss of effect thereafter. There were significant within group time effects for the general activity ($F=3.2$, $P=.009$), sleep ($F=3.9$, $P=.003$), and enjoyment of life ($F=2.3$, $P=.044$) domains of BPI 23.

Conclusions: Subacromial corticosteroid injection is associated with significant reduction in poststroke shoulder pain in patients with evidence of supraspinatus impingement, supraspinatus tendonitis, or subacromial bursitis. However, there is a gradual loss of effect with time. Controlled trials are needed to show a cause and effect relationship.

Keywords

Injections; Rehabilitation; Shoulder pain; Stroke

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NEARLY ONE THIRD OF ALL stroke survivors experience shoulder pain at some time during their recovery.^{1,2} In the majority of cases, pain is moderate to severe and significantly limits ADLs.² Postulated causes of poststroke shoulder pain include adhesive capsulitis, glenohumeral subluxation, complex regional pain syndrome, spasticity, and brachial plexopathy.³ However, in 1 case series, approximately half of the patients reported significant reduction in pain after a single subacromial injection of a short-acting anesthetic.⁴ Given the role of the rotator cuff in maintaining glenohumeral stability, supraspinatus impingement, supraspinatus tendonitis, and associated subacromial bursa inflammation may be important causes of shoulder pain in the stroke population. Among able-bodied persons, subacromial corticosteroid steroid injection has been shown to be effective as part of a comprehensive treatment for shoulder pain.^{5,6} A recent retrospective case series reported that subacromial corticosteroid injection is associated with significant reduction in poststroke shoulder pain.⁷ Patients experienced an absolute mean reduction of 2.6 ± 3.7 (95% confidence interval, 1.7-3.6; $P < .001$) on a 0 to 10 pain NRS. This corresponded to a mean relative reduction of $32.9 \pm 53.6\%$. Fifty-three (45%) patients satisfied the minimum clinically important 2-point absolute and 30% relative pain reduction criteria, respectively.⁸ However, due to the retrospective nature of the study, there was only 1 postinjection assessment and the time of this assessment varied significantly with a mean of 7.4 ± 3.0 weeks. There are no prospective studies that evaluate the effectiveness of subacromial corticosteroid injections for poststroke shoulder pain. Thus, the primary objective of this exploratory prospective case series was to describe the time course of pain reduction after a single subacromial corticosteroid injection for poststroke shoulder pain. A secondary objective was to describe the time course of pain interference with ADLs.

METHODS

Participants

We recruited participants from a stroke rehabilitation ambulatory clinic of an academic medical center. Inclusion criteria included duration of shoulder pain of at least 1 month, age older than 18 years old, ability to follow commands, ability to recall 3 items after 5 minutes, clinical evidence of supraspinatus impingement, supraspinatus tendonitis or subacromial bursitis, shoulder pain on the BPI 12⁹ of 3 or greater, and access and willingness to participate in regular telephone interviews for 3 months postinjection. Clinical evidence of supraspinatus impingement, supraspinatus tendonitis, or subacromial bursitis included shoulder pain with overshoulder activities on history and presence of subacromial tenderness on palpation, tenderness at the deltoid insertion on palpation, pain with passive abduction (Neer sign), or pain with passive abduction and internal rotation of the shoulder (Hawkin sign) on physical examination.¹⁰ Exclusion criteria included inability to communicate pain levels due to severe aphasia or cognitive deficits, shoulder pain prior to the stroke, radiographic evidence of a fracture (scapula, clavicle, or humerus), or subacromial corticosteroid injection within the previous 3 months. Participants were characterized with respect to demographics, stroke characteristics, and stroke manifestations.

Injection Procedure

All injections were performed by an established academic physiatrist who performs approximately 50 subacromial injections per year in the context of a comprehensive outpatient stroke rehabilitation program. All injections were performed in a sterile manner using a posterolateral approach.¹¹ The posterior-lateral aspect of the acromion was identified by palpation. The needle was angled approximately 30° anterior to the coronal plane and slightly superior to the transverse plane, and inserted just below the angle of the acromion to the depth of approximately 1.5 to 2cm. After a negative aspiration for blood, all participants received a mixture of triamcinolone acetonide and 1% lidocaine. All participants were nominally prescribed 60mg of triamcinolone acetonide and 2.5cc of 1% lidocaine. However, diabetic patients were prescribed 40mg of triamcinolone acetonide to reduce the risk of hyperglycemia.

Outcomes Assessment

We assessed participants at preinjection, weekly for the first 4 weeks postinjection, and then at 8 and 12 weeks. A 3-day window was allowed for the completion of each outcomes assessment. Outcomes were assessed with the BPI questionnaire, a multiple-question self-reported metric, which assesses both pain intensity (sensory dimension) and the interference (reactive dimension) of pain in everyday life. The BPI has shown both reliability and validity across cultures and languages.^{9,12} As per the recommendations of the developers of the BPI, we selected BPI 12, the “pain worst” rating scale, as the primary response variable. The question asks participants to rate their worst shoulder pain in the last week on an 11-point NRS of 0 to 10, where 0 indicates “no pain” and 10 indicates “pain as bad as you can imagine.” BPI 12 assesses pain during the past week without specifying or simulating a level of activity so that the pain score is most relevant to participants’ real life activities.

Secondary measures included BPI 15 and BPI 23. BPI 15 asks participants to rate their present shoulder pain on the same 11-point NRS that is used for BPI 12. BPI 23 assesses the degree to which pain interferes with several daily activities, including general activity (ADLs), mood, walking ability, normal work, interpersonal relationships, sleep, and enjoyment of life. Interference is assessed on an 11-point NRS, where 0 indicates “does not interfere” and 10 indicates “completely interferes.”

Concomitant Therapies

All participants were prescribed shoulder specific therapies as per routine clinical practice, unless indicated otherwise. Prescribed treatments included gentle range of motion exercise within the pain-free range in all planes. For those with volitional movement, strengthening of the rotator cuff and scapular stabilizers were prescribed. Treatments also included the incorporation of the hemiparetic upper limb for functional tasks as well as repetitive task-specific movement therapy.¹³ For ethical reasons, the use of TENS and pain medication were allowed, but only if they were already prescribed prior to enrollment in the study.

Analysis

BPI 12, BPI 15, and all 7 domains of BPI 23 were assessed using repeated measure ANOVA. If repeated measure ANOVA yielded a significant time effect, post hoc pair-wise

comparisons relative to baseline adjusted for multiple comparisons (Sidak) were carried out. Missing data were handled by last value forward imputation.

Outcomes were also assessed with respect to individual success. Treatment success was defined according to the following criteria: participants must first experience a minimum of 30% pain reduction on the primary outcome measure (BPI 12) relative to baseline by 4 weeks postinjection.⁸ They must then maintain this minimum pain reduction at 8 and 12 weeks. A participant who satisfies the 30% pain reduction criterion at 4 weeks, but not at 8 or 12 weeks, likely represents an initial responder with nonsustaining therapeutic effect. Meeting the 30% criterion at 8 or 12 weeks, but not at 4 weeks, likely represents a phenomenon unrelated to the intervention, such as natural recovery, response to another factor, or random noise.

RESULTS

Ten stroke survivors enrolled in the study. One subject was inadvertently enrolled without satisfying the memory inclusion criterion. However, pain scores on telephone interviews corroborated with pain scores recorded during prescribed therapy sessions. One diabetic participant received 40mg of triamcinolone, but all others received the 60mg dose. All participants tolerated the injection procedure without complications. All but 1 participant (participant 5) also exhibited significant pain reduction immediately after the injection consistent with a positive Neer test.¹⁰ Demographic, stroke characteristics, stroke manifestations, and baseline outcome measures are shown in table 1. Five of 10 participants exhibited glenohumeral subluxation. However, four of these exhibited only half finger breadth subluxation, whereas one exhibited 1 finger breadth subluxation. All but 1 participant exhibited hyper-reflexia in the affected upper limb. One participant exhibited hand edema with pain on palpation of the metacarpophalangeal joints and passive extension of the wrist and fingers. Of 140 possible BPI 12 and 15 data points, 12 or 9% were missing. Of 490 possible BPI 23 data points, 47 or 10% were missing. The primary reason for missing data was the inability to contact the participant after multiple attempts within the 3-day window.

Repeated measure ANOVA revealed significant within group time effect for BPI 12 ($F=7.7$, $P<.001$). The time course for BPI 12 is shown in figure 1. Based on absolute means, the largest therapeutic benefit was seen by the second week postinjection. Thereafter, there was partial, but gradual loss of effect up to 8 weeks postinjection. At 12 weeks, pain level again decreased. Post hoc analysis (table 2) revealed significant reduction in BPI 12 relative to baseline at 2, 3, 4, and 12 weeks postinjection, but not at 8 weeks postinjection.

There were 7 treatment successes and 3 treatment failures. Individual BPI 12 data normalized to baseline for the treatment success and failure participants are shown in figures 2 and 3, respectively. All treatment success participants experienced substantial reduction in BPI 12 by 3 weeks postinjection. Two participants experienced some worsening of pain at 8 weeks, but pain levels were still within the treatment success criterion. Two of 3 treatment failure participants experienced pain reduction initially, but pain returned back toward baseline by week 4. The third treatment failure participant did not experience substantial

pain reduction at any time during the study period. All treatment failure participants exhibited subluxation. Thus, only 2 of 5 participants with subluxation experienced treatment success, whereas all 5 participants without subluxation experienced treatment success ($P=.04$, chi-square).

Repeated measure ANOVA also showed significant within group time effects for general activity ($F=3.2$, $P=.009$), sleep ($F=3.9$, $P=.003$), and enjoyment of life ($F=2.3$, $P=.044$) domains of BPI 23. The time courses for these measures are shown in figures 4 through 6, respectively. In general, these time courses were similar to that of BPI 12. However, post hoc analyses revealed less robust findings. Significant reduction in pain interference with general activity was observed only at 4 weeks postinjection with a trend toward significant reduction at 3 weeks. There was a trend toward significant reduction in pain interference with sleep at 2 and 3 weeks postinjection, but not at any other time periods. Post hoc analysis did not detect any significant or trend toward significant reduction in pain interference with enjoyment of life. There were no significant within group time effects for BPI 15 or the mood, walking ability, normal work, and relationship domains of BPI 23.

Table 3 shows the concomitant therapies for all participants and their respective treatment success designations. Six of 10 participants received shoulder specific therapies. Participants 2 and 6 were prescribed therapies, but elected not to attend. Participant 4 was not prescribed therapies because there was a prior history of excellent long-term response to subacromial corticosteroid injection without concomitant therapies. Therapy was inadvertently not prescribed for participant 9.

DISCUSSION

Subacromial corticosteroid injection was associated with significant reduction in poststroke shoulder pain in patients with evidence of supraspinatus impingement, supraspinatus tendonitis, or subacromial bursitis. The therapeutic benefit peaked at 2 weeks postinjection, but gradually waned thereafter. Seven of 10 participants experienced clinically important reduction in shoulder pain as reflected by BPI 12. Participants also showed reduction in pain interference with general activity, sleep, and enjoyment of life based on BPI 23. To the best of our knowledge, this represents the first prospective study exploring the effectiveness of subacromial corticosteroid injection for poststroke shoulder pain.

There are several reasons for an increased risk of supraspinatus impingement, supraspinatus tendonitis, and associated subacromial bursitis in the hemiplegic shoulder. Degenerative changes in the supraspinatus are already highly prevalent in this population, independent of their stroke.^{14,15} For those with severe hemiparesis and glenohumeral subluxation, traction forces in the glenohumeral joint may further contribute to supraspinatus tears.¹⁶ Several mechanisms that normally protect the supraspinatus from impinging below the acromion are also lost in hemiplegia. These include the loss of rotator cuff function in conjunction with spasticity of the supraspinatus and deltoids; loss of scapular rotation during abduction due to loss of trapezius and serratus function in conjunction with increased tone of the latissimus dorsi, levator scapulae and rhomboid muscles; and loss of external rotation of the humerus during abduction due to weakness of the lateral rotators and hypertonia of the medial

rotators.¹⁷ Given these risks, it is reasonable to postulate that trauma due to traction on lifting, handling, and over-aggressive exercises damages the rotator cuff, causes inflammation and tendonitis, and results in shoulder pain. Pre-existing rotator cuff injuries may be also exacerbated by the loss of these protective mechanisms.

Data suggest a time dependent therapeutic effect of subacromial corticosteroid injections. Based on absolute means of BPI 12 (see fig 1), participants experienced rapid pain reduction in pain, peaking at 2 weeks postinjection. Even with median duration of pain of 4.5 months, corticosteroids appear to have a therapeutic effect, suggesting that an inflammatory mechanism has a role in the genesis and persistence of poststroke shoulder pain. After the peak effect, the mean pain level gradually increased such that post hoc analysis failed to detect a statistically significant therapeutic effect at 8 weeks postinjection relative to baseline. Although the nonsignificance at 8 weeks could be an artifact of inadequate power and conservative adjustment for multiple post hoc comparisons, the time dependent reduction of therapeutic effect of triamcinolone is likely to be real. Nevertheless, at 12 weeks, there was an unexpected reduction in BPI 12. Given the time from the initial injection, this is unlikely to be directly related to the corticosteroid; however, this may still represent natural recovery “piggy-backed” on the initial therapeutic effect of the corticosteroid.

Three of 10 participants did not experience treatment success based on the criterion of minimum of 30% reduction in BPI 12 relative to baseline at weeks 4, 8, and 12. Success rate of 70% is higher than the rate reported in the retrospective case series.⁷ However, due to the small sample size and differences in study design, direct comparisons would not be valid. There are several plausible explanations for the 3 treatment failures. All 3 exhibited glenohumeral subluxation. All 5 participants without subluxation experienced treatment success compared with only 2 of 5 participants with subluxation. Thus it is possible that the primary source of pain was the joint capsule rather than the subacromial bursa in these 3 participants. Participant 5 showed no evidence of the short-term benefit that had been shown by all others (see fig 3). In addition to the shoulder pain, this participant exhibited hand edema, metacarpophalangeal tenderness on palpation, pain with passive wrist and finger extension, and a negative Neer test. It is likely that this participant had complex regional pain syndrome I and the shoulder pain was due to pathology distinct from supraspinatus impingement, supraspinatus tendonitis, or subacromial bursitis. Participants 2 and 10 both exhibited short-term effects, but reverted back to baseline shortly thereafter. Participant 10 had a previously documented partial thickness tear of the supraspinatus, which may have contributed to the nonsustained therapeutic benefit of the corticosteroid. Participant 2 had a more recent stroke with shoulder pain of only 2-month duration. She was prescribed shoulder specific therapies, but chose not to attend. Possible explanations for treatment failure include incorrect placement of the injection solution,¹⁸ failure to comply with therapies, and as noted above, pain source other than the subacromial bursa.

Although there was significant reduction in BPI 12 scores, there was no effect on BPI 15. This was likely due to the fundamental differences between these measures. BPI 15 assesses present pain or pain at rest. Most stroke survivors experience shoulder pain only when they are performing over-shoulder activities, completing ADLs, or sleeping. BPI 15 in the

context of the present study would not capture these experiences. In contrast, BPI 12 assesses the worst pain during the past week without specifying the level of activity so that the pain score is most relevant to participants' actual experience during their routine daily activities.

Data suggest that shoulder pain has a negative impact on QOL, and pain reduction associated with subacromial corticosteroids may improve pain related QOL. QOL is generally referred to as a multidimensional construct involving the physical, emotional, functional, and social domains, which allows us to view the impact of disability, illness, or pain on the person as a whole.¹⁹ BPI 23, which assesses pain interference with general activity, walking ability, mood, vocation, relationships, sleep, and general enjoyment of life is consistent with this construct. Based on the data shown in table 1, shoulder pain appears to interfere most with sleep, general activity, and enjoyment of life. Not surprisingly, repeated measure ANOVA identified these 3 domains as exhibiting significant within-group time effect in association with the subacromial corticosteroid injection. However, post hoc analyses were able to detect statistically significant differences relative to baseline with only the general activity domain and only at 4 weeks. This inability to detect differences is primarily a power issue and was likely due to the higher variance of BPI 23 scores, especially at 8 and 12 weeks, the small sample size, and the conservative post hoc adjustment for multiple comparisons.

The treatment for poststroke shoulder pain should be rational and based on understanding of pathophysiology and etiology. In their review of treatment options for poststroke shoulder pain, Snels et al²⁰ proposed 3 approaches according to postulated etiology: normalization of muscle tone; reduction of subluxation; and treatment of shoulder capsule. There is now emerging evidence that suggest that tone reduction, especially of the subscapularis, is effective in reducing poststroke shoulder pain.^{21,22} The effectiveness of neuromuscular electrical stimulation to reduce subluxation is also supported by multiple RCTs.^{13,23} Although the review was optimistic regarding the effectiveness of intra-articular corticosteroid injections for the treatment of adhesive capsulitis, an earlier RCT by these same authors failed to show this.²⁴ Snels et al²⁰ did not consider the treatment of other extracapsular causes, such as supraspinatus impingement, supraspinatus tendonitis, and subacromial bursitis in their review because there was very little in the literature to justify their inclusion. In all likelihood, 2 or more etiologies contribute to an individual stroke survivor's shoulder pain. Rather than treating poststroke shoulder pain in a step-wise approach, consideration should be given to postulated etiology based on history, examination, and diagnostic studies. The specific etiology should then be addressed with appropriate measures. This study suggests that supraspinatus impingement, supraspinatus tendonitis, and subacromial bursitis should be considered in the differential diagnosis for poststroke shoulder pain.

Study Limitations

From a methodologic perspective, this prospective case series is a significant improvement over the previously published retrospective case series; nevertheless, the study has several limitations. Most importantly, as a case series, there was no control group. Spontaneous

recovery could be responsible for the observed reduction in shoulder pain. However, in 1 RCT of neuromuscular electrical stimulation for poststroke shoulder pain, the control group experienced a mean pain reduction of 2.3 points on BPI 12 over a 12-month postintervention period.²⁵ Thus, in the present study, it seems highly unlikely that the mean reduction of 5.3 points on BPI 12 only 2 weeks postinjection could be explained by natural recovery alone. Second, placebo effect and observer bias cannot be ruled out. Third, concomitant therapies were allowed, which may have confounded the study results, but although this is a possibility, there is little in the data that suggest that this occurred. Four of 6 participants who received shoulder specific therapies were treatment successes, but 3 of 4 participants who did not receive therapies were also treatment successes. Four of 7 participants who received pain medications were treatment successes, but all 3 participants who did not receive pain medications were also treatment successes. Three participants used TENS; however, 2 of these were treatment failures. Fourth, 1 participant who did not meet the memory inclusion criterion enrolled in the study; this participant was deemed a treatment success. Given the small sample size, this could have influenced the overall results of the study. Thus, we repeated the analysis without this participant's data. Repeated measure ANOVA continued to exhibit significant within group time effect ($F=6.3$, $P<.001$). Post hoc analysis also revealed similar results except that pain reduction at week 4 converted to a trend toward significance ($P=.086$) from significance. Fifth, follow-up was limited to 3 month postinjection. It is possible that at longer follow-up, there would be no therapeutic benefit relative to baseline. Finally, as an exploratory case series the sample size was small and our ability to generalize the results of this study to the broader stroke population and clinical practice is limited. Nevertheless, this case series provides an initial estimate of effect size for the design of a pilot RCT.

CONCLUSIONS

To the best of our knowledge, this is the first prospective report evaluating the effectiveness of subacromial corticosteroid injection for poststroke shoulder pain. The study suggests that the onset of therapeutic effect is relatively rapid and peaks at 2 weeks postinjection. The effect wanes thereafter, with the majority of participants experiencing a gradual reduction in effect, although a few experience rapid reduction. The therapeutic effect of the corticosteroid is associated with reduction in pain interference with general activities, sleep, and enjoyment of life. However, this study lacked the power to delineate a specific pattern of effect. Although a positive exploratory case series suggests effectiveness of an intervention, cause and effect cannot be invoked due to the lack of a control. A pilot, controlled RCT is the next step to guide the design of a definitive placebo-controlled RCT.

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List of Abbreviations

ADLs activities of daily living

ANOVA	analysis of variance
BPI	Brief Pain Inventory
BPI 12	Brief Pain Inventory question 12
BPI 15	Brief Pain Inventory question 15
BPI 23	Brief Pain Inventory question 23
NRS	Numeric Rating Scale
QOL	quality of life
RCT	randomized controlled trial
TENS	transcutaneous electrical nerve stimulation

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Fig. 1. Time course of BPI 12 scores (mean $\pm$ SE)

Fig. 2. Time course of individual BPI 12 scores of treatment success participants

Fig. 3. Time course of individual BPI 12 scores of treatment failure participants

Fig. 4. Time course of BPI 23-General activity (mean $\pm$ SE)

Fig. 5. Time course of BPI 23-Sleep (mean $\pm$ SE)

Fig. 6. Time course of BPI 23-Enjoyment of life (mean $\pm$ SE)

Table 1**Participant Characteristics**

Characteristic	Data
Age (y)	56.3±11.1
Sex	7/10 women
Time from stroke (mo)	22.4±33.1 (median, 13; IQR, 1–21)
Stroke type	9/10 nonhemorrhagic
Duration of shoulder pain (mo)	8.6±8.7 (median, 4.5; IQR, 2.8–14.8)
Subluxation	5/10
Neglect	0/10
Side of hemiparesis	8/10 left
Abduction pain-free ROM	65.0±26.2
Sensory impairment	6/10
Manual muscle examination	3.9±0.7 (median, 4.0; IQR, 3.8–4.0)
BPI 12	7.8±2.2
BPI 15	2.4±3.5
BPI 23-general activity	6.2±2.5
BPI-23 mood	4.0±3.7
BPI-23 walking	2.5±4.1
BPI 23 normal work	3.6±2.6
BPI 23 relations	1.9±3.3
BPI 23 sleep	6.8±3.2
BPI 23 enjoyment of life	4.4±4.1

NOTE. Continuous variables are means ± SD unless otherwise indicated.

Abbreviations: IQR, interquartile range; ROM, range of motion.

Table 2

Post Hoc Pair-Wise Comparisons Relative to Baseline

Measure	Assessment Period (wk)	Difference Relative to Baseline	<i>P</i>	95% CI
BPI 12	1	2.9±0.9	0.224	0.9 to 6.7
	2	5.3±0.9	0.007	1.4 to 9.2
	3	4.3±0.9	0.018	0.6 to 8.0
	4	3.9±0.9	0.042	0.1 to 7.7
	8	3.3±1.0	0.172	−0.9 to 7.5
	12	4.6±0.8	0.006	1.2 to 7.9
BPI 23	1	2.9±1.1	0.412	−1.6 to 7.4
General activity	2	4.1±1.1	0.109	−0.6 to 8.8
	3	4.0±1.0	0.052	−0.0 to 8.0
	4	3.3±0.8	0.044	0.0 to 6.5
	8	3.1±0.8	0.106	−0.4 to 6.6
	12	2.9±1.0	0.317	−1.3 to 7.1
BPI 23	1	3.2±1.4	0.642	−2.6 to 9.0
Sleep	2	4.5±1.1	0.058	−0.1 to 9.1
	3	4.1±1.1	0.090	−0.4 to 8.6
	4	3.7±1.3	0.310	−1.6 to 9.0
	8	2.7±1.3	0.746	−2.6 to 8.0
	12	2.8±1.3	0.697	−2.5 to 8.1
BPI 23	1	2.5±1.5	0.950	−3.8 to 8.8
Enjoyment of life	2	3.1±1.2	0.496	−2.0 to 8.2
	3	3.2±1.1	0.278	−1.3 to 7.7
	4	2.7±1.0	0.436	−1.6 to 7.0
	8	0.5±1.6	1.000	−6.3 to 7.3
	12	2.2±1.0	0.647	−1.8 to 6.2

NOTE. Values are mean ± SE unless otherwise indicated.

Abbreviation: CI, confidence interval.

Table 3

Concomitant Therapies for Each Participant

Participant	Therapy (h)	Pain Medications	TENS	Treatment Success
1	10	no	no	yes
2	0	yes	no	no
3	4	yes	yes	yes
4	0	no	no	yes
5	15	yes	yes	no
6	0	yes	no	yes
7	11	yes	no	yes
8	7	yes	no	yes
9	0	no	no	yes
10	5	yes	yes	no