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RESEARCH ARTICLE

Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: a blinded, controlled study

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Abstract Our aim was to describe the differences in the presence of trigger points (TrPs) in the shoulder muscles and to investigate the presence of mechanical hypersensitivity in patients with unilateral shoulder impingement and healthy controls. Twelve patients with strictly unilateral shoulder impingement and 10 matched controls were recruited. TrPs in the levator scapula, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles were explored. TrPs were considered active if the local and referred pain reproduced the pain symptoms and the patient recognized the pain as a familiar pain. Pressure pain thresholds (PPT) were assessed over the levator scapulae, supraspinatus, infraspinatus, pectoralis major, biceps brachii, and tibialis anterior muscles. Both explorations were randomly done by an assessor blinded to the subjects' condition. Patients with shoulder impingement have a greater number of active (mean ± SD: 2.5 ± 1; $P < 0.001$) and latent (mean ± SD: 2 ± 1; $P = 0.003$) TrPs when compared to controls (only latent TrPs, mean ± SD: 1 ± 1). Active TrPs in the supraspinatus (67%), infraspinatus (42%), and subscapularis (42%) muscles were the most prevalent in the patient group. Patients showed a significant lower PPT in all muscles when compared to controls ($P < 0.001$). Within the patient group a significant positive correlation between the number of TrPs and pain intensity ($r_s = 0.578$; $P = 0.045$) was found. Active TrPs in some muscles were associated to greater pain intensity and lower PPTs when compared to those with latent TrPs in the same muscles ($P < 0.05$). Significant negative correlations between pain intensity and PPT levels were found. Patients with shoulder impingement showed widespread pressure hypersensitivity and active TrPs in the shoulder muscles, which reproduce their clinical pain symptoms. Our results suggest both peripheral and central sensitisation mechanisms in patients with shoulder impingement syndrome.

Keywords Shoulder impingement · Trigger points · Pressure pain · Sensitization

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Introduction

Shoulder pain is a common health problem that has a multifactorial underlying pathology and is associated with high societal cost and patient burden. The 1-year prevalence of shoulder disorders ranges from 20 to 50%, depending on the definition of the condition and socio-demographic

features (Pope et al. 1997; Luime et al. 2004). It is estimated that the incidence of shoulder disorders ranges from 7 to 25 per 1,000 consultations with general physicians (Van der Windt et al. 1995). A recent survey found that the prevalence of shoulder pain as reported by practitioners was 12%, with the most prevalent working diagnosis impingement syndrome (13%) (Pribicevic et al. 2009). In 2000, the direct costs for the treatment of shoulder disorders in the United States were $7 billion (Meislin et al. 2005).

Shoulder impingement syndrome is considered the most common intrinsic cause of shoulder pain and disability. The etiology of shoulder impingement is not completely understood, but there is evidence showing the role of shoulder muscles as a potential related factor to this condition (Tyler et al. 2005). For instance, patients with light to moderate shoulder impingement syndrome had late recruitment of scapular muscles during arm elevation (Moraes et al. 2008). Ludewig and Cook (2000) found an increased upper and lower trapezius muscle activity during shoulder abduction in patients with shoulder impingement syndrome. Due to this imbalance in muscle activation, some authors have suggested that myofascial trigger points (TrPs) may play a relevant role in shoulder impingement syndrome (Simons et al. 1999).

TrPs are defined as hypersensitive spots in a taut band of a skeletal muscle that are painful on contraction, stretching or manual stimulation and give rise to a referred pain distant from the spot. Muscle TrPs may be active or latent. Active TrPs are those in which both their local and referred pains are recognized by the patient as responsible for pain symptoms. Latent TrPs have the same clinical findings as active TrPs, but they are not causing clinical symptoms (Simons et al. 1999). This clinical distinction between active and latent TrPs is substantiated by histo-chemical findings because higher levels of algogenic substances and chemical mediators (i.e., bradykinin, substance P, or serotonin) have been found in active TrPs when compared with latent TrPs and non-TrPs (Shah et al. 2005, 2008).

Several studies have demonstrated that active TrPs are related to different pain syndromes such as mechanical neck pain (Fernández-de-las-Peñas et al. 2007a), chronic tension type headache (Fernández-de-las-Peñas et al. 2007b, c, d), lateral epicondylalgia (Fernández-Carnero et al. 2007), and migraine (Calandre et al. 2006; Fernández-de-las-Peñas et al. 2006). The referred pain elicited by active muscle TrPs reproduced pain patterns associated with these pathologies. There is preliminary evidence suggesting that TrPs may be implicated in the clinical picture of shoulder impingement syndrome. With a case design, Ingber (2000) described 3 patients with shoulder impingement syndrome who had not respond to traditional treatment who were successfully treated with TrPs injection of the subscapularis muscle. A recent study found the presence of active TrPs in the infraspinatus muscle in patients with unilateral shoulder pain (Ge et al. 2008). Additionally, 2 study designs promoting the relevance of TrP treatment in shoulder impingement syndrome have been published (Bron et al. 2007a; Perez-Palomares et al. 2009). Nevertheless, to the best of authors' knowledge, there are no studies investigating the presence of myofascial TrPs in patients with unilateral shoulder impingement syndrome.

The aims of the present study were: (1) to describe the differences in the presence of TrPs in the levator scapulae, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles between patients with strictly unilateral shoulder impingement and healthy controls; (2) to investigate the presence of pressure pain hyperalgesia in patients with shoulder impingement; (3) to assess the relationship between active or latent TrPs and pain intensity; and (4) to analyze if pressure pain thresholds were related to the presence of TrPs in the shoulder muscles.

Materials and methods

Participants

Patients diagnosed by an orthopedic surgeon with stage I (Frieman et al. 1994) unilateral impingement syndrome (acute inflammation and either tendonitis or bursitis) on the dominant-right side were recruited. Patients were eligible if they had unilateral shoulder complaints (described as pain felt in the shoulder or upper arm) with a duration of at least 3 months and an intensity of at least 4 on an 11-point numerical pain rating scale (NPRS) during arm elevation. Patients would need to report positive Neer and Hawkins tests for the diagnosis of shoulder impingement syndrome. The Neer test is positive when the patient reports pain during passive arm elevation (Neer 1983). The Hawkins test is positive when the patient reports pain when the arm is flexed at 90° and passively positioned in internal rotation (MacDonald et al. 2000). A recent meta-analysis revealed that the pooled sensitivity and specificity for the Neer test was 79 and 53%, respectively, and for the Hawkins test was 79 and 59%, respectively (Hegedus et al. 2008).

Patients were excluded if they exhibited any of the following criteria: 1, bilateral shoulder symptoms; 2, younger than 18 or older than 65 years; 3, history of shoulder fractures or dislocation; 4, cervical radiculopathy; 5, previous interventions with steroid injections; 6, fibromyalgia syndrome (Wolfe et al. 1990); 7, any systemic disease; 8, previous history of shoulder or neck surgery; or 9, any type of physical intervention for the neck–shoulder area during the previous year.

Additionally, age-matched right-handed controls were recruited from volunteers who responded to a local announcement. They were excluded if they exhibited a history of neck, shoulder or arm pain, history of trauma or diagnosis of any systemic disease. Both the Neer and Hawkin tests were negative. The study protocol was approved by the local ethic committee (UC 45) and conducted according to the Helsinki Declaration. All participants signed an informed consent prior to their inclusion.

Muscle trigger point examination

Muscle TrPs were explored in the levator scapulae, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles by an assessor who had more than 8 years' experience in muscle TrPs diagnosis and who was blinded to the subjects' condition. TrP diagnosis was performed following the criteria described by Simons et al. (1999) and by Gerwin et al. (1997): (1) presence of a palpable taut band in a skeletal muscle; (2) presence of a hyperirritable tender spot within the taut band; (3) local twitch response elicited by the snapping palpation of the taut band; and (4) presence of referred pain in response to TrP compression. These criteria, when applied by an experience assessor, have obtained a good inter-examiner reliability (kappa) ranging from 0.84 to 0.88 (Gerwin et al. 1997). Bron et al. (2007b) evaluated patients with shoulder pain and found that the most reliable feature of TrP was the referred pain (percentage of pair-wise agreement $\geq$70%, range 63–93%).

TrPs were considered active when both the local and the referred pain evoked by digital compression reproduced the pain symptoms (both in location and pain sensation) and the subject recognized the pain as familiar pain (Simons et al. 1999), whereas TrPs were considered latent when the local and referred pain elicited by digital compression did not reproduce symptoms familiar to the subjects. Figure 1 details the referred pain patterns evoked by TrPs in the examined shoulder muscles according to Simons et al. (1999).

TrP examination was performed in a blinded fashion. After TrP assessment in all the muscles, the participant was asked: "When I pressed these muscles, did you feel any pain or discomfort locally, and in other areas (referred

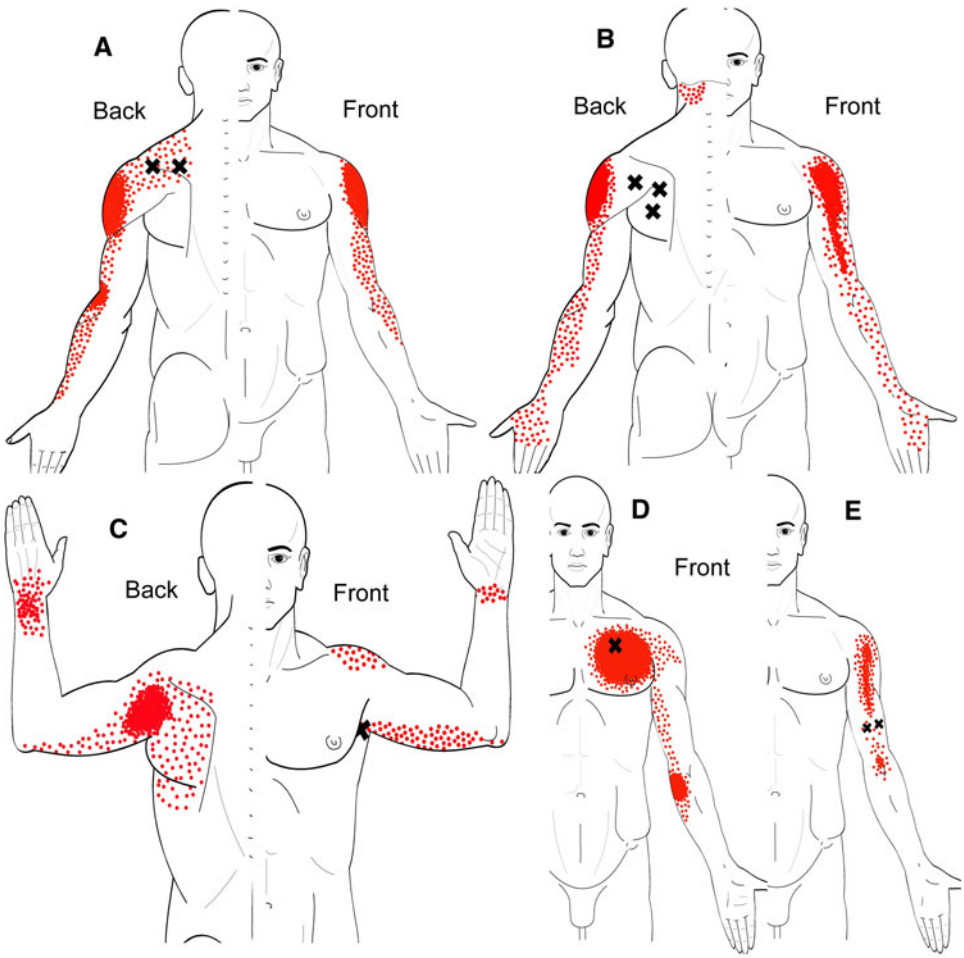

Fig. 1 Referred pain patterns from supraspinatus (**a**), infraspinatus (**b**) Subscapularis (**c**), pectoralis major (**d**), and biceps brachii (**e**) muscle TrPs as described by Simons et al. (1999)

pain). Please tell me whether the pain that you felt in the other area reproduced symptoms that you are suffering from". Participants had to indicate whether the pain elicited by palpation was located in the same area of their symptoms and reproduced the same pain sensation (active TrPs). If the elicited local or referred pain did not reproduce the same pain sensation than the patient suffered from, the TrP was considered latent.

Pressure pain threshold

Pressure pain threshold (PPT) is defined as the minimal amount of pressure where a sensation of pressure first changes to pain (Vanderweeen et al. 1996). A mechanical pressure algometer (Pain Diagnosis and Treatment Inc., Great Neck, NY) was used in this study. The device consists of a 1-cm^2 rubber disk attached to a pressure gauge, which displays values in kg/cm^2 (0–10 kg). The mean of 3 trials was calculated and used for the main analysis. A 30-s resting period was allowed between each trial. The reliability of pressure algometry has been found to be high the same day (ICC = 0.91 [95% CI 0.82–0.97]) (Chesterson et al. 2007) and between 4 separate days (ICC = 0.94–0.97) (Jones et al. 2007).

Study protocol

The study protocol was the same for shoulder patients and healthy controls. A 11-point numerical point rate scale (Jensen et al. 1999) (NPRS; 0 = no pain; 10 = maximum pain) was used to assess the intensity of current spontaneous pain and the pain experienced during arm elevation. Patients were asked to draw the distribution of their pain symptoms on an anatomic body map. None of the patients were taking any analgesic drug at the time the study was performed. Participants were asked to avoid any analgesic or muscle relaxant 72 h prior to the examination. Patients were examined when their rest pain intensity was less than 3 on a NPRS. All examinations were unilaterally conducted over the dominant-right arm, since all patients had the dominant-right shoulder affected.

PPT was first assessed over levator scapulae (2 cm superior to the superior angle of the scapula), supraspinatus (middle point over the fossa of the scapula), infraspinatus (muscle belly), pectoralis major (middle point under clavicle), major biceps (halfway between the coracoid process and the radial head), and tibialis anterior (halfway between the most superior attachment to the tibia and its tendon in the upper one-third of the muscle belly) muscles (Fig. 2). The order of point assessment was randomized between participants.

Secondly, myofascial TrPs in the levator scapulae, supraspinatus, infraspinatus, subscapularis, pectoralis major, and biceps brachii muscles were explored. The order of TrP evaluation was also randomized between participants. Both explorations were done by the same assessor who was blinded to the subjects' condition.

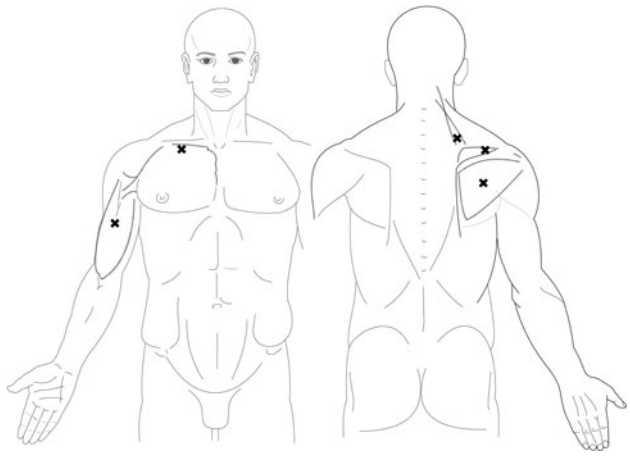

Fig. 2 Location of the points for pressure pain threshold assessment

Pressure pain threshold data management

In the current study, the magnitude of sensitization was investigated assessing the differences of absolute and relative PPT values between both groups. For relative values, we calculated a "PPT index" dividing PPT of each patient at each point by the mean of PPT score of the control group at the same point. A lower PPT Index (%) indicates greater degree of sensitization.

Statistical analysis

Data were analyzed with the SPSS statistical package (16.0 Version). Results are expressed as mean, standard deviation (SD), or 95% confidence interval (95% CI). The Kolmogorov–Smirnov test was used to analyze the normal distribution of the variables ($P > 0.05$). Quantitative data without a normal distribution (pain history, levels of pain, total number of muscle TrP, and number of latent or active TrPs) were analyzed with non-parametric tests and those data with a normal distribution (PPT) were analyzed with parametric tests. Differences in the number of myofascial TrPs (total, active, or latent TrPs) between groups were assessed with the non-parametric *U*-Man Whitney test. The chi square (χ^2) test was used to analyze the differences in the size of the distribution of muscle TrPs (active or latent) for each muscle within both study groups. Differences in PPT between both study groups were assessed with the unpaired Student's *t*-test. A one-way analysis of variance (ANOVA) test was used for assessing the differences in "PPT Index" between points. The non-parametric Kruskal–Wallis test was used to analyze the differences in the clinical pain

variables between patients with non-TrPs, latent TrPs, or active TrPs within each analyzed muscle. A one-way ANOVA test was used to analyze the differences in PPT between patients with non-TrPs, latent TrPs, or active TrPs within each analyzed muscle. The Bonferroni test was used as post hoc analysis in all multiple comparisons. The Spearman's rho (r_s) test was used to analyze the association between the number of TrPs (total, active,and latent) with those variables relating to pain symptoms and with PPT levels. Finally, the Spearman's rho (r_s) was also used to investigate the association between clinical variables and PPT over each point. The statistical analysis was conducted at 95% confidence level, and a P value less than 0.05 was considered statistically significant.

Results

Demographic and clinical data of the patients

Twelve patients, 7 men and 5 women, aged 20–38 (mean: 25 ± 9 years) diagnosed with unilateral shoulder impingement, and 10 matched controls, 5 men and 5 women, aged 20–38 (mean: 26 ± 8 years) were included ($P = 0.497$). All patients reported pain located in the anterior and posterior parts of the shoulder region, and 5 patients also reported pain in the dorso-lateral aspect of the forearm.

The mean duration of shoulder pain history was 8.5 months (95% CI 5–12). The mean spontaneous resting level of shoulder pain was 3.5 (95% CI 2.5–4.2), whereas the level of pain experienced during arm active elevation was 7 (95% CI 5.5–8). No correlation was found between shoulder pain history and the pain intensity.

Muscle TrPs in patients with shoulder impingement and healthy controls

The mean ± SD number of TrPs for each shoulder impingement patient was 4.5 ± 1 of which 2.5 ± 1 were active TrPs, and the remaining 2 ± 1 were latent TrPs. Healthy controls only had latent TrPs (mean ± SD: 1 ± 1). Therefore, the number of TrP between both groups was significantly different for both active TrPs ($z = -4.207$; $P < 0.001$) and latent TrPs ($z = -3.042$; $P = 0.003$).

The distribution of myofascial TrPs between patients and healthy controls was significantly different for the levator scapulae ($\chi^2 = 18.471$, $P < 0.001$), supraspinatus ($\chi^2 = 10.831$, $P = 0.004$), infraspinatus ($\chi^2 = 15.278$, $P < 0.001$), pectoralis major ($\chi^2 = 7.374$, $P = 0.03$), and biceps brachii ($\chi^2 = 6.926$, $P = 0.03$), but not for the subscapularis ($\chi^2 = 5.683$, $P = 0.07$), muscles. Active TrPs within the supraspinatus ($n = 8$, 67%), infraspinatus ($n = 5$, 42%), and subscapularis ($n = 5$, 42%) muscles were the most prevalent within the patient group. Table 1 summarizes the distribution of muscle TrPs for all muscles in both patients and healthy controls, and Table 2 details the number of active and latent TrPs in each patient or healthy control.

Pressure pain thresholds in patients with unilateral shoulder impingement

Patients with shoulder impingement showed significant lower PPT levels in all muscles when compared to controls: levator scapulae ($t = -6.665$; $P < 0.001$), supraspinatus ($t = -6.243$; $P < 0.001$), infraspinatus ($t = -6.984$; $P < 0.001$), pectoralis major ($t = -8.400$; $P < 0.001$), biceps brachii ($t = -4.277$; $P < 0.001$), and tibialis anterior ($t = -6.198$; $P < 0.001$) muscles (Table 3).

The ANOVA revealed significant differences for PPT indices between sites ($F = 6.215$; $P < 0.001$). The post hoc analysis revealed a greater PPT index (lesser degree of sensitization) in the biceps brachii muscle when compared to those indices of the levator scapulae ($P = 0.008$), supraspinatus ($P = 0.045$) infraspinatus ($P = 0.01$), and pectoralis major ($P = 0.01$) muscles, but not when compared to the tibialis anterior ($P = 0.9$) (Fig. 3).

Trigger point activity, shoulder pain, and PPT levels

Within the patient group, a significant positive correlation was found between the total number of TrPs and spontaneous pain intensity ($r_s = 0.578$; $P = 0.045$): the greater the

Table 1 : Distribution of myofascial trigger points (TrPs) in subjects with shoulder impingement and healthy controls

	Levator scapulae	Supraspinatus	Infraspinatus	Subscapularis	Pectoralis major	Biceps brachii
Patients with unilateral shoulder impingement syndrome						
Active TrPs (n)	5	8	5	5	2	2
Latent TrPs (n)	7	0	5	3	6	8
Non-TrPs (n)	0	4	2	4	4	2
Healthy control subjects						
Active TrPs (n)	0	0	0	0	0	0
Latent TrPs (n)	1	1	0	3	1	3
Non-TrPs (n)	9	9	10	7	9	7

Table 2 Number of active and latent myofascial trigger points in each subject with shoulder impingement and healthy control

		Number of active TrPs	Number of latent TrPs
Patients with unilateral shoulder impingement syndrome			
Patient	1	2	3
	2	2	3
	3	3	2
	4	3	3
	5	2	4
	6	2	2
	7	2	1
	8	2	2
	9	1	2
	10	2	2
	11	3	2
	12	2	3
Healthy control subjects			
Control	1	0	2
	2	0	0
	3	0	0
	4	0	2
	5	0	2
	6	0	1
	7	0	0
	8	0	2
	9	0	0
	10	0	0

pain intensity, the greater the total number of muscle TrPs. No correlation was found between duration of pain symptoms and number of TrPs ($P > 0.8$).

Further, the Kruskal–Wallis test revealed that pain experienced during arm elevation was related to the presence of TrPs in the biceps brachii ($F = 6.817$; $P < 0.015$) and subscapularis ($F = 4.379$; $P = 0.045$): those patients with TrPs, either active or latent in these muscles, showed greater

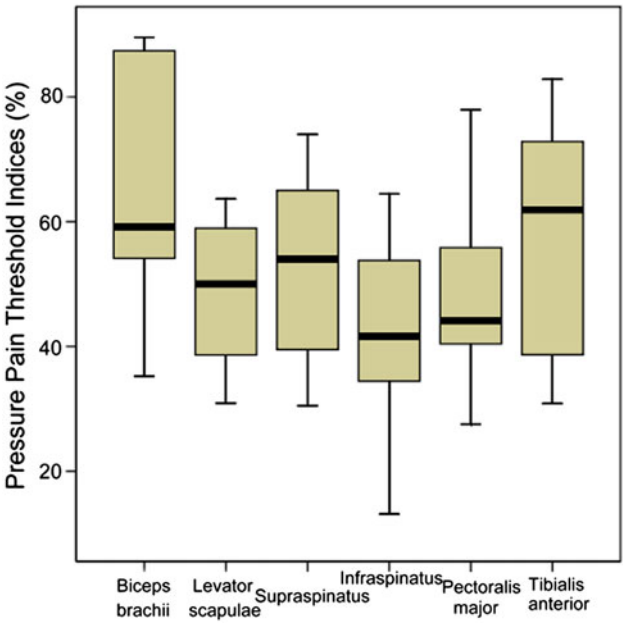

Fig. 3 Pressure pain threshold indices. The *boxes* represent the mean and the 25 and 75 percentile scores, and the *error bars* represent the standard deviation

levels of pain experienced during arm elevation than those patients not diagnosed with TrPs in the same muscles.

In addition, spontaneous pain intensity was related to the presence of active TrPs in the supraspinatus ($t = -2.257$; $P = 0.045$) and infraspinatus ($F = 4.259$; $P = 0.045$) muscles. In such a way, patients with active TrPs in these muscles showed greater levels of pain experienced during arm elevation than those not diagnosed with TrP in the same muscles. Table 4 summarizes clinical pain variables depending on TrP activity on each examined muscle.

Additionally, significant negative correlations were found between the total number of TrPs and PPT levels over the biceps brachii ($r_s = -0.759$; $P = 0.004$) and the pectoralis major ($r_s = 0.771$; $P = 0.003$) muscles. Similar correlations were also found between the number of active TrPs and PPT over the biceps brachii: ($r_s = -0.645$;

Table 3 Pressure pain thresholds (PPT, kg/cm^2) in patients with shoulder impingement syndrome and healthy controls

	Patients with unilateral shoulder impingement[#]	Healthy control subjects
Levator scapulae muscle	1.6 ± 0.4 (95% CI 1.4–1.9)	3.3 ± 0.8 (95% CI 2.8–3.9)
Supraspinatus muscle	2.1 ± 0.6 (95% CI 1.7–2.5)	4.0 ± 0.8 (95% CI 3.4–4.6)
Infraspinatus muscle	1.9 ± 0.6 (95% CI 1.5–2.3)	4.5 ± 1.1 (95% CI 3.7–5.2)
Pectoralis major muscle	1.2 ± 0.4 (95% CI 1.0–1.4)	2.4 ± 0.3 (95% CI 2.2–2.6)
Biceps brachii muscle	1.5 ± 0.4 (95% CI 1.2–1.8)	2.4 ± 0.5 (95% CI 2.0–2.6)
Tibialis anterior muscle	3.4 ± 1.1 (95% CI 2.7–4.1)	6.0 ± 0.9 (95% CI 5.4–6.6)

Values are expressed as means ± standard deviation (95% confidence interval)

[#] Significant lower PPT values when compared to healthy controls

Table 4 Shoulder pain characteristics depending on the presence of myofascial trigger points (trps) on each muscle within patients with shoulder impingement syndrome

		Spontaneous pain	Pain during shoulder movement
Levator scapulae muscle	Active TrPs ($n = 7$)	3.8 ± 1.3	7.8 ± 1.2
	Latent TrPs ($n = 5$)	2.4 ± 2.1	6.1 ± 3.7
	No TrPs ($n = 0$)	–	–
Supraspinatus muscle	Active TrPs ($n = 8$)	3.8 ± 1.9[#]	8.3 ± 1.3
	Latent TrPs ($n = 0$)	–	–
	No TrPs ($n = 4$)	1.5 ± 0.6	6.6 ± 2.9
Infraspinatus muscle	Active TrPs ($n = 5$)	4.0 ± 1[#]	8.6 ± 1.4
	Latent TrPs ($n = 5$)	4.0 ± 1.2	7.5 ± 1.2
	No TrPs ($n = 2$)	1.6 ± 0.5	5.5 ± 3.1
Subscapularis muscle	Active TrPs ($n = 5$)	4.0 ± 1.0	8.4 ± 1.2[#]
	Latent TrPs ($n = 3$)	3.4 ± 2.5	7.7 ± 1.4
	No TrPs ($n = 4$)	1.8 ± 1.0	4.3 ± 3.8
Pectoralis major muscle	Active TrPs ($n = 2$)	3.7 ± 2.5	8.1 ± 1.7
	Latent TrPs ($n = 6$)	3.0 ± 1.7	7.2 ± 0.2
	No TrPs ($n = 4$)	1.5 ± 0.7	5.6 ± 3.8
Biceps brachii muscle	Active TrPs ($n = 2$)	4.0 ± 1.4	8.5 ± 1.9[#]
	Latent TrPs ($n = 8$)	3.2 ± 2.0	7.9 ± 1.1
	No TrPs ($n = 2$)	1.0 ± 0.0	2.9 ± 4.0

Values are expressed as means ± standard deviation/NPRS = Numerical Pain Rate Scale (0–10)

[#] Significantly different between both TrPs subgroups and non-TrP subgroup (ANOVA test, Bonferroni, $P < 0.01$)

$P = 0.025$) and the pectoralis major ($r_s = 0.690$; $P = 0.015$) muscles. In such a way, the greater the total number of TrPs, particularly active TrPs, the lower was the PPT over the biceps brachii and pectoralis major muscles.

Finally, significant differences in PPT levels were also found to be dependent on TrP activity: (a) levator scapulae TrPs were related to lower PPT over the levator scapulae ($t = 2.606$; $P = 0.025$) and biceps brachii ($t = 3.970$; $P = 0.003$) muscles; (b) TrPs in the supraspinatus muscle were related to lower PPT over the levator scapulae ($t = 3.716$; $P = 0.004$), supraspinatus ($t = 2.236$; $P = 0.045$), pectoralis major ($t = 3.571$; $P = 0.005$), and biceps brachii ($t = 2.503$; $P = 0.03$) muscles; (c) infraspinatus muscle TrPs were related to lower PPT level over the levator scapulae ($F = 6.898$; $P = 0.015$) muscle; (d) subscapularis muscle TrPs were related to lower PPT levels over the levator scapulae ($F = 8.246$; $P = 0.009$), supraspinatus ($F = 5.606$; $P = 0.025$), pectoralis major ($F = 7.249$; $P = 0.015$), and biceps brachii ($F = 9.505$; $P = 0.001$) muscles; and (e) biceps brachii TrPs were related to lower PPT over the biceps brachii ($F = 4.825$; $P = 0.04$) and pectoralis major ($F = 4.368$; $P = 0.04$) muscles. Table 5 shows PPT depending on TrP activity on each examined muscle.

Pressure pain sensitivity and clinical features in unilateral shoulder impingement

Within the patient group, significant negative correlations between spontaneous pain intensity and PPT over the levator scapulae ($r_s = -0.637$; $P = 0.025$), supraspinatus ($r_s = -0.577$; $P = 0.045$), and biceps brachii ($r_s = -0.680$; $P = 0.015$) muscles were found: the greater the pain intensity, the lower the PPT levels.

Discussion

The current study showed the existence of active TrPs in the shoulder muscles in patients with unilateral shoulder impingement. Both the local and the referred pain areas elicited by manual exploration of active TrPs reproduced the pain pattern in all patients. In addition, patients with unilateral shoulder impingement showed lower PPT levels when compared to healthy controls. A greater number of TrPs and lower PPT were related to greater pain intensity: the greater the pain intensity, the greater the number of TrPs and the lower the PPT. Finally, PPT levels were lower in some muscles in patients with active TrPs when compared to those patients without TrPs. The current results suggest both peripheral and central sensitization is present in patients with shoulder impingement syndrome.

Muscle TrPs in shoulder impingement syndrome

The rotator cuff is formed by the supraspinatus, the infraspinatus, the teres minor, and the subscapularis (Keating et al. 1993). Active muscle TrPs in the supraspinatus, infraspinatus, and subscapularis muscles elicited a referred pain that mimicked the patients' usual shoulder pain. Further, active TrPs in the levator scapulae, biceps brachii, and pectoralis major were also found. When active TrPs were explored, patients spontaneously reported: "Yes, this is exactly the pain that I usually feel either spontaneously, but particularly during arm elevation". These findings support the view that active TrPs in the neck–shoulder musculature are involved in the pathophysiology of shoulder impingement syndrome and that the referred pain sensations may contribute directly to shoulder pain complaint.

Active TrPs, by definition, were not found in healthy controls, since they did not suffer from any pain symptoms. In addition, shoulder impingement syndrome subjects also showed latent TrPs in the examined muscles in a greater proportion than healthy controls. Lucas et al. found that latent TrPs disturb normal pattern of motor recruitment and movement efficiency suggesting the clinical relevance of latent TrPs (Lucas et al. 2004). Further, it has been proposed that latent TrPs may become active under the

Table 5 Pressure pain thresholds (PPT) depending on the presence of myofascial trigger points (TrPs) on each muscle within patients with shoulder impingement syndrome

		Levator scapulae	Supraspinatus	Infraspinatus	Pectoralis major	Biceps brachii	Tibialis anterior
Levator scapulae	Active TrPs ($n = 7$)	1.3 ± 0.3*	1.9 ± 0.5	1.6 ± 0.7	1.0 ± 0.2	1.1 ± 0.2*	3.5 ± 1.1
	Latent TrPs ($n = 5$)	1.8 ± 0.3	2.2 ± 0.6	2.1 ± 0.5	1.4 ± 0.4	1.8 ± 0.3	3.4 ± 1.1
	No TrPs ($n = 0$)	–	–	–	–	–	–
Supraspinatus	Active TrPs ($n = 8$)	1.4 ± 0.3#	1.9 ± 0.5#	1.8 ± 0.7	1.0 ± 0.2#	1.3 ± 0.4#	3.3 ± 1.1
	Latent TrPs ($n = 0$)	–	–	–	–	–	–
	No TrPs ($n = 4$)	2.0 ± 0.1	2.6 ± 0.4	2.2 ± 0.4	1.5 ± 0.4	1.9 ± 0.3	3.7 ± 1.2
Infraspinatus	Active TrPs ($n = 5$)	1.4 ± 0.2#	1.4 ± 0.3	1.7 ± 0.7	1.0 ± 0.2	1.3 ± 0.5	3.3 ± 1.3
	Latent TrPs ($n = 5$)	1.4 ± 0.3	2.4 ± 0.5	2.0 ± 0.4	1.4 ± 0.4	1.7 ± 0.4	3.5 ± 1.0
	No TrPs ($n = 2$)	1.9 ± 0.2	2.0 ± 0.5	2.2 ± 0.9	1.0 ± 0.1	1.5 ± 0.3	3.3 ± 1.3
Subscapularis	Active TrPs ($n = 5$)	1.3 ± 0.3#	1.8 ± 0.4#	1.5 ± 0.9	1.0 ± 0.1#	1.1 ± 0.3#	2.8 ± 1.1
	Latent TrPs ($n = 3$)	1.5 ± 0.3	1.8 ± 0.6	1.9 ± 0.5	1.0 ± 0.3	1.4 ± 0.2	4.0 ± 1.0
	No TrPs ($n = 4$)	2.0 ± 0.1	2.7 ± 0.2	2.1 ± 0.4	1.6 ± 0.4	2.0 ± 0.1	3.7 ± 1.1
Pectoralis major	Active TrPs ($n = 2$)	1.4 ± 0.2	1.7 ± 0.6	1.6 ± 0.5	1.1 ± 0.1	1.3 ± 0.3	3.9 ± 1.1
	Latent TrPs ($n = 6$)	1.7 ± 0.4	2.1 ± 0.5	2.4 ± 0.1	1.1 ± 0.4	1.4 ± 0.5	3.7 ± 0.9
	No TrPs ($n = 4$)	2.0 ± 0.1	2.5 ± 0.2	2.1 ± 0.7	1.7 ± 0.3	2.0 ± 0.1	4.0 ± 0.1
Biceps brachii	Active TrPs ($n = 2$)	1.3 ± 0.4	2.0 ± 0.8	1.5 ± 1.3	0.9 ± 0.3#	1.1 ± 0.3#	3.1 ± 1.1
	Latent TrPs ($n = 8$)	1.6 ± 0.3	2.0 ± 0.6	2.0 ± 0.5	1.1 ± 0.3	1.5 ± 0.4	4.0 ± 1.3
	No TrPs ($n = 2$)	2.1 ± 0.1	2.7 ± 0.1	2.1 ± 0.6	1.7 ± 0.3	2.0 ± 0.1	4.1 ± 0.5

Values are expressed as means ± standard deviation

* Significant differences between active and latent TrP subgroups (Student t-test, $P < 0.03$)

Significant differences TrP and non-TrP subgroups (ANOVA test, Bonferroni, $P < 0.01$)

influence of several factors such as repetitive and sustained shoulder activities (Simons 2004). Therefore, it may be that the presence of muscle TrPs, either active or latent, may be implicated in the sensory-motor disturbances often observed in patients with shoulder impingement syndrome (Ludewig and Cook 2000; Tyler et al. 2005; Moraes et al. 2008). In such a way, we do not know if inactivation of muscle TrPs can prevent recurrence of symptoms, which are very common in this patient population (Mitchell et al. 2005). Our results underline the importance of inspection and inactivating TrPs in the shoulder muscles in patients with shoulder impingement syndrome as they may contribute to the overall picture of pain. Two randomized trials are in progress in order to elucidate the role of inactivation of TrPs in patients with shoulder impingement syndrome (Bron et al. 2007a; Perez-Palomares et al. 2009).

An interesting finding was that the number of TrPs was related to a greater pain intensity of the symptoms. Further, the presence of active TrPs in different muscles was related to a greater intensity of spontaneous pain (supraspinatus/infraspinatus) and pain during arm elevation (biceps brachii/subscapularis). These findings further support the role of active TrPs within the shoulder musculature in shoulder impingement syndrome. Further, a greater number of muscle TrPs suggest the presence of spatial summation of TrP pain activity in shoulder impingement related to the intensity of the pain symptoms. Spatial summation of TrP pain activity has been also suggested in chronic tension type headache (Fernández-de-las-Peñas et al. 2007e). In fact, we do not know if the presence of numerous TrPs is responsible for shoulder pain symptoms in these patients or that muscle TrPs are activated due to pain. Although future longitudinal studies are needed to answer this question, it is more conceivable that TrPs would be responsible for pain symptoms.

Multiple active TrPs in the same muscle (i.e. infraspinatus) have been previously described in patients with shoulder pain (Ge et al. 2008). The present study is the first to report the presence of TrPs in multiple and different shoulder muscles, particularly those forming the rotator cuff. Nevertheless, it may be possible that the muscles examined in this study also showed multiple active TrPs. Moreover, Ge et al. (2008) found latent TrPs within the infraspinatus muscle on the asymptomatic side in patients with unilateral shoulder pain. It is not know if patients with unilateral shoulder impingement syndrome have muscle TrPs in the shoulder musculature within the unaffected side. Future studies investigating bilaterally the presence of multiple muscle TrPs in patients with unilateral shoulder impingement syndrome are needed.

Mechanical pain hypersensitivity in shoulder impingement syndrome

In this study, PPT levels were significantly decreased over the levator scapulae, supraspinatus, infraspinatus, biceps brachii, and pectoralis major muscles in patients with unilateral shoulder impingement syndrome when compared to healthy controls, which suggests a sensitization of muscle tissues in this patient population. This is expected since all examined muscles are involved in arm motion. These findings suggest the presence of segmental sensitization mechanisms as the examined muscles received innervation from the same segments of the cervical spine (C4–C6 segments). Consistent with a significant decrease in PPT levels over the shoulder muscles, we also found lower PPT levels in the tibialis anterior muscle suggesting multi-segmental sensory sensitization or sensitization of the central nervous system in unilateral shoulder impingement. However, we should recognize that we only investigated PPT levels over the affected side.

Nevertheless, it seems that there is a greater sensitization degree in the shoulder musculature, which is supported by the fact that the magnitude of PPT changes was higher for the levator scapulae ($49 \pm 11\%$), supraspinatus ($52 \pm 14\%$), infraspinatus ($42 \pm 13\%$), and pectoralis major ($49 \pm 15\%$) muscles when compared to the magnitude of PPT changes over the tibialis anterior ($58 \pm 18\%$) muscle.

Finally, there is no consensus about the differences in PPT levels that are needed to consider real changes between groups (Sterling 2008). Different studies conducted over the cervical spine (Chesterson et al. 2007; Ylinen et al. 2007) have suggested that differences ranging from 123 to 200 kPa (1.2–2 kg) are needed to consider real PPT differences. In the current study, differences between symptomatic (1.0–2.1 kg) and non-symptomatic (2.6 kg) regions were placed within this interval, so differences between both groups can be considered as real. However, we should consider that these studies investigating PPT changes were conducted over the cervical spine, so extrapolation of their results to the shoulder region should be done with caution.

Sensitization mechanisms associated with muscle TrPs in shoulder impingement

Pressure pain thresholds (PPT) (Chesterton et al. 2003; Rolke et al. 2005) are extensively used for investigating mechanical pain hypersensitivity in different localized pain conditions, e.g. whiplash (Sterling et al. 2003), unilateral migraine (Fernández-de-las-Peñas et al. 2008), repetitive strain injury (Greening and Lynn 1998), lateral epicondylalgia (Fernández-Carnero et al. 2009), chronic tension type headache (Fernández-de-las-Peñas et al. 2007f), low back pain (O'Neill et al. 2007), knee osteoarthritis (Bajaj et al. 2001), and carpal tunnel syndrome (Fernández-de-las-Peñas et al. 2009). These studies have consistently showed lower PPT levels in both painful and distant pain-free areas, suggesting both segmental and extra-segmental spreading of hyperexcitability.

The results of the current study reflect the presence of peripheral and central sensitization mechanisms in patients with unilateral shoulder impingement syndrome. The presence of active TrPs in the shoulder musculature suggests sensitization of muscle nociceptors since high levels of algogenic substances (Shah et al. 2005, 2008) and lower pressure pain thresholds (Ge et al. 2008) has been found in active muscle TrPs. Additionally, a study has recently demonstrated the existence of both nociceptive and non-nociceptive hypersensitivity at muscle TrPs (Li et al. 2009). These studies support that active, and also latent, muscle TrPs constitute a focus of peripheral nociceptive sensitization of both nociceptive and non-nociceptive nerve endings, evidencing the relevance of muscle TrPs for sensitization mechanisms.

We found that pressure pain hypersensitivity was negatively related to the number of active TrPs: the greater the number of active TrPs, the lower the PPT levels. Further, the presence of muscle TrPs within the shoulder muscles was also related to lower PPT in different muscles. Our findings suggest that the higher hyperalgesia may come from spatial summation of TrP-related pain in the shoulder musculature. This may also indicate that multiple active TrPs spatially increase the mechanical pain sensitivity peripherally and centrally, since PPT were not measured directly on the TrP, but on fixed points over the muscles. We could not assess PPT at TrPs since we did not know the existence of TrPs at the beginning of the study. Then, PPT levels were assessed at fixed points at the belly of the muscles in which we looked for the presence of TrPs, except for the subscapularis muscle (for practical reasons) and the tibialis anterior (non-painful point). Finally, Ge et al. reported that the association of multiple active muscle TrPs and the heterogeneity of mechanical pain hypersensitivity distribution suggest a crucial role of peripheral sensitization in unilateral shoulder pain (Ge et al. 2008).

Nevertheless, we can not exclude a role of central sensitization mechanisms in the presence of muscle TrPs. In fact, the existence of sensitization mechanisms in local pain syndromes suggests that sustained peripheral noxious input to the central nervous system plays a role in the initiation and maintenance of sensitization processes (Mendell and Wall 1965) since central sensitization is considered as a dynamic condition influenced by multiple factors including the activity of peripheral nociceptive inputs (Herren-Gerber et al. 2004). In the current study, the decrease in PPT levels was associated with the intensity of pain symptoms,

supporting a role of the peripheral nociceptive input as an important factor driving the development of spreading sensitization.

We found up to 3 active TrPs within each patient with shoulder impingement, supporting the assumption of spatial summation of TrP activity in these patients. Since active TrPs constitute a peripheral sensitization focus, the presence of multiple active TrPs may exert a spatial summation of nociceptive barrage to the dorsal horn neurons. Fernández-de-las-Peñas et al. formulated a pain model for patient with chronic tension type headache involving peripheral sensitization from active muscle TrPs and central sensitization mechanisms (Fernández-de-las-Peñas et al. 2007g). It is possible that similar sensitization mechanisms occur in shoulder impingement syndrome, although longitudinal studies are needed in order to further elucidate the role of muscle TrPs to the development of shoulder impingement syndrome.

Strengths and limitations of the study

Several methodological aspects of the current study should be mentioned. First, TrP examination was conducted by a blinded examiner ruling out of the chance of bias. Since manual palpation was done without any feedback of the participant about reproduction of pain symptoms, the examiner remained blinded until the end of the examination. This procedure has been used in previous studies (Fernández-Carnero et al. 2007; Fernández-de-las-Peñas et al. 2007a, b, c, d). Nevertheless, it is possible that a memory bias from any muscle can be present (Table 2). Second, we included a small sample size. Nevertheless, the results seem robust, which suggest that a greater sample size would not alter the direction of the results. Population-based epidemiological studies with greater sample sizes are now needed to permit a more generalized interpretation of these results. Finally, the third limitation of the current study was that we can not establish a cause-and-effect relationship between TrPs and shoulder impingement syndrome, because the design was not longitudinal and because the paper did not report the results of inactivating the active TrPs.

Conclusion

The current controlled study showed the existence of multiple active TrPs in the shoulder muscles in patients with unilateral shoulder impingement. Both local and referred pain elicited by manual exploration of active muscle TrPs reproduced the pain pattern in all patients. Patients showed pressure pain hyperalgesia in painful and non-painful distant areas, suggesting the presence of central sensitization. A greater number of TrPs and lower PPT levels were related to greater pain intensity: the greater the pain intensity, the greater the number of TrPs and the lower the PPT. Finally, active TrPs were related to lower PPT. Our results suggest both peripheral and central sensitization mechanisms in patients with shoulder impingement syndrome.

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