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CLINICAL RESEARCH

Determinants of Pain in Patients with Carpal Tunnel Syndrome

Fiesky Nunez MD, Ana-Maria Vranceanu PhD, David Ring MD, PhD

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Abstract

Background Carpal tunnel syndrome causes numbness, weakness, and atrophy. Pain without numbness is not characteristic of this disease.

Questions/purposes We tested the hypothesis that among patients with carpal tunnel syndrome confirmed by electrophysiologic testing, pain catastrophizing and/or depression would be good predictors of pain intensity at the time of diagnosis, whereas nerve conduction velocity would not.

Patients and Methods Fifty-four patients completed a measure of tendency to misinterpret pain, a measure of depressive symptoms, anxiety about pain, self-efficacy in response to pain, and a five-point Likert measure of pain intensity. One-tailed Spearman correlation was performed to find a correlation between pain and continuous variables. One-way ANOVA was performed to assess differences between categorical variables. For each group, all variables with significant correlations with pain intensity were included in a multiple linear regression analysis.

Results Sex, age, and electrophysiologic measures did not correlate with pain intensity. All measures of illness behavior correlated with pain intensity and were entered in a multiple linear regression model; only misinterpretation of nociception and depression were significantly associated and accounted for 39% of the variation in pain intensity.

Conclusions Illness behavior (specifically depression and misinterpretation of nociception) predicts pain intensity in patients with carpal tunnel syndrome.

Level of Evidence Level II, prognostic study. See Guidelines for Authors for a complete description of levels of evidence.

Introduction

Carpal tunnel syndrome is a common, discrete, incompletely understood pathologic process that can be verified objectively with electrophysiologic testing [20]. This pathologic process causes diminished sensibility in the distribution of the median nerve (the thumb, and the index, long, and radial half of the ring finger), weakness of palmar abduction, and atrophy of the intrinsic thenar muscles. The hallmark symptom of this disease is numbness that is intermittent initially and eventually becomes ever-present. The numbness can be so intense at times that it is painful, but pain without numbness is not characteristic of this disease process [20]. A recent study showed a diagnostic instrument based on numbness rather than pain correlates very well with electrophysiologic testing [5].

The diagnosis of carpal tunnel syndrome often is misapplied to patients with activity-related hand pain, pain with typing in particular [4, 15]. A study examining information regarding the etiology of carpal tunnel syndrome noted that popular media such as the Internet overemphasize the conception of carpal tunnel syndrome as pain resulting from damage to the nerve from hand use [15].

Pain is determined by nociception and illness behavior [11]. In some studies, illness behavior is a better predictor

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F. Nunez, A.-M. Vranceanu, D. Ring (✉)
Massachusetts General Hospital/Harvard Medical School, Yawkey 2100, 55 Fruit Street, Boston, MA 02114, USA
e-mail: dring@partners.org

of disabling musculoskeletal pain than objective pathophysiology or impairment [3, 8, 14, 18]. A better understanding of the influence of pathophysiology/nociception and illness behavior on pain in patients with carpal tunnel syndrome has the potential to (1) contribute to more accurate popular conceptions about the disease (ie, the misconception that carpal tunnel syndrome is "pain with typing" will be further exposed); (2) encourage further dissolution of the false mind-body dichotomy that hinders use of effective treatments (such as cognitive behavioral therapy) for patients with a predominant complaint of pain and either normal or mild electrophysiologic testing; and (3) encourage better studies of patients with a predominant complaint of pain who are offered carpal tunnel release, which may represent unnecessary or unwarranted surgery.

The purpose of this study was to assess the role of illness behavior (depression and coping strategies in particular) in reports of pain intensity in patients with electrodiagnostically confirmed carpal tunnel syndrome. This study addresses the null hypotheses that (1) there is no correlation between pain intensity and demographic variables such as age, sex, marital status, and employment; (2) there is no correlation between pain intensity and nerve conduction velocity measurements; and (3) there is no correlation between pain intensity and illness behavior variables such as misinterpretation and overinterpretation of nociception, symptoms of major depression, symptoms of anxiety related to pain, or the patient's belief in their ability to accomplish tasks despite feeling pain.

Patients and Methods

The setting was an orthopaedic hand and upper extremity practice in a tertiary care hospital. We used enrollment data from an Institutional Review Board-approved prospective cohort study of patients undergoing minor hand surgery that sought to find correlations between presurgery coping skills and self-reported upper extremity disability and pain after surgery [19]. All adult, English-speaking patients who requested surgery for carpal tunnel syndrome, trigger finger, or a benign hand tumor were invited to enroll; patients requesting revision of prior surgery for the identical problem were excluded. All of the patients with carpal tunnel syndrome in that study had confirmation of the diagnosis with electrodiagnostic testing. The electrodiagnostic criteria used for diagnosis was a value greater than 3.6 ms on distal sensory latency (DSL) or a value greater than 4.4 ms on distal motor latency (DML). The median sensory nerve conduction study (NCS) was performed across the wrist with a conduction distance of 13 cm. The median nerve was stimulated at the wrist, and the antidromic sensory nerve action potentials were recorded from Digit II. The median motor NCS was recorded from the abductor pollicis brevis and stimulated at the wrist 7 cm proximal to the recording electrodes.

The total cohort included 54 subjects, 19 men (35.2%) and 35 women (64.8%). Their average age (± SD) was 58 ± 14 years (range, 29–88 years). Ten subjects (18.5%) presented with right carpal tunnel syndrome, six (11.1%) had left carpal tunnel syndrome, and 38 (70.4%) had bilateral carpal tunnel syndrome. Sensory latency ranged from 3.3 ms to 6.5 ms with a mean of 5.34 ms and SD of 1.07. Motor latency at the wrist ranged from 4.25 ms to 14 ms with a mean of 6.5 ms and SD of 2.37 ms.

Marital status, current occupation, and number of years of academic instruction were recorded. Current occupation was stratified into professional, nonprofessional of hand labor, nonprofessional of nonlabor duties, unemployed, homemaker, retired, disabled, and workers' compensation (Table 1).

The presence of concomitant painful conditions (eg, arthritis, degenerative joint disease) and electrophysiologic testing results were used to represent the influence of pathophysiology on reports of pain intensity.

All patients completed the Pain Catastrophizing Score, the Patient Health Questionnaire–Depression Subscale, the Pain Anxiety Symptoms Scale, and the Pain Self-Efficacy Questionnaire. Pain intensity during the previous week was measured on a five-point Likert scale with 1 point

Table 1. Demographic and pathologic information

Parameter	Number (%)
Marital status	
Single	13 (24)
Living with partner	3 (5.6)
Married	24 (44.4)
Separated/divorced	7 (13)
Widowed	7 (13)
Current occupation	
Professional	9 (16.7)
Nonprofessional hand labor	8 (14.8)
Nonprofessional nonlabor	5 (9.3)
Unemployed	4 (7.4)
Homemaker	8 (14.8)
Retired	14 (25.9)
Workers' compensation	1 (1.9)
Disabled	5 (9.3)
Concomitant painful conditions	
Yes	28 (51.9)
No	26 (48.1)

	Mean	SD	Range
Years of academic instruction	13.5	2.7	4–18

representing no pain and 5 points representing extreme pain on the Disabilities of the Arm Shoulder and Hand Questionnaire.

Depression was measured with the Patient Health Questionnaire–Depression subscale (PHQ-D) [16], a reliable and valid measure of depressive symptoms within the past week. The PHQ-9 has nine questions taken directly from the Diagnostic and Statistical Manual of Mental Disorders, 4th edition [1], answered on a continuous scale from one to four.

The Pain Anxiety Symptoms Scale Short version (PASS) is a validated 20-question inventory designed to assess anxiety regarding pain [9, 10]. The inventory is a reliable measure of differentiable aspects of pain anxiety: (1) cognitive anxiety; (2) fear of pain; (3) escape and avoidance; and (4) physiologic anxiety. The cognitive–anxiety subscale evaluates symptoms such as the inability to concentrate and the frequency of unwanted thoughts when the patient is in pain. The fear-of-pain subscale measures the frequency of thoughts provoking fear and a profound dread of negative consequences when the patient is in pain. The escape and avoidance subscale rates the frequency of behaviors aimed at minimizing the severity and duration of pain. Finally, the physiological–anxiety subscale measures the patient's physical response to pain such as palpitations or feeling nauseous. With use of a six-point rating system (with 0 corresponding to never and 5 representing always), the Pain Anxiety Symptoms Scale short version has been shown to be useful for evaluation of generalized pain anxiety [9, 10].

Misinterpretation and overinterpretation of nociception was measured with the Pain Catastrophizing Scale (PCS) [17]. It has 13 items answered on a five-point scale from 0 equals not at all to 4 equals all of the time, which are subgrouped into three interrelated subscales: rumination, magnification, and helplessness [13, 17]. In addition, a combined total PCS score has been shown to be effective for assessment of generalized coping strategies [17].

The Pain Self-Efficacy Questionnaire (PSEQ) is a 10-item tool that measures the strength and generality of a patient's beliefs regarding his or her ability to accomplish a range of activities despite his or her pain [2]. Each item on the PSEQ is rated selecting a number on a seven-point scale in which 0 equals not at all confident and 6 equals completely confident. A total score is calculated by summing scores for all 10 items. A high score reflects strong self-efficacy beliefs [12].

Previous studies have provided means, SDs, and ranges for these questionnaires for reference populations (Table 2).

Statistical power calculated post hoc for this sample was 93.1% with alpha = 0.05 and large effect size f squared = 0.35. Power calculation was performed with G*Power software Version 3.0.10 (Kiel, Germany).

We conducted bivariate analyses among continuous demographic and study variables. One-tailed Spearman correlation was performed among age, pain intensity, DSL, DML, and the four measures of illness behavior. We used one-way ANOVA to assess whether there were any differences between categorical study variables such as sex and marital status. All variables with significant correlations with pain intensity ($p < 0.05$) were included in a multiple linear regression analysis.

Table 2. Measures of illness behavior

Measure	Reference scores	Mean	SD	Range
Misinterpretation of pain (pain catastrophizing score) [13]	Community: 13.9 ± SD 10; Pain clinic patients: 22.3 ± SD 10*	24.1	9.7	13–49
Symptoms of major depression (Patient Health Questionnaire–Depression Subscale) [7]	1–4 = Minimal depression; 5–9 = mild depression; 10–14 = moderate depression; 15–19 = moderately severe depression; 20–27 = severe depression†	13.8	4.6	9–27
Anxiety related to pain (Pain Anxiety Symptoms Scale)[9]	Chronic pain patients seeking help at a university center: 38.6 ± SD 20.4‡	21	19.8	0–78
Self-efficacy despite feeling pain (Pain Self-Efficacy Questionnaire)[12]	Low back pain patients attending pain management: 25.8 ± SD 12.4 Chronic pain patients awaiting behavioral therapy: 23 ± SD 12§	46.5	12.9	10–60
Pain intensity	Not applicable	2.9	1.0	1–5

* Osman A, Barios FX, Gutierez PM, Kopper BA, Merrifield T, Grittmann L. The Pain Catastrophizing Scale: further psychometric evaluation with adult samples. *J Behav Med.* 2000;23:351–365; †Kroenke K, Spitzer RL, Williams JB. The PHQ-9 validity of a brief depression severity measure. *J Gen Intern Med.* 2001;16:606–613; ‡McCracken LM, Dhingra L. A short version of the Pain Anxiety Symptom Scale (PASS_20): preliminary development and validation. *Pain Res Manag.* 2002;7:45–50; §Nicholas MK. The pain self-efficacy questionnaire: taking pain into account. *Eur J Pain.* 2007;11:153–163.

Table 3. Statistical analysis; correlation between variables and pain intensity

Parameter	Bivariate analysis: Spearman correlation (r)	Multivariable analysis: multiple linear regression (beta)
Misinterpretation of pain	.63*	.40*
Symptoms of major depression	.59*	.45*
Anxiety related to pain	.51*	−.05
Self-efficacy despite feeling pain	−.38*	.18
Distal sensory latency	−.142	Not entered
Distal motor latency	−.029	Not entered
		R-squared = .39**

* = $p < 0.05$; **the amount of variance in pain intensity explained by these variables (39%).

Results

Demographic variables do not correlate with pain intensity in carpal tunnel syndrome. There was no difference in pain intensity according to sex ($F < 0.001$; $p = 0.99$), marital status ($F = 2.37$; $p = 0.065$), job types ($F = 0.25$; $p = 0.97$), and no correlation with age ($r = 0.023$; $p = 0.43$) or number of years of academic instruction ($r = 0.016$; $p = 0.91$).

Pain correlated with other painful diseases but not with objective measures of median nerve dysfunction. Patients with associated painful conditions scored higher on the pain intensity scale ($F = 0.37$; $p = 0.007$). Electrophysiologic testing did not correlate with pain intensity: DSL ($r = 0.14$; $p = 0.19$) and DML ($r = 0.029$; $p = 0.43$) (Table 3).

There were significant correlations between pain intensity and all measures of illness behavior ($p < 0.05$) (Table 3). A multiple linear regression model including PCS, PHQ, PASS, and PSEQ as explanatory variables explained 39% of the variance in pain intensity ($F = 7.37$; $p < 0.001$; $R^2 = 0.39$). Only misinterpretation of nociception ($\beta = 0.4$; $p = 0.021$) and depressive symptoms ($\beta = 0.45$; $p = 0.007$) were significantly associated, accounting for 16% and 20.3% of the variances in pain intensity, respectively (Table 3).

Discussion

This study assessed the role of demographics, objective pathophysiology (eg, nerve conduction velocity), and illness behavior (depression and coping strategies) in reports of pain in two cohorts of patients with carpal tunnel syndrome verified by electrophysiologic testing. We found illness behavior is a better predictor of pain intensity than demographic factors and objective measures of pathophysiology (distal sensory and motor latency). Specifically, misinterpretation and overinterpretation of pain (pain catastrophizing) and depression are the best predictors of pain intensity. This finding is consistent with those of previous studies that conclude that psychosocial factors are as important or more important than pathophysiologic factors in reports of pain intensity in several conditions, including back pain, neck pain, joint pain, muscle soreness, and headaches [3, 14, 18]. Furthermore, our study was consistent in accounting for only 39% of the variation in pain intensity, emphasizing that pain is complex and incompletely understood.

The shortcomings of this analysis include (1) the use of a convenience sample of patients derived from another study; (2) retrospective analysis of prospective data; and (3) the fact that many potentially important factors such as atrophy, static numbness, and duration of symptoms are not accounted for.

Given that our data were collected prospectively in a consecutive series of patients electing operative treatment, these data are likely reproducible; however, given the shortcomings, our study is best considered pilot work meant to stimulate prospective research designed to better study the determinants of pain in carpal tunnel syndrome.

Our study shows the findings are consistent with those of previous studies in showing illness behavior is a better predictor of disabling musculoskeletal pain than demographic factors and objective measures of disease activity [3, 8, 14, 18], such as NCSs, and that pain is not a reliable measure of carpal tunnel severity [5]. It is increasingly clear that the wide variation in pain intensity and disability that healthcare providers observe for a given disease or impairment can be explained by mood, cognitions, emotions, and coping strategies. The intuitive concept that greater symptoms indicate greater pathophysiology is largely inaccurate, particularly when it comes to pain.

The myth that carpal tunnel syndrome is wrist pain with typing has a strong influence on illness behavior, health care, and society. This myth persists despite extensive evidence showing pain is not characteristic of carpal tunnel syndrome. As one example, a previous study concluded that the six most relevant clinical findings for diagnosis of carpal tunnel syndrome are thenar atrophy, a positive Phalen test, loss of two-point discrimination, present Tinel sign, nocturnal numbness, and numbness in the median nerve distribution [6]. These findings were used to create a diagnostic tool that later was validated and found to achieve equal probability of diagnosis when compared with electrodiagnosis [5].

Patients with substantial pain in association with carpal tunnel syndrome merit evaluation for other potential sources of pain such as arthritis, depression/psychologic

distress, and ineffective coping strategies. Current best evidence suggests numbness is the predominant symptom in idiopathic objectively verifiable median nerve dysfunction at the carpal tunnel (a description that is in many ways preferable to the misused, more vague term carpal tunnel syndrome) and that surgery to address intermittent (but not static) numbness is very effective. Better evidence may show the relief of pain observed in patients with activity-related pain diagnosed as carpal tunnel syndrome despite normal or mild electrophysiologic findings is merely the placebo effect, regression to the mean, or the natural course of the illness. In any case, patients with substantial pain in the context of carpal tunnel syndrome, or any disease for that matter, merit comprehensive care not hindered by a false mind-body dichotomy or faulty assumptions regarding the correlation between symptoms and disability and pathophysiology.

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