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ORIGINAL ARTICLE

What does pain sensitivity really predict in rheumatoid arthritis patients?

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SUMMARY

Objectives: The present study aimed to compare the pressure–pain threshold (PPT) values in patients with rheumatoid arthritis (RA) and age-gender matched controls with chronic nonspecific low back pain and to determine whether PPT values could be beneficial as a disease activity predictor after secondary fibromyalgia had been ruled out.

Methods: This study contained a cross-sectional observational study of participants with RA and chronic nonspecific low back pain controls without fibromyalgia. Visual analog scale (VAS), fatigue severity scale (FSS), pain catastrophizing scale (PCS), health assessment questionnaire (HAQ), hospital anxiety and depression scale (HADS), and disease activity score (DAS28) were administered. Pressure–pain threshold (PPT) values were measured with a baseline dolorimeter at the thumbnail bed, the dorsal aspect of the wrist, and the trapezius muscle on the dominant side.

Results: There were no differences in PPT scores at all points between RA and control groups. Female participants with RA had statistically lower PPT scores (high pain sensitization) at the wrist ($p<0.001$) and trapezius ($p<0.001$), but not at the nail bed ($p=0.084$). Multiple regression analysis identified only HADS-Depression as a determinant of the PPTs at all points.

Conclusion: The present study suggests that lower PPT is associated with depressive symptoms in participants with RA, and pressure algometry should be considered as an additional evaluation to detect pain/depression overlap.

Keywords: Catastrophization; central nervous system sensitization; depression; fibromyalgia; pain; pressure pain threshold; rheumatoid arthritis.

Introduction

Rheumatoid arthritis (RA) is a chronic, autoimmune, inflammatory disease of the synovial tissue of the joints, causing pain and functional disability. Pain in RA is the most important symptom that disrupts a patient's quality of life.[1] Although inflammation is often assumed to be the main determinant of pain intensity in patients with RA, more than one-tenth of patients with normal inflammatory markers still reported clinically meaningful pain degrees. The fact that pain remains despite the lack of evidence of inflammation indicates that factors, such as peripheral and central sensitization, play a substantial role in the pathogenesis of pain in RA.[2]

Pain in RA is usually evaluated using self-reported questionnaires by patients. However, inconsistencies between patients and their doctors were reported in assessing pain with these questionnaires. It was stated that devices, such as pressure algometry, which directly measure pain sensitivity, may be more objective. Although pressure algometry is not a completely objective test, it is a hybrid test that falls somewhere between subjective tests reported by the patient and objective techniques. Pain thresholds measured by a pressure algometer can be measured from different joints and non-articular areas to comprehensively assess pain sensitivity. While high pressure–pain threshold (PPT) shows low pain sensitivity, low thresholds indicate high sensitivity.[3,4]

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In a recently published systematic review including 1280 patients with different chronic pain diagnoses and 1463 healthy controls, it was stated that individuals with chronic pain had lower PPT values compared to healthy controls and that future studies were needed to clarify factors affecting the pain threshold.[5] Also, in the case of RA, there are a small number of studies regarding PPT and related parameters, and these have quite contradictory results. In some studies, PPT was found to be associated with subjective parameters, such as the number of sensitive joints, visual analog scale (VAS), and fatigue,[6] while Lee et al.[7] also found that it was associated with an objective inflammatory indicator, C-reactive protein (CRP).

When it comes to pain sensitivity and RA, fibromyalgia is an important confounder. FM is a phenotypic spectrum related to changes in central pain processing. Therefore, it significantly affects the PPT and is frequent in patients with RA (in the range of 12% to 48%, compared to 2–8% in the general population). Generally, participants with RA and concomitant FM have higher pain, higher disease activity scores, but also less joint destruction.[8]

We wanted to better understand the elements that may be associated with PPT and whether it will contribute to us in the follow-up of RA. Therefore, we excluded participants who satisfied FM criteria. First, we compared the pain thresholds of patients with RA with the control group with non-specific low back pain matched by age and sex. We also examined many objective and subjective parameters which may be associated with a low pain threshold. Given the strong association of fibromyalgia with low PPT, after excluding patients with concomitant fibromyalgia, we hypothesized that PPT scores would be similar across genders and associated with more objective parameters.

Material and Methods

The present study was planned as a controlled, cross-sectional study in accordance with the Helsinki Declaration and was approved by our local Ethical Committee (E17-1196). Patients over 18 years with RA diagnosed according to ACR/EULAR 2010 classification criteria[9] and age- and sex-matched patients with non-specific low back pain[10] were recruited from the Ankara Numune Training and Research Hospital Physical Medicine and Rehabilitation outpatient clinic between February 2017 and March 2018. Participants were informed about the study, and written approval was obtained from each participant.

Participants with RA and controls diagnosed with fibromyalgia according to the revised 2016 fibromyalgia diagnostic criteria,[11] with low 25(OH)-vitamin D levels (below 30 µg/l) and thyroid dysfunction, and a history of using opiate, antidepressant, gabapentin, and pregabalin in the past 3 months, and patients who had active arthritis in the wrist during the evaluation period were excluded.

Age, gender, body mass index (BMI), educational status, marital status, employment status, smoking, and alcohol use; duration of illness (years); duration of morning stiffness (minutes); biological treatment and/or synthetic disease-modifying antirheumatic drugs (DMARD) were recorded. BMI was calculated as kg/m^2.

Pain intensity was measured with the VAS. In this scale, no pain was determined as "0," and the most severe pain was determined as "10" in the 0–10 mm chart.[12] The "Pain Catastrophizing Scale (PCS)," which was validated and reliable in the Turkish population, was used to assess the cognitive character of the pain and the relationship between the severity of pain, cognitive and emotional factors, and disability.[13,14] This scale is a 13-part self-assessment scale used to determine patients' ineffective coping strategies about pain experience and their thoughts and feelings about pain.

Fatigue severity was evaluated using the Turkish version of the "Fatigue Severity Scale (FSS)."[15,16] The anxiety and depression symptoms of the patients were evaluated using the Turkish version of the "Hospital Anxiety and Depression Scale (HADS)."[17,18]

The functional capacities of the patients were analyzed using the "Health Assessment Questionnaire (HAQ)," which was validated and reliable in the Turkish population.[19,20] It consists of eight areas: dressing/grooming, arising, eating, walking, hygiene, reach, grip, and activity.

Table 1. Demographic characteristics of the study groups

	RA (n=100)	Control (n=80)	p
Age (years)			0.180
Mean±SD	55.93±10.81	54.50±9.47	
Median (min–max)	58.50 (24–72)	55.00 (31–70)	
Gender, women, n (%)	49 (49)	44 (55)	0.423
BMI (kg/m^2)			0.491
Mean±SD	27.66±4.89	28.02±5.04	
Median (min–max)	27.00 (19–48.40)	27.10 (18.30–47.80)	
Marital status - married, n (%)	84 (84)	66 (82.5)	0.788
Educational status - literate, n (%)	94 (94)	73 (91.3)	0.479
Employment status - working, n (%)	24 (24)	26 (32.5)	0.206
Smoking use, n (%)	24 (24)	21 (26.5)	0.729
Alcohol use, n (%)	1 (1)	4 (5)	0.105

RA: Rheumatoid arthritis; BMI: Body mass index; SD: Standard deviation.

The "Disease Activity Score (DAS28)" was used to evaluate the disease activity. It includes an assessment of the number of tender and swollen joints, a VAS for general health, and C-reactive protein (CRP) concentration or erythrocyte sedimentation rate (ESR).[21]

PPT was measured with a baseline dolorimeter. A baseline dolorimeter is a device with a 1 cm^2 pressure surface and a handgrip, showing the values obtained in kg/cm^2. PPT was measured with the patient in the sitting position. Three measurements were noted at the thumbnail bed, the dorsal aspect of the wrist, and the trapezius muscle on the dominant side, and the average values were recorded.[22]

Statistical Analysis

Statistical Package for Social Sciences (SPSS) Windows 18 package program was used for statistical analysis. Post hoc power analysis was performed using the G power 3.0.10 program, and the power of the study (1-β) was calculated as 0.95 with the number of patients and controls included. Data normality was tested using the Shapiro–Wilk test. Parametric and nonparametric tests were applied according to the normality of data distribution.

General descriptive statistics are summarized as mean±standard deviation and median (minimum–maximum) for continuous variables. Categorical data were defined by numbers and percentages. Independent sample T-test (for parametric variables), Mann–Whitney U test (for nonparametric variables), chi-square test (for nominal variables), and Fisher exact tests (nominal variables less than five in each group) were used for the comparison of groups.

Spearman correlation analysis was used to examine the relationship between PPT values and other variables. Variables correlated with PPT values were analyzed using multivariate linear regression analysis. Regression analysis met the assumptions (linear relationship between independent variables, covariance, independence of observations, and appropriate sample size). Statistical significance was determined as $p<0.05$.

Results

One hundred patients with RA and 80 controls were included; 49% of patients with RA were women, 84% were married, 94% were literate, and 24% were working. Of the control group, 55% were women, 82.5% were married, 91.3% were literate, and 32.5% were working. There was no significant difference in demographic characteristics between the patients with RA and the control group (Table 1).

While 86% of the patients in the RA group were using synthetic DMARD (sDMARD), 14% were receiving biological therapy and/or sDMARD treatment. The mean duration of the disease was 9.96±7.40

Table 2. Clinical and laboratory findings of the groups

	RA	Control	p
VAS			<0.001*
Mean±SD	3.03±3.04	4.88±2.15	
Median (min–max)	3.00 (3.00–10.00)	5.00 (1.00–9.00)	
PCS			0.060
Mean±SD	12.13±12.98	13.23±9.17	
Median (min–max)	6.00 (0.00–48.00)	12.00 (0.00–41.00)	
FSS			0.558
Mean±SD	3.47±1.94	3.65±2.05	
Median (min–max)	2.77 (1.00–7.00)	3.22 (1.00–7.00)	
HADS- Depression			0.389
Mean±SD	6.48±4.65	7.17±4.92	
Median (min–max)	6.00 (0.00–17.00)	6.50 (0.00–21.00)	
HADS- Anxiety			0.840
Mean±SD	6.99±4.24	7.16±4.33	
Median (min–max)	6.5 (0.00–18.00)	7.00 (0.00–17.00)	
HADS-Total			0.568
Mean±SD	13.47±8.07	14.33±8.46	
Median (min–max)	12.50 (0.00–30.00)	13.00 (1.00–37.00)	
HAQ			0.617
Mean±SD	0.61±0.64	0.59±0.49	
Median (min–max)	0.37 (0.00–2.62)	0.56 (0.00–1.87)	

RA: Rheumatoid arthritis; SD: Standard deviation; VAS: Visual Analog Scale; PCS: Pain Catasrophizing Scale; FSS: Fatigue Severity Scale; HADS: Hospital Anxiety and Depression Scale; HAQ: Health Assesment Questionnaire. *: Statistically significant value (p<0.05).

Table 3. Pain pressure threshold values of the groups

	RA	Control	p
Nail bed			0.346
Mean±SD	4.64±1.45	4.76±1.76	
Median (min–max)	4.75 (3.00–9.00)	4.5 (3.00–9.50)	
Wrist			0.447
Mean±SD	5.34±1.61	5.46±1.53	
Median (min–max)	5.00 (3.00–11.5)	5.37 (3.00–9.00)	
Trapezius muscle			0.280
Mean±SD	4.64±1.45	4.82±1.43	
Median (min–max)	4.25 (3.00–10.00)	4.87 (3.00–9.00)	

RA: Rheumatoid arthritis; SD: Standard deviation.

years. The mean morning stiffness duration was 29.05±1.70 minutes. The median count of tender and swollen joints was respectively 1 (0–24) and 1 (0–9). The mean DAS28-ESH and DAS28-CRP were 3.19±1.48 and 3.04±1.40, respectively. Forty-one patients with RA were in remission, 16 had low, 28 had moderate, and 15 had high disease activity. VAS values were statistically significantly higher in the controls than the participants with RA, but there was no significant difference in other parameters (Table 2).

Table 4. Pain pressure threshold values of the patients with RA

	Women (n=49)	Men (n=51)	p
Nail bed			
Mean	4.73±1.34	5.15±1.38	
Median (min–max)	4.50 (3.00–9.00)	4.75 (3.00–10.00)	0.084
Wrist			
Mean	4.80±1.54	5.85±1.52	
Median (min–max)	4.25 (3.00–11.00)	5.75 (3.00–11.50)	<0.001*
Trapezius muscle			
Mean	4.11±1.26	5.14±1.45	<0.001*
Median (min–max)	4.00 (3.00–8.50)	4.75 (3.00–10.00)	

RA: Rheumatoid arthritis; SD: Standard deviation; * Statistically significant value (p<0.05).

Table 5. Pain pressure threshold values of the patients with RA

	Nail bed PPT		Wrist PPT		Trapezius muscle PPT	
	p	r	p	r	p	r
Age	0.551	0.060	0.705	0.038	0.806	0.025
BMI (kg/m^2)	0.174	-0.137	0.066	-0.185	0.046*	-0.200
Disease duration (year)	0.247	-0.117	0.144	-0.147	0.364	0.092
Morning stiffness (minute)	0.415	-0.082	0.592	-0.054	0.542	-0.062
The number of tender joints	0.046*	-0.200	0.099	-0.166	0.361	-0.092
The number of swollen joints	0.109	-0.161	0.227	-0.122	0.522	-0.065
CRP	0.681	-0.042	0.144	0.147	0.076	0.178
ESH	0.952	-0.006	0.330	0.098	0.453	0.076
VAS	0.056	-0.192	0.069	-0.182	0.086	-0.173
PCS	0.013*	-0.248	0.013*	-0.249	0.034*	-0.212
FSS	0.107	-0.162	0.145	-0.147	0.422	-0.081
HADS- anxiety	0.157	-0.143	0.118	-0.157	0.285	-0.108
HADS- depression	0.010*	-0.256	0.005*	-0.282	0.003*	-0.293
HADS- total	0.026*	-0.223	0.014*	-0.245	0.024*	-0.225
HAQ	0.000*	-0.264	0.000*	-0.287	0.000*	-0.315
DAS28- ESH	0.013*	-0.248	0.091	-0.170	0.211	-0.126
DAS28- CRP	0.009*	-0.261	0.066	-0.185	0.220	-0.124

PPT: Pressure–pain threshold; RA: Rheumatoid arthritis; SD: Standard deviation; BMI: Body mass index; CRP: C-reactive protein; ESH: Erythrocyte sedimentation rate; VAS: Visual Analog Scale; PCS: Pain Catasrophizing Scale; FSS: Fatigue Severity Scale; HADS: Hospital Anxiety and Depression Scale; HAQ: Health Assesment Questionnaire; DAS28: Disease Activity Score; *: Statistically significant value (p<0.05).

No difference was found in terms of PPT values when compared with the control group (Table 3). When the PPT values of men and women in the RA group were compared, PPT values in all regions were lower in women, which was statistically significant in the wrist (p<0.001) and trapezius regions (p<0.001) (Table 4).

The relationship among PPT values of the nail bed, wrist, and trapezius muscle in the patients with RA and age, BMI, morning stiffness, disease duration, number of tender joints, number of swollen joints, CRP, ESR, VAS, PCS, FSS, HADS-Anxiety, and Depression were evaluated. PPT values in the nail bed were significantly associated with the number of sensitive joints, PCS,

Table 6. Multiple linear regression analysis of determinants associated with pressure pain threshold

	B	SE	p	95% CI	
Thumb nail bed					
Constant	5.669	0.367	0.000	4.940	6.397
HAQ	-0.106	0.265	0.690	-0.631	0.420
HADS-depression	-0.060	0.030	0.049*	-0.119	0.000
DAS28-ESH	-0.170	0.093	0.072	-0.355	0.016
Wrist					
Constant	5.952	0.203	0.000	5.517	6.304
PCS	-0.013	0.012	0.265	-0.037	0.010
HADS- depression	-0.093	0.033	0.006*	-0.159	-0.027
Trapezius muscle					
Constant	6.343	0.796	0.000	4.764	7.923
BMI	-0.047	0.029	0.105	-0.104	0.010
HADS-depression	-0.175	0.073	0.019*	-0.320	-0.029
HADS-Total	0.076	0.043	0.082	-0.010	0.162
HAQ	-0.485	0.248	0.054	-0.978	0.008

B: Regression coefficient; SE: Standard error; CI: Confidence interval; HAQ: Health Assesment Questionnaire; HADS: Hospital Anxiety and Depression Scale; DAS28: Disease Activity score; ESH: Erythrocyte sedimentation rate; PCS: Pain Catasrophizing Scale; BMI: Body mass index; *: Statistically significant value ($p<0.05$).

HADS-Depression, HADS-Total, HAQ, DAS28-ESH, and DAS28-CRP. Pain threshold values in the wrist were significantly associated with PCS, HADS-Depression, HADS-Total, and HAQ. Pain thresholds in the trapezius muscle were significantly associated with BMI, PCS, HADS-Depression, HADS-Total, and HAQ (Table 5).

When the parameters correlated with the pain threshold were examined by multiple linear regression analysis, depression was found to be the only parameter associated with pain thresholds at all points (Table 6).

Discussion

This study was mainly aimed at evaluating the possible association between PPT scores and many objective and subjective parameters in RA patients without FM. The major finding of this research was that the PPT in participants with RA correlates negatively with depression; higher depression scores yield lower PPTs or, in other words, increased pain sensitivity.

In this study, although VAS values were higher in the control group than in participants with RA, there was no difference in PPT scores. It has been determined in previous studies that participants with RA have lower PPTs in joint and extra-articular areas than the healthy population, patients with ankylosing spondylitis (AS), and osteoarthritis (OA).[23,24] Also, it has been demonstrated that participants with FM have the lowest PPTs, compared to participants with RA, AS, myofascial pain, osteoporosis, generalized OA, and healthy control participants.[24] Therefore, the reason for the similar PPTs among groups may be that patients with fibromyalgia, which is an important cause of low PPT, were not included in the present study.

The present study showed that female participants with RA have lower pain PPTs except at the thumbnail bed. The reason for no difference at only the nail bed between genders might be due to this region's extra protective function of the nail. Similar to our study, it has been shown in other studies that women have higher pain sensitivity.[3,25] Most of the studies on this matter have revealed that women and men differ in their response to pain and that pain increases in women.

Although the precise causes underlying these gender differences are unknown, many biological and psychosocial factors are thought to contribute. Emerging evidence indicates that endogenous opioid functioning and genotype play a causal role in these discrepancies and that sex hormones affect PPT.[26] Testosterone was

demonstrated to be more antinociceptive, although the influences of estrogen and progesterone on pain sensitivity are relatively complex.

Besides these biological mechanisms, various psychosocial mechanisms also play an important role. For example, it has been found that men and women have different strategies for coping with pain. While men tend to utilize behavioral distraction and problem-focused strategies to cope with pain, women tend to utilize a range of coping techniques, such as emotion-focused techniques, social support, and cognitive reinterpretation.[27] Consequently, despite excluding patients with FM, the PPT scores would still be lower in women due to the reasons mentioned above.

The literature showed that the PPT is associated with various disease-related markers, especially subjective parameters, in participants with RA.[6] The main goal of this study was to determine whether we could find more associations between PPT and objective data after excluding patients with FM. However, after multiple linear regression analysis, we showed that PPTs in all regions were not associated with objective or subjective parameters affecting disease activity but were only associated with depression.

Bagnato et al.[3] investigated the relationship between depression and PPT in individuals with psoriatic arthritis, RA, and AS. Similar to our study, they found that depression significantly lowered the pain threshold in all patient groups, while they did not find a correlation with VAS. The relationship between a high depression score and a low pain threshold has also been demonstrated in other groups of diseases.[28] However, it is unclear whether depression or pain sensitivity develops first.

It was suggested that neurotransmitter impairment in depression affects the perception of pain in two ways. The first is that it alters sensory pain thresholds by reducing spinal and subcortical processing of all sensory inputs. The second is that it increases pain by disrupting endogenous pain inhibition. Based on these mechanisms, it is not surprising that pain and depression are related.[29]

Depression comprises the most common comorbid condition associated with RA. Prevalence rates were shown to range from 14% to 48%.[30] The diagnosis and treatment of depression are often delayed because patients with depression seek medical attention for pain rather than emotional symptoms. It is estimated that if patients with pain were screened for depression, more than 60% would be diagnosed with depression.[31]

If the perception of pain is a manifestation of depression, the pain threshold can provide us with significant benefits for diagnosis and treatment. It was found that the diagnosis of major depression was delayed by up to 50%, as physicians focused only on pain symptoms.[30,31] Additionally, it should not be forgotten that untreated depression prevents the patient's adherence to the treatment process and perpetuates the vicious cycle of pain.

Moreover, there is no approved screening tool for comorbid pain and depression. Measuring the pressure–pain threshold may serve as a guide to determine whether depression and pain coexist in patients with suspected mood disorders who present only with pain complaints.

The limitation of our study is that its design is cross-sectional; this prevents the evaluation of causality. Also, although we evaluated many parameters in our study, we did not assess sleep problems. Therefore, additional studies with a higher number of participants are required to investigate the associations between pain sensitivity, disease activity, sleep, and depression in the future.

Conclusion

In conclusion, we found that the PPTs in patients with RA were similar to those in patients with non-inflammatory low back pain. PPTs in women were lower, and we did not find a significant relationship between PPT and any parameter other than depression, whether objective or subjective, including patient-reported pain.

For this reason, although we do not find that pressure algometry has a relationship that will guide the clinician in parameters related to disease activity in patients with RA, we believe that it may be helpful in identifying patients with depression who present with pain rather than mood symptoms. As a result, the utilization of pressure algometry in the assessment of chronic pain in patients with RA can be used as an additional evaluation to detect depression/pain overlapping.

Ethics Committee Approval: The Ankara Numune Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 25.01.2017, number: 1196/2017).

Authorship Contributions: Concept – HB; Design – HB, AÇU; Supervision – AÇU, FGY; Resource – AAG; Materials – FGY; Data collection and/or processing – AAG; Analysis and/or interpretation – FGY; Literature review – AAY; Writing – AÇU; Critical review – HB.

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