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Pain catastrophizing and its domains significantly impact rheumatoid arthritis disease activity

Damiano Currado[1,2], Lyubomyra Kun[2], Francesca Saracino[2], Leonardo Frascà[2], Francesca Trunfio[2], Onorina Berardicurti[1,2,9]✉, Erika Corberi[2], Annalisa Marino[1], Andrea Pilato[2], Ludovica Lamberti[2], Marta Vadacca[1], Amelia Rigon[1], Luisa Arcarese[1], Piero Ruscitti[3], Ilenia Pantano[4], Marta Vomero[2], Viktoriya Pavlych[3], Claudio Di Vico[4], Federica Camarda[5], Francesca Misceo[6], Francesco De Vincenzo[7], Addolorata Corrado[8], Francesco Paolo Cantatore[8], Federico Perosa[6], Giuliana Guggino[5], Paola Cipriani[3], Francesco Ciccia[4], Roberto Giacomelli[1,2] & Luca Navarini[1,2]

This study aims to evaluate the impact of pain catastrophizing (PC) on disease activity in patients with rheumatoid arthritis (RA) and to explore, in the same participants, if this association is related or not with anxiety and depression, which have been related to catastrophization in patients with chronic pain. A multi-center, observational study has been conducted on 158 RA patients from six Rheumatology Clinics. Participants were assessed using the Clinical Disease Activity Index (CDAI) and Simple Disease Activity Index (SDAI) and Disease Activity Score on 28 joints- C reactive protein (DAS28-CRP). Pain Catastrophizing, with its domains of Helplessness, Rumination and Magnification, was analyzed through Pain Catastrophizing Scale (PCS). Statistical analyses included univariable and multivariable regressions, to identify associations between disease activity and PC. Results revealed that higher PCS scores were significantly associated with increased CDAI, SDAI and DAS28-CRP values, indicating higher disease activity. Specifically, the domains of Rumination and Helplessness showed a strong correlation with disease activity, while Magnification did not. These associations persisted independently of anxiety and depression mood, as shown by multivariable regression analysis. Pain catastrophizing, particularly the domains of Rumination and Helplessness, significantly influences disease activity in RA patients, independent of mood disorders. These findings underscore the importance of addressing maladaptive cognitive perceptions of pain in the management of RA, to improve patient outcomes and facilitate disease remission.

Keywords Pain catastrophizing, Rheumatoid arthritis, Disease activity

Rheumatoid Arthritis (RA) is a debilitating chronic inflammatory disease characterized by symmetrical inflammation and erosion of small and large joints, leading to joint damage, severe pain and loss of function[1]. Extra-articular manifestations and comorbidities are also present in RA, leading to increased morbidity and

[1]Clinical and Research Section of Rheumatology and Clinical Immunology, Fondazione Policlinico Universitario Campus Bio-Medico, Rome, Italy. [2]Rheumatology and Clinical Immunology, Department of Medicine, School of Medicine, University of Rome "Campus Bio-Medico", Rome, Italy. [3]Rheumatology Unit, Department of Biotechnological and Applied Clinical Sciences, University of L'Aquila, L'Aquila, Italy. [4]Rheumatology Unit, Department of Precision Medicine, University of Campania L. Vanvitelli, Caserta, Italy. [5]Rheumatology Section, Department of Health Promotion, Mother and Child Care, Internal Medicine and Medical Specialties, University Hospital "P. Giaccone", Palermo, Italy. [6]Rheumatic and Systemic Autoimmune Diseases Unit, Department of Biomedical Science and Human Oncology (DIMO), University of Bari Medical School, Bari, Italy. [7]Department of Human Sciences, European University of Rome, Rome, Italy. [8]Rheumatology Clinic, Department of Medical and Surgical Sciences, University of Foggia, Rione Biccari, Foggia 71122, Italy. [9]Unit of Allergology, Clinical Immunology and Rheumatology, Department of Medicine, University Campus Bio-Medico di Roma, via Alvaro del Portillo 21, Rome 00128, Italy. ✉email: o.berardicurti@policlinicocampus.it

premature mortality[2–5]. In the last few years, increasing emphasis has been placed on the role of RA and its impact on the physical, psychological, and social domains, impairing health-related quality of life. Anxiety and depression have been shown to be associated with increased disease activity and poorer quality of life[6–8].

In this disease spectrum, chronic pain is a cornerstone symptom of musculoskeletal disorders and has significant predictive value regarding the course of the disease, the degree of disability, the frequency of healthcare utilization, and the socioeconomic and occupational consequences[9,10].

In response to chronic pain, patients have adopted a maladaptive coping strategy that involves a misperception of pain through qualitatively negative emotional and cognitive processes: pain catastrophizing (PC), an inclination to exaggerate in describing pain, to ruminate, or to feel helplessness about it[11]. The term catastrophizing was formally introduced by Albert Ellis and later adopted by Aaron Beck to refer to a maladaptive cognitive process originally described as a peculiar feature of patients with anxiety disorders or depressive disorders. It was based on the principle of an irrational negative anticipation of future events; when applied to present or potential painful experiences, this principle translates into a set of cognitive and emotional patterns that induce the individual to experience an amplified sense of threat and an inability to devise coping strategies[6–13].

The most commonly used psychometric tools in clinical practice for defining catastrophizing are the Coping Strategy Questionnaire (CSQ) and the Pain Catastrophizing Scale (PCS). The CSQ includes a subscale consisting of six items related to dimensions of helplessness and pessimism associated with pain. The domains investigated by the PCS are three: Rumination, Magnification, and Helplessness. Rumination refers to the frequent recurrence of thoughts related to experienced pain and the suffering it generates; attention is also focused on the desire for the painful condition to end. Magnification, on the other hand, involves an amplified sense of danger that the pain will worsen or that other adverse events will occur. Helplessness, finally, is a domain that refers to an irrational belief that there is no solution or hope for improvement in the painful experience[6–15].

In several studies, Pain Catastrophizing has been associated to an increased burden of the disease, with higher levels of pain, psychological disability, functional impairment, and poorer quality of life[16–18].

Furthermore, the proportion of catastrophizing patients remains substantially unchanged when comparing groups of patients with RA, axial spondyloarthritis (axSpA) and Psoriatic Arthritis (PsA), highlighting the importance and prevalence of this maladaptive cognitive perception of pain in different rheumatic diseases[7,8,17–22]. Moreover, other studies conducted in patients with PsA, as well as in those with chronic pain-related rheumatologic conditions such as osteoarthritis, fibromyalgia, and axSpA have confirmed the significant impact of pain catastrophizing on disease burden, psychological distress, and quality of life[7,23,24].

Despite its association with subjective disease perception (pain characteristic), PC does not align with inflammation biomarkers, such as swollen joint count, C-Reactive Protein serum level and ultrasound arthritis measures[25]. Furthermore, in RA patients PC impacts the drug retention rate; high levels of pain catastrophizing reduced the likelihood of achieving composite score remission[25–27].

In this context, it is possible that patients' perception of disease and psychological status could reduce the likelihood for achieving remission in RA and PsA[7,21,22].

However, nowadays the intricate pathways and mechanisms underlying this reciprocal influence between pain and biological, psychological and social factors remain not entirely elucidated, although different hypothesis have been postulated.

This study aims to explore the relationship between pain catastrophizing and disease activity in RA patients, by analyzing its specific domains, as assessed by the PCS. Furthermore, in the same participants, we analysed if this association is related or not with those psychometric domains which have been related to the catastrophization in patients with chronic pain. By clarifying whether pain catastrophizing correlates with disease severity independently of inflammatory markers, the study seeks to provide clinically meaningful insights for patient stratification and management.

Methods

A multi-center, cross-sectional study has been conducted on RA participants enrolled in six Italian Rheumatology Clinics. Consecutive participants have been enrolled from January 2021 to July 2021 during routine clinical visits; at baseline, RA participants fulfilled the American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) 2010 criteria[28]. The study received approval from the Ethics Committee of the University Campus Bio-Medico of Rome (approval no. 78.20 OSS) and was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. Inclusion criteria were: both genders, age > 18 years, and the fulfilment of ACR/EULAR 2010 criteria. The exclusion criteria were: history of any psychiatric disorder according to DSM-V prior the recruitment, history of any malignancy, pregnancy, age > 75 or inability to express informed consent to participate in the study.

At enrollment, the following RA disease activity scores were collected: CDAI, SDAI and DAS28-CRP. The use of Glucocorticoids (GCs), synthetic-, biological-, and target synthetic DMARDs were recorded. The HAQ was evaluated as measure of function. Validated questionnaires were administered to assess psychological, and social status, all of which have been previously employed and discussed in studies involving patients with inflammatory arthritis[7,27]: HADS[29,30], THS and its domain Agency and Pathway[31], Acceptance and Action Questionnaire (AAQ)[32] and Compassionate Engagement and Action Scales (CEAS)[33]. Patients fulfilling the 2016 American College of Rheumatology revised diagnostic criteria for fibromyalgia were further identified in our cohort[34]. Lung involvement was assessed by pulmonary function tests and high-resolution computed tomography of the thorax. Findings consistent with interstitial lung disease, such as usual interstitial pneumonia or nonspecific interstitial pneumonia patterns and requiring specific treatment for this condition were considered RA-ILD.

Pain Catastrophizing, with its domains of Helplessness, Rumination and Magnification, was analyzed through PCS. Continuous variables are described as median (interquartile range, 25–75th percentile), whilst categorical variables are described as percentages (%). The Shapiro-Wilk test was used to evaluate the normality of data.

Chi2 was used for the analysis of contingency tables, while Mann-Whitney test and Kruskal-Wallis with Holm's pairwise comparison corrections were used to compare ranks. To evaluate variables associated with SDAI, we performed univariable and multivariable regression analyses. Variables with $p < 0.1$ in univariate analysis were considered as candidates, but the final multivariable models also included clinically relevant variables (e.g., age, sex, and disease duration) regardless of their univariate significance. We tested multicollinearity using variance inflation factor (VIF) analysis.

Missing data were excluded from the relevant analyses using listwise deletion. The proportion of excluded cases was minimal (< 2%) and did not affect the overall results or conclusions. The whole statistical analysis was performed using Stata v.14 and p-values < 0.05 have been considered as significant.

Results

We enrolled 158 RA patients (median age of 61 (range 51–69) years, 70.9% women), with a median disease duration of 96 months (range 57.5–156); 63.6% of participants displayed the positivity for rheumatoid factor and 63% for anti-citrullinated protein antibodies. During the follow-up, 65.8% of participants were treated with conventional synthetic disease-modifying antirheumatic drugs (DMARDs), and 71.5% with biologic or targeted DMARDs. The main demographic, anthropometric, and clinical characteristics of the study population are reported in Table 1.

In this subset, we observed that the median Clinical Disease Activity Index (CDAI) value was 8.1 (range 2–14.5) and Simple Disease Activity Index (SDAI) 8.1 (range 2.3–14.7). Concomitant fibromyalgia was present in 18% and RA associated interstitial lung disease (RA-ILD) in 6.8% of participants.Furthermore, we observed a PCS median value of 17 (range 8–26) and about its domains, Helplessness was 6 (range 2–11), Rumination was 7 (range 3–11), Magnification was 2 (range 1–4).

By using the cut-off score of PCS = 30 to distinguish clinically significant from non-significant pain catastrophizing, we explored its relationship with age, disease activity, and psychological variables. Histograms (Fig. 1) were generated specifically to visualize the associations between PCS and age, CDAI, SDAI, DAS28, as well as anxiety and depression. Patients with PCS > 30 showed significantly higher values in tender joints ($p = 0.0007$), swollen joints ($p = 0.03$), VAS pain ($p = 0.008$), patients' global assessment ($p = 0.0002$), physician global assessment ($p = 0.01$), CDAI ($p < 0.0001$), SDAI ($p = 0.0001$), RA-ILD ($p = 0.013$), HAQ ($p < 0.0001$), HADS anxiety and depression ($p < 0.0001$), AAQ ($p < 0.0001$), and THS agency and pathway scores ($p = 0.006$ and $p = 0.004$, respectively).

To further evaluate the relationship between disease activity and PC, univariable and multivariable linear regression analyses have been performed, and the results are reported in Tables 2, 3 and 4.

Of note, the adjusted linear regression model showed a positive association between SDAI and PCS (b = 0.3, 95% CI 0.05 to 5, $p < 0.001$).

In the multivariable analysis, PCS was independently associated with SDAI (coefficient 0.25, 95% CI: 0.13 to 0.38, $p < 0.001$), whereas symptoms of anxiety and depression were not. Furthermore, in an additional multivariable linear regression model using DAS28 as the dependent variable (as shown in Table 4), PCS remained independently associated with disease activity (coefficient 0.04, 95% CI: 0.02 to 0.05, $p < 0.001$).

Furthermore, multivariable linear regressions showed significant association between the Helplessness component of PCS, (coeff 0.59, 95%CI 0.3 to 0.9, $p < 0.0001$) and between Rumination (coeff 0.49, 95%CI 0.2 to 0.8, $p = 0.001$) and SDAI. On the other hand, there was no significant association between magnification and SDAI disease activity ($p = 0.4$).

Discussion

In our study, we showed that PC, a self-maladaptive cognitive perception of pain, strongly influencing the patient's perception of the disease, was able to negatively impact disease activity. We demonstrate a strong correlation between PCS (considering both total PCS and the domains rumination and helplessness) and higher levels of SDAI, confirming that maladaptive cognitive PC significantly interferes with the achievement of low disease activity and remission.

We observed that RA patients with PCS scores > 30 exhibited a significantly higher number of tender and swollen joints, increased VAS pain, and higher patient and physician global assessments. These patients also showed greater disease activity, as measured by CDAI, SDAI and DAS28 scores, as well as more frequent LI. Moreover, they reported worse physical functioning, elevated levels of anxiety and depression, lower psychological flexibility, and reduced levels of hope.

These results are in line with our previous data just published, in which we described that, in axSpA and PsA patients, PC specifically correlated with the number of tender joints, patient-reported pain, and patient global assessment, thus confirming its predominant impact on the subjective dimension of the disease activity scores, independently of inflammation.

Of note, the correlation between PCS and the disease activity of other rheumatic diseases was already described in scientific literature[13,23–25,35–37], but for the first time we assessed the role of this maladaptive cognitive process in relation with anxious and depressive mood.

In our multivariable analysis, PCS is independently associated with SDAI, whilst anxious and depressive symptoms are not, indicating that the negative influence of maladaptive coping strategy on disease activity is not related to anxiety and depression. Conversely, anxious and depressive symptoms do not exhibit independent associations with SDAI.

Furthermore, multivariable linear regressions showed a significant association between the Helplessness and Rumination component of PCS and SDAI disease activity. In contrast, no significant association was observed

Variables	RA population ($N=158$)	RA participants with PCS < 30 ($N=127$)	RA participants with PCS > 30 ($N=31$)	p value
Age	61 (51–69)	59 (47–69)	63 (55–71)	0.004
Sex, female n(%)	112 (70.9)	90 (70.8)	22 (70.9)	0.9
Disease duration (months)	96 (57.5–156)	96 (52–168)	96 (60–144)	0.8
BMI	26.3 (23.1–29.8)	26.4 (23.31–30.5)	25.195 (22.8–28.35)	0.2
Fibromyalgia, n (%)	28 (18)	20 (15.8)	8 (25.9)	0.2
Smoke no/yes/former n(%)	81 (51.3)/36 (22.8)/41 (25.9)	68 (53.5)/26 (20.5)/33 (26.0)	13 (41.9)/10 (32.3)/8 (25.8)	0.3
RA-ILD, n (%)	11 (6.8)	5 (4.2)	5 (17.2)	**0.01**
Rheumatoid factor, n (%)	100 (63.6)	78 (61.8)	22 (71.4)	0.3
ACPA, n (%)	99 (63)	78 (61.8)	21 (67.7)	0.5
csDMARDs, n (%)	104 (65.8)	81 (63.8)	23 (74.2)	0.3
bDMARDs/tsDMARDs n (%)	45 (28.5)/113 (71.5)	37 (29.1)/90 (70.8)	8 (25.8)/23 (74.2)	0.7
CCS, n (%)	71 (44.9)	53 (41.5)	18 (58.6)	0.09
TJ	1 (0–4)	1 (0–3)	4 (2–7)	**0.0007**
SJ	0 (0–1)	0 (0–1)	1 (0–2)	**0.03**
VAS pain (0–10 cm)	5 (2–7)	4 (2–7)	7 (3.5–8)	**0.009**
PtGA	4 (2–7)	3 (1.5–6.5)	7 (4–8)	**0.0002**
PhGA	1 (0–3)	1 (0–2)	2 (1–3)	**0.01**
VAS GH (0–10 cm)	5.75 (3–7)	5 (3–7)	6 (5–7)	0.1
CRP (mg/dL)	0.23 (0.1–0.5)	0.2 (0.1–0.5)	0.2 (0.1–0.5)	0.7
HAQ	1 (0.3–1.6)	0.9 (0.1–1.5)	1.7 (1–2.3.3)	**< 0.0001**
CDAI	8.1 (2–14.5.5)	6.3 (2–12)	15 (8.1–18.1)	**< 0.0001**
SDAI	8.1 (2.3–14.7)	6.6 (2.0–12.6.0.6)	14.2 (8.7–18.9)	**0.0001**
DAS28 CRP	2.6 (1.66–3.72)	2.3 (1.65–3.57)	3.6 (2.27–4.29)	**0.001**
PCS	17 (8–26)	13 (7–21)	38 (34–39)	**< 0.0001**
Helplessness	6 (2–11)	5 (1–9)	16 (14.5–18)	**< 0.0001**
Rumination	7 (3–11)	6 (3–10)	15 (14–17)	**< 0.0001**
Magnification	2 (1–4)	2 (0–4)	5 (4.5–6.5)	**< 0.0001**
HADS anxiety	6 (2–9)	5 (2–8)	10 (7–13)	**< 0.0001**
HADS depression	6 (3–8)	5 (2–7)	9 (6–12)	**< 0.0001**
AAQ	17 (10–26)	14.5 (9–22.5.5)	25.5 (20–28.5.5)	**< 0.0001**
Hope THS	24 (21.5–28)	25 (22–29)	23 (20–24)	**0.003**
THS agency	12 (10–13.5.5)	12 (10–14)	10 (10–12)	**0.006**
THS pathway	13 (11.5–15)	13 (12–15)	12 (10–13)	**0.004**
Self compassion	72.5 (58–83)	74 (59–83)	70 (55–84.5.5)	0.5
Compassion from others	69 (53–86)	69 (53–83.5.5)	77 (52–84)	0.9

Table 1. Demographic and clinical characteristics of RA participants. Rheumatoid Arthritis (RA); Body Mass Index (BMI); Rheumatoid arthritis-associated interstitial lung disease (RA-ILD); Anti-citrullinated protein antibodies (ACPA); conventional synthetic disease-modifying antirheumatic drugs (csDMARDs); Biological Disease-Modifying Anti-Rheumatic Drugs (bDMARDs); targeted synthetic disease-modifying antirheumatic drugs (tsDMARDs); corticosteroids (CCS); tender joints (TJ), swollen joints (SJ), VAS pain (Visual Analogue Scale of pain), patients' global assessment (PtGA), physician global assessment (PhGA); Clinical Disease Activity Index (CDAI); Simple Disease Activity Index (SDAI); Disease Activity Score on 28 joints- C reactive protein (DAS28 CRP) Pain Catastrophising Scale (PCS); Health Assessment Questionnaire (HAQ), Hospital Anxiety and Depression Scale (HADS); Trait Hope Scale (THS); Acceptance and Action Questionnaire (AAQ); Compassionate Engagement and Action Scales (CEAS). Bold has been used to highlight statistically significant variables.

between the magnification component and SDAI disease activity. This discrepancy may be due to our decision to deliberately exclude individuals with a documented history of psychiatric disorders, as outlined in DSM-V. In fact, it has been reported that magnification shows a significant association with both physical and mental health-related quality of life and depressed mood[14,25,38,39].

Taking together all these data, we may assume that PC is not just a psychological response to pain but may have important consequences on the patient's ability to achieve remission, independently to anxious or depressed mood. We are aware that our study has some limitations including the relatively low number of participants, which does not allow us to derive robust conclusions; on the other hand, despite the limitations, the screening for any previous psychological intervention in our participants allow us to

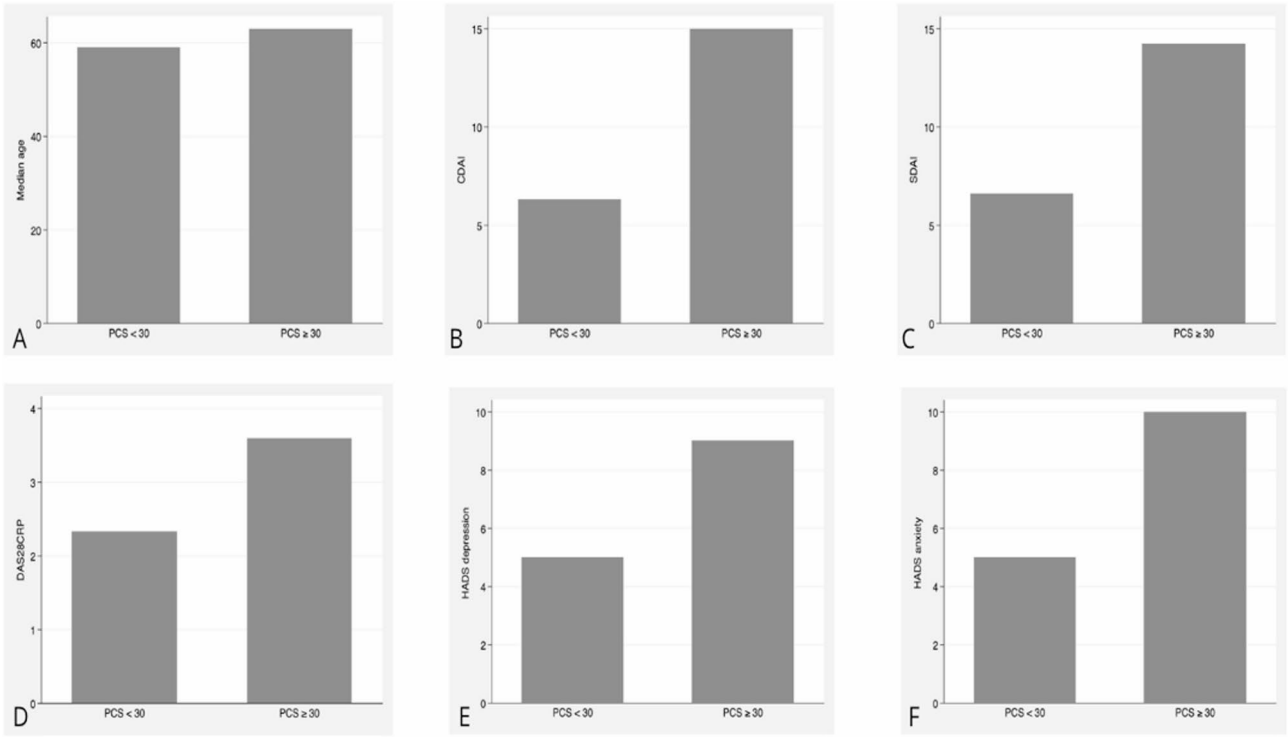

Fig. 1. Histogram representation of age, disease activity, and HADS depression and anxiety according to PCS levels. Clinical Disease Activity Index (CDAI); Simple Disease Activity Index (SDAI); Disease Activity Score on 28 joints- C reactive protein (DAS28 CRP); Hospital Anxiety and Depression Scale (HADS); Pain Catastrophising Scale (PCS).

select a well-defined cohort to assess the role of PC. Moreover, our Italian multicenter approach had the advantage to grant a sample representative of the whole national population.

In conclusion, this study demonstrates that PC affects the patient's perception of the disease indipendently of mood disorder, and significantly impacts the achievement of remission or low disease activity in rheumatoid arthritis. These findings shed a new light on the importance of both assessing and addressing PC in clinical practice.

Variables	Non-adjusted beta coeff.	95% CI	*p*
Age	0.1	0.05 to 1.8	0.07
Sex	1.6	1.5 to 1.1	0.2
BMI	0.1	0.14 to −0.9	0.4
Disease duration	0.01	0.01 to 1.2	0.2
Fibromyalgia	2.8	2. to 1.4	0.1
RA-ILD	6.3	2.7 to 2.3	**0.02**
Rheumatoid factor	1.1	1.4 to 0.8	0.4
ACPA	0.3	1.4 to 0.2	0.80
csDMARDs	1.9	1.4 to 1.4	0.2
bDMARDs/tsDMARDs	2.2	1.5 to 1.5	0.1
CCS	2.9	1.3 to 2.2	**0.03**
PCS	0.3	0.05 to 5	**< 0.0001**
Helplessness	0.6	0.1 to 5.8	**< 0.0001**
Rumination	0.6	0.1 to 4.7	**< 0.0001**
Magnification	0.6	0.3 to 2.2	**0.03**
HADS anxiety	0.4	0.1 to 2.7	**0.01**
HADS depression	0.3	0.2 to 2	**0.04**
AAQ	0.1	0.1 to 0.8	0.4
Hope THS	0.1	0.1 to −0.7	0.5
Self compassion	0.1	0.04 to 1.4	0.2

Table 2. Univariable regression; SDAI as dependent variable. Body Mass Index (BMI); Rheumatoid arthritis-associated interstitial lung disease (RA-ILD); Anti-citrullinated protein antibodies (ACPA); conventional synthetic disease-modifying antirheumatic drugs (csDMARDs); Biological Disease-Modifying Anti-Rheumatic Drugs (bDMARDs); targeted synthetic disease-modifying antirheumatic drugs (tsDMARDs); corticosteroids (CCS); tender joints (TJ), swollen joints (SJ), VAS pain (Visual Analogue Scale of pain), patients' global assessment (PtGA), physician global assessment(PhGA); Clinical Disease Activity Index (CDAI); Simple Disease Activity Index (SDAI); Pain Catastrophising Scale (PCS); Health Assessment Questionnaire (HAQ), Hospital Anxiety and Depression Scale (HADS); Trait Hope Scale (THS); Acceptance and Action Questionnaire (AAQ); Compassionate Engagement and Action Scales (CEAS). Bold has been used to highlight statistically significant variables.

Variable	Adjusted beta coeff	95% CI	*p*
Age	0.02	−0.07 to 0.012	0.65
Sex	1.17	−1.62 to 4.60	0.24
Disease duration	0.02	0.01 to 0.30	0.04
PCS	0.25	0.13 to 0.38	< 0.001
HADS anxiety	0.19	−0.17 to 0.55	0.30
HADS depression	−0.20	−0.63 to 0.23	0.36

Table 3. Multivariable regression; SDAI as dependent variable. Pain Catastrophising Scale (PCS); Hospital Anxiety and Depression Scale (HADS). R-squared 0.2.

Variable	Adjusted beta coeff	95% CI	*p*
Age	0.01	– 0.01 to 0.02	0.95
Sex	0.32	– 0.93 to 0.74	0.13
Disease duration	0.01	– 0.01 to 0.03	0.12
PCS	0.04	0.16 to 0.05	< 0.001
HADS anxiety	0.01	– 0.51 to 0.55	0.93
HADS depression	– 0.01	– 0.63 to 0.63	0.99

Table 4. Multivariable regression; DAS28 as dependent variable. Pain Catastrophising Scale (PCS); Hospital Anxiety and Depression Scale (HADS). R-squared 0.2.

Data availability

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

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Author contributions

All authors made substantial contributions to the conception or design of the work, the acquisition, and interpretation of data. All authors contributed to the critical review and revision of the manuscript and approved the final version. All the authors agreed to be accountable for all aspects of the work. DC study design, data acquisition, statistical analysis, interpretation of data, writing of the first draft of the paper; LK study design, data acquisition, statistical analysis, interpretation of data, writing of the first draft of the paper; FS study design, data acquisition, statistical analysis, interpretation of data, writing of the first draft of the paper; LF statistical analysis, interpretation of data; FT study design, data acquisition, interpretation of data; OB study design, interpretation of data, statistical analysis, interpretation of data, writing of the first draft of the paper; EC study design, data acquisition, interpretation of data; AM study design, data acquisition, interpretation of data; AP study design, data acquisition, interpretation of data; LL study design, data acquisition, interpretation of data; MV study design, data acquisition, interpretation of data; AR study design, data acquisition, interpretation of data; LA study design, data acquisition, interpretation of data; PR study design, data acquisition, interpretation of data; IP study design, data acquisition, interpretation of data; MV study design, interpretation of data; VP study design, data acquisition, interpretation of data; CDV study design, data acquisition, interpretation of data; FC study design, data acquisition, interpretation of data; FM study design, data acquisition, interpretation of data; FDV study design, data acquisition, interpretation of data; AC study design, data acquisition, interpretation of data; FPC study design, data acquisition, interpretation of data; FP study design, data acquisition, interpretation of data; GG study design, data acquisition, interpretation of data; PC study design, data acquisition, interpretation of data; FC study design, data acquisition, interpretation of data; RG study design, data acquisition, interpretation of data, interpretation of data, writing of the first draft of the paper; LN study design, data acquisition, interpretation of data, interpretation of data, writing of the first draft of the paper.

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Declarations

Ethical approval and consent to participate

The study complies with the Declaration of Helsinki, the Campus Bio-Medico University of Rome Ethics Committee has approved the research protocol (approval no. 78.20 OSS) and that informed consent has been obtained from the subjects.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Additional information

Correspondence and requests for materials should be addressed to O.B.

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