RESEARCH ARTICLE / *Araştırma Makalesi*

Neuropathic Pain in Patients with Psoriatic Arthritis: A Bystander or a Gamechanger?

Psoriatik Artritli Hastalarda Nöropatik Ağrı: Seyirci mi, Oyun Değiştirici mi?

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Abstract

Introduction	The relationship between clinical and laboratory parameters associated with the neuropathic pain presence in Psoriatic Arthritis is not well known and has not been adequately studied. Based on these assumptions, the aim of our study is to investigate how often neuropathic pain occurs in Psoriatic Arthritis patients and how much it is related to the clinical and laboratory parameters of the disease.
Materials and Methods	In the cross-sectional study, 45 Psoriatic Arthritis patients diagnosed according to The Classification Criteria for Psoriatic Arthritis were included. In our study, Pain Detect Questionnaire (PDQ) was used to assess the neuropathic pain characteristics. Presence of enthesitis was determined by SPARCC to ensure objective measurements. Functional status was evaluated with the Health Assessment Questionnaire (HAQ). The Short Form-36 (SF-36) questionnaire was used to evaluate the quality of life.
Results	A total of 45 patients were included in the study. The mean duration of symptoms was 78.91 ± 95.8 months. There are 16 patients receiving "NSAID" treatment, 28 patients receiving DMARD treatment, and 13 patients receiving biological therapy. Among the patients included in the study, 30 patients with neuropathic pain and 15 without neuropathic pain were found according to the Pain Detect questionnaire. A significant difference was observed between these two groups in the results of DAPSA, VAS movement, HAQ, morning stiffness, and SF-36 Body pain.
Conclusion	Our study has shown that neuropathic pain has a high prevalence in Psoriatic Arthritis patients. This association was observed to be related to functional limitation. Additionally, the DAPSA score was found to be significantly higher in patients with neuropathic pain due to pain sensation which suggests that it may be a factor reducing treatment success. It is conceivable that the recognition and treatment of neuropathic pain may increase the success of Psoriatic Arthritis treatment.
Keywords	Psoriatic Arthiritis, Neuropatic Pain, DMARD, Biological Treatment, Pain, Rheumatology

Öz

Amaç	*Psoriatik Artritte nöropatik ağrı varlığı ile ilişkili klinik ve laboratuvar parametreleri arasındaki ilişki iyi bilinmemektedir ve yeterince çalışılmamıştır. Bu varsayımlara dayanarak çalışmamızın amacı, Psoriatik Artrit hastalığı bulunan kişilerde nöropatik ağrı dediğimiz durumun ne sıklıkta olduğunu ve hastalığın klinik ve laboratuvar parametreleri ile ne kadar ilişkili olduğunu araştırmaktır.*
Yöntem ve Gereçler	*Kesitsel çalışmada The Classification Criteria for Psoriatic Arthritis'e göre tanı almış 45 Psöriatik Artrit hastası dahil edilmiştir. Çalışmamızda nöropatik ağrı özelliklerinin değerlendirilmesi için Pain Detect Anketi (PDQ) kullanıldı. Objektif ölçümler yapılması amacıyla SPARCC (Spondyloarthritis Research Consortium of Canada) aracılığıyla entezit varlığı belirlendi. Fonkiyonel durum, Sağlık Değerlendirme Anketi (HAQ) ile değerlendirilmiştir. Yaşam kalitesini değerlendirmek için Kısa Form-36 (SF-36) anketi kullanılmıştır.*
Bulgular	*Çalışmaya toplam 45 hasta (32 kadın [%71,1], 13 erkek [%28,9]) alınmıştır. Ortalama semptom süresi 78,91 ± 95,8 aydır. NSAID tedavisi alan 16 hasta (%35,6), DMARD tedavisi alan 28 hasta (%62,2), Biyolojik tedavi alan 13 hasta (%28,9) bulunmaktadır. Çalışmaya alınan hastalarda Pain Detect anketine göre nöropatik ağrısı olan olan 30 hasta (%66,7), Nöropatik ağrı olmayan 15 (33,3) saptanmıştır. Bu iki grup arasında DAPSA, VAS hareket, HAQ, sabah tutukluğu ve SF-36 Vücut ağrısı sonuçlarında anlamlı farklılık saptanmıştır.*
Sonuç	*Psoriatik Artritli hastalarda Nöropatik ağrının yüksek prevelansta bulunduğu çalışmamızla gösterilmiştir. Bu birlikteliğin fonksiyonel kısıtlılıkla ilişkili olduğu görülmüştür. Ayrıca Ağrı hissi nedeniyle DAPSA skoru nöropatik ağrı olan hastalarda anlamlı olarak yüksek bulunmuş ve bu da tedavi başarısını düşüren bir etken olabileceğini düşündürmektedir. Nöropatik ağrının tanınması ve tedavi edilmesinin Psoriatik Artrit tedavisindeki başarıyı arttırabileceği düşünülebilir.*

INTRODUCTION

Psoriatic arthritis (PsA) is a progressive, erosive, chronic, heterogeneous, and systemic inflammatory disease that develops in 30% of patients with psoriasis.[1] PsA can affect six clinical domains including peripheral arthritis, dactylitis, enthesitis, psoriasis, psoriatic nail disease, and axial disease.[2,3] PsA can be treated with DMARDs and biologics effectively.[4] However, although these treatments may lower inflammation in rheumatic diseases, some patients complain of decreased physical functions and quality of life due to pain.[5] Pain is the most common symptom in chronic inflammatory diseases, the occurrence of which is due to different mechanisms.[6] Pain in inflammatory diseases was considered as only a symptom until a few years ago, but there is now increasing evidence that chronic pain is a disease in itself.[7,8] Patients with inflammatory arthritis (IA), such as rheumatoid arthritis (RA), ranked pain as the most important symptom. In spite of the advances in RA treatment, many patients still complain of pain. Studies have shown us that occurrence of fibromyalgia with RA, a prototype of central sensitization, is associated with poorer improvement in both pain evaluation and disease activity scores of anti-inflammatory therapy.[9,10]

Pain is generally considered to occur due to inflammation in the synovium stimulating afferent sensory nerve C fibers in patients with RA, PsA, and spondyloarthritis (SpA), and thus it is accepted to be of nociceptive origin. On the other hand, pain hypersensitivity, an increased response of central and peripheral neurons, may continue after the cessation of inflammation due to maladaptive stimuli that lead to chronic pain.[11,12] Pain hypersensitivity leads to overestimation of joint sensitivity, pain, and the thought that health condition is worsening. For this reason, determining the possible underlying pain mechanisms may be important to help the treatment success.[13] In inflammatory joint diseases, accurate evaluation of pain is essential for treatment and follow-up because the main disease is a parameter evaluated in the calculation of activity indices. These are disease activity score-28 (DAS-28) for RA, ankylosing spondylitis disease activity score (ASDAS) for ankylosing spondylitis (AS), or disease activity index (DAPSA) for psoriatic arthritis for PsA.[14-16] As disease activity indices represent a very important variable both in daily clinical practice and in observational and clinical studies, this issue is important.[17]

Neuropathic pain is defined as pain caused or triggered by primary damage or dysfunction of the nervous system.[18] Neuropathic pain symptoms include burning, tingling, electric shock-like pain, hyperalgesia, and allodynia.[19] The relationship of the neuropathic pain with RA and Osteoarthritis (OA) has been examined in previous studies.[20-24] Regarding RA, it has been shown that at least 13% of patients had neuropathic pain features, that they could be detected in the early stage of the disease, and that their presence decreased remission success in the 6-month follow-up.[21] The prevalence of neuropathic pain in OA is estimated to be around 23%, and neuropathic pain was observed to persist even when invasive treatment strategies such as total knee replacement were used.[23,24]

In axial spondyloarthritis, both AS and non-radiographic axSpA, the presence of neuropathic pain is slightly over 30% and is related to lower quality of life, lower scores on patient assessment criteria, and higher functional limitation.[25,26]

The first data related to neuropathic pain in PsA were obtained from the DANBIO study.[27] The presence of neuropathic pain was evaluated with the PainDETECT questionnaire (PDQ) in Danish database. Researchers who participated in this study investigated pain prevalence in various rheumatological diseases. In the context of PsA, the presence of neuropathic pain characteristics was demonstrated in 28% of patients; this is a higher percentage compared to both RA and axSpA. As seen in this study, neuropathic pain characteristics are considered to be common in PsA patients.

The relationship between clinical and laboratory parameters associated with the neuropathic pain presence in PsA is not well known and has not been studied adequately. Based on these assumptions, the aim of our study was to research the frequency of neuropathic pain in people with PsA disease and its relation to the clinical and laboratory parameters of the disease.

MATERIALS and METHODS

Patient Selection

A total of 45 PsA patients diagnosed according to The Classification Criteria for Psoriatic Arthritis were included in this study, which was conducted at Bezmialem Foundation University Hospital between January 2022 and March 2022. Exclusion criteria from the study were presence of other rheumatic diseases, diseases that commonly cause neuropathic pain such as fibromyalgia, diabetes mellitus, chronic kidney failure or chronic liver disease, the presence of active skin conditions other than psoriasis, the presence of inflammatory joint comorbidities (such as gout or calcium pyrophosphate crystal arthropathy), entrapment neuropathies (e.g., carpal tunnel syndrome), cervical or lumbar radiculopathies, and polyneuropathies supported by any etiology. Fibromyalgia was excluded using the 2016 fibromyalgia diagnostic criteria.[12]

Patients underwent a cross-sectional evaluation, with an objective musculoskeletal examination on the day of admission by an experienced clinician who aimed to investigate the effect of PsA, assessing functional status and neuropathic features of pain. Demographic data, comorbidities, ongoing treatment, and acute phase reactants were also recorded for each patient.

PsA Measurements

For objective measurements, the number of tender joints (0-68 joints), the number of swollen joints (0-66 joints), and the presence of enthesitis were determined by SPARCC (Spondyloarthritis Research Consortium of Canada).

In addition to the number of tender and swollen joints, the clinician's overall assessment [0–10 visual rating scale (VAS)] of the patient's disease activity, the patient's VAS pain assessment (0–10), and the C-reactive protein value (CRP; mg/dl) were used to calculate the DAPSA.[16] DAPSA is a composite disease activity index specific to PsA and is an internationally accepted scale. It allows determination of disease activity status: ≤ 4 for remission, > 4 and ≤ 14 for low disease activity, > 14 and ≤ 28 for moderate disease activity, and >28 for high disease activity.[18]

Using the SPARCC clinical score, 8 bilateral enthesitis sites (medial and lateral humeral epicondyles, supraspinatus muscle tendon greater humeral tubercle, greater femoral trochanter, quadriceps tendon placed at the upper pole of the patella, patellar ligament placed on the lower pole of the patella or on the tibial calcaneal tubercle, Achilles tendon calcaneus and plantar fascia calcaneus), we evaluated the absence (0) or presence (1) of pain by palpation.

Functional status was assessed with the Health Assessment Questionnaire (HAQ). HAQ assesses the degree of difficulty in performing common daily activities in 8 areas compared to the previous week. For each activity, the patient is asked to respond on a 4-point scale (0 = no difficulty, 3 = impossible), and the highest value for each functional area is accepted. The final score is given by the average of 8 values.[28]

The Short Form-36 (SF-36) questionnaire was used to evaluate the quality of life. The SF-36 measures eight functions: physical function (PF), physical role "PR", bodily pain (BP), general health (GH), vitality (VT), social functioning (SF), emotional role (RE), and mental health (MH). Scoring was done in line with the suggestions of the developers of the questionnaire.[29]

Evaluation of Neuropathic Pain

Pain Detect Questionnaire (PDQ) was used to evaluate neuropathic pain characteristics in our study. It was

developed by the German Neuropathic Pain Research Group 10 years ago. The questionnaire was validated in different clinical entities such as post-thoracotomy pain, neoplasms, low back pain, OA, fibromyalgia syndrome (FMS), and in the field of inflammatory joint diseases. It is a self-administered questionnaire which can distinguish the nociceptive components from the neuropathic components of pain.[30] The questionnaire, which does not require a physical examination and evaluates the patient's symptoms, investigates sensations related to the presence of neuropathic pain such as allodynia, hyperalgesia, dysesthesia, and sudden pain. The PDQ assesses the qualitative characteristics of the painful sensations (burning, tingling or prickling, pain evoked by light touch, sudden pain attacks, pain at cold or warm stimulus, numbness, mild pressure that triggers pain), and areas where the pain radiates are indicated on the mannequin. There is also a question for the temporal course of pain (score from -1 to 1 depending on the selected pain course model). The final score can range from 1 to 38. Patients scoring between 0 and 12 were considered as negative. Patients with scores of 13-18 and ≥19 were considered as probable and highly probable neuropathic pain patients, respectively, as shown in previous studies.[31,32] Rifbjerg-Madsen et al. examined psychometric properties (Rasch analysis and test-retest analysis) of the PDQ in a large cohort of patients with inflammatory joint diseases (including PsA) and demonstrated that it was acceptable for pain classification.[33]

Statistical Analysis

All data were analyzed with Statistical Package for Social Sciences (SPSS 20.0; IBM, Armonk, NY, USA). The data of patients with PsA were evaluated in terms of normal distribution using the Kolmogorov-Smirnov test. Independent variables were age, body mass index (BMI), disease duration, use of biological agents, DMARD use, DAPSA, SPARCC, "HAQ", SF-36 score, VAS pain, erythrocyte sedimentation rate, and CRP. The demographic variables and clinical parameters of the patients were compared using the t-test or the $\chi 2$ test. Patients with PDQ values of <12 or ≥13 were grouped as NeP negative and NeP positive and compared using the t-test within each group. P value <0.05 was considered as statistically significant.

RESULTS

A total of 45 patients (32 females [71.1%], 13 males [28.9%]) were included in the study. Mean duration of symptoms was 78.91 ± 95.8 months. There were 16 patients (35.6%) receiving NSAID therapy, 28 patients (62.2%) receiving DMARD therapy, and 13 patients (28.9%) receiving biological therapy. Mean and median values of demographic and clinical variables and the minimum and maximum values of clinical parameters are given in Table 1.

Among the patients included in the study, according to the Pain Detect questionnaire, 30 patients with NP (66.7%) and 15 patients without NP (33.3%) were detected. DAPSA scores showing disease activity were significantly higher in patients with high PDQ scores (Table 2). In relation to disease activity, the number of sensitive and swollen joints, pain score, patient's global evaluation, dactylitis and enthesitis scores and measured disease activity were statistically significantly higher in NP group than non-NP group. Morning stiffness in patients with NP was statistically significantly higher than in patients without NP ($p<0.05$).

The VAS movement pain assessment test, which assesses the pain level of the patients during physical activity, was significantly higher in NP patients ($p<0.05$).

The HAQ questionnaire that shows the degree of physical limitation was statistically significantly higher in the NP group than in the non-NP group ($p<0.05$).

SF-36 body pain score, which assesses the perception of body pain, was statistically significantly higher in NP group ($p<0.05$) (Table 2).

Table 1. Mean and median values of demographic and clinical variables and the minimum and maximum values of clinical parameters

Parameter	Group	N (%)	Parameter	Mean ± SD	Median (Min-Max)
Education	Primary school	18 (40.0%)	Body Pain	37.78 ± 19.07	41 (0 - 84)
	Secondary school	8 (17.8%)	CRP	6.1 ± 12.66	1.2 (0.02 - 68)
	High school	11 (24.4%)	DAPSA	27.32 ± 13.81	22.78 (7.5 - 64.77)
	University	8 (17.8%)	DN4	4.16 ± 2.54	5 (0 - 9)
Type Of Disease	Axial spondyloar-thritis	10 (22.2%)	ESR	14.96 ± 10.55	15 (2 - 44)
	Oligoarticular	26 (57.8%)	General Health	42.98 ± 15.7	45 (10 - 77)
	Poliartricular	9 (20.0%)	HAQ	10.07 ± 8.46	8 (0 - 38)
Job	Not working	24 (53.3%)	Disease Duration (Months)	78.91 ± 95.8	36 (1 - 420)
	Traveling business	14 (31.1%)	Mental Health	53.78 ± 17.63	56 (12 - 80)
	Office	7 (15.6%)	Pain Detect	14.2 ± 5.84	14 (1 - 26)
Alcohol	Not consuming	39 (86.7%)	Physical function	65.33 ± 20.63	70 (20 - 100)
	Consuming	6 (13.3%)	Role emotional	35.56 ± 39.19	33.33 (0 - 100)
Biological Treatment	No	32 (71.1%)	Role physical	30.0 ± 36.38	25 (0 - 100)
	Yes	13 (28.9%)	Morning stiffness (mins)	32.58 ± 48.14	15 (0 - 240)
Gender	Male	13 (28.9%)	Social functioning	61.4 ± 24.27	62.5 (12.5 - 100)
	Female	32 (71.1%)	SPARCC	4.47 ± 3.35	4 (0 - 16)
DMARD	No	17 (37.8%)	VAS Movement	6.24 ± 1.94	6 (1 - 10)
	Yes	28 (62.2%)	VAS Rest	4.89 ± 2.32	5 (0 - 10)
Marital Status	Single	5 (11.1%)	Vitality	31.11 ± 17.12	30 (0 - 60)
	Married	40 (88.9%)	Age	44.24 ± 9.26	44 (22 - 66)
NSAID	No	29 (64.4%)			
	Yes	16 (35.6%)			
Smoke	Non-smoker	24 (53.3%)			
	Smoker	21 (46.7%)			

CRP:C-reactive protein, DAPSA: The Disease Activity Index for Psoriatic Arthritis, DN4: Douleur Neuropathique 4 Questions, ESR: Erythrocyte sedimentation rate, HAQ: Health Assessment Questionnaire, SPARCC: Spondyloarthritis Research Consortium of Canada Enthesitis Index, VAS: Visual Analogue Scale

Table 2:			
Parameter	NEUROPATHIC PAIN - PAIN DETECT QUESTIONNAIRE		
	Patients with NP (30)	Patients wihout NP (15)	P value
Body pain	33.07 ± 18.1	47.2 ± 17.93	0.017(S)
	32 (0 - 84)	51 (12 - 84)	
CRP	6.91 ± 14.85	4.46 ± 6.51	0.3(M)
	1.15 (0.02 - 68)	1.92 (0.2 - 26.13)	
DAPSA	31.66 ± 14.84	18.64 ± 4.72	0.001(M)
	28.11 (7.5 - 64.77)	17.78 (12.05 - 26.15)	
ESR	15.6 ± 10.8	13.67 ± 10.26	0.514(M)
	15 (2 - 44)	15 (4 - 43)	
General health	41.13 ± 15.72	46.67 ± 15.52	0.27(S)
	40 (10 - 72)	45 (15 - 77)	
HAQ	12.47 ± 8.89	5.27 ± 4.92	0.002(M)
	9.5 (1 - 38)	4 (0 - 19)	
Disease duration (Months)	78.13 ± 106.27	80.47 ± 73.82	0.322(M)
	24 (1 - 420)	60 (1 - 240)	
Mental health	50.13 ± 18.78	61.07 ± 12.69	0.066(M)
	52 (12 - 76)	64 (36 - 80)	
Pain Detect	17.4 ± 3.84	7.8 ± 3.3	<0.001(S)
	17 (10 - 26)	8 (1 - 12)	
Physical function	61.33 ± 21.17	73.33 ± 17.49	0.065(S)
	62.5 (20 - 100)	75 (30 - 100)	
Role emotional	35.56 ± 38.09	35.58 ± 42.68	0.99(M)
	33.33 (0 - 100)	0 (0 - 100)	
Role physical	22.5 ± 32.4	45.0 ± 40.31	0.065(M)
	0 (0 - 100)	50 (0 - 100)	
Morning stiffness(Mins)	44.2 ± 55.35	9.33 ± 8.42	0.003(M)
	22.5 (0 - 240)	10 (0 - 30)	
Social functioning	60.83 ± 22.44	62.53 ± 28.38	0.828(S)
	56.25 (25 - 100)	62.5 (12.5 - 100	
VAS movement	6.83 ± 1.97	5.07 ± 1.28	0.001(M)
	8 (1 - 10)	5 (3 - 8)	
VAS rest	5.23 ± 1.74	4.2 ± 3.14	0.163(M)
	5 (2 - 9)	3 (0 - 10)	
Vitality	27.67 ± 16.23	38.0 ± 17.3	0.055(S)
	25 (0 - 55)	35 (0 - 60)	
Age	45.17 ± 9.18	42.4 ± 9.46	0.351(S)
	46 (22 - 64)	44 (22 - 66)	
SPARCC	4.8 ± 3.12	3.8 ± 3.78	0.12(M)
	4 (0 - 16)	3 (0 - 12)	

CRP:C-reactive protein, DAPSA: The Disease Activity Index for Psoriatic Arthritis, ESR: Erythrocyte sedimentation rate, HAQ: Health Assessment Questionnaire, VAS: Visual Analogue Scale, SPARCC: Spondyloarthritis Research Consortium of Canada Enthesitis Index

DISCUSSION

Our study shows that the prevalence of neuropathic pain is high in PsA patients (66.7%). In these patients, pain severity and disease activity measurements were higher. In a study conducted with RA patients, it was observed that RA patients with NP had significantly higher pain severity and disease activity measurements.[5] Similar results were also obtained in our study. Additionally, depression rates were higher in patients with NP in this study.[5] In another study, neuropathic pain prevalence was researched in patients with RA, PsA and SpA using PDQ and it was found to be 28% in PsA patients. Additionally, NP was detected at a higher rate than the two types of arthritis researched.[13] Similar to our study, high disease activities were seen in NP patients in this study.

In that study, since fibromyalgia (FMS) patients were included in the study, distinction between FMS and NP is not clear.[13] It was shown that PainDETECT questionnaire could not distinguish between FMS and NP.[34] But in our study, patients with FMS were not included according to ACR Appropriateness Criteria, and this is considered to increase the importance of the study.

In another study conducted in PsA patients by using PDQ, NP prevalence was found to be 42%.[17] This study also shows that NP is seen at a high rate in PsA patients and affects disease activity.

In the field of both inflammatory and degenerative joint diseases, the mechanisms underlying pain symptoms have been the subject of intensive research in recent years. In particular, there is growing awareness that mechanisms including peripheral and central sensitization are involved as well as the nociceptive pathway.[35] Our results are consistent with those collected by Rifbjerg-Madsen et al., who first documented the significant prevalence of central pain from DANBIO registry data.[27]

In a prognostic study conducted on patients diagnosed with early RA, it was demonstrated that high PDQ scores at baseline resulted in low probability of Boolean remission at the 6th month evaluation.[36]

The strength of our study is that it researched demographic and disease-specific clinical variables associated with the presence of neuropathic pain characteristics in PsA patients. Additionally, it is considered important to determine the presence of NP in PsA patients. Pain severity and disease activity have been shown to be high in these patients. It was considered that this situation may lead to a low response to treatment and a decrease in the success of treatment. Apart from this, in the HAQ and VAS Movement results, which evaluate physical activity, a statistically significant difference was found. It can be thought that this situation reduces the patient's quality of life and physical activity and affects the treatment response and the patient's expectations. In this regard, it is considered that recognition of the presence of NP and treating it in patients with PsA may also affect the success of PsA treatment.

The limitations of our study are that the cross-sectional evaluation did not allow prognostic evaluation, the mild effect of psoriasis in our case study, and the effect of skin disease on certain pain descriptors of PDQ.

CONCLUSION

In conclusion, it has been shown that NP component is frequently seen in PsA patients, and the presence of this parameter may have a negative effect on physical limitation and treatment success. It has been considered that the recognition of NP and regulation of its treatment could increase the treatment success. It has been shown that presence of NP can cause severe disease.

Authorship contribution

Conceptualization; MSK, OVY. Data curation; MSK, OVY. Formal analysis; MSK, OVY. Investigation; MSK, MK, OVY. Methodology; MSK, MK, OVY. Project administration; MSK, MK, OVY Resources; MSK, OVY. Supervision;

MSK, OVY Validation; MSK. Roles/Writing-original draft; MSK. Writing-review & editing; MK

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Ethical consent

The study protocol was approved by the Bezmialem Foundation University Faculty of Medicine Ethics Committee (2020-06/95). Written informed consent was obtained from each patient. The study was carried out in accordance with the principles of the Declaration of Helsinki.

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