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ORIGINAL ARTICLE

Neuropathic pain in knee osteoarthritis: Prevalence, diagnosis, and clinical implications

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ABSTRACT

Objectives: This study aims to explore the prevalence of neuropathic pain (NP) in patients with knee osteoarthritis (KOA) and to assess its correlation with functional status.

Patients and methods: Between December 2023 and May 2024, a total of 193 patients (48 males, 145 females; mean age: 58.7±12.8 years; range, 22 to 89 years) who were diagnosed with KOA and had persistent knee pain for more than three months were included. The painDETECT and Douleur Neuropathique en 4 Questions (DN4) questionnaires were utilized to evaluate NP. The Visual Analog Scale (VAS) was used to assess pain severity, while the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) was utilized to assess functional status.

Results: The painDETECT indicated NP in 27.5% of patients, while the DN4 scale showed 30.6%. Patients with NP exhibited significantly elevated VAS and WOMAC scores to patients without NP, indicating a greater severity of pain and functional impairment in this subgroup ($p<0.05$). The agreement between the painDETECT and DN4 was moderate ($\kappa=0.472$). There were significant correlations between the painDETECT and DN4 scores with the WOMAC total score ($r=0.371$ and $r=0.242$, respectively, $p<0.001$).

Conclusion: Neuropathic pain is common in KOA patients and is associated with higher pain intensity and poorer functional outcomes. The moderate agreement between the painDETECT and DN4 scores may lead to a certain degree of diagnostic variation. Combining more than one method may increase the diagnostic accuracy.

Keywords: DN4 Questionnaire, knee, neuropathic pain, osteoarthritis, painDETECT, visual analog scale.

Knee osteoarthritis (KOA) is a prevalent musculoskeletal disorder, increasingly affecting the elderly due to an aging population and rising obesity rates, making it a significant cause of disability.[1] The disease begins with damage to the articular cartilage. Cartilage degeneration occurs due to the low healing capacity of chondrocytes. As KOA progresses, catabolic activities in articular cartilage are significantly accelerated. These catabolic factors cause inflammation in the subchondral bone, soft tissues, and synovium, leading to progressive joint damage.[2] Pain, as the primary symptom in KOA, is predominantly nociceptive, arising from local tissue damage. This type of pain is often provoked by joint movement and loading, particularly during the early stages of KOA. As the condition progresses, persistent pain at rest may develop, potentially leading to discomfort even during nighttime. However, it is still unclear at which point in the OA process the pain begins.[3,4]

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The complex pathophysiology of pain in KOA involves various factors beyond joint damage and synovitis, including periarticular and central sensitization. Recent studies have suggested both nociceptive and neuropathic pain (NP) mechanisms contribute to KOA, likely due to abnormalities in peripheral and central nervous system pathways.[5,6] Neuropathic pain often presents with sensory symptoms like burning, stinging, tingling, and chills.[7] Its pathogenesis involves ectopic activity, sensitization, and impaired modulation, which all contribute to increased neuronal excitability and hyperalgesia.[8] In KOA patients with severe pain, central sensitization is thought to increase, particularly when moderate or severe radiographic findings are absent. The potential mechanisms underlying NP in KOA remain unclear, highlighting the need for further research.

Current hypotheses have proposed that localized damage to structures such as the synovial capsule, ligaments around the joint, periosteum, and bone beneath the cartilage, coupled with inflammatory responses, may lead to peripheral nerve injury and the subsequent activation of nociceptors innervating these tissues.[9,10]

In the present study, we hypothesized that chronic joint pain in KOA might have a neuropathic component. We, therefore, aimed to explore the prevalence of NP symptoms in KOA and to assess the association between NP and functional status.

PATIENTS AND METHODS

This single-center, cross-sectional study was conducted at Antalya Training and Research Hospital, Department of Orthopedics and Traumatology between December 2023 and May 2024. Both inpatients and outpatients with chronic pain related to KOA who had persistent knee pain for more than three months were screened. The diagnosis of KOA was based on the American College of Rheumatology (ACR) classification criteria.[11] Individuals with inflammatory rheumatic diseases, prior knee surgeries, radiculopathies associated with infection or NP, those diagnosed with diabetes mellitus, B12 deficiency, cerebrovascular accidents, or participants who underwent treatment for neuropathy within the last six months were excluded from the study. Finally, a total of 193 KOA patients (48 males, 145 females; mean age: 58.7±12.8 years; range, 22 to 89 years) who met the inclusion criteria were recruited. A written informed consent was obtained from each patient. The study protocol was approved by the Antalya Training and Research Hospital Clinical Research Ethics Committee (Date: 23.11.2023, No: 16/8). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Anteroposterior (AP) knee radiographs were taken while standing under load and graded according to the Kellgren-Lawrence (K-L) scale, which is widely used to assess KOA severity, ranging from 0 (none) to 4 (severe).[12]

The age, sex, duration of symptoms, physical examination findings of the patients were recorded. Patients' current illnesses and medications were questioned. All individuals received a detailed clinical assessment to confirm their eligibility for participation. Body mass index (BMI), weight, and body composition were assessed using the Tanita MC 780 segmental body composition analyzer, with measurements taken while participants were barefoot through bioimpedance analysis. Additional evaluations included the Visual Analog Scale (VAS), where the patients rated their knee pain severity from 0 (no pain) to 10 (most severe pain),[13] and the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), consisting of 24 items evaluating stiffness, pain, and physical functions. A high score indicated an advanced stage of osteoarthritis.[14] The modified Charlson Comorbidity Index (CCI) was used to evaluate comorbidity burden, providing insights into overall health status and potential risk factors affecting KOA outcomes.[15]

Currently, there are no universally accepted standards, biomarkers, or imaging techniques that can definitively diagnose NP. The diagnosis is typically based on the patient's medical history, clinical examination, and the use of assessment tools to support the diagnosis, such as painDETECT and Douleur Neuropathique en 4 Questions (DN4). In this study, the painDETECT and DN4 were used together to assess NP. The painDETECT, a screening tool assessing temporal characteristics and pain distribution, provided scores ranging from 0 to 38, where scores ≤12 indicated NP was unlikely, 13-18 suggested possible NP, and ≥19 indicated a high likelihood of NP.[16] The DN4 included 10 Yes-or-No items, with a score of ≥4 indicating NP. Seven items assessed pain characteristics, while three assessed sensory abnormalities.[17]

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). Continuous data were expressed

in mean ± standard deviation (SD) or median (min-max), while categorical data were expressed in number and frequency. The distribution of continuous variables was assessed using histograms, probability plots, and the Kolmogorov-Smirnov test, revealing a non-normal distribution. The Mann-Whitney U test was used for comparisons. To determine the interobserver reliability of the K-L grading, the intraclass correlation coefficient (ICC) was utilized. Agreement between the painDETECT

TABLE I

Demographic and clinical characteristics of patients (n=193)

Variables	n	%	Mean±SD	Median	Min-Max
Age (year)			58.7±12.8	59.0	22.0-89.0
Sex					
Female	145	75.1			
Male	48	24.9			
BMI (kg/m²)			30.2±5.9	29.3	18.7-50.0
Normal	27	14.0			
Overweight	74	38.3			
Obesity	92	47.7			
Occupational status					
Housewife	98	50.8			
Not working	7	3.6			
Working	48	24.9			
Retired	40	20.7			
Duration of symptoms (year)			3.6±2.7	3.0	0.5-10.0
<1	43	22.3			
≥1	150	77.7			
Severity of pain					
VAS at rest			6.3±2.1	7.0	0-10.0
VAS with activity			6.4±1.9	7.0	1.0-10.0
Presence of neuropathic pain					
DN4 score			2.8±2.7	2.0	0-9
<3	134	69.4			
≥4	59	30.6			
Pain detect score			10.7±7.9	9.0	–1-30
≤18	140	72.5			
≥19	53	27.5			
Vitamin D level			23.5±13.1	22.1	6.6-76.0
<30	163	84.6			
≥30	30	15.5			
Side of knee pain					
Right	38	19.7			
Left	37	19.2			
Bilateral	118	61.1			
Dominant knee pain side					
Right	97	50.3			
Left	96	49.7			
Kellgren-Lawrence grade			2.7±1.0	3.0	1.0-4.0
1	22	11.4			
2	70	36.3			
3	44	22.8			
4	57	29.5			
WOMAC, total score			52.9±21.2	51.0	0-96.0
Pain			11.8±4.9	11.0	0-20.0
Stiffness			3.3±2.4	3.0	0-8.0
Physical function			37.7±15.7	38.0	0-68.0
Modified Charlson Index			2.4±1.6	2.0	0-7.0

SD: Standard deviation; BMI: Body mass index; VAS: Visual Analog Scale; DN4: Douleur Neuropathique en 4 Questions; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index.

TABLE II
Clinical characteristics of patients based on DN4 scores and painDETECT

	PainDETECT									DN4								
	Non-NP ≤18 (n=140)				NP ≥19 (n=53)					Non-NP ≤3 (n=134)				NP ≥4 (n=59)				
Variables	n	%	Median	Min-Max	n	%	Median	Min-Max	*p*	n	%	Median	Min-Max	n	%	Median	Min-Max	*p*
Age (year)			58.0	22.0-89.0			63.0	28.0-82.0	**0.028**			59.0	22.0-89.0			59.0	28.0-82.0	0.890
Sex									1.000									0.131
Female	105	75.0			40	75.5				96	71.6			49	83.1			
Male	35	25.0			13	24.5				38	28.4			10	16.9			
BMI (kg/m^2)			28.9	18.7-50.0			30.2	20.9-41.8	**0.007**			29.0	18.7-49.0			30.2	20.3-50.0	**0.048**
Duration of symptoms (year)			2.0	0.5-10.0			5.0	0.6-10.0	**0.001**			3.0	0.5-10.0			4.0	0.5-10.0	0.054
Severity of pain																		
VAS at rest			7.0	0-10.0			8.0	3.0-10.0	**0.005**			6.5	0-10.0			7.0	3.0-10.0	**0.003**
VAS with activity			6.0	1.0-10.0			7.0	3.0-10.0	**0.004**			6.0	1.0-10.0			8.0	3.0-10.0	**0.001**
Vitamin D level			21.9	6.6-76.0			23.6	6.7-58.4	0.805			22.1	6.6-76.0			17.3	6.7-59.1	0.762
Kellgren-Lawrence grade			2.0	1.0-4.0			3.0	1.0-4.0	**0.021**			2.0	1.0-4.0			3.0	1.0-4.0	**0.031**
WOMAC (total score)			47.0	0-96.0			68.0	0-96.0	**<0.001**			48.0	5.0-96.0			65.0	0-96.0	**0.001**
Pain			10.0	0-20.0			15.0	0-20.0	**<0.001**			10.0	3.0-20.0			14.0	0-20.0	**0.002**
Stiffness			2.0	0-8.0			5.0	0-8.0	**<0.001**			2.0	0-8.0			5.0	0-8.0	**<0.001**
Physical function			34.0	0-68.0			50.0	0-68.0	**<0.001**			34.0	0-68.0			47.0	0-68.0	**0.002**
Modified Charlson Index			2.0	0-6.0			3.0	0-7.0	**<0.001**			2.0	0-6.0			3.0	0-7.0	0.059

DN4: Douleur Neuropathique en 4 Questions; NP: Neuropathic pain; BMI: Body mass index; VAS: Visual Analog Scale; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index.

TABLE III
Correlation analysis results

	PainDETECT score		DN4 score	
	r	*p*	r	*p*
Age (year)	0.185	0.010	0.084	0.247
Body mass index (kg/m²)	0.211	0.003	0.196	0.006
Duration of symptoms (year)	0.242	0.001	0.221	0.002
Severity of pain				
VAS at rest	0.282	<0.001	0.226	0.002
VAS with activity	0.244	0.001	0.277	<0.001
Vitamin D level	0.001	0.997	–0.039	0.682
Kellgren-Lawrence grade	0.182	0.011	0.181	0.012
WOMAC				
Total score	0.371	<0.001	0.242	0.001
Pain	0.347	<0.001	0.227	0.001
Stiffness	0.409	<0.001	0.296	<0.001
Physical function	0.347	<0.001	0.235	0.001
Modified Charlson Index	0.280	<0.001	0.202	0.005
PainDETECT score	-	-	0.647	<0.001
DN4 score	0.647	<0.001	-	-

DN4: Douleur Neuropathique en 4 Questions; VAS: Visual Analog Scale; WOMAC: Western Ontario and McMaster Universities Arthritis Index; r: Spearman's rho.

and DN4 was assessed using the kappa (κ) coefficient. The κ values between 0.40 and 1.00 represent moderate to perfect agreement.[18] The overall agreement between the two questionnaires was expressed in percentage. The relationships between continuous variables were analyzed using the Spearman correlation test. Values below 0.40 represent weak correlation, while values between 0.40 and 1.00 represent moderate-to-strong correlation.[19] A *p* value of <0.05 was considered statistically significant.

RESULTS

The mean DN4 score was 2.8±2.7, with 30.6% of patients scoring ≥4. The mean painDETECT score was 10.7±7.9, with 27.5% of patients scoring ≥19. The number of patients diagnosed with bilateral knee involvement secondary to osteoarthritis was 118 (61.1%). Most patients were classified as Grade 2 (36.3%) or Grade 3 (29.5%) in osteoarthritis severity. The mean WOMAC total score was 52.9±21.2 (Table I). The κ value for the agreement between the painDETECT and DN4 questionnaires was 0.472 (p<0.001), indicating a significant overall agreement at 78%. Additionally, the interobserver reliability for KOA assessment showed an ICC of 0.922 (p=0.001).

There were significant differences in the VAS scores at rest and during activity, K-L KOA grade, and WOMAC scores (p<0.05 for all) between patients with NP and those without NP, as evidenced by both the painDETECT and DN4. However, vitamin D levels were similar in patients with and without NP (p=0.805 for painDETECT and p=0.762 for DN4) (Table II).

The correlation between the painDETECT and DN4 scores was strong (r=0.647). For painDETECT scores, significant correlations were found with BMI, duration of symptoms, VAS at rest and activity, K-L grade, WOMAC total score and subdomains, and CCI (p<0.05 for all) (Table III). For DN4 scores, significant correlations were found with BMI, duration of symptoms, VAS at rest and activity, K-L grade, WOMAC total and subdomains, and CCI (p<0.05 for all), indicating a weak correlation.

DISCUSSION

In the present study, we investigated the prevalence of NP symptoms in KOA patients and evaluated the association between NP and functional status. The main finding of this study is that NP significantly influences pain perception and functionality in KOA patients, aligning with broader observations

on the multifaceted nature of pain in chronic joint conditions.[20] In this study, the prevalence of NP among KOA patients was assessed using two validated tools. The painDETECT identified NP in 27.5% of patients, while the DN4 revealed a slightly higher prevalence of 30.6%. Previous studies have reported the prevalence of NP symptoms in 20 to 40% of KOA patients. These findings align with the literature and highlight the importance of considering both nociceptive and neuropathic components in understanding and managing pain in KOA patients.[21,22]

Although both painDETECT and DN4 provide useful results for assessing NP, their differences in accuracy show the importance of choosing the right tool for each patient.[16,17] Gölge et al.[23] found that WOMAC pain and VAS scores were significantly higher in KOA patients with NP compared to those without NP. In this study, patients with NP had significantly higher VAS (rest, activity) and WOMAC scores compared to patients without NP. These findings are consistent with those reported in previous studies.[24] In contrast to this result, Ohtori et al.[21] reported that WOMAC pain scores and painDETECT scores were correlated, however, there was no significant correlation with the WOMAC physical function scores. Many studies have suggested that NP symptoms may be affected by the duration of KOA symptoms.[24-27] Polat et al.[28] suggested that a longer duration of symptoms could be a risk factor for the development of NP. Supporting this hypothesis with further studies is of utmost importance for determining the NP risk factors in KOA, as identifying the character of pain in KOA is crucial in terms of treatment planning. In cases accompanied by NP, centrally acting drugs such as duloxetine, gabapentin, and pregabalin are usually utilized. Non-steroidal anti-inflammatory drugs (NSAIDs) and analgesics can be added to treatment to yield more favorable outcomes.[29]

To diagnose and treat NP early, it is of paramount importance to identify the local damage to the joint that occurs in KOA. Particularly in assessments using the K-L classification, increased radiographic severity has been suggested as a significant predictor of NP development.[24,30] In this context, numerous studies have investigated the relationship between knee pain and radiographic findings and shown that NP may be associated with radiographic severity in KOA patients.[31,32] However, some research has reported conflicting results.[33] To illustrate, Valdes et al.[33] observed an inverse relationship between radiographic severity and pain. However, these results showed a significant correlation between K-L grades and NP scores. This finding suggests that advanced radiographic changes may amplify NP symptoms in KOA, likely due to heightened inflammatory (cytokines, prostaglandins, etc.) and mechanical stress on peripheral nerves. Mechanical changes which lead to instability and impaired mobility in the joint can mechanically stimulate the nerves. Continuous pain stimuli in advanced KOA can lead to central sensitization and NP. Further studies are needed to clarify these contradictions between radiological grade and pain in the literature.

Detecting the presence of concomitant NP in KOA is important, as individuals with NP often report worse pain severity, concomitant anxiety-depression, and poorer quality of life.[34] In this study, we found a significant relationship between the CCI and painDETECT results, but not with DN4. This discrepancy may be due to the differences in how painDETECT and DN4 assess NP. The former evaluates a broader range of pain characteristics, while the latter focuses more on somatosensory tests and specific pain descriptions. Therefore, the painDETECT may be more sensitive in detecting NP in patients with a higher comorbidity burden. In a study conducted by Li et al.,[35] similar to this study, the comorbidity burden of patients was assessed using the CCI, and an increasing number of comorbidities elevated the risk of functional disability.

Nonetheless, the present study has several limitations. First, while patients from varying radiographic stages were included, the distribution of participants was not balanced, with a greater proportion represented in more advanced stages. This imbalance may have affected the representativeness of the findings and limited their applicability to a broader patient population. Second, the reliance on questionnaires, such as painDETECT and DN4, to assess NP may have introduced subjective bias or overstated the results. Although these tools are validated and widely used, they may not capture the full spectrum of NP mechanisms. Third, the cross-sectional nature of this study prevents us from establishing causal relationships between radiographic severity and NP symptoms. Addressing these limitations in future studies would be crucial for a more comprehensive understanding of the relationship between radiographic severity and NP in KOA patients.

In conclusion, NP is common in KOA patients and is associated with higher pain intensity and poorer functional outcomes. The moderate agreement between the painDETECT and DN4 scores may lead to a certain degree of diagnostic variation. Combining more than one method may increase the diagnostic accuracy. Further well-designed, large-scale, comparative studies are needed to confirm these findings.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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