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Types of Chronic Pain After Total Knee Arthroplasty and Their Relationship with Pain Intensity, Disability, and Quality of Life

Total Diz Artoplastisi Sonrası Kronik Ağrı Tipleri ve Bunların Ağrı Şiddeti, Engellilik ve Yaşam Kalitesi ile İlişkisi

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ABSTRACT

OBJECTIVE: To determine the prevalence of different pain mechanisms, including nociplastic pain, and to evaluate the associations of these mechanisms with pain intensity, disability, pain catastrophizing, and quality of life in patients with chronic pain after total knee arthroplasty (TKA).

MATERIALS and METHODS: This single-center cross-sectional observational study included 200 consecutive patients with chronic post-TKA pain between November 2022 and January 2023. Chronic post-TKA pain was defined as patient-reported pain in the operated knee persisting or recurring for more than three months after primary TKA, with a visual analog scale (VAS) score greater than 0 at the study visit. Neuropathic pain was identified using the painDETECT questionnaire. In patients not classified as possible or likely neuropathic pain, nociplastic pain affecting the musculoskeletal system was identified using the clinical criteria and grading system proposed by Kosek et al., including clinical history and bedside sensory examination for static mechanical allodynia, dynamic mechanical allodynia, thermal allodynia, and hyperalgesia. Patients were assigned to one dominant pain phenotype according to an a priori analytic hierarchy: neuropathic, nociplastic, and nociceptive pain. Pain intensity was assessed using the VAS, pain catastrophizing by the Pain Catastrophizing Scale, disability by the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), and quality of life by the 36-item Short Form Health Survey (SF-36).

RESULTS: According to the classification hierarchy, 29 patients (14.5%) had possible neuropathic pain, 22 (11.0%) had neuropathic pain, 35 (17.5%) had possible nociplastic pain, 61 (30.5%) had probable nociplastic pain, and 53 (26.5%) had nociceptive pain. VAS, WOMAC, and Pain Catastrophizing Scale scores differed significantly across pain phenotype groups (all p<0.001). Patients with possible neuropathic, neuropathic, and probable nociplastic pain generally showed greater pain intensity, disability, and pain catastrophizing than those with nociceptive pain. SF-36 physical and mental component scores also differed significantly between groups, with the lowest scores generally observed in the probable nociplastic and possible neuropathic groups.

CONCLUSION: Chronic pain after TKA is mechanistically heterogeneous, and approximately three quarters of the present cohort showed possible or likely neuropathic or nociplastic features. These findings support structured mechanism-based evaluation before and after TKA, while also emphasizing the need for prospective studies including preoperative pain phenotyping and standardized sensory testing.

KEYWORDS: Total knee arthroplasty, chronic post-surgical pain, nociplastic pain, neuropathic pain, nociceptive pain, painDETECT, WOMAC, quality of life

ÖZ

AMAÇ: Total diz artroplastisi (TDA) sonrası kronik ağrı gelişen hastalarda, nosiplastik ağrı da dahil olmak üzere farklı ağrı mekanizmalarının prevalansını (yaygınlığını) belirlemek ve bu mekanizmaların ağrı şiddeti, engellilik, ağrı felaketleştirme ve yaşam kalitesi ile olan ilişkilerini değerlendirmektir.

GEREÇ ve YÖNTEM: Bu tek merkezli, kesitsel ve gözlemsel çalışma; Kasım 2022 ile Ocak 2023 tarihleri arasında, TDA sonrası kronik ağrısı olan ardışık 200 hastayı dahil etmiştir. TDA sonrası kronik ağrı; primer TDA'dan sonra ameliyat edilen dizde üç aydan fazla süren veya tekrarlayan, çalışma vizitindeki görsel analog skala (VAS) skoru 0'dan büyük olan ve hasta tarafından bildirilen ağrı olarak tanımlanmıştır. Nöropatik ağrı, 'painDETECT' anketi kullanılarak belirlenmiştir. Olası veya muhtemel nöropatik ağrı olarak sınıflandırılmayan hastalarda, kas-iskelet sistemini etkileyen nosiplastik ağrı; Kosek ve ark. tarafından önerilen, klinik öykü ile statik mekanik allodini, dinamik mekanik allodini, termal allodini ve hiperaljeziye yönelik yatak başı duyusal muayeneyi içeren klinik kriterler ve derecelendirme sistemi kullanılarak tanımlanmıştır. Hastalar, önsel (a priori) bir analitik hiyerarşiye göre baskın bir ağrı fenotipine atanmıştır: nöropatik, nosiplastik ve nosiseptif ağrı. Ağrı şiddeti VAS ile, ağrı felaketleştirme 'Ağrı Felaketleştirme Ölçeği' ile, engellilik 'Western Ontario ve McMaster Üniversiteleri Osteoartrit İndeksi' (WOMAC) ile ve yaşam kalitesi ise '36 Maddelik Kısa Form Sağlık Taraması' (SF-36) ile değerlendirilmiştir.

BULGULAR: Sınıflandırma hiyerarşisine göre; 29 hastada (%14,5) olası nöropatik ağrı, 22 hastada (%11,0) nöropatik ağrı, 35 hastada (%17,5) olası nosiplastik ağrı, 61 hastada (%30,5) muhtemel nosiplastik ağrı ve 53 hastada (%26,5) nosiseptif ağrı saptanmıştır. VAS, WOMAC ve Ağrı Felaketleştirme Ölçeği skorları, ağrı fenotip grupları arasında anlamlı farklılık göstermiştir (tümü için p<0,001). Olası nöropatik, nöropatik ve muhtemel nosiplastik ağrısı olan hastalar; nosiseptif ağrısı olanlara kıyasla genel olarak daha yüksek ağrı şiddeti, engellilik ve ağrı felaketleştirme düzeyleri sergilemiştir. SF-36 fiziksel ve zihinsel bileşen skorları da gruplar arasında anlamlı derecede farklılık göstermiş olup, en düşük skorlar genellikle muhtemel nosiplastik ve olası nöropatik gruplarında gözlemlenmiştir.

SONUÇ: TDA sonrası kronik ağrı mekanistik olarak heterojendir ve mevcut kohortun yaklaşık dörtte üçü olası veya muhtemel nöropatik veya nosiplastik özellikler sergilemiştir. Bu bulgular, TDA öncesi ve sonrasında yapılandırılmış mekanizma temelli değerlendirmeyi desteklemekle birlikte, ameliyat öncesi ağrı fenotiplemesini ve standartlaştırılmış duyusal testleri içeren prospektif çalışmaların gerekliliğini vurgulamaktadır.

ANAHTAR KELİMELER: Total diz artroplastisi, kronik cerrahi sonrası ağrı, nosiplastik ağrı, nöropatik ağrı, nosiseptif ağrı, painDETECT, WOMAC, yaşam kalitesi

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INTRODUCTION

Total knee arthroplasty (TKA) is an effective treatment for end-stage knee osteoarthritis; however, a substantial minority of patients continue to report persistent pain after surgery. Chronic pain after TKA affects approximately one in five patients and can have a considerable negative impact on physical function, psychological well-being, and health-related quality of life (1). Chronic post-surgical pain is generally defined as pain that persists or recurs for longer than three months after surgery (2).

Persistent pain after TKA is mechanistically heterogeneous, and identifying the dominant pain mechanism is important for both clinical interpretation and treatment planning. According to the International Association for the Study of Pain (IASP), nociceptive pain arises from actual or threatened damage to non-neural tissue with activation of nociceptors, neuropathic pain is caused by a lesion or disease of the somatosensory nervous system, and nociplastic pain arises from altered nociception despite no clear evidence of ongoing tissue damage sufficient to activate peripheral nociceptors or of a lesion or disease of the somatosensory system (3,4). Because nociplastic pain may coexist with nociceptive or neuropathic features, structured clinical criteria are required to classify patients in a reproducible manner (5).

Central sensitization and nociplastic mechanisms may already be present in patients with knee osteoarthritis before surgery and may contribute to pain severity that is disproportionate to structural joint damage (7,15,16,21,22). Recent evidence also suggests that neuropathic-like symptoms and central pain mechanisms before TKA are associated with poorer postoperative outcomes (7). Therefore, evaluating pain mechanisms only after surgery may limit causal interpretation and clinical applicability, but postoperative phenotyping remains important for guiding treatment in patients with persistent pain.

Recent studies have shown that neuropathic symptoms are common in individuals with chronic pain after TKA (6,23), and that preoperative neuropathic-like pain and central pain mechanisms may predict poorer postoperative outcomes after knee replacement (7). Nevertheless, data on the relative distribution of nociceptive, neuropathic, and nociplastic pain phenotypes among patients with chronic pain after TKA remain limited. Therefore, this study aimed to determine the prevalence of different pain mechanisms, including nociplastic pain, and to evaluate their associations with pain intensity, disability, pain catastrophizing, and quality of life in patients with chronic post-TKA pain.

MATERIALS and METHODS

Study Design and Participants

This was a single-center, cross-sectional, hospital-based observational study reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations (20). The study was conducted after approval by the Shanghai Jiao Tong University Institutional Ethics Committee (approval number: SJTUM-2022-045). Written informed consent was obtained from all participants before enrollment.

A total of 200 consecutive patients who presented to the orthopedics outpatient clinic with chronic knee pain after primary TKA between November 2022 and January 2023 were enrolled. Chronic post-TKA pain was operationally defined as patient-reported pain in the operated knee that persisted or recurred for more than three months after primary TKA, together with a VAS score greater than 0 at the study visit. The presence of chronic post-TKA pain was therefore used as an inclusion criterion, whereas the VAS score was analyzed as a continuous outcome rather than as a binary threshold for phenotype classification.

Comprehensive medical histories and physical examinations were obtained for all participants. Patients with active uncontrolled systemic disease, malignancy, infection, generalized pain syndromes (e.g., fibromyalgia), or major psychiatric disorders were excluded. The exclusion of generalized pain syndromes and major psychiatric disorders was intended to reduce diagnostic confounding during pain phenotyping; however, this may also have influenced the estimated prevalence of nociplastic pain and is addressed as a limitation.

Pain Phenotyping

Pain phenotyping was performed using a two-stage clinical approach. The objective of the classification was to assign each patient to one dominant pain phenotype for group-based analysis; it was not intended to imply that pain mechanisms are mutually exclusive in individual patients. Because mixed-

mechanism pain is common, an a priori analytic hierarchy was used to avoid double counting: patients were first screened for possible or likely neuropathic pain, the remaining patients were assessed for possible or probable nociplastic pain, and those meeting neither classification were categorized as having nociceptive pain.

Neuropathic pain was assessed with the painDETECT questionnaire (PDQ). Scores of 19 or higher were classified as neuropathic pain, whereas scores of 13 to 18 were classified as possible neuropathic pain (9).

Nociplastic pain was assessed according to the clinical criteria and grading system proposed by Kosek et al. for chronic nociplastic pain affecting the musculoskeletal system (5). The clinical interview documented pain duration, distribution, disproportion between pain severity and identifiable peripheral pathology, history of hypersensitivity to touch, pressure, movement, heat or cold, and comorbid symptoms including sleep disturbance, fatigue, cognitive complaints, and hypersensitivity to environmental stimuli such as light, sound, odor, or temperature.

The bedside sensory examination focused on pain hypersensitivity in the painful knee/periarticular region. Static mechanical allodynia was considered present when light static touch or pressure over the painful region evoked pain that was clearly disproportionate to the stimulus. Dynamic mechanical allodynia was considered present when light stroking of the painful region with a cotton swab or soft brush evoked pain. Thermal allodynia was considered present when non-noxious cold or warm stimulation of the painful region evoked pain. Hyperalgesia was considered present when pinprick or pressure over the painful region produced exaggerated pain compared with the contralateral knee or a remote reference site. Findings were recorded as present only when the response was reproducible and judged clinically disproportionate to the stimulus.

Possible nociplastic pain was assigned when the patient had chronic regional pain that was not sufficiently explained by nociceptive or neuropathic mechanisms and had evoked pain hypersensitivity in the region of pain. Probable nociplastic pain required possible nociplastic pain plus a history of pain hypersensitivity in the painful region and relevant comorbid symptoms, in accordance with the Kosek grading framework (5). Patients who did not meet criteria for possible/likely neuropathic pain or possible/probable nociplastic pain were classified as having nociceptive pain.

Outcome Measures

The time elapsed since surgery was recorded for all participants. Pain intensity was evaluated using the VAS (0-10 cm) (10). Pain catastrophizing was assessed with the Pain Catastrophizing Scale (PCS; range 0-52) (11). Functional status and disability were measured using the WOMAC, with total scores normalized to a 0-100 scale, where higher scores indicate greater disability (12). Health-related quality of life was evaluated using the SF-36. The eight SF-36 domains were scored and transformed according to the standard SF-36 scoring algorithm, and physical component summary and mental component summary scores were calculated using the standard aggregation method described by Ware and Sherbourne (13).

Statistical Analysis

Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were examined for normality using the Shapiro-Wilk test. Normally distributed variables were compared across groups using one-way analysis of variance with Tukey post-hoc testing. Non-normally distributed variables were analyzed using the Kruskal-Wallis test followed by Dunn post-hoc pairwise comparisons with Bonferroni correction. Categorical variables were compared using the chi-square test or Fisher exact test, as appropriate. Data for normally distributed variables are expressed as mean +/- standard deviation; non-normally distributed variables are presented as median (interquartile range [IQR]). A two-tailed p-value of <0.05 was considered statistically significant.

Effect sizes were reported where appropriate to improve clinical interpretability. Eta-squared (eta-squared) was reported for Kruskal-Wallis analyses. Because no a priori sample size calculation was performed, a post hoc sensitivity power analysis was added for transparency. Assuming five groups, alpha=0.05, and a total sample size of 200, the study had approximately 80% power to detect a small-to-moderate omnibus group effect of f=0.25, equivalent to eta-squared approximately 0.058. This sensitivity analysis was interpreted descriptively because group sizes were unequal and the study was exploratory.

RESULTS

A total of 200 patients with chronic post-TKA pain participated in the study. The mean age of the participants was 56.54 +/- 12.08 years. No significant differences were observed between pain phenotype groups in age, body mass index (BMI), education, or time elapsed since surgery ($p > 0.05$) (Table 1).

Following the classification hierarchy, 29 patients (14.5%) were classified as having possible neuropathic pain, 22 (11.0%) as neuropathic pain, 35 (17.5%) as possible nociplastic pain, 61 (30.5%) as probable nociplastic pain, and 53 (26.5%) as nociceptive pain. Thus, 147 patients (73.5%) showed possible or likely neuropathic or nociplastic features, whereas 53 patients (26.5%) were categorized as having nociceptive pain.

Pain intensity, disability, and pain catastrophizing differed significantly across pain phenotype groups (Table 2). VAS scores were significantly higher in possible neuropathic, neuropathic, possible nociplastic, and probable nociplastic groups than in the nociceptive group. WOMAC scores were significantly higher in possible neuropathic, neuropathic, and probable nociplastic groups than in the nociceptive group, and the probable nociplastic group also had higher WOMAC scores than the possible nociplastic group. PCS scores were significantly higher in possible neuropathic, neuropathic, and probable nociplastic groups than in the nociceptive group; possible neuropathic pain also showed higher PCS scores than possible nociplastic pain.

SF-36 physical and mental component scores also differed significantly across groups (Table 3). The probable nociplastic group had significantly lower SF-36 physical and mental component scores than the nociceptive and possible nociplastic groups. The possible neuropathic group also had lower SF-36 component scores than the possible nociplastic group. These findings indicate that non-nociceptive pain phenotypes, particularly probable nociplastic and possible neuropathic pain, are associated with worse symptom burden and quality of life.

Table 1. *Demographic characteristics of participants*

Variables	Possible neuropathic (n=29)	Neuropathic (n=22)	Possible nociplastic (n=35)	Probable nociplastic (n=61)	Nociceptive (n=53)	p-value
Age (years)	54.62 +/- 10.48	52.31 +/- 12.02	57.65 +/- 10.47	56.50 +/- 12.75	58.64 +/- 12.87	0.257
BMI (kg/m²)	29.69 +/- 5.13	30.52 +/- 5.42	30.33 +/- 4.84	29.75 +/- 5.39	29.28 +/- 6.02	0.874
Education (years)	7.60 +/- 5.25	7.01 +/- 5.10	6.98 +/- 4.98	7.42 +/- 5.01	7.40 +/- 4.86	0.928
Values are presented as mean +/- standard deviation. One-way ANOVA. BMI: body mass index. Column order follows the analytic classification hierarchy						

Table 2. *Comparison of pain intensity, disability, and pain catastrophizing between groups*

Variables	Possible neuropathic	Neuropathic	Possible nociplastic	Probable nociplastic	Nociceptive	p-value
VAS	7 (5-8)[a]	7 (6-8)[a]	6 (4-6)[a]	7 (5-8)[a]	5 (3-7)	<0.001
WOMAC	44.4 (28-60)[a]	42.7 (25-55)[a]	22.2 (12-38)	33.3 (18-52)[ab]	24 (10-40)	<0.001
PCS	33.3 (18-42)[ab]	21 (8-35)[a]	3 (0-15)	16 (5-30)[a]	10 (3-20)	<0.001
Values are presented as median (IQR). Kruskal-Wallis test with Dunn post-hoc pairwise comparisons and Bonferroni correction. Superscript a: significant difference versus nociceptive pain (adjusted p<0.05). Superscript b: significant difference versus possible nociplastic pain (adjusted p<0.05). VAS: visual analog scale; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index, PCS: Pain Catastrophizing Scale						

Table 3. *Comparison of SF-36 component summary scores between groups*

Variables	Possible neuropathic	Neuropathic	Possible nociplastic	Probable nociplastic	Nociceptive	p-value
SF-36 Physical	33.75 (25-45)[b]	39.06 (28-50)	56.87 (42-68)	31.87 (22-48)[ab]	44.37 (30-58)	0.007
SF-36 Mental	33.87 (26-48)[b]	42.64 (30-56)	62.25 (48-72)	34.12 (22-50)[ab]	46.5 (32-60)	0.001
Values are presented as median (IQR). Kruskal-Wallis test with Dunn post-hoc pairwise comparisons and Bonferroni correction. Superscript a: significant difference versus nociceptive pain (adjusted p<0.05). Superscript b: significant difference versus possible nociplastic pain (adjusted p<0.05). SF-36: 36-item Short Form Health Survey						

DISCUSSION

The present study demonstrates that chronic pain after TKA is mechanistically heterogeneous. In this cohort, 25.5% of patients were classified as having possible or likely neuropathic pain and 48.0% as having possible or probable nociplastic pain, whereas 26.5% were classified as having nociceptive pain. These findings indicate that persistent post-TKA pain cannot be interpreted solely as ongoing nociceptive pain and support structured mechanism-based assessment in this population.

Patients classified as having neuropathic or probable nociplastic pain generally reported greater pain intensity, disability, and pain catastrophizing than those with nociceptive pain. These findings are consistent with previous reports showing that neuropathic or neuropathic-like pain after TKA is associated with worse clinical status and poorer patient-reported outcomes (6,8,14,23). They are also compatible with evidence indicating that pain sensitization is common in knee osteoarthritis and may contribute to less favorable outcomes after knee replacement (7,15,16,21,22).

The high proportion of non-nociceptive pain phenotypes is clinically important. Approximately three quarters of patients with persistent pain after TKA in this cohort had possible or likely neuropathic or nociplastic features. Although the present cross-sectional study cannot determine whether these mechanisms were present before surgery or developed afterward, previous literature suggests that central sensitization and neuropathic-like pain can precede TKA and may influence postoperative recovery (7,21,22). Therefore, screening for central sensitization and neuropathic-like pain before surgical decision-making may warrant consideration; however, this recommendation is based on indirect evidence from the present cross-sectional data and requires validation through prospective studies before implementation in clinical practice. Similarly, postoperative pain phenotyping may help guide individualized treatment when pain persists after surgery, although evidence from intervention trials targeting specific pain phenotypes is currently lacking.

Quality-of-life outcomes also differed significantly across pain phenotypes. The lower SF-36 physical and mental component scores observed in the probable nociplastic and possible neuropathic groups suggest that pain mechanisms are associated not only with local pain severity and disability, but also with broader health-related quality of life. This is compatible with earlier literature showing that chronic pain after TKA has multidimensional effects extending beyond local joint symptoms (1).

PCS scores differed significantly across groups, suggesting that cognitive and emotional responses to pain may vary according to the dominant pain phenotype. This finding is clinically relevant because pain catastrophizing and related psychosocial factors have been associated with persistent postsurgical pain after joint replacement (17,18). Therefore, assessment of persistent post-TKA pain should not be limited to structural or implant-related explanations; maladaptive pain processing and psychosocial contributors should also be considered.

The classification strategy used in this study should be interpreted carefully. The hierarchy was an analytic rule designed to assign a single dominant phenotype and avoid double counting; it should not be interpreted as a validated diagnostic hierarchy or as evidence that pain mechanisms are mutually exclusive. Because possible or likely neuropathic pain was classified before nociplastic pain, the prevalence of nociplastic pain is unlikely to have been inflated by patients with high PDQ scores. However, the use of a single-label classification may simplify mixed-mechanism pain, and some patients classified as nociplastic may also have had residual nociceptive contributors not fully captured by the study protocol.

Several limitations should be acknowledged. First, this was a single-center cross-sectional study including only patients who already had chronic pain after TKA; therefore, the observed distribution of pain phenotypes should not be generalized to all TKA recipients or to other populations. Second, no preoperative assessment of pain phenotype, central sensitization, psychological status, or pain distribution was available. This is a major limitation because pre-existing central sensitization or neuropathic-like symptoms may contribute to persistent postoperative pain. Third, the exclusion of patients with generalized pain syndromes such as fibromyalgia and major psychiatric disorders may have introduced selection bias and may have underestimated the prevalence of nociplastic features in the broader post-TKA population. Fourth, no a priori sample size calculation was performed; although a post hoc sensitivity analysis and effect sizes were added, the smaller neuropathic (n=22) and possible neuropathic (n=29) subgroups may have limited statistical precision, increasing the risk of both type I and type II errors in pairwise comparisons involving these groups. Fifth,

painDETECT was used to screen for neuropathic pain; DN4 was not administered and could have provided complementary clinical information. Sixth, nociplastic pain was assessed using clinical criteria and bedside sensory examination rather than quantitative sensory testing, and inter-rater reliability could not be evaluated because assessments were performed by a single physician. Seventh, implant-related and radiographic factors were not systematically analyzed, and correlation analyses between continuous outcome variables (e.g., VAS, WOMAC, PCS) were not performed because the primary aim was group-based comparison of pain phenotypes rather than exploration of continuous associations; this choice may have limited the granularity of the findings. Eighth, only SF-36 physical and mental component summary scores were analyzed as prespecified quality-of-life outcomes; individual domain scores were collected but were not examined because domain-level analysis was not part of the original study protocol and would have substantially increased the number of comparisons without a clear a priori hypothesis, thereby inflating the risk of type I error. These limitations should be addressed in future prospective studies.

CONCLUSION

Chronic pain after TKA is frequently associated with non-nociceptive pain features. In this cohort, possible or likely neuropathic and nociplastic phenotypes were common and were associated with greater pain burden, functional impairment, pain catastrophizing, and poorer quality of life than nociceptive pain. These findings support routine mechanism-based pain assessment in patients with persistent post-TKA pain and underscore the potential value of preoperative screening for central sensitization and neuropathic-like symptoms, pending validation in prospective cohorts. Future longitudinal studies should include preoperative and postoperative phenotyping, standardized sensory testing, and evaluation of targeted treatment strategies for different pain mechanisms.

Ethics: The study was conducted after approval by the Shanghai Jiao Tong University Institutional Ethics Committee (approval number: SJTUM-2022-045).

Etik: Bu çalışma, Shanghai Jiao Tong Etik Kurulu tarafından onaylanmıştır (Karar No: SJTUM-2022-045).

Author contribution status; The concept of the study; WC, JZ, LW. design; WC, JZ, LW. literature review; WC, JZ, LW. collecting and processing data; WC, JZ, LW. statistics; WC, JZ, LW. writing phase; WC, JZ, LW.

Yazar katkı durumu; Çalışmanın konsepti; WC, JZ, LW. dizaynı; WC, JZ, LW. Literatür taraması; WC, JZ, LW. verilerin toplanması ve işlenmesi; WC, JZ, LW. istatistik; WC, JZ, LW. yazım aşaması; WC, JZ, LW.
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